Linking Family and Intimate Partner Relationships to Chronic Pain: An Application of the Biobehavioral Family Model

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Introduction: Research is needed to determine mechanisms of effect linking family relationships and chronic pain for adults. Guided by the Biobehavioral Family Model (BBFM), the present study examined indirect effects between a negative family emotional climate and chronic pain disease activity, as mediated by biobehavioral reactivity. *Method:* Data for this study are from the Midlife Development in the United States; specifically, a subsample of participants who reported experiencing chronic pain ($n = 1,461$, ages 32–84). Participants self-reported family strain, biobehavioral reactivity (i.e., anxiety, depression), and chronic pain disease activity (i.e., pain interference, global health). A subsample of participants ($n = 1,070$) completed an intimate partner strain measure, indicating they were married/in a committed relationship. Structural equation models were tested with maximum likelihood estimation and bootstrapping. *Results:* Family strain was indirectly associated with chronic pain disease activity via biobehavioral reactivity—Model 1; $\chi^2(10) = 40.75$, $p < .000$, root mean square error of approximation [RMSEA] $= .07$, comparative fit index [CFI] $= .96$, standardized root-mean-square residual [SRMR] $= .04$; partial mediation occurred for partnered participants. This finding was replicated when modeling family strain simultaneously with intimate partner strain, though intimate partner strain was not associated with chronic pain disease activity—Model 2; $\chi^2(5) = 8.29$, $p = .14$, RMSEA $= .03$, CFI $= .99$, SRMR $= .01$. *Discussion:* These findings add to the growing literature that emphasizes the role of family relationships in chronic pain. Future research is needed to replicate our use of the BBFM to specify pathways of effect, incorporating relational and observational data, with diverse samples.

Keywords: family relationships, intimate partner relationships, chronic pain, depression, anxiety

Greater than 30% of U.S. adults suffer from acute or chronic pain; statistics are worse for older adults (Dahlhamer et al., 2018; Johannes, Le, Zhou, Johnston, & Dworkin, 2010) and more staggering than diabetes, heart disease, and cancer, collectively (American Academy of Pain Medicine, n.d.). Despite the drastic increase of pain reported in the United States, and the rash of opioid research seeking to address an addiction crisis, pain remains misunderstood and challenging to treat (Pergolizzi et al., 2013). Individuals struggling with chronic pain are challenged with financial burden (Campbell, Jordan, & Dunn, 2012), emotional distress (De Souza & Frank, 2011), and physical disability (West, Usher, & Foster, 2011). They also experience systemic effects on their family relationships, which both impact and are impacted by pain experiences (Campbell et al., 2012; Kiecolt-Glaser, Gouin, & Hantsoo, 2010; Reese, Somers, Keefe, Mosley-Williams, & Lumley, 2010).

Existing research has begun to document associations between family and intimate partner

relationships and pain (Barr, Culatta, & Simons, 2013; Reese et al., 2010). However, studies rarely specify mechanisms of effect whereby relationships impact the health of adults experiencing chronic pain (Robles, Slatcher, Trombello, & McGinn, 2014). Because of these complex and not yet understood pathways, a biopsychosocial understanding (Engel, 1977) of how chronic pain is related to and affected by close family relationships and mental health is critical in furthering our understanding of the etiology, maintenance, and treatment of the condition. The present study will use an empirically supported theoretical model, the Biobehavioral Family Model (BBFM; Wood, 1993), as a guide to posit family pain pathways.

BBFM

The BBFM is an evidence-based, systemic, multilevel, biopsychosocial model (Wood, Miller, & Lehman, 2015) that posits a mediation relationship among three constructs, whereby biobehavioral reactivity is the mechanism through which family emotional climate impacts and potentiates disease activity (Wood et al., 2008). The BBFM has been repeatedly substantiated (Woods, Bridges, & Carpenter, 2019) using nationally representative data sets (e.g., Priest & Woods, 2015; Priest et al., 2015), with primary care patients (Woods & Denton, 2014), and in observational studies (Wood et al., 2008). This evidence supports the use of the model in elucidating pathways whereby family relationships affect adult health (Wood et al., 2015; Woods, 2019).

