

## EXAMINING DIFFERENTIATION OF SELF AS A MEDIATOR IN THE BIOBEHAVIORAL FAMILY MODEL

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*The Biobehavioral Family Model (BBFM) is a biopsychosocial model of health. This model proposes that biobehavioral reactivity mediates the association between the family emotional climate and disease activity. To improve the clinical relevance of the BBFM, variables that mediate the association between family emotional climate and biobehavioral reactivity need to be tested. This study examined differentiation of self as a mediator. Using data from the Midlife Development in the United States study (n = 854), results suggested that differentiation of self mediated the association between the family and intimate partner emotional climate and mental health symptoms. These findings suggest that including differentiation of self into the BBFM may help interventions based on the model target factors that could improve health outcomes.*

The Biobehavioral Family Model (BBFM; Wood, 1993) is a biopsychosocial model of health (e.g., Lim, Wood, & Miller, 2008; Lim, Wood, Miller, & Simmens, 2011; Priest & Woods, 2015; Priest et al., 2015; Woods & Denton, 2014; Woods, Priest, & Roush, 2014). The BBFM examines how negative family emotional climate contributes to individual family members experiencing greater biobehavioral reactivity (i.e., depression, anxiety, and psychophysiological stress) resulting in increased disease activity (i.e., chronic health conditions). Originally developed to explain associations between family relationships and health for children (Wood, 1993), recent research has shown the model's utility in examining association between family relationships and health for adults (e.g., Priest & Woods, 2015; Priest et al., 2015; Woods & Denton, 2014; Woods et al., 2014).

Although research using the BBFM with children has demonstrated its clinical relevancy (Wood, Miller, & Lehman, 2015; Woods & McWey, 2012), research using the BBFM with adults has yet to examine variables that could potentially explain the association between a negative family emotional climate and greater biobehavioral reactivity. In other words, for an adult in a highly negative family emotional climate, what factors could a therapist address in session that could mitigate the effect of the family emotional climate on the individual's biobehavioral reactivity? And if this person is able to reduce their biobehavioral reactivity, could this potentially reduce disease activity? In order to improve the clinical relevancy of the BBFM, it is necessary to incorporate variables rooted in family therapy theories that could potentially mediate the association between family emotional climate and biobehavioral reactivity. The purpose of this article is to test differentiation of self (Kerr & Bowen, 1988) as a potential mediator between family emotional climate and biobehavioral reactivity variables in the BBFM. Specifically, using data from the Midlife Development in the United States Survey (Love, Seeman, Weinstein, & Ryff, 2010), this study replicates hypotheses from previous research using the BBFM with adults (Priest et al., 2015), but extends this model by incorporating a measure of differentiation of self as a potential mediator between family emotional climate and biobehavioral reactivity.

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### *The Biobehavioral Family Model*

The BBFM is a biopsychosocial model of health that integrates family functioning, and mental and physical health into a comprehensive model (Wood, 1993). Specifically, the BBFM posits three main constructs: family emotional climate, biobehavioral reactivity, and disease activity. The family emotional climate is hypothesized as the “the overall intensity and valence of emotional exchange (Wood et al., 2015) that occurs in a family system.” Recent research has shown the importance of exploring the family emotional climate and intimate partner emotional climate as distinct factors for adults (Woods et al., 2014). The family emotional climate represents the level, intensity, frequency, and balance of positive and negative emotional exchanges that occur with parents, children, or other relatives (Wood et al., 2015); whereas the intimate partner emotional climate represents the level, intensity, and frequency of positive and negative exchanges that occur within an intimate partnership (Woods et al., 2014). The hypotheses of the BBFM suggest that if the family and intimate partner emotional climates are more negative then the relational processes that occur within this system may produce or exacerbate stress dysregulation (Wood et al., 2015).

The second construct of the BBFM is biobehavioral reactivity. This variable is conceptualized as the degree or intensity with which a person emotionally and physiologically responds to family or intimate partner emotional climate (Wood et al., 2015). Specifically, Wood (1993) hypothesized that biobehavioral reactivity is comprised of the “behavioral, emotional, and physiological response” that a person has to their family emotional climate. Previous research has used measures of depression and anxiety symptoms (Lim et al., 2008, 2011; Priest & Woods, 2015; Woods & Denton, 2014; Woods et al., 2014) as well as allostatic load (Priest et al., 2015) to operationalize this construct. Allostatic load is the “wear and tear that results from chronic over activity or under activity” of physiological systems (e.g., the cardiovascular system; McEwen, 1998, p. 171). These measures capture the emotional and physiological responses that an individual has in the family emotional climate. In the BBFM, biobehavioral reactivity is the construct which links family emotional climate to disease outcomes.

The final construct of the BBFM is disease activity. This variable is conceptualized as the activation of pathophysiological processes (Wood, 1993) or as the severity of illness or chronic health conditions (Woods & Denton, 2014). In research with adults, this variable has been operationalized as the number of chronic illness an individual is diagnosed with, the number of prescription medications an individual takes weekly, and self-reported health measures (e.g., Priest et al., 2015; Woods et al., 2014).

The BBFM hypothesizes that biobehavioral reactivity mediates the association between family (or intimate partner) emotional climate and disease activity. If the family emotional climate is negative, then an individual will be emotionally or physiologically dysregulated (e.g., greater depression, anxiety, or allostatic load). This dysregulation will lead to increased disease activity. Similarly, if the family emotional climate is positive, then an individual will not be as emotionally or physiologically dysregulated, resulting in less disease activity. Researchers have found support for the hypotheses of the BBFM. For example, using a large, representative dataset, Woods et al. (2014) found that those with more negative family and intimate partner emotional climates reported greater biobehavioral reactivity, and this was associated with greater reported disease activity. Similar findings were found for Latino Americans (Priest & Woods, 2015) and primary care patients (Woods & Denton, 2014).

Although research lends support to the hypotheses of the BBFM, the clinical applicability of the model with adults remains limited. Specifically, research using the BBFM with adults often points to the need to use couple and family interventions to treat mental and physical health problems (e.g., Woods & Denton, 2014); however, it has yet to specify how therapists could intervene. In order to improve the clinical applicability of the BBFM with adults, it would also be important to identify variables that could potentially mediate the associations between family and intimate partner emotional climate and biobehavioral reactivity.

