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Family emotional climate and health: Testing conveyance of effects via psychobiological mediators

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Abstract

Using the Biobehavioral Family Model and data from five Midlife in the United States projects ($N = 793$), this study tests whether allostatic load and negative affect reactivity convey the effects of categorizations of family emotional climate on health appraisal and morbidity (chronic conditions) across 20 years. Results indicated that negative family emotional climate (high strain, low support/parental affection) was indirectly associated with health appraisal 20 years later via negative affect reactivity at 10 years. Ambivalent family emotional climate (high strain/support) was directly associated with greater morbidity. Allostatic load did not serve as a significant mediator in the models tested. Findings suggest that family emotional climates marked by strain and intensity may be especially problematic for health, partly by exacerbating negative affect reactivity.

KEYWORDS

daily diary methods, families, health, psychophysiology

1 | INTRODUCTION

Overwhelming evidence has established that close relationships impact health. Among types of close relationships, family relationships have the strongest effects. Family members provide greater emotional support than friends and nonrelatives (Shor & Roelfs, 2015; Shor, Roelfs, &

Yogev, 2013). Family members are also greater sources of negative affect and relational strain (Priest, Roberson, & Woods, 2019; Yang, Schorpp, & Harris, 2014). The quality of family relationships is repeatedly demonstrated as the crux of how these persons affect our health. In other words, family relationships that are positive and supportive are associated with improved health outcomes (Holtfreter, Reisig, & Turanovic, 2016; Shor et al., 2013), whereas stressful, strained family relationships are linked to worse mental and physical health and mortality (Guevara & Murdock, 2019; Holt-Lunstad, Smith, & Layton, 2010; Priest, 2013).

Yet, research is only beginning to explore the mutual effects of positivity and negativity in relationships (Ross, Rook, Winczewski, Collins, & Dunkel Schetter, 2019). Studies most often test either positive or negative qualities of close relationships or assess both but compare their relative effects on health (Ross et al., 2019). However, people frequently experience both positivity *and* negativity in their relationships. Uchino, Holt-Lunstad, Uno, and Flinders (2001) provided initial evidence of the unique effect of what they term "ambivalent" relationships (i.e., relationships characterized by high positivity and high negativity) on cardiovascular reactivity to stress (p. 363). Ambivalent relationships have since been associated with greater functional health limitations than solely negative relationships, which may be more strongly associated with worse psychological well-being (Rook, Luong, Sorkin, Newsom, & Krause, 2012). Uchino et al. (2012) found that the number of ambivalent relationships, but not the number of negative relationships, was associated with more rapid aging as indicated by shorter telomeres. In addition, ambivalence in relationships has been tied to greater inflammation (Uchino et al., 2013) and coronary–artery calcification (Uchino, Smith, & Berg, 2014). Overall, Holt-Lunstad and Uchino (2019) suggest that, when an individual's constellation of relationships includes ambivalent connections, the result is great interpersonal, and intra-individual, stress.

However, a limitation of the ambivalence–health literature, as well as the relationships and health literature more broadly, is its focus on marital relationships (Woods, Bridges, & Carpenter, 2019). This is despite the continued decline in married adults and rise in adults who never marry or wait to do so until later in life, as well as an increase in adults who do not live with a spouse but increasingly with other relatives (Carr, 2019; Woods, Bridges, et al., 2019). Recent research has demonstrated that the effects of family relationships, with family members *other* than a spouse or intimate partner, have potentially *greater* effects on long-term health outcomes than the quality of intimate partner relationships (Woods, Priest, & Roberson, 2019). As adults age, their intergenerational ties are at least as important as their romantic ones, and there is important growth in the literature documenting the effects of parent–child, –stepchild, and –grandchild relationships on adults' health (Carr & Utz, 2020). Furthermore, given the longitudinal impact of these nonintimate family relationships (e.g., parents, siblings, children), especially those occurring early in life, examining both childhood and adulthood family relationship quality (i.e., support and strain) may provide a meaningful window into how families affect health.

In addition, gaps remain in how we understand the specific mediating pathways that link family relationships and physical health over time (Farrell, Imami, Stanton, & Slatcher, 2018; Uchino, Bowen, Kent de Gray, Mikel, & Fisher, 2018). Two specific pathways that have garnered support include psychological and biological mechanisms. However, few studies have examined specific psychological pathways that may link close relationships and health (Uchino et al., 2018), despite the promise of affective processes as a mechanism of effect (Farrell et al., 2018). Recently, research has found support for negative affect reactivity (i.e., negative affect increase in response to stressor exposure), specifically as a mediator linking change in

perceived partner responsiveness over 10 years to later mortality (Stanton, Selcuk, Farrell, Slatcher, & Ong, 2019). Furthermore, allostatic load (i.e., chronic physiological reactivity across multiple systems in response to stress; McEwen, 1998) has been linked to family strain (Priest et al., 2015), although evidence is still needed for this composite measure of biological dysregulation as a mediator linking families and health within a comprehensive, longitudinal model (Priest et al., 2019; Wiley, Bei, Bower, & Stanton, 2017). Finally, although theoretical models posit both psychological and biological pathways, few studies have simultaneously tested these mechanisms of effect. As Pietromonaco and Collins (2017) recommend, "testing integrated models including multiple mediators will facilitate an understanding of how social relationships may translate into health outcomes" (p. 537).

1.1 | The Biobehavioral Family Model

The current study uses the Biobehavioral Family Model (BBFM; Wood, 1993; Wood et al., 2008) as a theoretical framework guiding the development of mediational hypotheses. This systemic model includes three constructs: *family emotional climate* (the emotional intensity and valence of family relationships), *biobehavioral reactivity* (individual family members' psychophysiological responses to stress), and disease activity (frequency and intensity of illness). The BBFM is the most explicit and empirically supported biopsychosocial theoretical model (Woods, 2019), and it has been substantiated with lab-based family interaction studies (Wood et al., 2008) and multiple adult populations (Priest et al., 2015; Priest et al., 2019; Woods & Denton, 2014).

1.1.1 | Family emotional climate

Prior research testing the BBFM's pathways for adults has conceptualized family emotional climate (FEC) as the quality of nonintimate family relationships, intimate partner relationship quality, social support received from friends and relatives, and adverse childhood experiences including abuse and neglect (Priest et al., 2019; Roberson, Shorter, Woods, & Priest, 2018; Signs & Woods, 2020; Woods & Denton, 2014; Woods, Priest, & Roush, 2014). As the construct specifies that the emotional climate of a family is marked by its positivity and negativity, as well as the intensity of each (Wood, 1993), it aligns closely with the model of social relationships specified by Uchino et al. (2001), thus accounting for positive (supportive) and negative (aversive) relationships, as well as ambivalent and indifferent ones.

In addition, the BBFM is applied across the developmental lifespan, theorizing pathways to health for children and adults (Wood & Miller, 2005; Woods & Denton, 2014). Similarly, Holt-Lunstad and Uchino (2019) specify the contributions of early family environment to the development of ambivalence in relationships. Recent research has supported testing both the effects of early family relationships and concurrent relationships on adult health outcomes, using the BBFM (Priest et al., 2019). This study and others (Signs & Woods, 2020) testing the BBFM have used both observed variables and latent constructs (Priest et al., 2015; Woods & Denton, 2014) to operationalize FEC. Thus, the present test would be the first to our knowledge to simultaneously model how these markers of FEC cluster and combine in unique latent classes to reflect a more authentic, multi-dimensional representation of close family relationships.

1.1.2 | Biobehavioral reactivity

Importantly for this study, biobehavioral reactivity operates as the mediating construct through which family emotional processes affect disease outcomes. Specifically, Wood, Miller, and Lehman (2015) specify biobehavioral reactivity is "the degree or intensity with which an individual responds physiologically, emotionally, and behaviorally to emotional challenge" (p. 382). In the context of warm, emotionally supportive, and responsive family relationships, biobehavioral reactivity is expected to be well-moderated, and the effects of negative interactions and distress, when present, will be buffered by regulated emotion and physiological stress reactions. However, individuals embedded in hostile, critical, and stressful families become increasingly dysregulated, which potentiates the effects of external, relational stress on disease processes.

Though often operationalized as depression, anxiety, and emotion dysregulation (Wood et al., 2015; Woods & Denton, 2014), Wood (2019) asserts that this mediating construct is also reflective of allostasis/allostatic load. Allostasis represents the body's physiological response to stress with the goal of maintaining homeostasis and balance, a process that is often protective and especially helpful in the face of immediate or emergent stressors (McEwen, 1998, 2005). These adaptations are conveyed via chemical mediators, such as cortisol and adrenaline, as well as glucocorticoids and blood pressure, as examples; each prime the body to respond to challenge. However, when the processes of allostasis are chronically activated, and dysregulated, these stress responses can be detrimental to physical health. Allostatic load, as defined by McEwen (1998) refers to the result of repeated stress reactivity across the body's many systems (e.g., immune, cardiovascular, sympathetic nervous system) that accumulates and manifests as physiological wear and tear. In reflecting the totality of the body's stress reactivity, the construct of allostatic load also provides an advantage over measuring solitary biological parameters to model health risk (Seeman, Singer, Ryff, Love, & Levy-Storms, 2002). As such, it represents a prime operationalization of biobehavioral reactivity, which specifies the conveyance of family impacts on health via, in part, physiological reactions to emotional distress. Testing allostatic load as a biobehavioral reactivity variable has only recently garnered evidence in support, and, along with emotion dysregulation, been found to link FEC and disease activity (Priest et al., 2015; Priest et al., 2019).

Overall, the BBFM provides an apt overlay to the current study as its conceptualization of FEC accounts for both the quality and intensity of the relationships. Furthermore, the model posits psychological and physiological links between close family relationships and physical health outcomes, providing a specific, theorized mechanism of effect.

1.2 | Present study

Few studies have tested the full indirect relationship between FEC, biobehavioral reactivity, and physical health longitudinally (Farrell et al., 2018).

We will do so with three time points across 20 years through the use of the Midlife in the United States project (MIDUS; Brim et al., 2018). This study will first test a composite of negative and positive qualities of family relationships and perform a second test on how these FEC composites operate to impact midlife health outcomes via specific psychobiological mediators. We will do so guided by the BBFM, an evidence-based theoretical approach to specifying how the emotional climate of families gets "under the skin" of individual family members to impact their disease activity. We will investigate psychological and biological mediators (i.e., allostatic

load and negative affect reactivity) individually, and in conjunction, as an advance on current science. As specified by the BBFM, we will test the following longitudinal, mediational hypotheses:

Hypothesis 1 *A more negative FEC (including those with high strain and low support and ambivalent climates with high strain and high support) will predict significantly greater biobehavioral reactivity (i.e., greater allostatic load, greater negative affect reactivity).*

Hypothesis 2 *Greater biobehavioral reactivity will predict significantly worse health outcomes.*

Hypothesis 3 *FEC will produce a nonsignificant direct effect on long-term health outcomes, such that the effect of family relationships on health is indirect, through biobehavioral reactivity.*

Findings may provide the advantage of identifying how unique multifactorial composites of family relationships can influence health, incorporating support and strain and family of origin, as well as current family relationship quality. This test allows us to examine whether positive, negative, or ambivalent family relationships have a greater impact on health in adulthood, and in what configuration. Moreover, our purpose is to provide evidence of how the quality of these relationships is conveyed across adulthood to affect physical health.