Family Emotional Climate

Family emotional climate is the construct in the BBFM that describes the balance of emotional exchanges within the family that are either positive (e.g., warm, supportive, affirming) or negative (e.g., hostile, critical, attacking), and varying in intensity (Wood et al., 2015). Problematic family functioning is consistently linked to chronic pain severity and disability (Woods, Priest, Kuhn, & Signs, 2019), in part through pain catastrophizing and resulting depressive symptoms (Akbari, Dehghani, Khatibi, & Vervoort, 2016); healthy family functioning is related to fewer pain occurrences (Palermo, Putnam, Armstrong, & Daily, 2007) and decreased pain interference (Woods, Priest, et al.,

2019). Further, marital conflict is associated with an increase in pain (Burns et al., 2013; Grant, Long, & Willms, 2002), whereas greater marital quality has been associated with less pain among rheumatoid arthritis patients (Reese et al., 2010) and less pain-related disability (Robles et al., 2014).

Biobehavioral Reactivity

Biobehavioral reactivity, the link between family emotional climate and disease activity, describes the individual family member's psychophysiological response to emotional stimuli. This mediating construct represents the intensity with which an individual reacts to stress and their capability to regulate emotions. Biobehavioral reactivity is most often operationalized as anxiety or depressive symptoms (Wood et al., 2008; Wood et al., 2015; Woods & Denton, 2014), as these are psychological experiences with physiological components (e.g., psychomotor change, fatigue) reflecting expressions of chronic stress and reactivity to stress (Priest, Roberson, & Woods, 2019; Roberson, Shorter, Woods, & Priest, 2018). Specific to chronic pain, depressed (Carroll, Cassidy, & Cote, 2004) and anxious (Roy-Byrne et al., 2008) individuals are at higher risk for developing intense and disabling pain, whereas longitudinal evidence highlights the development of back pain as an effect of depression (Larson, Clark, & Eaton, 2004).

Disease Activity

Disease activity, the endogenous variable of the BBFM, represents physical health processes contributing to illness that may be activated or exacerbated by experiences of stress (Wood et al., 2015). Recent BBFM research has operationalized disease activity as morbidity, prescription medication use (Priest et al., 2019), and self-rated health (Roberson et al., 2018). To date, no research has expanded the application of the BBFM to a specific health condition for adult family members.

Present Study

Researchers and advocacy groups continue to call for a biopsychosocial approach to examining and treating chronic pain (e.g., National

Institutes of Health Office of Disease Prevention, 2014). The constructs of the BBFM provide an apt overlay to conceptualizing the complex processes that affect the pain experiences of adult family members. Research substantiates the individual associations proposed by the BBFM for this population (Campbell et al., 2012; Vivekanantham, Campbell, Mallen, & Dunn, 2014). The purpose of the present study is to enhance our understanding of the impact of family and intimate partner relationships on chronic pain through the use of the BBFM to guide the specification of a priori mediation hypotheses. Similar to prior research using the BBFM (e.g., Priest et al., 2019; Woods & Denton, 2014), we hypothesize:

1. a significant, direct pathway between negative family emotional climate and biobehavioral reactivity;
2. a significant, direct pathway between biobehavioral reactivity and disease activity (i.e., chronic pain experiences); and
3. a nonsignificant pathway between negative family emotional climate and chronic pain experiences, such that the effects of a negative family emotional climate are fully mediated through the construct of biobehavioral reactivity.

Method

Sample

Data for this study were from the Midlife Development in the United States (MIDUS) study, a longitudinal project to determine biopsychosocial pathways to health and aging (Ryff et al., 2012). Specifically, we use data from MIDUS 2 (collected in 2004–2006), a follow-up to the original MIDUS project (1995–1996; Brim et al., 2011). The present study used data from the project's self-administered questionnaires; the completion rate for this portion of MIDUS II was 81% (Ryff et al., 2012).

Chronic pain subsample. MIDUS 2 included a total of 4,963 English-speaking, U.S. residents aged 32 to 84 years (M age = 55 years, 53% female, 67% reported having a higher level of education than having graduated from high school; Ryff et al., 2012). This study used a subsample of MIDUS 2 participants who

reported experiencing chronic pain (i.e., "pain that persists past the normal time of healing and has lasted anywhere from a few months to many years"; n = 1,461, M age = 58 years, 58% female, 91.4% White). A total of 82.8% (n = 1,210) of these participants reported having seen a physician about their pain. Of the full pain subsample, most participants (n = 1,070) completed the intimate partner strain measure, indicating they were married or living with an intimate partner. Therefore, Model 1 tested our hypotheses with the full chronic pain subsample (n = 1,461, single and partnered) using family strain as the independent variable, and a partnership grouping variable (described below). Model 2 is tested using solely those who reported being in a current intimate partnership (n = 1,070) and included both family strain and intimate partner strain as independent variables.

