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The Biobehavioral Family Model: Close relationships and allostatic load

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ABSTRACT

Rationale: This study tested the inclusion of allostatic load as an expansion of the biobehavioral reactivity measurement in the Biobehavioral Family Model (BBFM). The BBFM is a biopsychosocial approach to health which proposes biobehavioral reactivity (anxiety and depression) mediates the relationship between family emotional climate and disease activity.
Methods: Data for this study included a subsample of $n = 1255$ single and married, English-speaking adult participants (57% female, M age = 56 years) from the National Survey of Midlife Development in the United States (MIDUS II), a nationally representative epidemiological study of health and aging in the United States. Participants completed self-reported measures of family and marital functioning, anxiety and depression (biobehavioral reactivity), number of chronic health conditions, number of prescribed medications, and a biological protocol in which the following indices were obtained: cardiovascular functioning, sympathetic and parasympathetic nervous system activity, hypothalamic pituitary adrenal axis activity, inflammation, lipid/fat metabolism, and glucose metabolism.
Results: Structural equation modeling indicated good fit of the data to the hypothesized family model ($\chi^2 = 125.13$ $p = .00$, SRMR = .03, CFI = .96, TLI = .94, RMSEA = .04) and hypothesized couple model ($\chi^2 = 132.67$, $p = .00$, SRMR = .04, CFI = .95, TLI = .93, RMSEA = .04). Negative family interactions predicted biobehavioral reactivity for anxiety and depression and allostatic load; however couple interactions predicted only depression and anxiety measures of biobehavioral reactivity.
Conclusion: Findings suggest the importance of incorporating physiological data in measuring biobehavioral reactivity as a predicting factor in the overall BBFM model.

Links between adult intimate partner and family relationships and physical health are well documented in the literature (Carr and Springer, 2010; Cohen, 2004; Woods et al., 2014). Higher reports of negative intimate partner and family functioning are linked to increased anxiety and depression symptoms (Priest, 2013; Whisman, 2007), and higher reports of anxiety and depression symptoms are associated with chronic diseases (Woods et al., 2014). Additionally, there is an increased focus in research on testing pathways tying relational variables to health outcomes (e.g., Kiecolt-Glaser et al., 2010; Kiecolt-Glaser and Newton, 2001; Kouvonen et al., 2011). This study attempted to ameliorate gaps in the literature, including a need for an increased focus on precise pathways by which negative family and intimate partner functioning stresses biological systems, and the need for specific physiological risk factors and outcomes using population-level data and biomarker studies (Carr and Springer, 2010; Wood and Miller, 2005). Specifically, we investigated and expanded the applicability of the Biobehavioral Family Model (BBFM; Wood, 1993), a multilevel biopsychosocial theoretical model explaining the effects of close relationships on health. Close relationships, in particular, are important to investigate as they can both buffer and potentiate risk factors related to health (Wood and Miller, 2002), in part due to the higher level of emotional intensity that these relationships tend to have compared to other social relationships, as well as their continued duration over the lifespan (Weihs et al., 2002).

The BBFM has been substantiated with lab-based family interaction studies (e.g., Wood et al., 2008) and findings suggest that the

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model is useful in highlighting family-psycho-biological pathways by which relational stress affects health outcomes (Wood et al., 2015). However, research evaluating the BBFM with adult family members has yet to incorporate objective physiological data (e.g., Woods and Denton, 2014; Woods et al., 2014; Priest and Woods, 2015), thereby limiting the measurement of the biobehavioral reactivity construct and the applicability of the model to guide clinical intervention. Specifically, subjective measures of depression and anxiety are often used to measure the biobehavioral reactivity construct. In this study, we included measures of allostatic load (McEwen, 1998) to expand the biobehavioral reactivity construct. We first tested whether allostatic load and self-reported measures of depression and anxiety provided better measurement as a single construct or as two separate but related constructs. We then used the best fitting measurement to reexamine the hypothesized pathways of the BBFM.

1. The Biobehavioral Family Model

The Biobehavioral Family Model (BBFM) is a biopsychosocial approach (Engel, 1977) to health that has integrated family functioning, psychological health, and physical health outcomes into a comprehensive model (Wood, 1993). The goal of this model was to connect principles of general systems theory (von Bertalanffy, 1969) with Minuchin's psychosomatic family model (Minuchin et al., 1978) to account for the impact of psychosocial factors on biological processes and disease activity (Wood and Miller, 2002). In the BBFM, family relationships serve as integral aspects of individual family member functioning that can serve to improve or aggravate health outcomes (Wood and Miller, 2002). The model theorizes the reciprocal nature of social, emotional, and physical influence on the experience of illness. In other words, the BBFM posits that there is responsivity at both the interpersonal (family) and individual levels and that individual responsivity/reactivity is "a pivotal factor and bidirectional pathway by which family patterns and disease processes influence one another" (Wood, 1993, p. 266). Wood (1993) suggests that individual and interpersonal responsivity interact, accounting for the risk of disease activity in individual family members (e.g., greater reactivity in individual family members may incur greater interpersonal, relational responsivity and higher levels of negative affect may be detrimental to family members' health). Although the BBFM was developed to explain global connections between family processes, individual family member reactions to relational stress, and disease activity across the lifespan and for all health conditions and outcomes, its emphasis on stress-related health outcomes meant that it was initially tested for children experiencing pediatric asthma and their families (e.g., Wood et al., 2008). Only recently has the BBFM been expanded and adapted to explain the connections between close relationships and health for adults (e.g., Woods and Denton, 2014).