2 | METHOD

2.1 | Sample

MIDUS (Brim et al., 2018) data collection began in 1995 and continues today, with waves released at 10-year intervals. The project is designed to examine biopsychosocial impacts on aging and is uniquely suited to test the present pathways due to a rich collection of relational measures and the inclusion of biomarker and daily diary projects in MIDUS 2. This study used data from five MIDUS projects, including the main surveys at each of the three waves (i.e., MIDUS 1, 2, and 3), as well as the Biomarker Project (Ryff, Seeman, & Weinstein, 2019) and the Daily Stress Project 2 (Ryff & Almeida, 2017). A full timeline of MIDUS data collection can be accessed via midus.wisc.edu/data/timeline.php. As the data are deidentified, and publicly available, this project did not require institutional review board approval.

MIDUS 1 (Brim et al., 2018) data were collected during 1995–1996 and comprised 7,108 participants, including a random digit dialing sample, siblings, an oversampling of five metropolitan areas, and a random digit dialing sample of twin pairs. Respondents were 51% female, with an average age of 46.38 years ($SD = 13.0$). MIDUS 2 (Ryff et al., 2017) was a follow-up study, conducted in 2004–2006, and comprised 70% of MIDUS 1 participants ($n = 4{,}963$; 53% female, M age = 55.43 [$SD = 12.45$]). MIDUS 3 (Ryff, Almeida, et al., 2019) data were collected during 2013–2014, as a follow up to MIDUS 1 and 2. MIDUS 3 is comprised of 3,294 (46%) of the original MIDUS 1 respondents (66% of MIDUS 2).

The Biomarker Project, or Project 4 (Ryff, Seeman, et al., 2019), was a subproject of MIDUS 2 with the intention of integrating comprehensive biological assessments into the MIDUS portfolio. Data were collected between 2004 and 2009, at three General Clinical Research Centers via a lab-based protocol, including a fasting blood draw, 12-hr urine collection, and saliva

specimens. This project was comprised of 1,054 (or, 36%) of MIDUS 1 participants (56.8% female, *M* age = 54.52 years [*SD* = 11.71]).

Finally, the Daily Stress Project 2 (Ryff & Almeida, 2017) was a follow up to the MIDUS 1 National Study of Daily Experiences to examine day-to-day experiences of stress. The project collected participant data across eight evenings and consists of daily coded telephone interviews. The project is comprised of 1,842 core MIDUS participants and is 57.2% female, with an average age of 56.24 years.

Use of participants who completed these five MIDUS projects resulted in a sample size of 793 for this study. Of these, 55.5% were female, and the majority were White (91.2%; 2.6% Black/African American, 1.8% Asian or Pacific Islander, all other races reported <1%) and non-Hispanic (96.0%), with an average age of 45.81 years (range 25–74; *SD* = 10.96). This sample additionally reports a median household income (including wages/personal income, pension, social security, and other government assistance) of $68,500 (*M* = $82,708.01, *SD* = $59,758.41), and 73.1% reported having completed some college or greater education.

2.2 | Measures

2.2.1 | Independent variables

We operationalized the BBFM's FEC construct in MIDUS 1 using participant reports of family strain and support and maternal and paternal affection during childhood. The former two measures reflect the quality of current family relationships (*other* than intimate partnerships; Walen & Lachman, 2000); the latter two measures reflect the quality of parental relationships experienced in childhood (Rossi, 2001). A latent class analysis (LCA) of the four FEC measures was conducted (described below) to categorize participants according to overall FEC valence and intensity.

Each of these four measures was completed via the MIDUS 1 self-administered questionnaire. MIDUS researchers used mean imputation to accommodate missing responses; participants were assigned scale scores if they answered at least one item on a measure (Ryff, Almeida, et al., 2019). Descriptive statistics for each are presented in Table 1.

Family strain

The family strain measure (Walen & Lachman, 2000) includes four items preceded by the prompt, "Other than your spouse/partner, how often do your family members": (a) make too many demands on you; (b) criticize you, (c) let you down when counting on them; and (d) get on your nerves. Responses ranged from 1 (*often*) to 4 (*never*). Responses were reversed coded and averaged such that higher scores reflect greater family strain. Previous research using this measure with MIDUS data has shown it to be reliable (Priest et al., 2019).

Family support

The family support measure (Walen & Lachman, 2000) similarly includes four items assessing how much members of participants' families (excluding their spouse/partner) (a) care about them; (b) understand the way they feel; (c) can be relied upon; and (d) how much they can be opened up to emotionally. Responses ranged from 1 (*often*) to 4 (*never*). Responses were reverse coded and averaged such that a higher average reflects greater family support. This measure has also been shown to be reliable in previous research (Priest et al., 2019).

TABLE 1 Participant reports of family emotional climate and intimate partner relationship quality: Correlations and descriptive statistics ($N = 793$)

Variables	1	2	3	4	5	6	7	8	9	10	11	12	13	14	15
1. Family strain	—														
2. Family support	−0.441***	—													
3. Maternal affection	−0.273***	0.391***	—												
4. Paternal affection	−0.204***	0.398***	0.452***	—											
5. Allostatic load	0.030	0.019	0.019	0.023	—										
6. Negative affect[a]	0.219***	−0.151***	−0.118**	−0.113**	0.008	—									
7. T3 health appraisal	−0.181***	0.155***	0.090*	0.110**	−0.217***	−0.168***	—								
8. T3 morbidity	0.179***	−0.055	−0.061	−0.061	0.195***	0.264***	−0.458***	—							
9. T1 health appraisal	−0.089*	0.121**	0.122**	0.083*	−0.165***	−0.117**	0.430***	−0.332***	—						
10. T1 morbidity	0.203***	−0.168***	−0.179***	−0.137**	0.149***	0.237***	−0.276***	0.482***	−0.355***						
11. Sex[b]	0.194***	0.027	−0.196***	−0.044	−0.009	0.044	0.000	0.129**	−0.081*	0.178***	—				
12. Age	−0.168***	0.129***	0.055	0.061	0.347***	−0.192***	−0.017	0.089*	−0.021	0.063	−0.036	—			
13. Education[c]	0.002	−0.032	0.064	0.117**	−0.130**	−0.037	0.134***	−0.100**	0.137***	−0.024	−0.116**	−.054	—		
14. Intimate partner strain[d]	0.309***	−0.186***	−0.140**	−0.147***	0.046	0.231***	−0.059	0.063	−0.037	0.154***	0.059	−.055	.053	—	
15. Intimate partner support[d]	−0.202***	0.262***	0.183***	0.137**	−0.073	−0.125**	0.096*	−0.022	0.016	−0.115**	−0.099*	.091*	−.051	−0.676***	—

TABLE 1 (Continued)

Variables	1	2	3	4	5	6	7	8	9	10	11	12	13	14	15
M	2.12	3.47	3.08	2.65	0.27	0.17	3.52	2.75	3.77	2.17	0.55	45.81	.73	2.19	3.63
SD	0.59	0.59	0.71	0.80	0.14	0.18	1.03	2.56	0.90	2.15	0.50	10.97	.44	0.59	0.52
α	.78	.82	.91	.93	—	—	—	—	—	—	—	—	—	.87	.90

Note: T1 = Time 1; T3 = Time 3.
[a]Computed as an average of daily means for the purposes of this table;
[b]Sex: 0 = *male*, 1 = *female*.
[c]Education: 0 = *graduated high school or less*, 1 = *some college or more*.
[d]Measures completed solely by those in an intimate partnership ($n = 605$). α = Cronbach's alpha.
$^{*}p < .05$.
$^{**}p < .01$.
$^{***}p < .001$.

Maternal affection

Maternal affection (Rossi, 2001) was assessed using seven items, the first of which asked, "How would you rate your relationship with your mother during the years you were growing up?" on a scale of 1 (*excellent*) to 5 (*poor*). This item was reverse coded and multiplied by 0.75 to ensure continuity with the remaining items, which were assessed on a scale of 1 (*a lot*) to 4 (*not at all*; Brim et al., 2018). The remaining six items asked, "How much did she understand your problems and worries?," "How much could you confide in her about things that were bothering you?," "How much love and affection did she give you?," "How much time and attention did she give you when you needed it?," "How much effort did she put into watching over you and making sure you had a good upbringing?," and "How much did she teach you about life?" Each item was reverse coded such that higher scores indicated greater maternal affection. The measure's scale score was calculated using the mean of the seven items. The measure demonstrates good reliability at MIDUS 1 ($\alpha = .91$; Brim et al., 2018).

Paternal affection

The paternal affection measure also included seven items, which replicated the language and scoring of the maternal affection measure above, although replacing gender pronouns (Rossi, 2001). The paternal affection measure's scale score was calculated using the mean of the seven items. The paternal affection measure demonstrates good reliability at MIDUS 1 ($\alpha = .93$; Brim et al., 2018).

2.2.2 | Mediating variables

We operationalized biobehavioral reactivity, the BBFM's mediating construct, using allostatic load and negative affect reactivity, reflecting two critical dimensions of psychophysiological distress and dysregulation. Our mediators were assessed at MIDUS 2 via Biomarker Project data (i.e., allostatic load) and the Daily Stress Project 2 (i.e., negative affect reactivity).

Allostatic load

As outlined above, allostatic load reflects a measure of cumulative biological risk in the face of chronic physiological stress reactivity. As such, allostatic load was measured in this study using risk scores across seven physiological systems (Brooks et al., 2014). These systems include inflammation, sympathetic nervous system, parasympathetic nervous system, metabolic glucose, lipid metabolism, cardiovascular system, and hypothalamic pituitary adrenal axis. Each system was assessed using multiple physiological indicators via the MIDUS 2 Biomarker Project (a total of 25 indicators across the seven systems; Ryff, Seeman, et al., 2019). Each system's indicators have clinical cutoffs that have been established whereby participants' physiological measurements can be classified as below or above that cutoff. Participants' dichotomous indicators of risk in each system were tabulated according to prior research (Brooks et al., 2014; Gruenewald et al., 2012). Within each system, indicators' cutoff risk scores of 0 (below high-risk cutoff) or 1 (at or above high-risk cutoff) were then averaged to create an overall risk for each of the seven systems (thus, each of the seven physiological systems' risk scores ranged from 0 to 1).