Measures

Negative family emotional climate was assessed using two separate measures: family strain and intimate partner strain, both developed by Schuster, Kessler, and Aseltine (1990) and Walen and Lachman (2000).

Family strain was measured using four items that asked participants to rate how often members of their family, not including their spouse or partner, "make too many demands," "criticize," "let you down," and "get on your nerves."

Intimate partner strain was measured with six items that asked participants to rate how often their spouse or partner, "makes too many demands," "argues with you," "makes you feel tense," "criticizes," "lets them down," and "gets on your nerves."

Items for both strain measures were coded using a 4-point Likert scale ranging from 1 (*often*) to 4 (*never*). Responses were reverse coded and averaged to create a scale score. The family strain (α = .79) and intimate partner strain (α = .87) measures were reliable for the present sample.

Biobehavioral reactivity was constructed to represent emotion dysregulation, including symptoms of depression and anxiety. Depression and anxiety were both measured using the World Health Organization's (WHO) Composite International Diagnostic Interview-Short Form (CIDI-SF; Kessler et al., 1998). The psychometric properties, including test–retest reli-

ability and diagnostic accuracy, of the CIDI and CIDI-SF, have been broadly substantiated, including with the MIDUS 2 dataset (Busseri & Peck, 2015; Walker & Druss, 2015). The nature of the CIDI-SF scale prevents calculating reliability estimates (Vittengl, 2017) for the present sample.

Depression was assessed using the seven items of the major depression subscale, and included questions such as, "During two weeks in past 12 months, when you felt sad, blue, or depressed, did you feel down on yourself, no good, or worthless?" or ". . . lose your appetite?" Participants' answered 0 (*no*) or 1 (*yes*). "Yes" responses were summed to create a scale score ranging from 0 (*lowest depressed affect*) to 7 (*highest depressed affect*; Ryff et al., 2012).

Anxiety was assessed using the 10 items of the generalized anxiety disorder subscale, and included questions such as, "How often, over the past 12 months, were you restless because of your worry?" or ". . . had trouble falling asleep?" These items were coded using a 4-point Likert scale ranging from 1 (*most days*) to 4 (*never*). Participant responses of "most days" were summed to create a continuous anxiety disorder scale score, ranging from 0 (*lowest anxiety score*) to 10 (*highest anxiety score*).

Chronic pain disease activity was measured using two sets of questions. The first assessed pain interference using five items from the Brief Pain Inventory Short Form (Cleeland, 2009). Items used a 10-point Likert scale ranging from 0 (*did not interfere*) to 10 (*completely interfered*) and assessed pain interference with general activity, mood, relations with other people, sleep, and enjoyment of life. Item responses were averaged to create a pain interference score (Ryff et al., 2012). The scale was reliable for the present sample ($\alpha = .91$).

The second set of questions assessed global health through participants' responses to a single item that asked, "In general, would you say your physical health is excellent, very good, good, fair, or poor?" Responses were coded using a 5-point Likert scale ranging from 1 (*excellent*) to 5 (*poor*).

Analyses

Structural equation modeling (SEM) was used to test the hypothesized mediation relationship between family strain (Models 1 and 2) and intimate partner strain (Model 2) and chronic pain disease activity, through biobehavioral reactivity. Biobehavioral reactivity and disease activity were both included in the models as latent constructs. Given the impact of age on physical health and pain (Briggs et al., 2016; Meisner, Linton, Séguin, & Spassiani, 2017), we included age as a control variable in both models, regressing the effects of age onto disease activity.