Research examining the BBFM with children has examined the role of attachment as a mediator between family emotional climate and disease activity (e.g., Wood, Klebba, & Miller, 2000; Wood et al., 2006; Wood et al., 2008). Moreover, research has shown that attachment processes are associated with health outcomes for adults (e.g., McWilliams & Bailey, 2010). However, in clinical practice, adult attachment processes are often addressed only in the romantic partnership. For

example, Emotionally Focused Couples Therapy, an attachment-based approach, focuses exclusively on intimate partners' attachment processes (Wiebe & Johnson, 2016). Research examining the BBFM with adults has shown that the adult family emotional climate often has stronger associations with biobehavioral reactivity than the intimate partner emotional climate (e.g., Priest & Woods, 2015; Priest et al., 2015). Since clinical work with adult attachment is typically operationalized only as an intimate partner process, including it in the BBFM may neglect the important role that the family emotional climate plays in adult health. Therefore, in order to expand the clinical application of the BBFM with adults, it would be necessary to incorporate variables that could potentially mediate the association between family *and* intimate partner emotional climate and biobehavioral reactivity. Differentiation of self is one such variable.

### *Differentiation of Self*

Differentiation of self is a central tenant of Bowen's family systems theory (Kerr & Bowen, 1988). Differentiation of self is described as the ability to emotionally self-regulate and to balance individuality and togetherness in relationships. Kerr and Bowen (1988) suggest that individuals with poor differentiation have strong emotional reactions which can lead them to fuse or cutoff from partners or other family members. Individuals with better differentiation of self are able to manage their emotions in times of high stress or conflict, and they are able to maintain close connections without giving up their sense of self. Differentiation of self is also hypothesized as a construct that is present both in the intimate partner relationship and in other family relationships. Specifically, Bowen would suggest that a person's level of differentiation would be constant across relationships—a person's level of differentiation would be the same whether that person was interacting with a romantic partner or with another family member (Kerr & Bowen, 1988).

### *The BBFM and Differentiation of Self*

Although researchers have not yet included differentiation of self into empirical tests of the BBFM, Theodoratou-Bekou, Andreopoulou, Andriopoulou, and Wood (2012) proposed a theoretical conceptualization and case example of how differentiation of self could add to the BBFM. They argued that although the BBFM presented specific pathways about how the family emotional climate was linked to health, infusing Bowen's theory into the BBFM model could help interventions be more "precisely targeted to family-specific relational factors (Theodoratou-Bekou et al., 2012, p. 4)." Specifically, in the case example, they demonstrated that targeting an individual's level of differentiation could reduce the effects of a negative family emotional climate and that differentiation of self could potentially reduce biobehavioral reactivity for an adolescent with asthma. They suggested that even though family relationships might be a source of conflict, by increasing differentiation of self, an individual could sustain physical and emotional well-being without cutting off from family members (Theodoratou-Bekou et al., 2012).

One way to test the assumptions put forth by Theodoratou-Bekou et al. (2012) would be to include differentiation of self as a mediator between intimate partner and family emotional climate in a test of the BBFM model. Research has already substantiated links between intimate partner and family emotional climate and biobehavioral reactivity (e.g., Priest et al., 2015; Wood et al., 2015). Therefore, it would be necessary to test a variable that has been hypothesized to be related to both family and intimate partner relationships. Bowen has argued that differentiation of self is present in all relationships (Kerr & Bowen, 1988) and as such, it could potentially be a mediator between both intimate partner and family emotional climate and biobehavioral reactivity.

### *Present Study*

This study aims to expand the clinical relevancy of the BBFM by including differentiation of self as a potential mediator between intimate and family emotional climate and biobehavioral reactivity. To accomplish this, the following hypotheses of the BBFM were tested first:

- (1) A more negative intimate partner or family emotional climate will be related to greater biobehavioral reactivity.
- (2) Greater biobehavioral reactivity will be associated with increased disease activity.
- (3) Biobehavioral reactivity will mediate the association between the intimate partner and family emotional climates and disease activity.

After these hypotheses were tested, differentiation of self was introduced into the model and the following mediation hypotheses were tested:

- (4) A direct association will be found between intimate partner and family emotional climate and differentiation of self.
- (5) A direct association will be found between differentiation of self and biobehavioral reactivity.
- (6) An indirect association will be found between intimate partner and family emotional climate and biobehavioral reactivity such that differentiation of self will mediate the association between intimate partner and family emotional climate and biobehavioral reactivity.

Overall, these hypotheses aim to expand upon a previous test of the BBFM (Priest et al., 2015) with MIDUS data by including differentiation of self as an additional mediator. By doing so, the goal of this study is to improve the clinical utility of the BBFM by testing the ideas Theodoratou-Bekou et al. (2012) have put forth.

## METHOD

Data for this study came from the respondents of Midlife Development in the United States Survey (MIDUS II; Ryff, Seeman, & Weinstein, 2012). Specifically, data for this study came from those who also participated in the MIDUS II Biomarker Project (Love et al., 2010). The goal of the Biomarker Project was to examine biopsychosocial pathways that contribute to health. To accomplish this, the Biomarker Project respondents participated in a 2-day clinic visit. During this visit, respondents' vital signs, functional capacities, heart rate variability, blood pressure, and cortisol levels were assessed. Additionally, respondents completed self-reported health assessments that asked about current chronic health conditions, major health events, and their family medical history. A complete description of the clinic visit protocol can be found in Love et al. (2010).

Since the goal of this study was to examine both the intimate partner and family emotional climate, only those participants who reported being partnered were included in this sample. The Biomarker Project had originally included  $n = 1,255$  respondents. Of those, only  $n = 854$  (68%) were partnered and completed the questions asking about their intimate partner emotional climate. This sample was used in this study. The average age of the partnered respondents was 54.51 ( $SD = 11.46$ ), and 50% of the sample was female. The sample was 86.8% White, 9.1% of the sample was Black/African American, and the remaining 4.1% reported another racial/ethnic group.