Missing data were imputed for metabolic lipids, metabolic glucose, parasympathetic nervous system, and cardiovascular and inflammation systems as each had greater than two indicators comprising their system risk score. Specifically, for participants missing solely one (for systems

with three or four indicators in total) or two (for systems with five indicators, i.e., metabolic lipids and inflammation) indicator risk scores, the remaining indicator risk scores were averaged to provide a total system risk score.

The seven systems' risk scores were then summed to create an overall allostatic load risk score ranging from 0 to 7 (Brooks et al., 2014). Higher scores indicate greater allostatic load and thus greater risk of physical health issues.

Negative affect reactivity

We incorporated reports of both daily negative affect and daily stressors to calculate within-person and between-person negative affect reactivity. Specifically, on each of the 8 days of the Daily Stress Project 2, a negative affect measure (Watson, Clark, & Tellegen, 1988) assessed the frequency of 14 negative emotional states such as nervous, irritable, and anxious on a scale from 0 (*none of the time*) to 4 (*all of the time*). Item responses were averaged within each day for a daily negative affect score. Participants' average negative affect scores ranged from a sample mean of 0.14 on day 8 ($SD = 0.23$; $n = 730$) to a sample mean of 0.27 on day 1 ($SD = 0.25$; $n = 730$). Across all 8 days, for a total of 5,987 reports, participants' average negative affect score equaled 0.18 ($SD = 0.29$), with a range of 0.00–2.60. In addition, participants reported whether they had experienced any of a list of multiple stressors (e.g., work/school problems, interpersonal conflict, perceived discrimination). Stressors were reported on 2,544 participant days for the current sample.

Daily negative affect reactivity was calculated as a within-person slope using a two-level random-effects model in Mplus (Muthén & Muthén, 2017): Level 1 represented daily negative affect regressed onto stress exposure, with the random slope representing change in negative affect from a nonstressor day to a stressor day. Level 2 represented the average slopes when adjusting for between-person stress exposure in average negative affect. This level 2 slope variable is how negative affect reactivity is operationalized in this study. In other words, our multilevel model first accounted for changes in negative affect following the experience of a stressor for each individual participant (i.e., within-person slopes, across the eight daily diary days) and then estimated variance in change in negative affect across the full sample (i.e., differences between participants). Thus, Models 2a/2b and 3a/3b, which include negative affect reactivity as a mediating variable, test this variable in level 2 in order to reduce bias (or error) in estimating the parameters caused by exporting the individual participants' slope parameters as an observed variable.

2.2.3 | Dependent variables

We include health appraisal and morbidity at MIDUS 3, collected via the project's self-administered questionnaire, as dependent variables.

Health appraisal

Participants rated their overall health on a scale from 1 (*excellent*) to 5 (*poor*). Responses were reverse coded, such that lower scores represent worse self-rated health. The average rating for the present sample equaled 3.52 ($SD = 1.03$) or a rating of physical health between "good" and "very good."

Morbidity

Participants answered dichotomous items regarding the presence of multiple chronic conditions experienced or treated in the past 12 months. At MIDUS 3, a total of 29 chronic conditions were

assessed (which were also assessed at MIDUS 1 and 2); we used participants' summed "yes" responses to this full list of chronic conditions. This morbidity count variable has been validated in multiple studies exploring health outcomes of MIDUS participants (Elliot, Turiano, Infurna, Lachman, & Chapman, 2018; Priest et al., 2019; Woods et al., 2019). The average number of chronic conditions for the present sample was 2.75 ($SD = 2.56$).

2.2.4 | Covariates

Covariates included in our models are MIDUS 1 measures of dependent variables, as well as self-reported sex (0 = *male*, 1 = *female*), age, and education (0 = *high school or less*, 1 = *some college or more*) at MIDUS 1. In addition, we include measures of intimate partner strain and intimate partner support at baseline, regressed onto our models' dependent variables. MIDUS researchers similarly used mean imputation to account for missing data, assigning scale scores for participants who completed at least one item on each of these scales (Ryff, Almeida, et al., 2019). Descriptive statistics for these measures are included in Table 1.

Intimate partner strain

This is a six-item measure, and this measure of intimate partner strain mirrors items found in the family strain measure, above, with two additional items asking respondents, "How often does [your spouse or partner] argue with you" and "How often does he or she make you feel tense?" (Walen & Lachman, 2000). Participants used a scale ranging from 1 (*often*) to 4 (*never*), which was recoded such that higher scores indicated greater strain. Responses to the measure's six items were averaged to calculate a scale score.

Intimate partner support

Similarly, the intimate partner strain measure reflects the four items of the family support measure, above, but includes two additional items asking, "How much does [your spouse or partner] appreciate you?" and "How much can you relax and be yourself around him or her?" (Walen & Lachman, 2000). Item response options ranged from 1 (*a lot*) to 4 (*not at all*) and were reverse coded in order for higher scores to represent greater support. Participant responses were averaged to create an intimate partner support scale score.

2.3 | Analyses

2.3.1 | Latent class analysis

In order to meet the first aim of this project (to test a composite of negative and positive qualities of family relationships), an LCA of the four FEC measures was used to categorize participants at MIDUS 1. The LCA evaluates underlying patterns occurring across these aspects of relational quality, identifying subgroups, or classes, within the sample that are naturally occurring. This finite-mixture modeling approach provides a rich measurement and conceptualization of FEC, affording us the opportunity to investigate how aspects of family relationship quality cluster together. Prior research has tested how individual family relationship measures affect biobehavioral reactivity, directly and via multiplicative (interaction) effects. However, we argue that this approach investigates effects at the measurement level, as opposed to first

understanding how responses on these measures occur in tandem and then using these person-centered taxonomies (i.e., clusters; Dyer & Day, 2015) to reflect individuals' full emotional climates. In addition, using this type of modeling better reflects the theoretical construct of FEC. The emotional climate of the family is comprised of multiple facets. By including and clustering multiple aspects of the FEC together, we are better able to examine the theoretical constructs of the BBFM.

Thus, using a median split of the four measures described above (i.e., family support, family strain, maternal affection, and paternal affection), we loaded each of the participants' dichotomized scores (i.e., below the median, at or above the median) into the class analysis model, tested using Mplus 8.0 (Muthén & Muthén, 2017). Analysis type was mixture, and models with one, two, three, four, and five classes were compared. Model fit was assessed using Akaike's Information Criterion (AIC), Bayesian Information Criterion (BIC), entropy, the Vuong-Lo–Mendell–Rubin Likelihood Ratio Test (VLMR), and the parametric bootstrapped likelihood ratio test (BLRT; McLachlan & Peel, 2000). VLMR and BLRT assess fit between nested models that differ by one profile and provide a significance test that specifies whether the tested model, or tested model minus one class, provides a better fit to the data. The best-fitting LCA model will have the lowest AIC and BIC compared to other models tested, as well as entropy closer to 1 and a significant VLMR and BLRT ($p < .05$).

Following the LCA, we assigned posterior probabilities for each MIDUS participant, representing the probability of a participant being in each latent class. In other words, after identifying naturally occurring subgroups of FEC in our full sample, each participant's probability of belonging to each class was assessed, and individual class membership was assigned based on the highest probability. Thus, each participant was assigned to one of the classes resulting from the best-fitting LCA model, based on the likelihood they were in the specific class.

Finally, using analysis of variance and χ^2 tests, we examined covariate and dependent variable values of participants in the resulting classes to test for between-class differences.

In order to enter participants' FEC as the exogenous variable in our structural path models, class membership (resulting from the LCA) was dummy coded. Each dummy-coded group was entered as independent variables, and the remaining FEC class that was not specified served as the reference category for the specified classes.

2.3.2 | Multilevel structural path modeling

Overall, we tested six structural path models in Mplus 8.0 (Muthén & Muthén, 2017), each testing our three hypotheses and estimating the indirect effects of FEC on health (i.e., health appraisal and morbidity in separate models) via allostatic load (Models 1a/1b), negative affect reactivity (Models 2a/2b), and the full range of psychobiological mediators tested in the prior two iterations (Models 3a/3b). In other words, we tested the pathways of the BBFM in each model, examining what effects membership in each of our FEC latent classes (i.e., results of our LCA, above) had on biobehavioral reactivity (Hypothesis 1), as well as the effects of biobehavioral reactivity on disease activity (Hypothesis 2), predicting a nonsignificant direct association between FEC class and physical health (Hypothesis 3). As Models 1a and 1b are structural path models with observed variables, each was estimated using Monte Carlo integration and full information maximum likelihood to account for missing data.

Models 2a–3b, which include negative affect reactivity, are multilevel structural path models. Multilevel modeling is necessary in order to account for the dependence of the repeated

TABLE 2 Probability of class members scoring at or above the median on each family emotional climate measure for each latent class ($N = 793$)

	Latent class			
Measure	**Ambivalent (30.2%)**	**Positive (21.5%)**	**Negative (35.8%)**	**Indifferent (9.8%)**
Family support	0.77	0.93	0.38	0.39
Family strain	0.85	0.18	0.99	0.01
Maternal affection	0.78	0.84	0.18	0.18
Paternal affection	0.75	0.81	0.20	0.14

TABLE 3 Means and *SD* of family climate scores, health, and demographic variables by latent class

Variable	**Ambivalent** $n = 240$	**Positive FEC** $n = 171$	**Negative FEC** $n = 284$	**Indifferent** $n = 78$	F (3)
T1 health appraisal	3.81 (0.85)	3.91 (0.80)	3.67 (0.94)	3.77 (1.02)	2.71*
T3 health appraisal	3.60 (1.00)	3.70 (1.01)	3.38 (1.03)	3.60 (0.96)	4.21**
T1 morbidity	1.98 (2.06)	1.79 (1.83)	2.66 (2.41)	1.83 (1.75)	8.08***
T3 morbidity	3.49 (3.23)	2.55 (2.33)	3.41 (2.99)	2.97 (3.31)	3.87**
Age	45.76 (10.88)	47.76 (12.43)	44.33 (9.85)	48.73 (10.64)	5.42**
Sex[a]	0.58 (0.49)	0.49 (0.50)	0.61 (0.49)	0.42 (0.50)	4.39**
Education[b]	0.75 (0.75)	0.76 (0.43)	0.70 (0.46)	0.64 (0.48)	1.20

Note: FEC = family emotional climate; T1 = MIDUS 1; T3 = MIDUS 3.
[a]Sex: 0 = *male*, 1 = *female*.
[b]Education: 0 = *high school or less*, 1 = *some college or more*.
*$p < .05$.
**$p < .01$.
***$p < .001$.

measures of the daily diary on level 1 (i.e., within subjects) and to estimate random-effect slopes to operationalize negative affect reactivity. The random slope is included in level 2 (i.e., between subjects) as a mediator to test indirect effects between FEC and health. Models that included health appraisal, a continuous variable, used linear regression to estimate pathways; models that included morbidity, a count variable, used Poisson regression. Models 2a and 3a were estimated using Bayesian estimation, with multiple imputation to account for missing data. Models 2b and 3b used Monte Carlo integration and full information maximum likelihood to account for missing data. Control variables were included to account for biased estimates due to missing data. Specifically, we included intimate partner strain and support as control variables to account for the known impact of marital quality on health outcomes. Individuals who were not in a relationship and had missing data on the marital quality variables were retained in the sample through the full information maximum likelihood estimation process in Mplus.