The BBFM incorporates three variables: family emotional climate, biobehavioral reactivity, and disease activity (Wood, 1993). The model anticipates a mediation effect of biobehavioral reactivity on the association between family emotional climate and physical health. The construct of family emotional climate includes: relationship quality, interpersonal responsivity and reactivity, the positive and negative emotional processes within the family, as well as the intensity of those processes (Wood et al., 2008). Biobehavioral reactivity is proposed as the emotional and physiological ways in which an individual family member reacts to the family emotional climate (Wood et al., 2008). Biobehavioral reactivity is the construct of the BBFM that ties family process to health outcomes (Wood, 1993) and, as detailed in Wood et al. (2008), biobehavioral reactivity "is best understood … as reflecting the degree of emotion/physiological regulation or dysregulation" (p. 23). Disease activity is often operationalized as self-reported health and the presence of illness. The BBFM predicts that, in families where the emotional climate is marked by negativity and conflict, individuals will exhibit more biobehavioral reactivity (psychophysiological responsiveness to stress), which will lead to increased disease activity, or, worsened physical health (Wood, 1993; Wood and Miller, 2002; Wood et al., 2008).

Though the applicability of the BBFM's constructs and pathways are demonstrated in the literature, the measurement of the biobehavioral reactivity construct has been a consistent limitation. Specifically, prior tests of the model using adult samples use subjective, self-report measures of depression and/or anxiety to operationalize the biobehavioral reactivity construct (e.g., Priest and Woods, 2015; Woods et al., 2014). Though depression and anxiety were hypothesized as manifestations of high levels of biobehavioral reactivity, the original intent of this construct was to examine physiological reactivity in biological systems (e.g., the autonomic nervous system, the hypothalamic-pituitary-adrenal axis, etc.) and in emotional systems (e.g., depression and anxiety). The biobehavioral reactivity construct was viewed as the psychophysiological link between family emotional climate and disease activity. In other words, for the family emotional climate to affect disease activity, biological and emotional systems would need to be stressed. As the family emotional climate stressed the systems of a family member, this family member would be more susceptible to disease (Wood, 1993).

2. Allostatic load

Measuring and testing the applicability of the BBFM without physiological data does not encapsulate the original intent of the biobehavioral reactivity construct and therefore limits the applicability of the model. One way to improve the measurement of the biobehavioral reactivity construct would be to include the objective physiological measure of allostatic load. Allostatic load has been defined as "wear and tear that results from chronic over activity or under activity of allostatic systems" (McEwen, 1998, p. 171). The physiological or allostatic systems activated by stress are somewhat contradictory processes: when activated by stress, these systems can both protect and damage the body. If these systems repetitively respond to stress, their continual activation can damage the body and result in poor health (Seeman et al., 2002).

Research has identified seven physiological systems pertinent to the body's stress response. These physiological processes, also referred to as allostatic process, includes: cardiovascular functioning, the sympathetic and parasympathetic nervous systems, the hypothalamic pituitary adrenal axis, inflammation, lipid/fat metabolism, and glucose metabolism (Brooks et al., 2014; Buckwalter et al., 2011). This multi-system measure of allostatic load has been shown to predict more variance in health (e.g., depression, anxiety, and medical outcomes) compared to single weighted measures of allostatic load (Buckwalter et al., 2011).

3. BBFM and allostatic load

There are several areas of research that indicate the potential inclusion of allostatic load in the BBFM as a factor mediating the connection between family emotional climate and disease activity. Specifically, research suggests that problematic family functioning is linked to mental and physical health, which in turn has been linked to mental illness (Afifi et al., 2009; Priest, 2013; Whisman, 2007) and chronic diseases (Friedmann et al., 2006; Uchino, 2006; Zhang et al., 2007). Aspects of social relationships such as social support, social negativity, and contact frequency are shown to be associated with allostatic load, in that higher levels of spousal

and family negativity are associated with higher allostatic load levels (Brooks et al., 2014). Conversely, higher levels of spousal support are linked to lower allostatic load, independent of influences such as age and other sociodemographic factors (Brooks et al., 2014).

Additionally, research substantiates the connection between close relationships and biological measures of stress. Several studies found that positive social relationships are associated with lower levels of allostatic load (e.g., Juster et al., 2010; Seeman et al., 2002; Singer and Ryff, 1999). Similarly, increased social involvement and spousal presence is also linked with lower allostatic load (Seeman et al., 2004). On the contrary, negative relationships with spouses and between family members are related to higher scores of allostatic load (Brooks et al., 2014; Singer and Ryff, 1999), as are elevated levels of demands and criticism from spouses (Seeman et al., 2002). Lastly, specific to the present study, Brooks et al. (2014), using MIDUS data, found that several aspects of social relationships, including higher levels of spousal negativity, are associated with higher allostatic load.

At an individual level, a family member's response to stress can impact the physiological systems of the body and overall health outcomes depending on the stress level in the environment (e.g., family emotional climate). In most circumstances, the body's natural tendency after a stress-related response is to return to homeostasis, a process called allostasis (Buckwalter et al., 2011). If an individual is repeatedly exposed to a stressful atmosphere (i.e., high degree of conflict, negative relationship processes, etc), the body's ability to return to allostasis may be impacted, resulting in possible dysfunction in the individual's physiological systems (McEwen, 1998). Research consistently supports the notion that chronic stress corresponds with physiological reactivity, and that this process accelerates disease activity (e.g., Juster et al., 2010). The allostatic load model, which hypothesizes these connections, is a close corollary of the BBFM. In addition, the BBFM posits that the ways by which family members respond to stress are connected not only to their physiological coping mechanisms (required to maintain allostasis), but also the family emotional climate (Wood, 1993).