### Measures

*Family emotional climate.* The Family Strain composite from MIDUS II was used to measure family emotional climate. This composite is created by averaging the respondents score across four items. These items asked the respondents to think about members of their family excluding their spouse/partner and to indicate how much these family members: (a) make too many demands; (b) let them down when counting on them; (c) get on their nerves; (d) and criticize them. Responses to these questions were on a scale from 1 to 4, with higher numbers representing greater strain. This scale demonstrated reliability for this sample (Cronbach's  $\alpha = .76$ ). This composite has been used in previous tests of the BBFM with MIDUS data (e.g., Priest et al., 2015). This composite was included as an observed variable in the structural equation models.

*Intimate partner emotional climate.* The Partner Strain composite from MIDUS II was used to measure the intimate partner emotional climate. This composite is created by averaging the respondents score across six items. These six items ask the respondents to report how much: (a) their spouse/partner makes too many demands on them; (b) their spouse/partner argues with them; (c) their spouse/partner makes them feel tense; (d) their spouse/partner criticizes them; (e) their spouse/partner lets them down; and (f) their spouse/partner gets on their nerves. Responses to the questions ranged from 1 to 4, with higher numbers representing greater strain. This scale demonstrated reliability for this sample (Cronbach's  $\alpha = .86$ ). This composite has also been used in previous test of the BBFM (e.g., Priest et al., 2015). This composite was included as an observed variable in the structural equation models.

*Biobehavioral reactivity.* This study replicated previous researchers' operationalization of the BBFM with MIDUS data. Specifically, Priest et al. (2015) demonstrated how a two-factor model of biobehavioral reactivity provided good fit for the MIDUS Biomarker Project data. This two-factor model, with one factor that examines mental health symptoms and another that examines allostatic load, was replicated for this study. This two-factor model represents the emotional and physiological dysregulation of the biobehavioral reactivity construct.

The mental health symptoms variable was measured using the Anxious and Depressive Symptoms subscales of the Mood and Anxiety Questionnaire (Keogh & Reidy, 2000). The Anxious Symptoms subscale consists of 11 items that ask the respondents to indicate if they have experienced the following symptoms during the past week: feeling afraid, had diarrhea, felt nervous, felt uneasy, had a lump in the throat, had an upset stomach, felt keyed up or on edge, was unable to relax, felt nauseous, felt tense or high-strung, and muscles were tense or sore. Responses were on a scale from 1 to 5, with higher scores representing greater presence of anxiety symptoms. This scale demonstrated reliability for this sample (Cronbach's alpha = .80). The Depressive Symptom Subscale included 12 items that asked respondents to indicate if they have experienced the following symptoms during the past week: felt sad, felt discouraged, felt worthless, felt depressed, felt like a failure, blamed myself for a lot of things, felt inferior to others, felt like crying, was disappointed in myself, felt hopeless, felt sluggish or tired, and felt pessimistic about the future. Responses for scale also ranged from 1 to 5, with higher scores representing greater presence of depressive symptoms. This scale demonstrated reliability for this sample (Cronbach's alpha = .90). For both scales, responses to each question were summed to create a total score for each participant. These total scores were used as indicators for the mental health symptoms latent variable.

Allostatic load was measured using five biomarkers. Specifically, respondents' risk score in their cardiovascular functioning, lipid metabolism, metabolic glucose, inflammation, and parasympathetic nervous system were each used as indicator on the latent allostatic load variable. Respondents' cardiovascular functioning was determined by their systolic and diastolic blood pressure and pulse. Respondents' lipid metabolism was measured by their body mass index, weight-height ratio, triglycerides, HDL and LDL cholesterol levels. Respondents' glucose metabolism was measured by their insulin, glucose, and hemoglobin levels. Respondents' inflammation was measured by their blood C-reactive protein, serum IL 6, fibrinogen, serum soluble E-selectin, and serum soluble ICAM-1. Respondents' parasympathetic nervous system was measured by four different heart rate variability measures, standard deviation of R-R, root mean squared successive differences in heart rate, low frequency heart rate variability, and high frequency heart rate variability.

Following a similar process of Brooks et al. (2014), risks scores were calculated for each of the five biomarkers. These risk scores were used as the indicators for the latent allostatic load variable. In order to create a risk score for each biomarker, high-risk cut points as reported by Gruenewald et al. (2012) were used. If a score on one of the measures of a biomarker exceeded the high-risk cut point, it was assigned a value of 1. This was done for each of the biomarker measures. Then the total score for each measure was divided by the number of measures to create a risk score for each biomarker. For example, for the cardiovascular functioning measures the high-risk cut point for systolic blood pressure was 143, for diastolic blood pressure the cut point was 82, and for pulse it was 77. If a respondent had a systolic blood pressure score of 150, and a diastolic blood pressure score of 85, and a pulse of 65, two of the measures would be coded as high risk and be given a value of 1. These two scores would be added together and divided by three to give the respondent a risk score of 0.66. A similar process was used for each biomarker and these five risk scores were used as indicators of the allostatic load latent variable. Cut points for each of the indicators used to create biomarker risk scores are reported in Table 1.

*Disease activity.* The disease activity latent variable was measured using two indicators. The first indicator asked the respondents to indicate the number of chronic conditions they experienced in the past month. Respondents could choose from 39 possible chronic conditions including headaches, backaches, stomach trouble, high blood pressure, stroke, ulcer, or high blood sugar. Each chronic condition that the respondent endorsed was added together to form a composite chronic conditions score. The second indicator was a question that asked the respondent to rate their

Table 1  
*High Risk Cut Points For Each Biomarker Indicator*

Physiological system	Indicators of system dysregulation	Cut point
Cardiovascular	Pulse	≥77
	Systolic blood pressure	≥143
	Diastolic blood pressure	≥82
Lipid/Fat metabolism	HDL	≤41.37
	LDL	≥128
	Body mass index	≥32.31
	Weight-height ratio	≥0.97
	Triglycerides	≥160
Glucose metabolism	Glucose	≥105
	Insulin	≥4.05
	Hemoglobin	≥6.10
Inflammation	Serum soluble E-selectin	≥50.58
	Serum soluble ICAM-1	≥329.65
	Blood C-reactive protein	≥3.18
	Serum IL 6	≥3.18
	Fibrinogen	≥390
Parasympathetic nervous system	SDRR	≤23.54
	RMSSD	≤11.83
	Low frequency spectral	≤113.96
	High frequency spectral	≤54.16

health on a scale from 1 to 5; 1 representing “excellent health” and 5 representing “poor health.” These two indicators were used as observed indicators of the disease activity latent variable.