Model fit for all continuous outcome models was evaluated by examining the χ^2 test for model fit whereby a nonsignificant χ^2 ($p \geq .05$) indicates that the estimated model fits the data. If χ^2 is significant ($p < .05$), which is probable given the present sample size (Kline, 2016), we

examined alternative indicators of fit, including root mean square error of approximation (<0.08) and comparative fit index (>0.90). Model fit statistics are not calculated in Mplus for count outcome models (i.e., using Poisson regression), such as our Models 1b, 2b, and 3b using morbidity as the dependent variable.

Tests of between-group differences were also completed to evaluate whether the effects of each FEC class significantly differed from the reference group, and from one another; results are presented via Wald χ^2 tests of parameter constraints. Finally, mediating indirect paths (i.e., the effects of FEC on health indirectly via biobehavioral reactivity, as hypothesized) were calculated for Models 1a and 1b using bootstrapping (5,000 resamples). As bootstrapping is not available for multilevel mediation models estimated in Mplus, given the limitations in resampling when considering the hierarchical structure of nested data, we used Bayesian estimation methods for Models 2a and 3a. This procedure calculates nonsymmetric confidence intervals for each model's indirect effects. Finally, as Bayesian estimation is not available for analyzing indirect effects of count-dependent variables (i.e., with a Poisson distribution, such as our morbidity variable), we calculated the indirect effects of Models 2b and 3b outside of Mplus using the Sobel Test (MacKinnon, Warsi, & Dwyer, 1995).

The results of each of the six models are presented in Figures 1–6; additional results specifying standard errors, 95% confidence intervals, parameter estimates for Models 3a and 3b, and indirect effects for each model are presented in Tables 4–6.

3 | RESULTS

3.1 | Classes of family emotional climate

We found evidence of four unique classes of FEC (AIC = 3,113.11, BIC = 31,261.37; entropy = 0.577). Specifically, each LCA testing model, representing one, two, three, and then

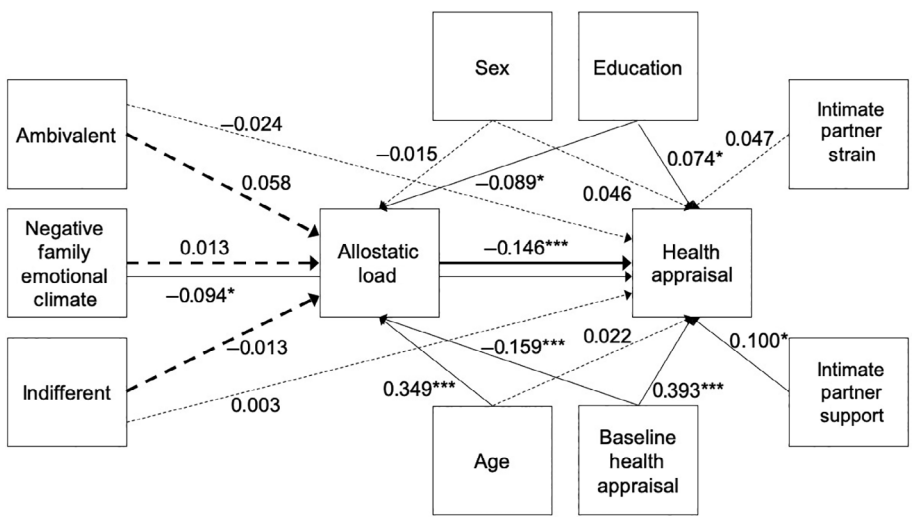

FIGURE 1 Structural equation model 1a of family emotional climate, allostatic load, and health appraisal (standardized; $N = 771$). $\chi^2 = 89.52$, $p < .000$, CFI = 0.751, root mean square error of approximation = 0.077, SRMR = 0.057. *$p < .05$, ***$p < .000$. Hypothesized pathways indicated in bold; nonsignificant pathways further signified by dashed line

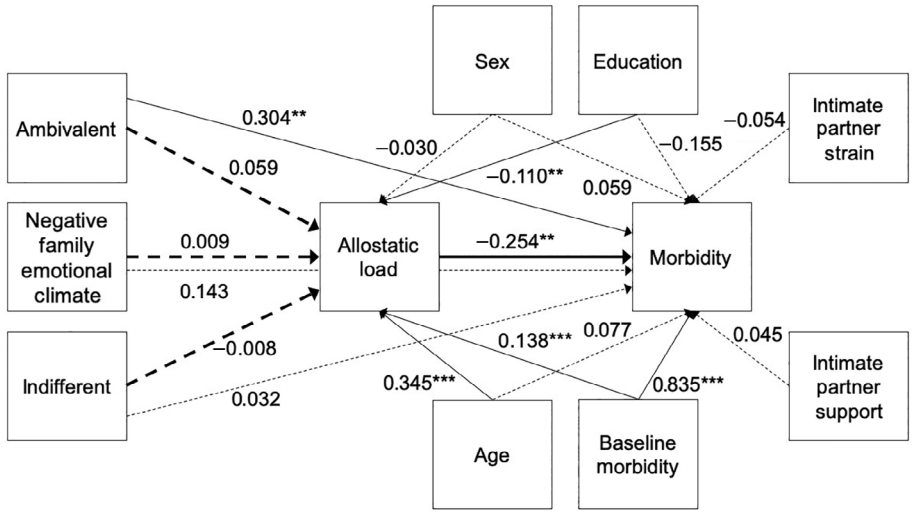

FIGURE 2 Structural equation model 1b of family emotional climate, allostatic load, and morbidity (standardized; $N = 771$). Akaike's Information Criterion = 3,677.13, Bayesian Information Criterion = 3,793.28. *$p < .05$, **$p < .01$, ***$p < .000$. Hypothesized pathways indicated in bold; nonsignificant pathways further signified by dashed line

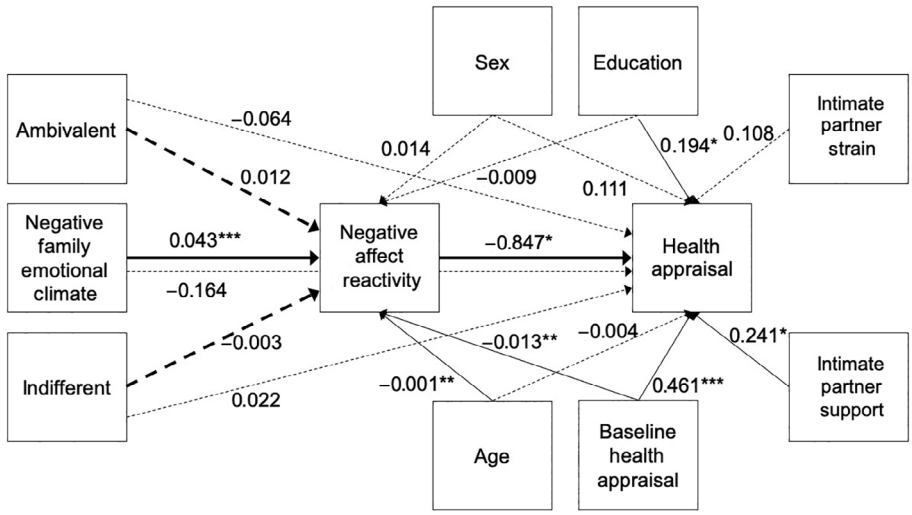

FIGURE 3 Multilevel structural path model 2a of family emotional climate, negative affect reactivity, and health appraisal (unstandardized; $N = 770$). Akaike's Information Criterion = 4,270.45, Bayesian Information Criterion = 4,450.54. *$p < .05$, **$p < .01$, ***$p < .000$. Hypothesized pathways indicated in bold; nonsignificant pathways further signified by dashed line. Negative affect reactivity equals the slope of daily negative affect regressed onto daily stress

four underlying FEC classes, demonstrated improved model fit (i.e., improvements in AIC, BIC, and entropy), whereas no such improvement (or, worse fit) was demonstrated in models testing greater than four classes of FEC. In addition, the VLMR of the four-class model was significant ($p < .001$), as was the BLRT ($p < .001$), suggesting that this model fit the data significantly better than the three-class model we tested.

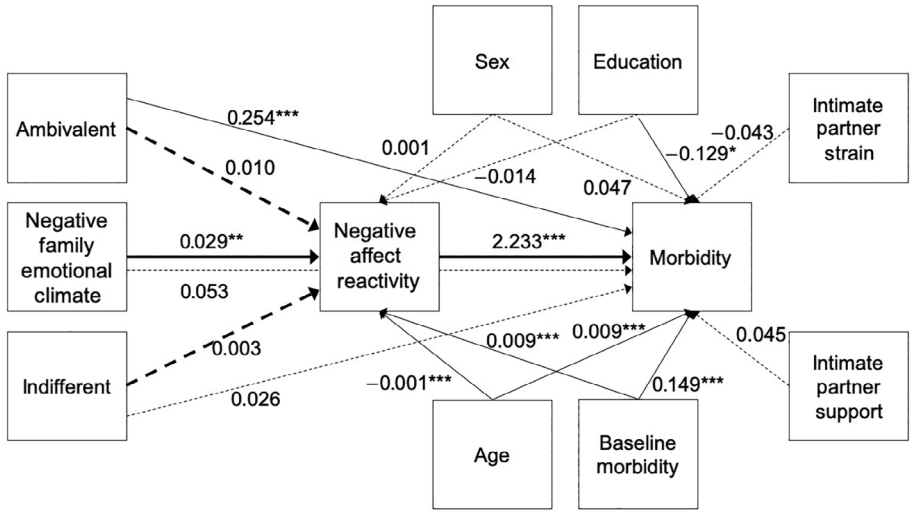

FIGURE 4 Multilevel structural path model 2b of family emotional climate, negative affect reactivity, and morbidity (unstandardized; $N = 771$). Akaike's Information Criterion = 7,699.45, Bayesian Information Criterion = 7,872.89. $^{*}p < .05$, $^{**}p < .01$, $^{***}p < .000$. Hypothesized pathways indicated in bold; nonsignificant pathways further signified by dashed line. Negative affect reactivity equals the slope of daily negative affect regressed onto daily stress