4. Present study

The purpose of this study was to further test the BBFM with a nationally representative sample of adults, and to test allostatic load as a physiological measure of the biobehavioral reactivity construct. First, we tested whether a one factor measurement model with allostatic load, depression, and anxiety all used to create the latent construct of biobehavioral reactivity would provide worse fit than a two factor measurement model where allostatic load was measured separately from depression and anxiety. We predicted that, because the BBFM suggests that the biobehavioral construct includes individual family members' physiological *and* emotional reactivity to emotional stimuli in the family (Wood et al., 2008), the two factor measurement model would provide a better fit to the data. This hypothesis is also reflective of allostatic load research in general; in other words, allostatic load is conceptualized as a distinct representation of individuals' physiological responses to stress, separate from subjective experiences of distress (i.e., depression and anxiety) (Brooks et al., 2014; McEwen, 1998).

Following the measurement test, we then used the best fitting measurement model of biobehavioral reactivity to test the pathways of the BBFM. Specifically, we hypothesized the following mediation relationship endemic to the BBFM:

(1) A direct pathway between family emotional climate and biobehavioral reactivity. Specifically, increases in family and intimate partner strain a will be associated with increases in allostatic load, depression, and anxiety.
(2) A direct pathway between biobehavioral reactivity and disease activity. Specifically, increases in biobehavioral reactivity (as measured by allostatic load, depression, and anxiety) will be associated with increases in disease activity (as measured by the presence or absence of chronic conditions and number of prescription medications).
(3) A nonsignificant pathway between family emotional climate and disease activity. In other words, we predict an indirect relationship between family emotional climate and disease activity that is mediated by biobehavioral reactivity (thereby rendering the pathway between family emotional climate and disease activity nonsignificant).

Overall, the present study adds to the current literature on families and health in three ways. First, it is novel because it models mental and physical health together in one comprehensive model. Much research investigating family relationship and health explores these health outcomes separately (Carr and Springer, 2010; Woods et al., 2014). Second, the present study adds to the families and health literature by investigating precise (mediation) pathways by which relational functioning affects health. Moreover, Carr and Springer (2010) have suggested that the use of biomarker is important to the advancement in understanding associations between families and health. This study examines the potential mediation role of allostatic load between family emotional climate and disease activity. Finally, the present study uses a systemic biopsychosocial model (assessing for both family and individual functioning), an approach that is recommended for investigating the impact of relational patterns on stress and health outcomes. This approach can be used for organizing and fostering future research and clinical interventions (Wood et al., 2015).

5. Method

5.1. Sample

The data for this study are from the National Survey of Midlife Development in the United States (MIDUS II; Ryff et al., 2012). MIDUS II is follow-up to the original MIDUS study completed between 1995 and 1996. The purpose of MIDUS studies were to "delineate the biopsychosocial pathways through which converging processes contribute to diverse health outcomes" (Singer and Ryff, 1999, p. 18.). This study focuses specifically on those in the sample that completed the bio-indicators and health protocol of MIDUS II also known as Project 4 (Love et al., 2010). A subsample of $n = 1255$ single (divorced, widowed, or never married) and married adults participated in this project. This included $n = 1054$ who participated in the original MIDUS study and an additional 201 sample recruited specifically for MIDUS II. In total, 54.9% of those eligible to participate in Project 4 choose to participate. Those who choose to participate in this study were brought in for a 2-day clinic visit. During this visit, blood, urine, and saliva samples were drawn, and participants underwent clinical assessments that included measuring blood pressure, medication usage, heart rate variability assessment, and a comprehensive physical exam. Additionally, participants completed self-reported health assessments including family medical history, major health events, subjective health, and symptoms and conditions. For a complete description of the protocol, see Love et al. (2010).

5.2. Demographic characteristics

The MIDUS II Project 4 sample is characterized as the following:

57% female, 78% White, 69% married, 52% with a high school degree or some college (42% college graduates or higher), and with a mean age of 56 and 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).

5.3. Measures

5.3.1. Family emotional climate

Family emotional climate was measured by the two family strain composites from the MIDUS II Project 4 data. Each of these composites ask the respondent to report how much strain the respondent receives from the family and how much strain the respondent gives to the family. Specifically, the strain to family composite is comprised of four questions that ask how frequently the respondent makes too many demands, criticizes, let down, or get on her/his family's nerves. The strain from family composite is also comprise of four question, but these questions as how often the respondent's family makes too many demands, criticizes, let down, or gets on the respondents nerves. Responses were coded, summed, and averaged so that higher totals on this composite represented greater family strain. Question from both composites used a Likert response scale with four responses ranging from "not at all" to "a lot." These composites were used as latent indicators of the family emotional climate variable.

5.3.2. Intimate partner emotional climate

Intimate partner emotional climate was measured by the two partner strain composites from the MIDUS II Project 4 data. Similar to the family strain composite, strain from partner composites ask the respondent to report how much strain the respondent receives from the partner and the strain to partner composite ask the respondent to report how much strain the respondent gives to the partner. Each composite had six questions that asked about demands in the relationship, tense feelings, arguing, criticizing, letting down and getting on nerves. Responses were coded, summed, and averaged so that a higher score represented more partner strain. Question from both composites used a Likert response scale with four responses ranging from "not at all" to "a lot." These composites were used as latent indicators of the intimate partner emotional climate variable.