*Differentiation of self.* No established measure of differentiation of self was included in the MIDUS II. However, measures were collected that reflected a respondent’s level of individuality, togetherness, and emotional reactivity. Three measures from MIDUS II—the Autonomy scale, Positive Relationship with Others scale, and the Stress Reactivity scale—were used to operationalize the concept of differentiation of self. The Autonomy scale was used to operationalize the individuality construct. The Positive Relationship with Others scale was used to operationalize the togetherness construct, and the Stress Reactivity scale was used to operationalize the emotional reactivity construct.

The Autonomy scale asks respondent to indicate how much they agreed with the following 7 items: “I am not afraid to voice my opinions, even when they are in opposition to the opinions of most people,” “My decisions are not usually influenced by what everyone else is doing,” “I tend to be influenced by people with strong opinions,” “I have confidence in my opinions, even if they are contrary to the general consensus,” “It’s difficult for me to voice my own opinions on controversial matters,” “I tend to worry about what other people think of me,” and “I judge myself by what I think is important, not by the values of what others think is important.” Responses were measured on a 1–7 scale, with a higher number representing greater autonomy. All items were coded so that higher scores reflect greater autonomy. This scale demonstrated reliability for this sample (Cronbach’s alpha = .69). These questions are similar to other established measures of differentiation which assess a person’s individuality. For example, the Crucible Differentiation Scale (Schnarch & Regas, 2012) asks questions such as, “I have held onto principles and values when it did not make me popular” and “I tell people what I think they want to hear,” to measure a person’s level of individuality.

The Positive Relationship with Others Scale is a 7-item measure that asks how much “Maintaining close relationships has been difficult and frustrating for me,” “Most people see me as loving

and affectionate,” “I often feel lonely because I have few close friends with whom to share my concerns,” “I enjoy personal and mutual conversation with family members and friends,” “I know that I can trust my friends, and they know they can trust me,” and “I have not experienced many warm and trusting relationships with others.” Similar to the Autonomy scale, responses ranged from 1 to 7, with higher scores reflecting more positive relationships with others. This scale demonstrated reliability for this sample (Cronbach’s alpha = .78). These questions reflect aspects of the togetherness construct of differentiation of self. Specifically, Kerr and Bowen (1988) suggest that those with poor differentiation of self find it difficult to develop and maintain close relationships, while those with greater differentiation can sustain and create meaningful close relationships.

The Stress Reactivity Scale consists of three questions that asked respondents to indicate how true the following statements were of them: “My mood goes up and down;” “I sometimes get myself into a state of tension and turmoil as I think of the day’s events;” and “Minor setbacks sometimes irritate me too much.” Responses were coded on a scale from 1 to 4 and coded such that a higher number reflected greater emotional reactivity. This scale demonstrated reliability for this sample (Cronbach’s alpha = .75). These items are similar to emotional reactivity questions used in other established differentiation of self measures. For example, the Differentiation of Self Inventory (Skowron & Friedlander, 1998) use statements such as, “I wish I wasn’t so emotional,” “At times I feel like I’m riding an emotional roller coaster,” and “If I have an argument with my spouse/partner, I tend to think about it all day,” in order to measure emotional reactivity.

### *Data Analysis*

Data were analyzed using a measurement model and two structural equation models. The measurement model examined the fit of the latent variables used in the subsequent models. Specifically, this model tested the fit of the differentiation of self latent variable with three indicators (the Autonomy, Positive Relationship with Other, and the Stress Reactivity scales), the mental health symptoms latent variable with two indicators (the Depressive and Anxiety Symptoms subscales of the MASQ), the latent allostatic load variable with five indicators (cardiovascular, metabolic lipids, metabolic glucose, inflammation, and parasympathetic nervous system functioning), and the disease activity latent variable with two indicators (chronic conditions and self-reported health).

The first structural model (The BBFM Model) replicated previous tests with the MIDUS data and tested the first three hypotheses of the study. Specifically, this first model tested whether there was an association between the family and intimate partner emotional climate observed variables and the biobehavioral reactivity latent variables (mental health symptoms and allostatic load); whether there was an association between the biobehavioral reactivity latent variables and disease activity latent variable; and whether biobehavioral reactivity mediated the association between the family and intimate partner emotional climates and disease activity.

The second structural model (The BBFM and Differentiation of Self Model) was a replication of the first model, but included the differentiation of self latent variable. This model tested whether there were associations between the family and intimate partner emotional climates and differentiation of self; whether there was an association between differentiation of self and the biobehavioral reactivity variables; and whether differentiation of self mediated the association between the family and intimate partner emotional climate variables and the biobehavioral reactivity variables.

All models and mediation analyses were evaluated in Mplus 7.11 (Muthén & Muthén, 2012). Maximum likelihood with robust standard errors (MLR) was used as the estimator. This estimator can account for the non-normality of the data (Asparouhov, 2005). Model fit was evaluated using five fit statistics. Specifically, the chi-square test, the root mean square error approximation (RMSEA), the comparative fit index (CFI), the Tucker-Lewis index (TLI), and the standardized root mean square residual (SRMR) were all used to evaluate the fit of the model to the data. A model that fits the data well will typically have a small, non-significant chi-square statistic, a RMSEA less than 0.05, a CFI and TLI greater than 0.90, and a SRMR less than 0.10 (Kline, 2011).

Mediation analyses for both models were conducted using the MODEL INDIRECT statement in Mplus. Since MLR was used as the estimator, the only mediation method available in Mplus is the delta method (Olkin & Finn, 1995). This method is similar to the Sobel test and has

been recommended as a mediation test that can be used in path analysis that can produce accurate standard errors for non-normal data. (MacKinnon, 2008).