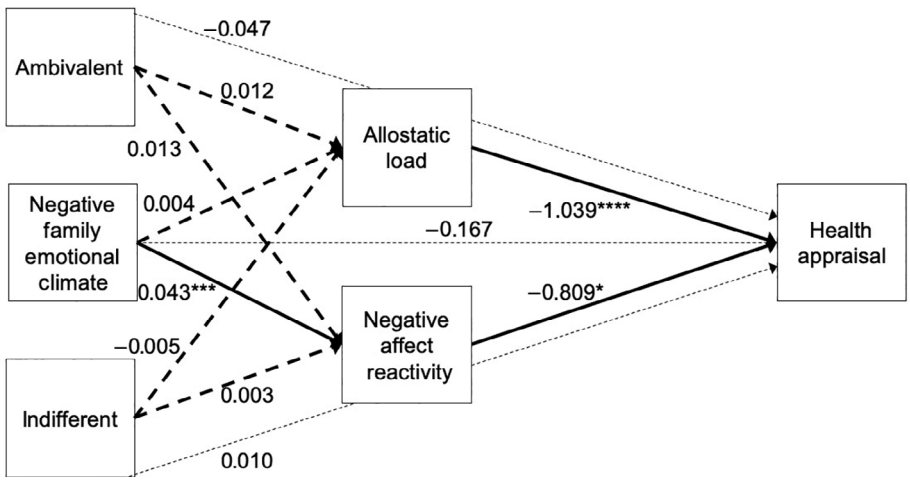

FIGURE 5 Multilevel structural path model 3a of family emotional climate, allostatic load, negative affect reactivity, and health appraisal (unstandardized; $N = 770$). Akaike's Information Criterion = 3,407.49, Bayesian Information Criterion = 3,660.96. $^{*}p < .05$, $^{***}p < .000$. Hypothesized pathways indicated in bold; nonsignificant pathways further signified by dashed line. Negative affect reactivity equals the slope of daily negative affect regressed onto daily stress. Control variables include age, sex, education, intimate partner strain, and intimate partner support, not currently presented in this figure for ease of interpretation

Based on these results, we labeled the classes as follows: (a) Ambivalent ($n = 240$), (b), Positive Family Emotional Climate ($n = 171$), (c) Negative Family Emotional Climate ($n = 284$), and (d) Indifferent ($n = 78$). Participants with Ambivalent family relationships were characterized by high probabilities of being at or above the median on each of the four FEC measures (Table 2). In

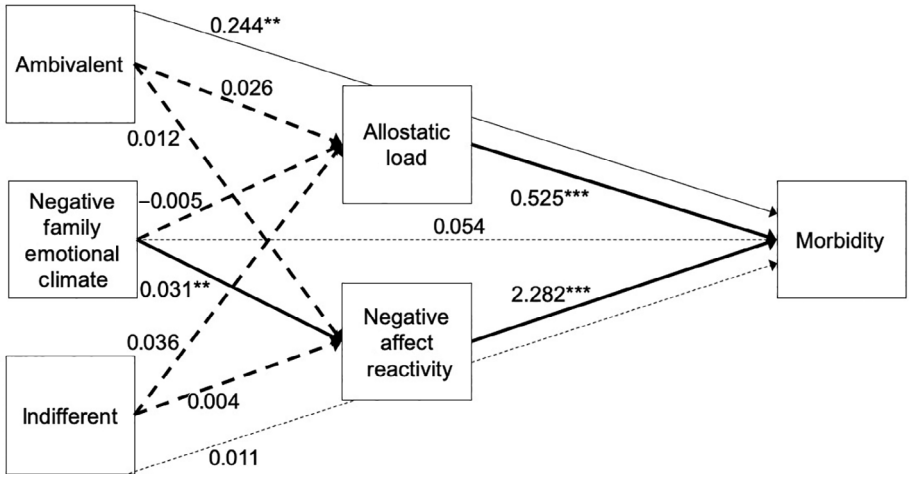

FIGURE 6 Multilevel structural path model 3b of family emotional climate, allostatic load, negative affect reactivity, and morbidity (unstandardized; $N = 771$). Akaike's Information Criterion = 7,417.371, Bayesian Information Criterion = 7,664.20. *$p < .05$, **$p < .01$, ***$p < .000$. Hypothesized pathways indicated in bold; nonsignificant pathways further signified by dashed line. Negative affect reactivity equals the slope of daily negative affect regressed onto daily stress. Control variables include age, sex, education, intimate partner strain, and intimate partner support, not currently presented in this figure for ease of interpretation

other words, they were more likely to endorse having high family support, high family strain, high maternal affection, and high paternal affection. A Positive Family Emotional Climate was characterized by the having the highest probability of endorsing strong levels of family support, a low probability of endorsing high levels of family strain, and the highest probability of having strong maternal and paternal affection. A Negative Family Emotional Climate, in contrast, was characterized by the lowest probability of endorsing positive family support, highest probability of having above-the-median strain, and low probabilities of reporting maternal and paternal affection at/above the median. Finally, our fourth class, which we label Indifferent, reflects moderate probabilities of having above-the-median family support and the lowest probability of above-the-median family strain, maternal affection, and paternal affection. These classifications align with prior specifications of FEC by valence and intensity (Holt-Lunstad & Uchino, 2019; Ross et al., 2019; Uchino et al., 2001).

After assigning each participant to one of the four classes of FEC, we then dummy-coded their membership across the classes. In other words, we assigned the Positive Family Emotional Climate class as our reference group and indicated for each of the remaining classes whether a participant was, or was not, a member. For example, participants with a Negative Family Emotional Climate were coded as a 0 in the Ambivalent and Indifferent classes and as a 1 in the Negative Family Emotional Climate class. This process allowed for a comparison between each of these classes and participants in the Positive Family Emotional Climate class. Participants' class of FEC was used as the exogenous variable in each of our structural equation models.

3.2 | Latent class descriptives

We then tested between-class mean differences in our covariates and dependent variables (Table 3). The four classes significantly differed regarding age: members of the Indifferent group were the oldest, and those with a Negative Family Emotional Climate were the youngest. The

TABLE 4 Standardized coefficients and significance levels for allostatic load models (*SE* in parentheses)

Parameter estimate	Model 1a health appraisal ($N = 771$)			Model 1b morbidity ($N = 770$)		
	Standardized	*p*	95% CI	Standardized	*p*	95% CI
Ambivalent → allostatic load	0.059 (0.044)	.183	−0.028, 0.144	0.059 (0.044)	.174	−0.026, 0.145
Negative FEC → allostatic load	0.013 (0.045)	.771	−0.075, 0.102	0.009 (0.044)	.829	−0.077, 0.096
Indifferent → allostatic load	−0.013 (0.040)	.752	−0.090, 0.065	−0.008 (0.040)	.833	−0.086, 0.069
Allostatic load → health	**−0.146 (0.039)**	**.000**	−0.222, −0.069	**0.254 (0.079)**	**.001**	0.100, 0.409
Ambivalent → health	−0.024 (0.042)	.570	−0.105, 0.058	**0.304 (0.100)**	**.002**	0.107, 0.500
Negative FEC → health	**−0.094 (0.043)**	**.031**	−0.149, −.009	0.143 (0.092)	.119	−0.037, 0.323
Indifferent → health	0.003 (0.035)	.933	−0.066, 0.072	0.032 (0.086)	.706	−0.136, 0.201
Indirect effects[a]						
Ambivalent → AL → health	−0.009 (0.007)	.205	−0.026, 0.003	0.015 (0.012)	.203	−0.002, 0.048
Negative FEC → AL → health	−0.002 (0.007)	.785	−0.017, 0.012	0.002 (0.011)	.828	−0.019, 0.026
Indifferent → AL → health	0.002 (0.006)	.738	−0.010, 0.016	−0.002 (0.010)	.834	−0.024, 0.017
Control variables						
Sex → Allostatic load	−0.015 (0.035)	.664	−0.085, 0.054	−0.030 (0.036)	.410	−0.100, 0.041
Sex → health	0.046 (0.032)	.152	−0.017, 0.108	0.059 (0.091)	.516	−0.119, 0.237
Education → allostatic load	**−0.089 (0.036)**	**.014**	−0.159, −0.018	**−0.110 (0.035)**	**.002**	−0.180, −0.041
Education → health	**0.074 (0.033)**	**.023**	0.010, 0.139	−0.155 (0.083)	.061	−0.318, 0.007
Age → allostatic load	**0.349 (0.033)**	**.000**	0.284, 0.414	**0.345 (0.033)**	**.000**	0.280, 0.410
Age → health	0.022 (0.036)	.541	−0.048, 0.092	0.077 (0.077)	.316	−0.074, 0.227
T1 health → allostatic load	**−0.159 (0.037)**	**.000**	−0.233, −0.086	**0.138 (0.039)**	**.000**	0.062, 0.214
T1 health → T3 health	**0.393 (0.033)**	**.000**	0.329, 0.457	**0.835 (0.048)**	**.000**	0.741, 0.930
T1 IP strain → T3 health	0.047 (0.051)	.355	−0.052, 0.146	−0.054 (0.135)	.691	−0.319, 0.211
T1 IP support → T3 health	**0.100 (0.049)**	**.043**	0.003, 0.196	0.045 (0.122)	.712	−0.194, 0.284

Note: Significant pathways indicated in bold.

Abbreviations: AL, allostatic load; CI, confidence interval; FEC, family emotional climate; IP, intimate partner; T1, MIDUS 1; T3 = MIDUS 3.

[a]Calculated using bootstrapping (5,000 resamples).