5.3.3. Biobehavioral reactivity

Biobehavioral reactivity was operationalized with measures examining anxiety, depression, and allostatic load. Subscales from the Mood and Anxiety Symptom Questionnaire (Clark and Watson, 1991) were used to measure depression and anxiety. Specifically, the Anxious Symptoms and the Depressive Symptoms subscales were used in these analyses. The Anxious Symptoms subscale includes questions that ask respondents questions such as how often during the past week they "startled easily," "felt nervous," "felt dizzy," "felt tired," "hands were cold or sweaty," or "was unable to relax." Responses ranged from "not at all" to "extremely". Responses from each of the Anxious Symptoms subscale questions were coded, summed, and averaged so that a higher score represented higher levels of anxiety. Similarly, the Depressive Symptoms subscale included questions such as how often during the past week respondents "felt sad" "felt like a failure," "felt nothing was enjoyable," "heart was racing or pounding," or "was disappointed in myself." This subscale has the same response categories as the Anxious Symptoms subscale, and responses were coded, summed, and averaged so that a higher score represented more depressive symptoms.

Since the measures of depression and anxiety include somatic indicators of these disorders (e.g., heart racing, feeling dizzy, etc.), we also constructed scales of depression and anxiety that did not include the somatic indicators. The scales without the somatic symptoms were highly correlated with the original MASQ scales ($r > .9$), and when used in the data analysis did not meaningfully change the model fit (and the small changes were toward worse model fit) or the direction and significance of the pathways. Moreover, the scales without somatic symptoms only slightly change the magnitude of the pathways (the greatest change in any pathway was $\beta < .04$) Due to these findings, and because the MASQ scales have already been used in previous research, the results reported here include the original MASQ depression and anxiety scales with the somatic symptoms.

5.3.4. Allostatic load

Allostatic load was used as an objective physiological measure of biobehavioral reactivity in this study. This multi-system measure was designed to summarize physical health symptoms through calculating a composite index of indicators of cumulate strain on the body's organs and tissues. In this study, we replicated the measure of allostatic load recommended and used by Brooks et al. (2014) in a study of MIDUS data. Specifically, we used a compilation of seven biological systems (Table 1) to compute biomarker indicators for which participants fell into high-risk quartile ranges, including: cardiovascular functioning, parasympathetic nervous system activity, inflammation, lipid/fat metabolism, and glucose metabolism were used to (Brooks et al., 2014). This methodology is similar to previous allostatic load research (e.g., Gruenewald et al., 2012; Seeman et al., 2002).

Following the process of Brooks et al. (2014), a system risk score was calculated for each of the seven separate physiological systems (i.e., sympathetic nervous system, parasympathetic nervous system, cardiovascular, glucose metabolism, lipid, and inflammation). Each system included scores from a number of biomarkers for each participant (Table 5). These scores were continuous and ranged from 0 to 1 (0–100% of system biomarkers). Scores were computed by coding respective system measures as either 0 or 1 according to high-risk cut-off points (see Gruenewald et al., 2012 for overview), summing the individual measures for each system, and dividing

Table 1
Allostatic load biological systems and biomarker indicators.

System	Indicators
Cardiovascular	Systolic Blood Pressure
	Diastolic Blood Pressure
	Pulse
Metabolic Lipids	Body Mass Index
	Weight–Height Ratio
	Triglycerides
	HDL
	LDL
Metabolic Glucose	Hemoglobin
	Glucose
	Insulin
Inflammation	Blood C-Reactive Protein
	Serum IL 6
	Fibrinogen
	Serum Soluble E-Selectin
	Serum Soluble ICAM-1
Sympathetic Nervous System	Epinephrine
	Norepinephrine
Parasympathetic Nervous System	Heart Rate Variability:
	Standard Deviation of R–R
	Root Mean Squared Successive Differences of Heart Rate
	Low Frequency Heart Rate Variability
	High Frequency Heart Rate Variability
Hypothalamic Pituitary Adrenal Axis	Cortisol Level
	Blood DHEA-S

Table 2
Descriptive statistics for study variables (N = 1255).

Variables	*M*	Min.	Max.	n
Family Emotional Climate				
Strain from Family	2.03	1.00	4.00	1051
Strain to Family	1.74	1.00	4.00	1249
Intimate Partner Climate				
Strain from Partner	2.17	1.00	4.00	810
Strain to Partner	2.01	1.00	4.00	810
Biobehavioral Reactivity				
Anxiety	16.73	11.00	47.00	1251
Depression	18.63	12.00	60.00	1252
Allostatic Load				
Cardiovascular	.27	.00	1.00	1253
Lipids	.25	.00	1.00	1241
Glucose	.49	.00	1.00	1228
Inflammation	.28	.00	1.00	1233
Sympathetic NS	.24	.00	1.00	1233
HPA Axis	.24	.00	1.00	1239
Parasympathetic NS	.25	.00	1.00	1153
Disease Activity				
Prescription medication	2.81	.00	22.00	1255
Chronic conditions	4.10	.00	20.00	1255

Note. NS = nervous system; HPA = hypothalamic-pituitary-adrenal axis.

Table 3
Unstandardized and standardized factor loadings for biobehavioral reactivity and allostatic load (N = 1255).