Mplus was used to conduct SEM and test indirect effects in both models, using maximum likelihood to account for missing data (Mplus 8, Version 1.6; Muthén & Muthén, 2018). This process estimates standardized path coefficients, standard errors, and model fit. To ensure results were robust, we used bootstrapping (1,000 resamples), and report 95% confidence intervals (CIs) using bootstrap standard errors for pathway estimates and indirect effects, accounting for nonnormality. We avoid using and reporting bias-corrected bootstrapping given concerns regarding increased rates of Type I error (Biesanz, Falk, & Savalei, 2010). In addition, specific to Model 1, we used a multigroup model, entering intimate partnership status as a grouping variable (0 = *single,* 1 = *partnered*). This process estimates Model 1's pathways for both groups, simultaneously, allowing us to ascertain whether the associations between family strain and disease activity occur uniquely for those with, and without, an intimate partnership. These two groups did not significantly differ in their average family strain scores, $F = 1.25$, $p = .265$.

The χ^2 test, the root mean square error approximation (RMSEA), the comparative fit index (CFI), and the standardized root-mean-square residual (SRMR) were used to assess model fit. Good model fit was determined if the RMSEA and SRMR were less than .05, and the CFI value was greater than .95 (Kline, 2016). Although a nonsignificant χ^2 statistic typically indicates a well-fitting model, it is highly sensitive to sample size (Kline, 2016); hence, our use of multiple fit statistics. Empirically based model trimming (i.e., the deletion of statistically nonsignificant pathways; Kline, 2016) was used, as necessary, to achieve parsimony and best model fit.

Results

Model 1: Family Strain

The first model used the full chronic pain sample ($N = 1,443$; 18 were dropped from model estimation due to missing family strain data). Model 1 demonstrated weak fit, $\chi^2(16) = 127.71$, $p < .000$, RMSEA $= .10$, CFI $= .86$, SRMR $= .06$. Although each of our hypothesized pathways were significant for both single ($n = 372$) and partnered ($n = 1,089$) participants, the association of age with pain was nonsignificant ($\beta = .07$, $p = .38$ for single participants; $\beta = .05$, $p = .53$ for partnered participants). Thus, we trimmed age as a control variable.

Trimming age improved model fit, $\chi^2(10) = 40.75$, $p < .000$, RMSEA $= .07$, CFI $= .96$,

SRMR $= .04$, though the χ^2 value differed by partnership status (i.e., $\chi^2 = 28.38$ for single participants; $\chi^2 = 12.37$ for partnered participants). For single individuals, family strain was significantly associated with biobehavioral reactivity ($\beta = .24$, $p = .027$, CI [.06, .49]), which, in turn, was associated with disease activity ($\beta = .67$, $p < .000$, CI [.46, 1.01]). In accordance with our hypothesis, the direct association between family strain and pain was nonsignificant ($\beta = .14$, $p = .34$, CI [$-.15$, .28]). For single adults, Model 1 explained 51% of the variance in the chronic pain latent construct ($R^2 = .51$, $p = .039$; Figure 1). Similarly, for partnered individuals, family strain and biobehavioral reactivity were significantly associated ($\beta = .30$, $p < .000$, CI [.17, .41]), as were biobehavioral reactivity and disease activity ($\beta =$
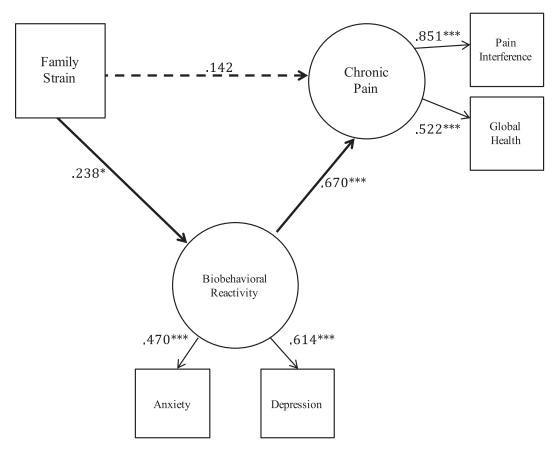

Figure 1. Standardized path coefficients for Model 1, family strain, for single participants ($n = 372$). Model fit indices: $\chi^2(10) = 28.38$, $p < .000$, RMSEA $= .07$, CFI $= .96$, SRMR $= .04$. * $p < .05$. *** $p < .001$.

.37, p < .000, CI [.24, .54]). However, contrary to our hypothesis, family strain was directly, significantly associated with the chronic pain latent variable for this partnered subsample (β = .12, p = .012, CI [.03, .22]). Model 1 explained 18% of the chronic pain variance observed for part-nerned adults (R^2 = .18, p = .000; Figure 2). Sample covariance for both groups is presented in Table 1.