TABLE 5 Coefficients and significance levels for negative affect reactivity multilevel structural path models (*SE* in parentheses)

Parameter estimate	Model 2a health appraisal ($N = 770$)			Model 2b morbidity ($N = 771$)		
	Unstandardized	***p***	**95% CI**	**Unstandardized**	***p***	**95% CI**
Intercept[a]	0.466 (0.011)	.000	0.445, 0.486	0.509 (0.010)	.000	0.489, 0.528
Residual variances[a]	0.049 (0.001)	.000	0.047, 0.051	0.054 (0.001)	.000	0.052, 0.056
Ambivalent → affect reactivity	0.012 (0.012)	.310	−0.011, 0.034	0.010 (0.009)	.255	−0.007, 0.027
Negative FEC → affect reactivity	**0.043 (0.011)**	**.000**	0.020, 0.065	**0.029 (0.009)**	**.001**	0.012, 0.046
Indifferent → affect reactivity	0.003 (0.016)	.829	−0.027, 0.034	0.003 (0.012)	.822	−0.021, 0.026
Affect reactivity → health	**−0.847 (0.360)**	**.019**	−1.553, −0.141	**2.233 (0.416)**	**.000**	1.417, 3.049
Ambivalent → health	−0.064 (0.093)	.488	−0.246, 0.117	**0.254 (0.068)**	**.000**	0.121, 0.388
Negative FEC → health	−0.164 (0.092)	.076	−0.345, 0.017	0.053 (0.072)	.457	−0.087, 0.194
Indifferent → health	0.022 (0.124)	.859	−0.221, 0.265	0.026 (0.097)	.788	−0.164, 0.216
Indirect effects[b]						
Ambivalent → NAR → health	−0.007 (0.012)	.140	−0.033, 0.010	0.022 (0.020)	.276	—
Negative FEC → NAR → health	**−0.034 (0.018)**	**.015**	−0.073, −0.004	**0.065 (0.024)**	**.006**	—
Indifferent → NAR → health	−0.003 (0.015)	.340	−0.037, 0.027	0.007 (0.027)	.803	—
Control variables						
Sex → affect reactivity	0.014 (0.008)	.099	−0.003, 0.030	0.001 (0.007)	.857	−0.012, 0.014
Sex → health	0.111 (0.067)	.096	−0.020, 0.241	0.047 (0.050)	.349	−0.051, 0.146
Education → affect reactivity	−0.009 (0.010)	.333	−0.028, 0.009	−0.014 (0.007)	.059	−0.028, 0.001
Education → health	**0.194 (0.075)**	**.010**	0.046, 0.342	**−0.129 (0.052)**	**.014**	−0.231, −0.026
Age → affect reactivity	**−0.001 (0.000)**	**.001**	−0.002, 0.001	**−0.001 (0.000)**	**.000**	−0.002, 0.001
Age → health	−0.004 (0.003)	.204	−0.010, 0.002	**0.009 (0.002)**	**.000**	0.004, 0.013

TABLE 5 (Continued)

Parameter estimate	Model 2a health appraisal ($N = 770$)			Model 2b morbidity ($N = 771$)		
	Unstandardized	*p*	95% CI	Unstandardized	*p*	95% CI
T1 health → affect reactivity	**−0.013 (0.005)**	**.006**	−0.022, −0.004	**0.009 (0.002)**	**.000**	0.006, 0.012
T1 health → T3 health	**0.461 (0.037)**	**.000**	0.388, 0.533	**0.149 (0.010)**	**.000**	0.129, 0.169
T1 IP strain → T3 health	0.108 (0.088)	.217	−0.064, 0.280	−0.043 (0.037)	.244	−0.115, 0.029
T1 IP support → T3 health	**0.241 (0.098)**	**.014**	0.048, 0.433	0.045 (0.026)	.079	−0.005, 0.095
Between level						
Intercept—Health	0.596 (0.550)	.278	−0.481, 1.674	0.551 (0.155)	.000	0.247, 0.855
Intercept—NAR	−0.090 (0.030)	.003	−0.148, −0.031	−0.178 (0.018)	.000	−0.214, −0.142
Residual variances—Health	0.800 (0.041)	.000	0.719, 0.881	—	—	—
Residual variances—NAR	0.010 (0.001)	.000	0.009, 0.012	0.005 (0.000)	.000	0.004, 0.006

Note: Significant pathways indicated in bold.

Abbreviations: CI, confidence interval; FEC, family emotional climate; IP, intimate partner; NAR, negative affect reactivity; T1, MIDUS 1; T3, MIDUS 3.

[a]Within level, negative affect.

[b]Model 2a calculated using Bayesian estimation, Model 2b calculated using the Sobel test.

groups also differed with regard to sex, such that participants with Ambivalent and Negative Family Emotional Climates were skewed toward having proportionally more female than male participants. The four classes did not significantly differ regarding level of education.

The four classes significantly differed regarding baseline health appraisal, such that those with a Positive Family Emotional Climate reported, on average, global health approximately equating a rating of "very good," whereas those with a Negative Family Emotional Climate reported the worst average health appraisal ratings (Table 3). This difference was exacerbated at MIDUS 3. Similarly, the Positive Family Emotional Climate group reported the lowest number of chronic conditions when assessing baseline morbidity, whereas participants in the Negative Family Emotional Climate class reported the greatest number among the four classes. However, at MIDUS 3, participants with an Ambivalent FEC reported the greatest number of conditions (Table 3).

3.3 | Models 1a and 1b: Allostatic load

We first tested the indirect effects of baseline FEC on health outcomes 20 years later via a 10-year allostatic load. In Model 1a, we entered health appraisal as the observed dependent variable (Figure 1). Although we failed to find a significant effect of FEC on allostatic load (our

TABLE 6 Coefficients and significance levels for full multilevel structural path models (*SE* in parentheses)

Parameter estimate	Model 3a health appraisal ($N = 770$)			Model 3b morbidity ($N = 771$)		
	Unstandardized	***p***	**95% CI**	**Unstandardized**	***p***	**95% CI**
Intercept[a]	0.466 (0.011)	.000	0.445, 0.486	0.502 (0.010)	.000	0.482, 0.521
Residual variances[a]	0.049 (0.001)	.000	0.047, 0.051	0.054 (0.001)	.000	0.052, 0.056
Ambivalent → affect reactivity	0.012 (0.012)	.310	−0.011, 0.034	0.012 (0.009)	.199	−0.006, 0.029
Negative FEC → affect reactivity	**0.043 (0.011)**	**.000**	0.020, 0.065	**0.031 (0.009)**	**.001**	**0.013, 0.048**
Indifferent → affect reactivity	0.003 (0.016)	.828	−0.027, 0.034	0.004 (0.012)	.760	−0.020, 0.027
Ambivalent → allostatic load	0.018 (0.014)	.184	−0.009, 0.045	0.026 (0.058)	.656	−0.088, 0.139
Negative FEC → allostatic load	0.004 (0.013)	.780	−0.023, 0.030	−0.005 (0.055)	.922	−0.114, 0.103
Indifferent → allostatic load	−0.005 (0.019)	.775	−0.043, 0.031	0.036 (0.057)	.524	−0.076, 0.149
Affect reactivity → health	**−0.818 (0.357)**	**.022**	−1.517, −0.119	**2.282 (0.415)**	**.000**	**1.468, 3.096**
Allostatic load → health	**−1.039 (0.267)**	**.000**	−1.562, −0.516	**0.525 (0.088)**	**.000**	**0.353, 0.698**
Ambivalent → health	−0.047 (0.092)	.608	−0.227, 0.133	**0.244 (0.073)**	**.001**	**0.100, 0.388**
Negative FEC → health	−0.167 (0.092)	.067	−0.347, 0.012	0.054 (0.075)	.473	−0.093, 0.201
Indifferent → health	0.010 (0.123)	.933	−0.230, 0.251	0.011 (0.079)	.886	−0.143, 0.166
Indirect effects[b]						
Ambivalent → NAR → health	−0.008 (0.011)	.170	−0.033, 0.010	0.027 (0.021)	.195	—
Ambivalent → AL → health	−0.019 (0.016)	.084	−0.052, 0.010	0.014 (0.030)	.654	—
Negative FEC → NAR → health	**−0.032 (0.019)**	**.012**	−0.076, −0.003	**0.071 (0.024)**	**.003**	—
Negative FEC → AL → health	−0.005 (0.014)	.394	−0.031, 0.025	−0.003 (0.029)	.928	—
Indifferent → NAR → health	−0.003 (0.015)	.394	−0.032, 0.026	0.009 (0.027)	.739	—
Indifferent → AL → health	0.007 (0.019)	.320	−0.032, 0.049	0.019 (0.030)	.530	—

TABLE 6 (Continued)

Parameter estimate	Model 3a health appraisal ($N = 770$)			Model 3b morbidity ($N = 771$)		
	Unstandardized	*p*	95% CI	Unstandardized	*p*	95% CI
Control variables						
Sex → affect reactivity	0.014 (0.008)	.099	−0.003, 0.030	0.002 (0.007)	.778	−0.011, 0.015
Sex → Allostatic load	−0.005 (0.010)	.591	−0.028, 0.009	−0.006 (0.047)	.905	−0.097, 0.086
Sex → health	0.103 (0.066)	.118	−0.026, 0.232	0.046 (0.054)	.393	−0.059, 0.151
Education → affect reactivity	−0.009 (0.010)	.333	−0.028, 0.009	−0.014 (0.007)	.061	−0.028, 0.001
Education → Allostatic load	**−0.030 (0.011)**	**.009**	−0.052, −0.007	−0.023 (0.051)	.656	−.122, 0.077
Education → health	**0.161 (0.075)**	**.032**	0.014, 0.309	**−0.113 (0.057)**	**.045**	**−0.224, −0.002**
Age → affect reactivity	**−0.001 (0.000)**	**.001**	−0.002, −0.001	**−0.001 (0.000)**	**.000**	**−0.002, −0.001**
Age → Allostatic load	**0.005 (0.000)**	**.000**	0.004, 0.005	**0.004 (0.002)**	**.026**	**0.001, 0.008**
Age → health	0.001 (0.003)	.772	−0.005, 0.007	**0.006 (0.002)**	**.011**	**0.001, 0.011**
T1 health → affect reactivity	**−0.013 (0.005)**	**.006**	−0.022, −0.004	**0.009 (0.002)**	**.000**	**0.006, 0.012**
T1 health → Allostatic load	**−0.025 (0.006)**	**.000**	−0.036, −0.014	0.008 (0.017)	.667	−0.027, 0.042
T1 health → T3 health	**0.435 (0.037)**	**.000**	0.361, 0.508	**0.145 (0.012)**	**.000**	**0.122, 0.168**
T1 IP strain → T3 health	0.105 (0.087)	.229	−0.066, 0.275	−0.061 (0.037)	.101	−0.134, 0.012
T1 IP support → T3 health	**0.208 (0.098)**	**.033**	0.016, 0.400	**0.056 (0.026)**	**.032**	**0.005, 0.106**
T2 Allostatic load corr. T2 NAR	0.000 (0.001)	.552	−0.001, 0.001	0.000 (0.001)	.833	−0.003, 0.003
Between level						
Intercept—Health	0.916 (0.552)	.097	−0.166, 1.998	0.516 (0.157)	.001	0.209, 0.823
Intercept—NAR	−0.090 (0.030)	.003	−0.148, −0.031	−0.176 (0.019)	.000	−0.212, −0.139
Intercept—AL	0.182 (0.034)	.000	0.115, 0.249	0.049 (0.099)	.617	−0.144, 0.243
Residual variances—Health	0.784 (0.040)	.000	0.704, 0.863	—	—	—

(Continues)

TABLE 6 (Continued)

Parameter estimate	Model 3a health appraisal ($N = 770$)			Model 3b morbidity ($N = 771$)		
	Unstandardized	*p*	95% CI	Unstandardized	*p*	95% CI
Residual variances—NAR	0.010 (0.001)	.000	0.009, 0.012	0.005 (0.000)	.000	0.005, 0.006
Residual variances—AL	0.017 (0.001)	.000	0.015, 0.018	0.125 (0.001)	.000	0.122, 0.127

Note: Significant pathways indicated in bold.