## RESULTS

The means, standard deviations, and range of each of the variables used in the study are reported in Table 2. The correlations of each of the variables with all of the other study variables are reported in Table 3.

### *Measurement model*

The measurement model, which examines the fit of the latent variables used in the subsequent structural models, had good fit for the data ( $\chi^2(48) = 125.55, p < .001$ ; RMSEA = 0.04; CFI = 0.95; TLI = 0.93; SRMR = 0.04). Factor loadings for each of the latent variables are reported in Table 4. The factor loadings of each of the indicators of the differentiation of self variable were greater than 0.50 and all loaded significantly ( $p < .001$ ). The factor loadings of each of the indicators of mental health symptoms variable were greater than 0.70 and all loaded significantly ( $p < .001$ ). The factor loadings for each of the indicators of the allostatic load variable were greater than 0.30 and all loaded significantly ( $p < .001$ ). The factor loadings for each of the indicators of the disease activity variable were greater than 0.40 and all loaded significantly. These results suggested that each of the latent variables provided good fit for the data and could be used to reflect the constructs being tested in the subsequent structural models.

### *BBFM Model*

The first structural model, which tested the first three hypotheses of the study, demonstrated good fit for the data ( $\chi^2(36) = 71.77, p < .001$ ; RMSEA = 0.03; CFI = 0.97; TLI = 0.95; SRMR = 0.03). All fit statistics, with the exception of the chi-square statistic, indicated that the model fit the data well. Standardized beta coefficients for each of the significant pathways are presented in Figure 1. A significant association was found between both the family and intimate

Table 2  
*Descriptive and Distribution Statistics For All Variables*

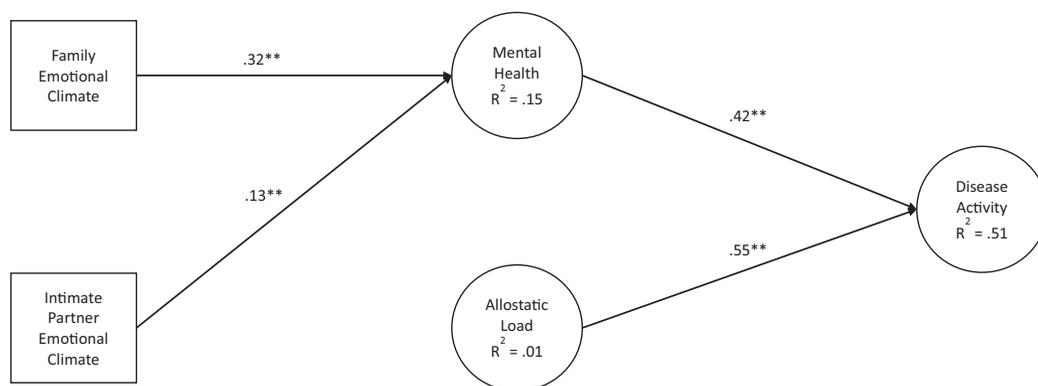
	Mean	Standard deviation	Range
Family emotional climate variables			
Family strain	2.00	0.67	1.00–4.00
Partner strain	2.20	0.61	1.17–4.00
Biobehavioral reactivity variables			
Cardiovascular	0.24	0.27	0.00–1.00
Metabolic lipid	0.24	0.25	0.00–1.00
Metabolic glucose	0.24	0.25	0.00–1.00
Inflammation	0.26	0.26	0.00–1.00
Parasympathetic nervous system	0.26	0.37	0.00–1.00
Anxiety symptoms	16.53	4.54	11.00–42.00
Depression symptoms	18.10	6.34	12.00–60.00
Disease activity variables			
Chronic illnesses	3.95	2.84	0.00–18.00
Self-reported health	2.33	.94	1.00–5.00
Differentiation variables			
Autonomy	37.27	6.69	14.00–49.00
Positive relationships	41.15	6.83	18.70–49.00
Reactivity	6.08	2.25	3.00–12.00

Table 3  
Correlation Matrix For All Variables

	1	2	3	4	5	6	7	8	9	10	11	12	13	14
1. Family strain														
2. Partner strain	.31**													
3. Cardiovascular	-.04	.001												
4. Metabolic lipid	.03	.02	.19**											
5. Metabolic glucose	.02	-.04	.17**	.31**										
6. Inflammation	.09**	.08*	.18**	.26**	.23**									
7. Parasymphathetic nervous system	.01	.02	.16**	.12**	.22**	.20**								
8. Anxiety	.30**	.19**	.07*	.01	-.01	.02	.03							
9. Depression	.31**	.20**	.02	.05	.01	.08*	.01	.68**						
10. Chronic illnesses	.08*	.03	.08*	.07*	.13**	.21**	.21**	.20**	.18**					
11. Self-reported health	.14**	.07*	.12**	.18**	.17**	.23**	.13**	.21**	.28**	.33**				
12. Autonomy	-.15**	-.17**	.07*	.09	.01	.05	.07	-.19**	-.25**	.06	-.06			
13. Positive relationships	-.27**	-.30**	.02	-.06	-.01	-.04	.03	-.27**	-.35**	-.02	-.20**	.37**		
14. Reactivity	.28**	.18**	-.02	.04	.02	.01	-.04	.38**	.41**	.11**	.20**	-.35**	-.42**	

**Table 4**  
*Standardized and Unstandardized Factor Loadings for the Differentiation of Self, Biobehavioral Reactivity, and Disease Activity Latent Variables*

Item	B (SE)	B (SE)
Differentiation of self		
Reactivity	1.00 (–)	0.70 (0.05)
Autonomy	–2.16 (0.22)	–0.51 (0.04)
Positive relationship	–2.71 (0.28)	–0.63 (0.04)
Mental health symptoms		
Anxiety	1.00 (–)	0.75 (0.03)
Depression	1.67 (0.12)	0.90 (0.03)
Allostatic load		
Cardiovascular	1.00 (–)	0.35 (0.04)
Metabolic lipids	1.25 (0.17)	0.50 (0.04)
Metabolic glucose	1.50 (0.23)	0.51 (0.04)
Inflammation	1.40 (0.22)	0.53 (0.04)
Parasympathetic nervous system	1.46 (0.27)	0.39 (0.05)
Disease activity		
Chronic conditions	1.00 (–)	0.49 (0.05)
Self-reported health	0.46 (0.12)	0.67 (0.05)