Tests of indirect effects demonstrated a significant mediation relationship for single participants, and a partial mediation relationship (Frazier, Tix, & Barron, 2004) for participants in an intimate partnership. For the single group, we observe an asymmetrical confidence interval distribution, such that 0 is not within the interval, indicating a significant effect despite the nonsignificant p value. For the partnered

group, although the direct association between family strain and disease activity remained significant, the indirect effect of family strain on chronic pain, through the mediating variable of biobehavioral reactivity, was also significant (see Table 2).

Model 2: Family and Intimate Partner Strain

Model 2 used the portion of the sample that reported being in an intimate partner relationship (n = 1,065; five participants were dropped from model estimation due to missing family strain data). Similar to Model 1, Model 2 demonstrated a moderate fit to the data, $\chi^2(8)$ = 61.86, p < .000, RMSEA = .08, CFI = .90, SRMR = .04. Age was nonsignificant (β =
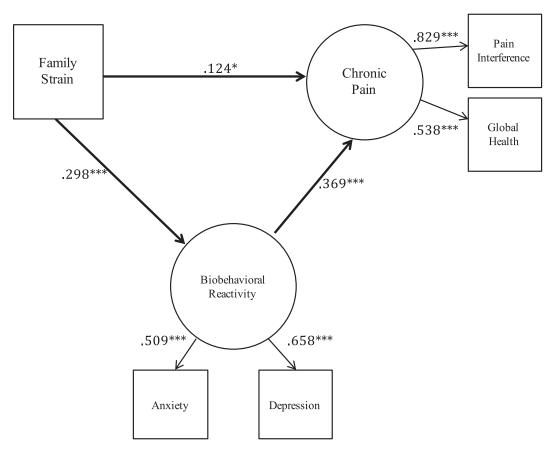

Figure 2. Standardized path coefficients for Model 1, family strain, for partnered partici-pants (n = 1,089). Model fit indices: $\chi^2(10)$ = 12.37, p < .000, RMSEA = .07, CFI = .96, SRMR = .04. * p < .05. *** p < .001.

Table 1

Sample Covariance for Model 1 (N = 1,443)

Variables	1	2	3	4	5
Single participants (*n* = 372)					
1. Global health	1.134				
2. Depression	.624	4.526			
3. Anxiety	.207	.753	1.519		
4. Pain interference	1.146	1.892	.803	5.860	
5. Family strain	.022	.197	.093	.453	.417
Partnered participants (*n* = 1,089)					
1. Global health	1.077				
2. Depression	.258	3.351			
3. Anxiety	.178	.654	1.125		
4. Pain interference	1.130	.970	.433	5.961	
5. Family strain	.058	.216	.103	.306	.376

.006, *p* = .63) and therefore trimmed from the model.

Model trimming greatly improved fit, $\chi^2(5) = 8.29$, *p* = .14, RMSEA = .03, CFI = .99, SRMR = .01; 18% of chronic pain variance was explained (R^2 = .18, *p* = .001; Figure 3). Results supported our hypotheses, such that family strain (β = .26, *p* < .000, CI [.15, .38]) and intimate partner strain (β = .11, *p* = .022, CI [.02, .20]) were both significantly associated with biobehavioral reactivity, though the latter to a lesser extent. Biobehavioral reactivity was, in turn, associated with the latent chronic pain dependent variable (β = .37, *p* < .000, CI [.24, .53]). As family and partner strain increase, depression and anxiety also increase; an increase in reactivity is associated with greater

chronic pain. The direct pathway between family strain (β = .11, *p* = .019, CI = [.006, .19]) and chronic pain was significant, as was true for these participants in Model 1, while the direct pathway between intimate partner strain (β = .03, *p* = .574, CI [−.07, .11]) and chronic pain was nonsignificant. Sample covariance is presented in Table 3.

Tests of indirect effects confirmed full mediation for family strain as the independent variable, whereas the indirect association of intimate partner strain with pain via biobehavioral reactivity is conservatively judged to be nonsignificant (i.e., though the confidence interval did not include 0, the lower limit neared 0, and the *p* value was > .05; Table 4).