Abbreviations: AL, allostatic load; CI, confidence interval; FEC, family emotional climate; IP, intimate partner; NAR, negative affect reactivity; T1 = MIDUS 1; T2 = MIDUS 2; T3 = MIDUS 3.

[a]Within level, negative affect.

[b]Model 3a calculated using Bayesian estimation, Model 3b calculated using the Sobel test.

first hypothesis), results indicate a direct effect of FEC on health appraisal, such that participants with a Negative Family Emotional Climate had worse health appraisal at MIDUS 3, an effect that significantly differed from participants with a Positive Family Emotional Climate (our reference group). Indirect effects were nonsignificant for each class (Table 4).

In Model 1b, we estimated the same mediation relationship when entering morbidity as the observed dependent variable (Figure 2). Results indicate that participants reporting Ambivalent family relationships demonstrate a significant direct association between class membership and number of chronic conditions, a pathway estimate significantly greater than that of participants with a Positive Family Emotional Climate. Although allostatic load was directly associated with morbidity, it was not predicted by baseline family climate, contrary to our second hypothesis. Thus, we find no evidence of an indirect pathway from FEC to morbidity via allostatic load and fail to find support for our third hypothesis when testing these specific operationalizations of biobehavioral reactivity and disease activity (Table 4).

3.4 | Models 2a and 2b: Negative affect reactivity

Next, we tested the indirect pathway from FEC to health through MIDUS 2 negative affect reactivity (using multilevel modeling). Model 2a utilized health appraisal as the dependent variable, as with Model 1a (Figure 3). In support of our first hypothesis, results show that participants with a Negative Family Emotional Climate experience greater negative affect reactivity, an effect that significantly differs from those with a Positive Family Emotional Climate, as well as those with an Ambivalent climate and an Indifferent climate (Table 7). We also found a significant association between greater negative affect reactivity and worse health appraisal, as hypothesized. The indirect effect of a Negative Family Emotional Climate on health appraisal via negative affect reactivity was significant (Table 5). Direct associations between each class of FEC and health appraisal were nonsignificant (in support of Hypothesis 3).

Model 2b estimated the effects of FEC on morbidity as the endogenous variable, via negative affect reactivity. Similar to Model 2a and in accordance with the pathways of the BBFM, we found a significant mediation effect: baseline Negative Family Emotional Climate was significantly associated with greater negative affect reactivity, which in turn was directly associated with a greater number of chronic conditions at MIDUS 3 (Figure 4; Table 5). The Negative

TABLE 7 Wald χ^2 tests of parameter differences between family emotional climate classes

	Model 1a		Model 1b		Model 2a		Model 2b		Model 3a		Model 3b	
Pathway	χ^2	*p*	χ^2	*p*	χ^2	*p*	χ^2	*p*	χ^2	*p*	χ^2	*p*
FEC → Allostatic load												
Ambivalent = negative	3.361	.067	3.166	.075	—	—	—	—	0.399	.528	0.874	.350
Ambivalent = indifferent	0.311	.577	2.981	.084	—	—	—	—	0.027	.870	2.064	.151
Negative = indifferent	3.642	.056	0.443	.506	—	—	—	—	0.693	.405	0.416	.519
FEC → negative affect reactivity												
Ambivalent = negative	—	—	—	—	**9.130**	**.003**	**9.070**	**.003**	**10.049**	**.002**	**9.630**	**.002**
Ambivalent = indifferent	—	—	—	—	0.309	.578	0.459	.498	0.286	.593	0.448	.504
Negative = indifferent	—	—	—	—	**7.100**	**.008**	**7.787**	**.005**	**7.527**	**.006**	**5.474**	**.019**
FEC → health												
Ambivalent = negative	1.325	.250	1.626	.202	1.520	.218	**12.700**	**.000**	2.186	.139	**9.904**	**.002**
Ambivalent = indifferent	1.725	.189	1.479	.224	0.519	.471	**6.473**	**.011**	0.227	.634	**8.178**	**.004**
Negative = indifferent	0.299	.585	0.141	.708	2.498	.114	0.065	.799	2.266	.132	0.235	.628

Note: Significant differences indicated in bold.

Family Emotional Climate–negative affect reactivity association significantly differed from associations between each of the other FEC classes and affect reactivity (Table 7). We again found that participants with Ambivalent relationships reported greater morbidity at MIDUS 3, a direct association significantly differing from the association found for participants with a Positive Family Emotional Climate (our reference group), as well as those in the Indifferent and Negative Family Emotional Climate classes (Table 7).

3.5 | Model 3a and 3b: Full model

Our third and final set of models incorporated both allostatic load and negative affect reactivity as mediators. Therefore, FEC classification at baseline/MIDUS 1, allostatic load and negative affect reactivity at MIDUS 2, and health outcomes at MIDUS 3 were included in these models to test the effects of each of the psychobiological mediators as reflected in the BBFM.

Using health appraisal as the dependent variable, Model 3a found a significant association between FEC and negative affect reactivity but not with allostatic load, replicating findings from prior models (Figure 5; Table 6). Specifically, a Negative Family Emotional Climate was associated with significantly greater negative affect reactivity 10 years later, compared to a Positive Family Emotional Climate. This association also significantly differed from the effects of Ambivalent and Indifferent FECs on negative affect reactivity (Table 7). Greater negative affect reactivity, in turn, was associated with worse health appraisal, as was allostatic load, as hypothesized. However, allostatic load was not associated with concurrent negative affect reactivity, despite prior research suggesting an association (Sin, Graham-Engeland, Ong, & Almeida, 2015). In addition, the indirect effect of a Negative Family Emotional Climate on health appraisal via negative affect reactivity was not significant ($p > .05$).

Our sixth and final model (Model 3b) utilized morbidity as the dependent variable, as with Models 1b and 2b. We again found that a Negative Family Emotional Climate was significantly associated with greater negative affect reactivity at MIDUS 2 and found a direct association between negative affect reactivity and MIDUS 3 morbidity, in support of our first and second hypotheses (Figure 6; Table 6). As with the prior models testing affect reactivity, the Negative Family Emotional Climate–negative affect reactivity association significantly differed between each of the other classes of family climate (Table 7). In accordance with our third hypothesis, the indirect effect of a Negative Family Emotional Climate on morbidity 20 years later, via negative affect reactivity at 10 years, was significant.

Although allostatic load was associated with later morbidity, it was not associated with any of the baseline FEC classes. Nor was allostatic load significantly, concurrently associated with negative affect reactivity. Similar to Models 1b and 2b, we found that Ambivalent family relationships were directly associated with worse morbidity at MIDUS 3 compared to participants with a Positive Family Emotional Climate, as well as those with Indifferent and Negative FECs (Table 7).

4 | DISCUSSION

Across each of the models tested, we build toward identifying that negative affect reactivity, but not allostatic load, serves as a potentially important mediator between FEC and health. This is especially the case for persons classified as having a negative FEC, which in this study was

characterized by low levels of family support and parental affection and high levels of family strain. In other words, in comparison to adults who report a positive, ambivalent, or indifferent FEC, those who report a negative FEC experience a significantly stronger association with greater negative affect reactivity and, in turn, worse health. In Models 3a and 3b, we find evidence of this indirect effect when accounting for allostatic load risk across seven physiological systems, as well as baseline health. As a result of these models, we are able to identify that participants with a negative FEC not only have worse health outcomes (self-rated, as well as number of chronic conditions) than those in other types of FECs, but we are also able to tease out a potential pathway by which this effect occurs. This indirect pathway aligns with the hypothesized mediation effect of the BBFM, which posits that a more negative FEC is associated with worse disease activity through greater biobehavioral reactivity.

In Models 1a/1b and 3a/3b, however, we failed to find a significant association between FEC and allostatic load. It may be that there is a half-life of the effect of relational stress on physiological measures of stress, such that measurements of these two constructs taken 10 years apart are too distant to find an effect. Conversely, it may be that our measurement of family strain did not account for the intensity of relational stress that may affect physiological changes, such as adverse childhood experiences of abuse and neglect (Priest et al., 2019). We also failed to find a significant association between negative affect reactivity and allostatic load, both assessed at MIDUS 2. This may indicate that individual physiological systems comprising the overall allostatic load calculation (e.g., inflammation, cardiovascular reactivity), rather than an overall risk score, may serve as more meaningful mediating pathways. In other words, the composite measure used here may hide variance in individual physiological systems that reflect physiological stress reactivity resulting from family strain through the inclusion of nonreactive systems.

4.1 | Relationship ambivalence

Interestingly, participants in the Ambivalent class, whose FEC was characterized by high family support and strain, as well as high maternal and paternal affection, demonstrated direct associations with greater morbidity 20 years later that were especially unique. Although high family strain is the quality that most closely aligns the Ambivalent and Negative Family Emotional Climate classes, it is not the sole driver of our found connections as the Ambivalent–morbidity association significantly differed from direct associations between each of the other FEC classes. An alternative hypothesis is that, as our family support and strain measures do not assess specifically which family relationships participants are considering when rating their quality, those ranking high in each are capturing a variety of intense relationships of both types—warm/understanding and critical/demanding. Thus, opposing intensities across family ties may be especially potent.

In addition, prior researchers have posited that ambivalent close relationships exacerbate the effects of stress on health due to high levels of quality fluctuation and a lack of reliability, which buffers the benefits of high support (Ross et al., 2019; Uchino et al., 2012). This lack of stability—reliable and unreliable support, caring, and hostility—characterizes family relationships marked by chaos and enmeshment. Wood's (1993, 2019) BBFM breaks the concept of enmeshment into three dimensions, each included in the FEC construct. These variables include proximity (i.e., cohesion and closeness defined by interpersonal boundaries), generational hierarchy (i.e., the extent of generational boundaries that support coparent teams and

prevent cross-generational coalitions; Wood, Klebba, & Miller, 2000), and responsivity (i.e., degree to which family members are responsive to one another). Families maladaptive in these areas—characterized by intrusiveness, demanding overinvolvement, and extreme emotional reactions in response to family interactions—could prove to be especially stressed and vulnerable to disease (Wood, 2019) and could be characterized as being ambivalent (i.e., both highly supportive and strained): cohesive relationships with minimal boundaries (i.e., those an individual might be most likely to open up to and who would respond emotionally) and a good deal of negativity.