*Figure 1.* The BBFM Structural Equation Model,  $\chi^2(36) = 71.77, p < .001$ ; RMSEA = 0.03; CFI = 0.97; TLI = 0.95; SRMR = 0.03. \*\* $p < .01$ ; only significant pathways are shown.

partner observed variables and the mental health symptoms latent variable such that greater partner and familial strain was associated with higher levels of depression and anxiety; however, no significant associations were found between the family and intimate partner variables and allostatic load. In other words, greater partner or family strain was not linked to worse allostatic load. Significant associations were found between both biobehavioral reactivity variables and the disease activity variable, such that greater depression, anxiety, and allostatic load was linked to greater disease activity. The family and intimate partner emotional climate variables accounted for 15% of the variance of the mental health symptoms variable ( $R^2 = .15, p < .01$ ) and less the 1% of the variance in allostatic load ( $R^2 < .01, p = .40$ ). All of the explanatory variables in the model accounted for about 50% of the variance of the disease activity variable ( $R^2 = .51, p < .001$ ).

The results of the mediation analysis for the first model are presented in Table 5. These results suggested that the association between family emotional climate and disease activity was mediated

Table 5

Results of the Mediation Analysis for the BBFM Structural Equation Model

	Estimate	Standard error	p-value
Family emotional climate → Disease activity			
Total	0.18	0.05	.00
Indirect			
FEC → BBR → DA	0.14	0.03	.00
FEC → AL → DA	0.04	0.03	.17
Direct			
FEC → DA	0.001	0.05	.98
Intimate partner emotional climate → Disease activity			
Total	0.04	0.05	.44
Indirect			
IPEC → BBR → DA	0.06	0.02	.01
IPEC → AL → DA	0.01	0.03	.64
Direct			
IPEC → DA	-0.03	0.05	.569

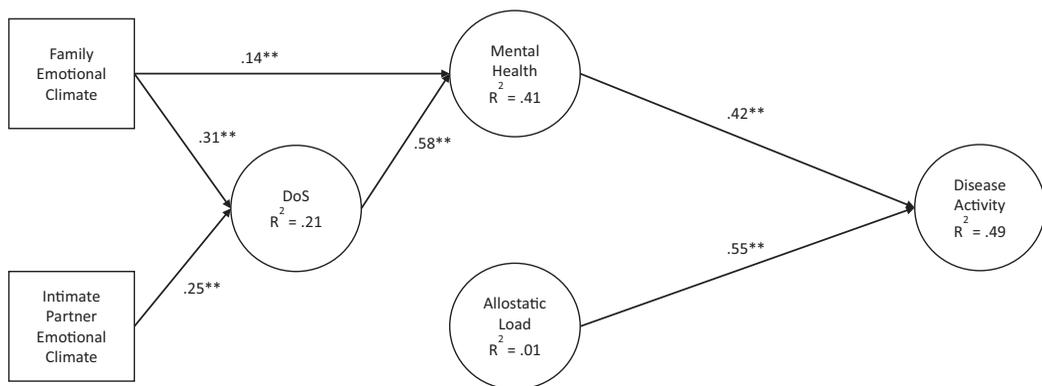


Figure 2. The BBFM and Differentiation of Self Structural Equation Model,  $\chi^2(64) = 156.33$ ,  $p < .001$ ; RMSEA = 0.04; CFI = 0.94; TLI = 0.92; SRMR = 0.04.  $**p < .01$ ; DoS, Differentiation of Self; only significant pathways are shown.

through the mental health symptoms variable, but not through the allostatic load variable. Similarly, the association between the intimate partner emotional climate and disease activity was mediated through the mental health symptoms variable, but not through allostatic load.

*BBFM and Differentiation of Self Model*

The second structural model, which included differentiation of self as a latent variable and tested the last three hypotheses of the study, also demonstrated good fit for the data ( $\chi^2(64) = 156.33$ ,  $p < .001$ ; RMSEA = 0.04; CFI = 0.94; TLI = 0.92; SRMR = 0.04). All fit statistics, with the exception of the chi-square statistic, suggested that the model fit the data well. Standardized beta coefficients for each of the significant pathways are presented in Figure 2. Significant associations were found between both the family and intimate partner emotional climate variables and the differentiation of self variable such that greater conflict was associated with greater reactivity, less autonomy, and poorer togetherness. A direct significant association was found between the differentiation of self variable and the mental health symptoms variable such

Table 6  
*Results of the Mediation Analysis for the BBFM and Differentiation of Self Structural Equation Model*

	Estimate	Standard error	p-value
Family emotional climate → Mental health symptoms			
Total	0.32	0.05	.00
Indirect			
FEC → DOS → BBR	0.18	0.03	.00
Direct			
FEC → BBR	0.14	0.05	.01
Intimate partner emotional climate → Mental health symptoms			
Total	0.13	0.04	.01
Indirect			
IPEC → DOS → BBR	0.15	0.04	.00
Direct			
IPEC → BBR	-0.02	0.04	.71

that less differentiation of self was associated with greater mental health symptoms. However, there was no significant effect between the differentiation of self variable and allostatic load. The family and intimate partner emotional climate variables accounted for 21% of the variance of the differentiation of self variable ( $R^2 = .21, p < .01$ ). The family and intimate partner emotional climate and the differentiation of self variables account for about 41% of the variance in the mental health symptoms variable ( $R^2 = .41, p < .01$ ), but for less than 1% of the variance of allostatic load ( $R^2 < .01, p = .37$ ). All of the explanatory variables in the model accounted for about 50% of the variance of the disease activity variable ( $R^2 = .49, p < .001$ ).