Discussion

Findings provide support for an indirect pathway of negative family emotional climate, as measured by family strain, on pain via depression and anxiety, as posited by the BBFM. This was observed in Model 1, for single and partnered participants, though the association was solely partially mediated for the partnered subsample. However, despite replicating this finding in Model 2, the intimate partner strain pathway was minimally impactful. In other words, though intimate partner strain was associated with biobehavioral reactivity, the size of the effect was weaker than family strain, and the indirect association between partner strain and

Table 2

Mediation of the Effect of Family Strain on Chronic Pain Through Biobehavioral Reactivity in Model 1 (N = 1,443)

Group	Point estimate	*SE*	*p*	95% CI	
				Lower	Upper
Single participants (*n* = 372)					
Total	.302	.058	.000	.179	.406
Direct	.142	.149	.339	−.196	.269
Family strain → pain					
Indirect	.160	.145	.270	.041	.494
Family strain → BBR → pain					
Partnered participants (*n* = 1,089)					
Total	.234	.040	.000	.160	.323
Direct	.124	.050	.012	.028	.222
Family strain → pain					
Indirect	.110	.037	.003	.053	.195
Family strain → BBR → pain					

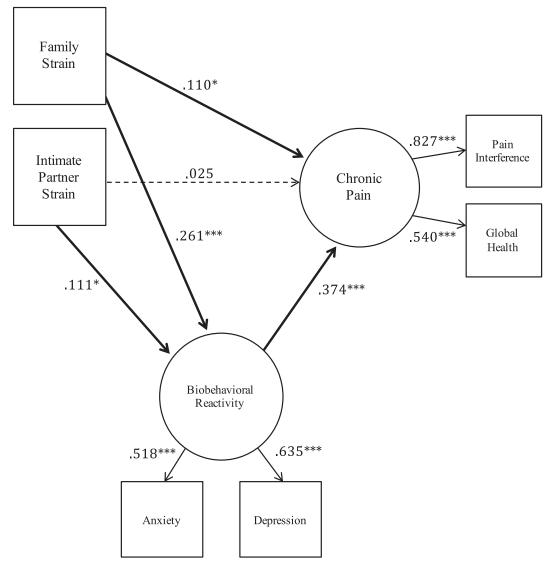*Note.* CI = confidence interval; BBR = biobehavioral reactivity; 1,000 bootstrap samples.

Figure 3. Standardized path coefficients for Model 2, family and intimate partner strain ($n = 1,065$). Model fit indices: $\chi^2(5) = 8.29$, $p = .14$, RMSEA $= .03$, CFI $= .99$, SRMR $= .01$. * $p < .05$. ** $p < .01$. *** $p < .001$.

pain via depression/anxiety was deemed non-significant.

Though contrary to our hypothesis when deciding to operationalize negative family emotional climate, in part, using intimate partner strain, these findings align with similar effects found in the literature. Despite an emphasis in the literature on intimate partner relationship quality in influencing adult health (Woods,

Bridges, et al., 2019), newer research has begun to demonstrate stronger associations between nonmarital family relationships and adult health as compared to intimate partnerships (e.g., Priest et al., 2019; Woods et al., 2019). Further, though the BBFM theorizes a mediation relationship among these variables, we found a significant association between family strain and chronic pain for partnered participants. Though

Table 3
Sample Covariance for Model 2 (N = 1,065)

Variable	1	2	3	4	5	6
1. Global health	1.081					
2. Depression	.262	3.315				
3. Anxiety	.183	.631	1.113			
4. Pain interference	1.131	.952	.424	5.938		
5. Family strain	.059	.211	.101	.299	.370	
6. Intimate partner strain	.041	.162	.062	.188	.142	.398

these effects should be interpreted tentatively, given the cross-sectional nature of the data, it is possible results indicate a longitudinal association between pain and family strain. Given the life span trajectories of family relationships *other* than an intimate partner/spouse (e.g., parents, siblings, adult children), the present findings may indicate a reciprocal relationship over time between persistent pain and family relationship quality that serves to strengthen the association found here. Additional research is needed to test temporal and causal effects between family strain and pain, using repeated measures over multiple timepoints.