	Factor loadings	
Item	B (SE)	β (SE)
Biobehavioral reactivity		
Anxiety	1.000 (–)	.642 (.147)
Depression	2.362 (1.074)	1.108 (.249)
Allostatic load		
Cardio	1.000 (–)	.349 (.038)
Lipids	1.347 (.176)	.536 (.036)
Glucose	1.536 (.211)	.523 (.035)
Inflammation	1.255 (.179)	.468 (.037)
Parasympathetic nervous system	1.100 (.177)	.302 (.040)

Table 4
Mediation results for the family emotional climate model (n = 1255).

FEC → DA	Estimate	Standard error	*p*
Total	.08	.05	.14
Indirect			
FEC → BBR → DA	.11	.04	<.01
FEC → AL → DA	.08	.03	<.01
Direct			
FEC → DA	–.11	.06	.07

FEC – Family emotional climate, BBR – Biobehavioral reactivity, AL – Allostatic load, DA – Disease activity.

Table 5
Mediation results for the intimate partner emotional climate model (n = 810).

IPEC → DA	Estimate	Standard error	*p*
Total	.06	.07	.40
Indirect			
IPEC → BBR → DA	.08	.03	<.01
IPEC → AL → DA	.05	.03	.13
Direct			
FEC → DA	–.06	.08	.40

IPEC – Intimate partner emotional climate, BBR – Biobehavioral reactivity, AL – Allostatic load, DA – Disease activity.

this score by the number of measures in that given system. For instance, cardiovascular functioning (i.e., the first biological system used to compute AL) was measured by (a) resting systolic blood pressure, (b) diastolic blood pressure, and (c) pulse rate for each participant. Each of these measures were given a score of 0 or 1, depending on cut-off point (i.e., 1 = high risk; 0 = no risk), summed, and divided by 3 (i.e., number of measures used to determine score for cardiovascular functioning). This method was repeated for each of the seven biological systems. An allostatic load summary score was then computed as the sum of the seven individual risk scores, which ranged from 0 to 7. Higher scores indicated higher risk for physical health symptoms.

Previous allostatic load research has used multiple specifications of physiological systems included in allostatic load in order to test for robustness (e.g., Hill, Rote, Ellison and Burdette, 2014). The present study utilizes the methodology outlined by Brooks et al. (2014) in order to replicate previous measurement of allostatic load in research using the current dataset. In addition, using multiple specifications of allostatic load is prohibited in the present study as we are using multiple indicators for each physiological system prior to using each system as an observed variable, part of the latent variable of allostatic load.

5.3.5. Disease activity

Disease activity was measured using two separate questions, similar to previous research (e.g., Woods et al., 2014). The first question asked about the number of reported chronic conditions. Specifically, respondents were asked if they ever had heart disease, stroke, diabetes, asthma, cancer, COPD, ulcers, or arthritis. The number of present conditions was summed for each respondent and ranged from 0 to 7. The second question asked about the number of reported prescription medications for each participant. Responses for this question ranged from 0 to 22. These two variables were used as indicators for the disease activity latent variable.

5.4. Data analysis

Data analysis was conducted in two steps. First, confirmatory factor analyses (CFAs) were used to test whether a one factor model of biobehavioral reactivity provided better fit than a two factor model. Specifically, we compared the fit statistics of a one factor model where the 7 risk systems of allostatic load and the depression and anxiety subscales of the MASQ were all used as observed indicators of the latent variable of biobehavioral reactivity. The two factor model included one factor where just the allostatic load risk scores were used as indicators of an objective measure of biobehavioral reactivity, and a second factor where just the depression and anxiety subscales of the MASQ were used as indicators of a subjective measure of biobehavioral reactivity.

Once the better fitting measurement model of the biobehavioral reactivity construct was identified, the second step was to evaluated full structural models testing the pathways of the BBFM. Following the examples of previous research (e.g., Priest and Woods, 2015), we tested two models. One that examined the associations between family emotional climate, biobehavioral reactivity, and disease activity, and one that tested the associations of intimate partner emotional climate, biobehavioral reactivity, and disease activity. The family emotional climate model included the entire sample (n = 1255) and used the family support and strain scales as indicators of family emotional climate variable, the best fitting measurement model of biobehavioral reactivity, and the number of reported chronic conditions and prescription medications as indicators of disease activity. The intimate partner emotional climate model included only those who reporting currently being married or cohabiting (n = 810). This model used the intimate partner support and strain scales as indicators of romantic partner emotional climate, the best fitting measurement model of biobehavioral reactivity, and the same

indicators of disease activity. Additionally, mediation analyses were conducted on both models, using the delta method (Olkin and Finn, 1995) to evaluate whether the relationship between family and intimate partner emotional climate and disease activity were mediated by biobehavioral reactivity. The delta method is similar to the Sobel test (MacKinnon, 2008) and is recommended as a test of mediation that produces accurate standard errors (MacKinnon et al., 2002). The delta method is common for estimating standard errors when using path analysis to model mediation effects (MacKinnon, 2008).

Analyses were conducted in M*plus* (Muthen and Muthen, 2012). Since the outcome variable for all models used count variables as indicators of the disease activity variable, the models were first specified to allow for the use of count data. However, when using count data in M*plus*, model fit statistic are not produced and mediation analyses cannot be conducted. Therefore, we reran the models so that the indicators of the disease activity variable were estimated as continuous variables. We then compared the standardized estimates from the count and continuous models and found that for both the family emotional climate model and the intimate partner emotional climate model these estimates were nearly identical. Therefore, since the estimates were similar and because using count data does not allow for mediation analysis, we report on the models with continuous indicators.