The results of the mediation analysis for the second model are presented in Table 6. These results suggested that the association between family emotional climate and the mental health symptoms variable was mediated through the differentiation of self variable. Similarly, the association between the intimate partner emotional climate and mental health symptoms was also mediated through differentiation of self. Since no significant associations were found between the family and intimate partner emotional climate nor the differentiation of self and allostatic load variables, mediation tests were not conducted for these pathways.

## DISCUSSION

Research applying the BBFM with adults has often lacked focus on clinical application. Theodoratou-Bekou et al. (2012) proposed that differentiation of self could expand the clinical applicability of the BBFM. Specifically, they argued that having greater differentiation of self could reduce the effect that a negative family or intimate partner emotional climate has on biobehavioral reactivity and disease activity. This study sought to test these assumptions by examining if differentiation of self mediated the association between family and intimate partner emotional climate and biobehavioral reactivity. To accomplish this, a model that examined the original pathways of the BBFM was tested first (The BBFM Model). This was then followed by an examination of a second model that included differentiation of self as a mediator between family and intimate partner emotional climate and biobehavioral reactivity (The BBFM and Differentiation of Self Model).

### *The BBFM Model*

Similar to previous tests of the BBFM with MIDUS data (e.g., Priest et al., 2015) the original pathways of the BBFM tested in this study provided good fit for the data and most of the hypothesized pathways were in the predicted direction. It should be noted, however, that contrary to

previous findings of the BBFM (e.g., Priest et al., 2015), no association was found between family or intimate partner emotional climate variables and the allostatic load variable. When Priest et al. (2015) used MIDUS data to test associations between family and intimate partner emotional climate and allostatic load, they found a significant association between family emotional climate and allostatic load, but not between intimate partner emotional climate and allostatic load. However, Priest et al. (2015) examined two models. The first model only explored how the family emotional climate was related to biobehavioral reactivity and disease activity; this model includes both individuals who were single and individuals who were partnered. The second model only explored how intimate partner emotional climate was related to biobehavioral reactivity and disease activity; this model included those individual who were married or partnered. This study combined both family and intimate partner emotional climates into the same model. This was done in order to explore the mediating effect of differentiation of self on the association between family and intimate partner emotional climates and biobehavioral reactivity simultaneously. The combining of both variables into the same model may explain the lack of significance found between the family emotional climate variables and allostatic load.

### *Differentiation of Self Model*

In expanding the BBFM to include a measure of differentiation of self, the goal of this study was to improve the clinical application of the model with adults. Theodoratou-Bekou et al. (2012) argued that the concepts of Bowen's family systems theory were highly compatible with the BBFM. Specifically, they suggested that differentiation of self could be a key mechanism that could reduce the effect that negative intimate partner or family emotional climate could have on an individual's mental and physical health. In other words, if a person is able to balance the pull of togetherness and individuality within a close relationship or relationships and regulate their emotional reactions, even if their family system remain stressful, this individual could avoid negative health outcomes (Kerr & Bowen, 1988; Priest, 2015).

The findings of this study lend support for the inclusion of differentiation of self as a mediating construct in the BBFM. The results suggested that differentiation of self partially mediated the association between family emotional climate and the mental health symptoms variable of biobehavioral reactivity, and between intimate partner emotional climate and mental health symptoms. This is similar to other work that has examined the mediating role of differentiation of self between family factors and mental health (e.g., Priest, 2015). The results here suggest that, for adults, the inclusion of differentiation of self within the BBFM may help expand the clinical utility of the model.

### *Clinical Implications*

As Theodoratou-Bekou et al. (2012) argued, and the results of this study support, including differentiation of self into the BBFM may help inform interventions that use the BBFM as a guide. By targeting differentiation of self, individuals with negative intimate partner and family emotional climates may be able to reduce some of the effects these climates have on their mental and physical health.

Bowen (Kerr & Bowen, 1988) claimed that those with greater levels of differentiation of self are better able to manage conflictual and emotionally charged situations without becoming emotionally reactive. On the other hand, those with less differentiation of self were more likely to fuse or cutoff partners or family member, or triangulate others into conflict in emotionally charged situations. Moreover, those with less differentiation were expected to have higher levels of clinical symptoms such as depression, anxiety, or other physical health problems. Bowen suggested that in order to increase differentiation of self, a person must increase their ability to, "be in emotional contact with a difficult, emotionally charged problem and not feel compelled to preach about what others 'should do', not rush to fix the problem, and not pretend to be emotionally detached by emotionally insulating oneself (Kerr & Bowen, 1988, p. 108)."

In their case example, Theodoratou-Bekou et al. (2012) encouraged increases in differentiation of self for their client in similar ways. For example, Theodoratou-Bekou et al. (2012) encouraged their client, a female adolescent, to set boundaries with family members, to observe herself and her interactions with her family, to question her maladaptive interactions, and to find ways to

become aware of her own emotional reactivity and strengthen her emotion regulation ability. The results of this study lend support to these types of interventions and others aimed to increase a person's differentiation of self. Specifically, the results of this study suggest that if a person has greater autonomy, and less emotional reactivity, they may be able to mitigate the effects of their negative intimate partner and family emotional climates on their mental and physical health.

### *Limitations*

The findings of this study need to be interpreted in the context of the study's limitations. First, the vast majority of the sample was White (86.8%). Moreover, those in the Biomarkers Project tended to be more educated and have higher incomes than the rest of the MIDUS data or the general population (Love et al., 2010). The demographic characteristics of the sample, therefore, limit the generalizability of the findings. Additionally, this was not a clinical sample, and the responses regarding the intimate partner or family emotional climate only reflect one person's view of these relationships. Future research would benefit from studying the BBFM's pathways with multiple family members that are seeking clinical help.

Second, the Biomarkers Project did not include an established measure of differentiation of self. Although the items used to operationalize this variable were similar to established measures of differentiation of self (e.g., Schnarch & Regas, 2012; Skowron & Friedlander, 1998) and other operationalizations of differentiation of self using other large datasets (e.g., Priest, 2015), future research would benefit from exploring the hypotheses tested in this study with an established measure of differentiation of self.