Overall, the present findings highlight that the BBFM may help to explain health outcomes for adults experiencing chronic pain. Specifically, for this sample, feeling like one's family members are demanding or criticizing is associated with greater depression and anxiety which is, in turn, associated with worse global health and pain interference. This finding is an important next step in exploring pathways between close family relationships and chronic pain.

Limitations and Future Research

Although this study addressed several gaps in the literature, it also has limitations for which future research is necessary. First, though the use of secondary data is beneficial (Smith et al., 2011), researchers are restricted in utilizing specific samples and measures. For example, this study was limited to self-reported relationship strain. The use of other types of relational measures (e.g., assessing relational process, observational measures, dyadic assessments, etc.) could further tease out in which ways families affect and are affected by pain. Further, the family strain measure specified participants should report on their family relationships *other* than their intimate partnership. However, participants did not specify which family relationships they were evaluating, despite evidence this impacts participant responses on family assessments (Priest, Parker, & Woods, 2018). In addition, MIDUS is limited in the diversity of its sample; as a result, generalizability is limited by a mostly White sample. Further research is needed to account for race/ethnicity in family

Table 4
Mediation of the Effect of Family and Intimate Partner Strain on Chronic Pain Through Biobehavioral Reactivity in Model 2 (N = 1,065)

Variable	Point estimate	SE	p	95% CI Lower	95% CI Upper
Total: Intimate partner strain	.066	.041	.104	−.056	.454
Total: Family strain	.207	.040	.000	.415	.964
Direct					
Intimate partner strain → pain	.025	.044	.574	−.204	.345
Family strain → pain	.110	.047	.019	.019	.669
Indirect					
Intimate partner strain → BBR → pain	.042	.022	.054	.025	.279
Family strain→ BBR → pain	.098	.032	.003	.170	.537

Note. CI = confidence interval; BBR = biobehavioral reactivity; 1,000 bootstrap samples.

pain associations. Lastly, this study used cross-sectional data to explore the quality of close family relationships on health, thus limiting our ability to determine the temporal ordering of our variables. Longitudinal research is needed to better understand the possible underlying effects of relational quality on health (Robles et al., 2014), and more specifically, the causal pathways between close family relationships, biobehavioral reactivity, and chronic pain experiences over time.

Clinical Implications

The theoretical foundation of the BBFM, as well as the present findings, indicate that treatment for adults with chronic pain may benefit from including family-based interventions. Further, the patterns observed presently suggest it may be more important to consider the role of family, other than intimate partners, in considering the associations between strained close relationships and pain. This is an assertion supported by recent research identifying family level variance in chronic pain outcomes (Woods, Priest, et al., 2019). As one example of BBFM-guided intervention, the family emotional climate construct includes proximity, triangulation, and family responsivity (Wood, 1993). Each are specific areas where clinicians can observe and intercede. Wood et al. (2015) suggested that the BBFM indicates the need for providers to view a patient's family relationships, to assess for both emotional distress and chronic illness, and to openly discuss the impact of stress and family members' own depression and anxiety on health in order to create change within the family. Using the BBFM as a clinical guide may also help to highlight which family members are needed for specific aspects of care (Wood et al., 2015) and would serve to address the need for therapeutic approaches based in theory (Shields, Finley, Chawla, & Meadors, 2012). Given the powerful role of family for health, and, presently, for adults with chronic pain, medical family therapists (McDaniel, Doherty, & Hepworth, 2014) may be especially useful in this arena.

Conclusion

Despite the fact that 70 to 100 million individuals, or more, suffer from chronic pain in the United States (Dahlhamer et al., 2018; Institute of Medicine, 2011), pain continues to be diffi-cult to treat and manage (Pergolizzi et al., 2013). Because of the complexity of this condition, individuals experience financial burden (Campbell et al., 2012), and emotional (De Souza & Frank, 2011) and physical distress (West et al., 2011). In contrast, individuals in healthy close relationships report fewer chronic pain experiences (Reese et al., 2010). To best understand connections between close family relationships and pain, a systemic biopsychosocial model is necessary. The present findings add to the growing body of literature that emphasizes the role of family relationship quality in chronic and persistent pain. Our results highlight a significant pathway whereby family strain is associated with chronic pain disease activity, through individuals' depression and anxiety symptoms. These findings support the need for further research elucidating mechanisms of effect linking family relationships and pain, using the BBFM to specify mediational pathways.

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