Maximum likelihood with robust standard errors (MLR) and chi-square was used as the estimator. MLR is robust to non-normality and non-independence of observations (Asparouhov, 2005). To assess model fit, the chi-square test, the standardized root mean square residual (SRMR), the comparative fit index (CFI), and the root mean square error approximation (RMSEA) were evaluated. If the model is a good fit for the data, the chi-square statistic will be small and non-significant, the SRMR will be less than .10, the CFI value will be greater than .95, and the RMSEA will be less than .05 (Kline, 2011). However, it should be noted that the chi-square statistic often perform poorly with large samples and non-normally distributed data, whereas the other statistic used to evaluate fit do not have the same sample size based limitations as the chi-square statistic.

6. Results

Means, standard deviations, ranges, and alpha coefficients for major study variables are presented in Table 2.

6.1. Confirmatory factor analyses

The one factor model of biobehavioral reactivity, with all seven allostatic load system indicators and both MASQ subscales, demonstrated poor fit (χ^2 = 1289.73, df = 27, p = .000, SRMR = .10, RMSEA = .19, CFI = .02). The two factor model, with allostatic load (objective biobehavioral reactivity) and the MASQ subscale (subjective biobehavioral reactivity) loading as separate but correlated factors, demonstrated better but relatively poor fit (χ^2 = 130.03, df = 26, p = .000, SRMR = .04, RMSEA = .06, CFI = .92). Upon inspection of the factor loadings of two factor model, the sympathetic nervous system and hypothalamic pituitary adrenal axis risk scores were not significantly related to the allostatic load latent variable. These variables were trimmed from the model, and the two factor model was run again. This model demonstrated good fit (χ^2 = 23.75, df = 13, p = .03, SRMR = .02, RMSEA = .03, CFI = .99), and was used in the subsequent structural models. Unstandardized and standardized factor loadings for the final two factor model are reported in Table 3.

6.2. The family emotional climate model

The family emotional climate model (n = 1255) demonstrated good fit (χ^2 = 125.13, df = 39, p = .00, SRMR = .03, RMSEA = .04, TLI = .94, CFI = .96). Standardized path coefficients were significant in the hypothesized directions. Specifically, family emotional climate was significantly associated with both the objective (allostatic load) and subjective (depression and anxiety) biobehavioral reactivity. Moreover, objective and subjective biobehavioral reactivity were both significantly associated with disease activity, and the association between family emotional climate and disease activity was not significant (see Fig. 1).

The results of the mediation analysis partially supported the hypotheses of the BBFM. Specifically, there was a non-significant total effect between family emotional climate and disease activity.

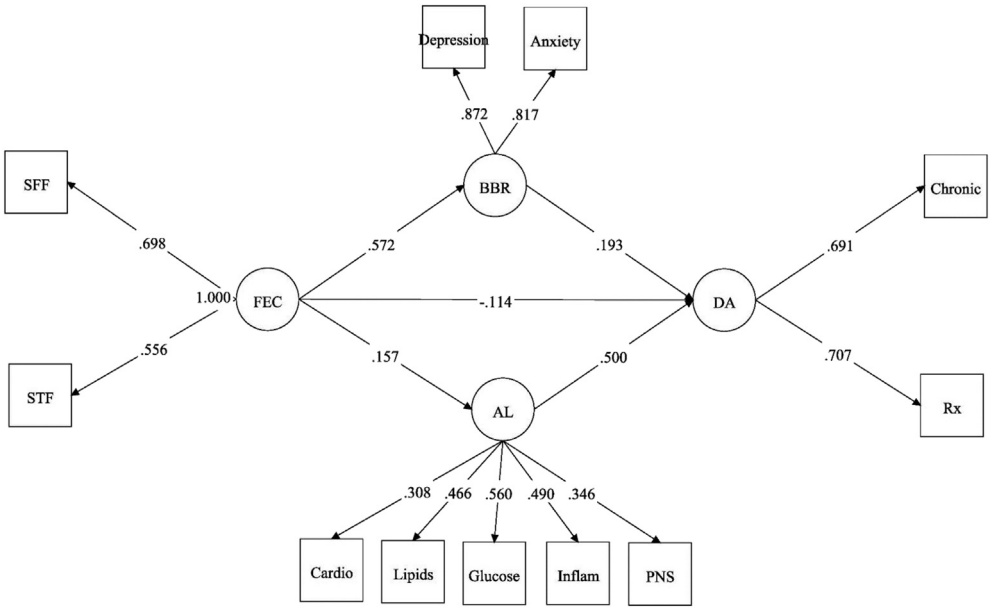

Fig. 1. Family Emotional Climate Model. χ^2 = 125.13 p = .00, SRMR = .03, CFI = .96, TLI = .94, RMSEA = .04. SFF – strain from family, STF – strain to family, FEC – family emotional climate, AL – allostatic load, PNS – parasympathetic nervous system, BBR – biobehavioral reactivity, DA – disease activity, Rx – prescriptions.

This effect was mediated both by the objective and subjective biobehavioral reactivity variables and biobehavioral reactivity (see Table 4). In other words, family emotional climate was related to disease activity through objective and subjective reports of biobehavioral reactivity.