This potentially suggests that FEC and relationship ambivalence are not only solely marked by valence and intensity but also intra- and interpersonal relationship processes reflecting continua of cohesion, structure, flexibility, boundaries, and family-level responsiveness. In fact, prior research demonstrates that differentiation of self (i.e., characterized as the ability to regulate emotion and balance individuality and togetherness in relationships, which in an extreme reflects enmeshment; Kerr & Bowen, 1988) serves as a possible mediator linking FEC and biobehavioral reactivity in the BBFM (Priest, 2017). As we suggest that what has been termed "ambivalent" relationships (most recently in the Social Ambivalence and Disease model; Holt-Lunstad & Uchino, 2019) is indeed a specific type of FEC, as theorized by the BBFM, it is possible to extend tests of both models by measuring the qualities of family relationships beyond support and strain. Specifically, we recommend that it may be meaningful to capture families' processes of connecting, adapting, specifying roles, and emotionally responding to one another via self-report (e.g., using the FACES-IV; Author; Olson, 2011) and observational (e.g., using the Family Process Assessment Protocol; Wood et al., 2008) or experimental (Holt-Lunstad & Clark, 2014) methods. Furthermore, in our FEC LCA, we collapsed measures of concurrent and childhood measures of family relationship quality, including current family support and strain, and retrospective reports on parental affection. However, a limitation of our approach is that we may only have been able to assess ambivalence in adult family relationships, measuring solely love and positive attention received from mothers and fathers during youth. As such, we also recommend that future studies consider assessing the full spectrum of relationship valence—support and strain—across the life course in order to more fully capture ambivalence in family relationships at unique points in time, as well as trajectories of ambivalent FECs.

Finally, the present tests do not assess stress-coping skills and health behaviors, which may be more negatively impacted by ambivalence, and thus associate our Ambivalent class with morbidity. In order to assess an ambivalence–health link of support interference, it will be necessary for future tests of the BBFM to incorporate health behaviors as a potential mediating variable. Prior research has found support for the use of food to cope and physical activity as mechanisms of effect in the BBFM, although in the context of romantic relationship strain alone (Roberson, Shorter, et al., 2018).

4.2 | Indifferent relationships

There is little research examining indifferent close relationships (Holt-Lunstad & Uchino, 2019; Ross et al., 2019). Although originally posited as potentially reflecting relationships with low contact frequency, such as casual coworkers (Uchino et al., 2001), Ross et al. (2019) more recently described this category as emotionally disengaged, such as a partner with whom there is little conflict or intimacy. Uchino, Holt-Lunstad, Smith, and Bloor (2004) go so far as to

suggest indifferent relationships should not be considered close, and theorize that they have little effect on health. In contrast, indifference in intimate partnerships that reflects withdrawal or disengagement could quite possibly be stressful, with a negative association with long-term health (Ross et al., 2019).

The results of our LCA demonstrated a category aligning with the characterizations of low strain/low support but was specific to family relationships. Thus, a more helpful description of what has been previously termed "indifferent" may be Wood's (2019) characterization of a "flat family emotional climate" characterized by neither positive nor negative expressed emotion, distance and avoidance, and a lack of responsiveness (p. 5). Wood theorizes that this type of family configuration is potentially most closely linked to high biobehavioral reactivity and, possibly, behavioral disorders. However, we found that participants in the Indifferent FEC did not have significantly worse allostatic load risk or negative affect reactivity than participants with a Positive Family Emotional Climate. Given that our Indifferent class was significantly older than the other groups, these low-intensity ratings of positivity and negativity may reflect an expected process of aging, whereby family relationships tend to become less conflictual and less intense (English & Carstensen, 2014; Woods et al., 2019). Thus, the association between our Indifferent class and biobehavioral reactivity may not significantly differ from the Positive Family Emotional Climate association.

Participants classified as having an Indifferent FEC may also be otherwise buffered from the worse health outcomes that negative, low-quality family relationships impart via stress and strain. Emotional disengagement or reserve may serve as a protective factor, whereby strain and requests for support are minimal and thus nonimpactful for long-term health. Overall, this is the first study, to our knowledge, to categorize family relationships (including current and parental relationships in childhood) as indifferent in quality. Additional research is needed to tease out the qualities of family ties characterized by low positivity and negativity and how these relationships affect health.

Finally, although prior social scientists have characterized relationships with high/high and low/low strain and support as *ambivalent* and *indifferent*, respectively, we suggest, based on this study, as well as the FEC construct of the BBFM, that these labels are oxymoronic. In other words, they convey internal, intrapersonal emotional states that reflect contradiction or apathy, although they are characterized by relational qualities, processes, and behaviors. Instead, we suggest that these two types of FEC may reflect a shared underlying process—a third dimension of close relationships, aside from gradations (intensity) of valence. As discussed above, this dimension may reflect enmeshment and the family-level boundaries and emotional responsiveness that, when blurred, diffuse, chaotic, and overreactive, indicate ambivalent relationships and when disengaged, distant, or rigid, indicate indifferent ties. It will be important to continue to refine these taxonomies, how their characteristics cluster, possible demographic variations (e.g., by age, gender, and culture), and how they may be associated with health.

4.3 | Limitations and future research

Although this study improves upon prior research by testing a multidimensional conceptualization of FEC and psychobiological mechanisms of effect over 20 years, the project is limited in its generalization. Specifically, although MIDUS provides an innovative approach to examining longitudinal, biopsychosocial health, the characteristics of the core MIDUS sample are such that participants are mostly White, with high levels of education. These qualities are skewed in

the present sample as the Biomarker Project and Daily Stress Project 2 utilize data collection strategies requiring time- and effort-intensive research participation. Therefore, participants with greater resources, who are more likely to be White, employed, and highly educated, had a greater likelihood of completing participation in the five MIDUS studies presently sampled. As such, the effects found in this study's models may not be generalized to dissimilar samples. Although prior research has demonstrated the applicability of the BBFM to underserved and underrepresented samples (Priest, McNeil Smith, Woods, & Roberson, 2020; Priest & Woods, 2015; Woods & Denton, 2014), this study does not add to that line of research. Testing the BBFM's pathways longitudinally should consider and intentionally include more diverse samples. These future research studies could incorporate contextual stressors that may impact both the quality of family relationships and biobehavioral reactivity to affect health (e.g., discrimination, neighborhood safety, healthcare access).

In addition, by incorporating participants who completed all three waves of MIDUS, we exclude participants whose health effects may have been more severe, resulting in death. Prior research has found significant effects of relationship support and affect reactivity on mortality using MIDUS data (Stanton et al., 2019). Thus, modeling morbidity and mortality in trajectory models that estimate the shape of effects over time may be an indicated next step.

Previous studies using the BBFM as a theoretical guide have regularly incorporated measures of emotion regulation and/or depression/anxiety in their operationalizations of biobehavioral reactivity (Priest et al., 2019; Roberson, Woods, Priest, & Miller, 2018; Woods et al., 2014; Woods & Denton, 2014). This study sought to advance this line of research by testing allostatic load and negative affect reactivity as mediating links between family and health. However, it is possible that negative affect reactivity and depression or anxiety, for example, are associated and that incorporating each of these mediating variables in categorizations of psychophysiological regulation (similar to our FEC LCA used presently) could be a meaningful next step. In seeking to avoid issues with incremental validity testing, such as introducing an issue of multicollinearity (Wang & Eastwick, 2020), we did not include these measures (although assessed in MIDUS) in this study. In other words, it is unclear whether aspects of neuroticism (e.g., moody, worrying, nervous), depression (e.g., sadness, irritability, feeling worthless), or anxiety (e.g., feeling irritable due to worry, low on energy), for example, are distinct from negative affect (e.g., feeling nervous, sad, irritable, worthless), as presently measured in MIDUS (Ryff et al., 2017). Including each of these measures would require additional hypothesizing about intertwined trajectories of personality, family-related distress, biobehavioral reactivity, and mood over time. Furthermore, given our findings regarding ambivalence and morbidity, it is possible that health behaviors or other interim pathways linking relationships and health require subsequent exploration (Roberson, Shorter, et al., 2018). Additional research exploring the biobehavioral reactivity construct of the BBFM is required.

Finally, although we intentionally focus on the effects of nonintimate family relationships, the present family strain and support measures used in MIDUS do not ask participants to specify which family members they are considering when completing the measure. The families and health literature suffers as a whole from lacking specification in this regard (Woods, Bridges, et al., 2019). However, prior research demonstrates that who a participant considers when completing family relationship measures may affect responses and thus assessment results (Priest, Parker, & Woods, 2018). In addition, as Holt-Lunstad and Uchino (2019) suggest, the specific type of family relationship may have implications for ambivalence in relationships, as well as family-based interventions. Future attempts to investigate pathways linking close

relationships and health should indicate the limits of family members included by stipulating a priori or by inviting respondents to clarify. Given the importance of variables, including proximity (Wood, 2019) and contact frequency (Holt-Lunstad & Uchino, 2019), in conceptualizing FEC, unique types of family relationships, and family composition, may have varying effects on stress reactivity.

4.4 | Conclusion

In summary, we conclude that it is meaningful and theoretically important to assess clusters of relationship characteristics to define individuals' FECs. The results of our present LCA appear to validate prior two-dimensional classifications of relationships (Uchino et al., 2001), which align with the FEC of the BBFM (Wood, 1993; Wood et al., 2008). In our present test of the BBFM, we find that a negative FEC is linked to health outcomes—health appraisal and morbidity—via negative affect reactivity. Furthermore, ambivalent relationships are linked to morbidity by mechanisms heretofore undefined, whereas associations between an indifferent FEC and biobehavioral reactivity do not appear to appreciably differ from positive FEC associations. Finally, allostatic load was not meaningfully incorporated into the present test of the BBFM. It will be important in future research to continue to refine the theoretical measurement of FEC and to refine mechanisms of effect by which unique climates affect long-term health outcomes for adults.

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DATA AVAILABILITY STATEMENT

The data that support the findings of this study are openly available at ICPSR at http://doi.org/10.3886/ICPSR02760.v15, http://doi.org/10.3886/ICPSR04652.v7, http://doi.org/10.3886/ICPSR26841.v2, http://doi.org/10.3886/ICPSR29282.v9, and http://doi.org/10.3886/ICPSR36346.v7.

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