Finally, it is important to note the limitations of doing mediation analyses when using cross-sectional data. Maxwell and Cole (2007) and Maxwell, Cole, and Mitchell (2011) have discussed the biases that arise when using cross-sectional data to examine potential mediating variables. Specifically, they have noted, that in some cases, cross-sectional analysis can yield indirect effects that might not be present in a longitudinal analysis. In other words, a variable that is shown to be a mediator in cross-sectional data may not have an effect when using longitudinal data. Maxwell and Cole (2007) and Maxwell et al. (2011) have recommended the use of autoregressive models to examine the longitudinal effect of mediators. Although there are currently three waves of MIDUS data, they do not all contain the variables of interest used in this study, and therefore using a longitudinal autoregressive model would not be possible. Future waves of MIDUS data collection are expected to include the variables used in this study. When those waves are collected and released, it would be necessary to test the hypotheses presented in this study. However, even with the potential biases of cross-sectional mediation, similar processes have been used in previous tests of the BBFM with children and adults to establish potential mediators, and is common practice to establish potential mediators for subsequent tests of longitudinal mediation (e.g., Lim et al., 2008, 2011; Woods & Denton, 2014).

## REFERENCES

- Asparouhov, T. (2005). Sampling weights in latent variable modeling. *Structural Equation Modeling, 12*(3), 411–434.
- Brooks, K. P., Gruenewald, T., Karlamangla, A., Hu, P., Koretz, B., & Seeman, T. E. (2014). Social relationships and allostatic load in the MIDUS study. *Health Psychology, 33*(11), 1373.
- Gruenewald, T. L., Karlamangla, A. S., Hu, P., Stein-Merkin, S., Crandall, C., Koretz, B., et al. (2012). History of socioeconomic disadvantage and allostatic load in later life. *Social Science & Medicine, 74*(1), 75–83.
- Keogh, E., & Reidy, J. (2000). Exploring the factor structure of the Mood and Anxiety Symptom Questionnaire (MASQ). *Journal of Personality Assessment, 74*(1), 106–125.
- Kerr, M. E., & Bowen, M. (1988). *Family evaluation*. New York, NY: WW Norton & Company.
- Kline, R. B. (2011). *Principles and practice of structural equation modeling* (3rd ed.). New York, NY: The Guilford Press.
- Lim, J., Wood, B. L., & Miller, B. D. (2008). Maternal depression and parenting in relation to child internalizing symptoms and asthma disease activity. *Journal of Family Psychology, 22*(2), 264.
- Lim, J., Wood, B. L., Miller, B. D., & Simmens, S. J. (2011). Effects of paternal and maternal depressive symptoms on child internalizing symptoms and asthma disease activity: Mediation by interparental negativity and parenting. *Journal of Family Psychology, 25*(1), 137.

- 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, 22*(8), 1059–1080.
- MacKinnon, D. P. (2008). *Introduction to statistical mediation analysis*. Mahwah, NJ: Lawrence Erlbaum.
- Maxwell, S. E., & Cole, D. A. (2007). Bias in cross-sectional analyses of longitudinal mediation. *Psychological Methods, 12*(1), 23.
- Maxwell, S. E., Cole, D. A., & Mitchell, M. A. (2011). Bias in cross-sectional analyses of longitudinal mediation: Partial and complete mediation under an autoregressive model. *Multivariate Behavioral Research, 46*(5), 816–841.
- McEwen, B. S. (1998). Protective and damaging effects of stress mediators. *New England Journal of Medicine, 338*(3), 171–179.
- McWilliams, L. A., & Bailey, S. J. (2010). Associations between adult attachment ratings and health conditions: Evidence from the National Comorbidity Survey Replication. *Health Psychology, 29*(4), 446.
- Muthén, L. K., & Muthén, B. O. (2012). *Mplus user's guide: The comprehensive modeling program for applied researchers*. Los Angeles, CA: Muthén & Muthén.
- Olkin, I., & Finn, J. D. (1995). Correlations redux. *Psychological Bulletin, 118*(1), 155.
- Priest, J. B. (2015). A Bowen family systems model of generalized anxiety disorder and romantic relationship distress. *Journal of Marital and Family Therapy, 41*(3), 340–353.
- 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.
- Ryff, C. D., Seeman, T., & Weinstein, M. (2012). *National Survey of Midlife Development in the United States (MIDUS II): Biomarker Project, 2004–2009. ICPSR29282-v6*. Ann Arbor, MI: Inter-university Consortium for Political and Social Research [distributor], 2013-12-20. <https://doi.org/10.3886/icpsr29282.v6>
- Schnarch, D., & Regas, S. (2012). The crucible differentiation scale: Assessing differentiation in human relationships. *Journal of Marital and Family Therapy, 38*(4), 639–652.
- Skowron, E. A., & Friedlander, M. L. (1998). The Differentiation of Self Inventory: Development and initial validation. *Journal of Counseling Psychology, 45*(3), 235.
- Theodoratou-Bekou, M., Andreopoulou, O., Andriopoulou, P., & Wood, B. (2012). Stress-related asthma and family therapy: Case study. *Annals of General Psychiatry, 11*(1), 28.
- Wiebe, S. A., & Johnson, S. M. (2016). A review of the research in emotionally focused therapy for couples. *Family Process, 55*(3), 390–407.
- Wood, B. L. (1993). Beyond the “psychosomatic family”: A biobehavioral family model of pediatric illness. *Family Process, 32*(3), 261–278.
- Wood, B. L., Klebba, K. B., & Miller, B. D. (2000). Evolving the biobehavioral family model: The fit of attachment. *Family Process, 39*(3), 319–344.
- Wood, B. L., Lim, J., Miller, B. D., Cheah, P., Zwetsch, T., Ramesh, S., et al. (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.
- Wood, B. L., Miller, B. D., Lim, J., Lillis, K., Ballow, M., Stern, T., et al. (2006). Family relational factors in pediatric depression and asthma: Pathways of effect. *Journal of the American Academy of Child & Adolescent Psychiatry, 45*(12), 1494–1502.
- 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., & McWey, L. M. (2012). A biopsychosocial approach to asthma in adolescents encountering child protective services. *Journal of Pediatric Psychology, 37*(4), 404–413.
- 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.