6.3. The intimate partner emotional climate model

Using a subsample of married and cohabitating adults ($n = 810$), the intimate partner emotional climate model also demonstrated good fit ($\chi^2 = 132.67$, df = 39 $p = .00$, SRMR = .04, RMSEA = .04, TLI = .93, CFI = .95). Similar to results in Model 1, the model's χ^2 was significant, which may be influenced by the sample size. In accordance with our hypotheses, standardized path coefficients were significant in the hypothesized directions (see Fig. 2) with the exception of intimate partner emotional climate and the objective measure of measure of biobehavioral reactivity (allostatic load). There was a significant pathway between intimate partner emotional climate and the subjective measure of biobehavioral reactivity (depression and anxiety), and there were significant pathways between the objective and subject measures of biobehavioral reactivity and disease activity. There were nonsignificant pathways between intimate partner emotional climate and the objective measure of biobehavioral reactivity, and a nonsignificant pathway between intimate partner emotional climate and disease activity.

The results of the mediation analysis partially supported the hypothesis of the BBFM. Specifically, the total effect between intimate partner emotional climate and disease activity was nonsignificant, as was the direct effect between intimate partner emotional climate and disease activity. The only significant mediation pathway for this model was the indirect effect between intimate partner emotional climate, the subjective biobehavioral reactivity variable (depression and anxiety), and disease activity (see Table 5).

7. Discussion

The purpose of this study was to incorporate objective physiological measurements into the biobehavioral reactivity construct of the BBFM. Specifically, we tested the hypothesis that objective (allostatic load) and subjective (self-reported depression and anxiety) reports of biobehavioral reactivity would be best measured as two separate constructs, and that using this two factor measurement model of biobehavioral reactivity, the mediational pathways of the BBFM would be replicated in both the family emotional climate and intimate partner emotional climate models of the BBFM.

As predicted and in accordance with BBFM and allostatic load literature, we found that objective/physiological and subjective/emotional reports of biobehavioral reactivity were best measured as two distinct factors. In research with adults using the BBFM (e.g., Priest and Woods, 2015; Woods and Denton, 2014; Woods et al., 2014), the biobehavioral reactivity construct has frequently been measured with subjective reports of anxiety and depression. The results of the present study suggest that biobehavioral reactivity is best measured by both physiological and emotional variables as distinct factors.

It is also important to note that the best fitting measurement model excluded the sympathetic nervous system and hypothalamic pituitary adrenal axis risk scores. When these scores were removed from the measurement model, the fit improved. This finding may suggest that these seven systems impact overall biobehavioral reactivity differently. In other words, it may be that instead of measuring allostatic load as a latent variable, future research may benefit from examining how family emotional climate and intimate partner emotional climate are linked to each individual allostatic load system. For example, it may be that family emotional climate may be linked to dysregulation in cardiovascular functioning but not to glucose metabolism or vice-versa. Further, these types of studies can help to determine and highlight the directionality of specific pathways tested in the BBFM, which could provide additional knowledge about meaningful differences between close relationship types and different biological systems for adult health.

Our second hypothesis stated that, when including the best fitting measurement model of biobehavioral reactivity into a test of the BBFM, the pathways between the variables and the mediation found in other tests of the BBFM would be replicated. This hypothesis was supported for the family emotional climate model.

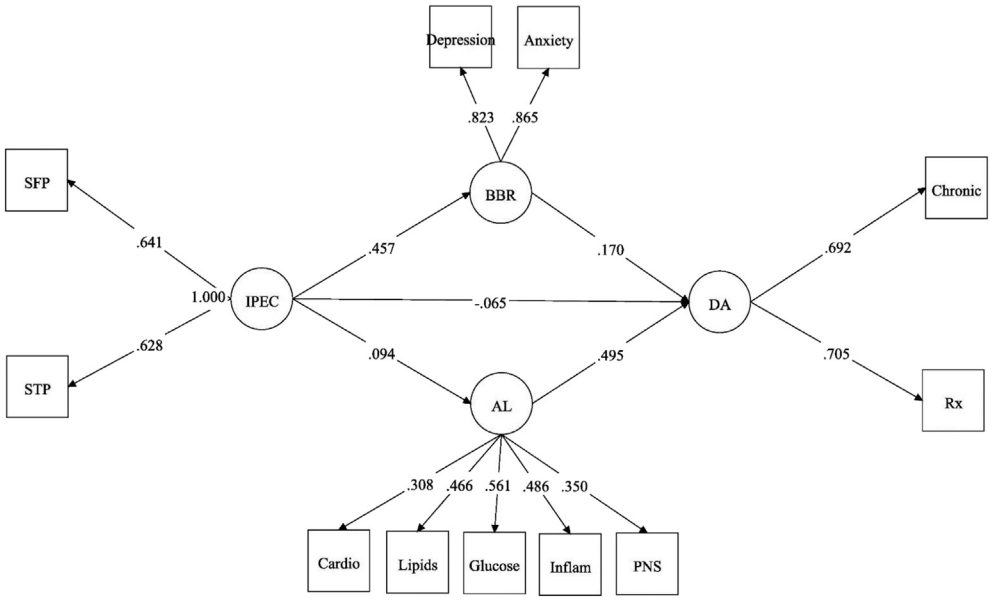

Fig. 2. Intimate Partner Emotional Climate Model. $\chi^2 = 132.67$, $p = .00$, SRMR = .04, CFI = .95, TLI = .93, RMSEA = .04 SFP – strain from partner, STP – strain to partner, IPEC – intimate partner emotional climate, AL – allostatic load, PNS – parasympathetic nervous system, BBR – biobehavioral reactivity, DA – disease activity, Rx – prescriptions.

Specifically, significant pathways were found between family emotional climate and both the objective and subjective measures of biobehavioral reactivity, and these two measures were both significantly linked to disease activity. Family emotional climate was also linked to disease activity but this association was mediated by the biobehavioral reactivity measures. In other words, all of the hypothesized pathways and relationships of the BBFM were substantiated for the family emotional climate model, similar to previous research (e.g., Priest and Woods, 2015; Woods et al., 2014; Woods and Denton, 2014).

However, the same was not found for the intimate partner climate model. Though intimate partner climate was linked to the subjective measure of biobehavioral reactivity (depression and anxiety), it was not linked to the objective measure (allostatic load), contrary to our hypotheses. Moreover, this finding is dissimilar to other findings using the MIDUS data (e.g., Brooks et al., 2014). Although we used risk scoring of biomarker indicators similar to Brooks et al. (2014), the authors used a different measurement model as a final representation of allostatic load. Specifically, Brooks et al. (2014) computed a composite overall risk score of all seven risk systems to create an observed variable of allostatic. In the present study, the allostatic load measurement model only included five of the seven allostatic load systems and used a latent variable to represent systems' risk scores. Brooks et al. (2014) found that spouse negativity was linked allostatic load, and in this study the latent variable of intimate partner emotional climate was not linked to the latent variable of allostatic load. It may be that the discrepancy in the results stems from the different measurement models (i.e., a compilation of seven systems as an observed variable versus a latent variable using five of the seven systems as observed variables) used in the two studies.

Another possible explanation for the absence of a significant pathway between intimate partner emotional climate and allostatic load may be that intimate partners may form after patterns of stressful interactions are established in the family–of-origin. In other words, since allostatic load is a measure of chronic over- or under-functioning of allostatic systems, family relationships may be more salient to this construct than intimate partners. Family relationships begin in childhood and generally have a longer duration than intimate partners. As the source of initial secure, supportive emotional relationships (or conflicted, problematic ones), families-of-origin "are crucial determinants of the capacity for regulation of emotional and physiological processes" (Weihs et al., 2002, p. 9) and therefore may relate uniquely to a construct such as biobehavioral reactivity. Future research would benefit from asking respondents to indicate what relationships they considered when answering questions about family emotional climate. By having a clearer understanding of how respondents define family relationships, it may be easier to determine how different types of family relationships are related to biobehavioral reactivity and allostatic load, similar to suggestions made by previous researchers (e.g., Uchino et al., 1996). The findings may also indicate the necessity of assessing for both family-of-origin and intimate partner effects for adults, despite the frequent practice of assessing only marital relationships for adult research participants with the assumption that marriage is the most important, meaningful relationship for this population (Carr and Springer, 2010; Woods et al., 2014). Overall, a developmental approach to understanding how different types of family relationships affect adult family members may be warranted. Social support researchers suggest that how individuals perceive support may be established early on, and that early social processes in families-of-origin can long affect individuals' health, into adulthood (Uchino, 2006). Therefore, highlighting different relationship types and relational complexity throughout the lifespan, and teasing out differences in associations with health outcomes, could help to design specific intervention studies (Uchino, 2006) using the BBFM as a guiding theoretical model.

7.1. Limitations and future directions

The results from this study should be viewed in consideration of its limitations. First, the fact that our variables were measured at one point in time precludes our ability to examine the longitudinal patterns of associations that could inform our understanding of the temporal and directional patterns between family relationships, biobehavioral reactivity, and disease activity throughout the life course. This limitation is important as longitudinal evidence demonstrates that marriages exhibiting distress and conflict tend to decline in self-reported health over time (Umberson et al., 1996). In addition, because the present study is cross-sectional, it may be that the relationships among the BBFM variables are reciprocal (as is theorized by Wood, 1993). In other words, family experiences may produce stressful experiences for individual family members, but this experienced stress (in the form of biobehavioral reactivity) may also contribute to a negative perception of one's family, thereby influencing statistical associations between these constructs. As there is a possibility of endogeneity between family emotional climate and biobehavioral reactivity (i.e., depression and anxiety may underlie or give rise to problematic relational functioning), future research would benefit from testing the BBFM using studies with longitudinal or experimental design.

Second, we were limited in various ways due to the use of secondary data. Only one spouse's responses were examined which restricted our ability to examine the potential influences of spousal dyads or family unit interactions. Studies applying the BBFM to adult samples have yet to use dyadic data, limiting the understanding of how these effects occur in a relational context. Further, a necessary next step in understanding the impact of family relationships on health would be to collect data from both spouses and/or multiple family members to further investigate the BBFM dyadically. Additionally, when asking about the family emotional climate, it not clear from the items collected in the MIDUS data who each respondent was thinking about when answering questions about family. It is likely that different family compositions (e.g., families with children in the home versus families with grown children no longer at home) could result in higher or lower levels of involvement with family members which could contribute to more or less family stain. Future research examining the relationship between families and health would benefit by asking the respondents who they were thinking about when answering questions about family strain. If this type of question was asked, it would be possible to explore how family composition may moderate the relationship between family strain and health outcomes.

A final consideration of the present study would be to increase the ethnic and socioeconomic diversity in the participant sample and explore potential variations in the health profiles (e.g., allostatic load, depression/anxiety) to further improve generalizability. Moreover, it would be important for future research to examine how gender, race/ethnicity and socioeconomic factors influence the pathways of the BBFM.

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