Journal of Psychosomatic Research 202 (2026) 112509

Contents lists available at ScienceDirect

Journal of Psychosomatic Research

journal homepage: www.elsevier.com/locate/jpsychores

Short communication

Family emotional climate, depressive symptoms, and pain prevalence: Testing mediation pathways among midlife and older Black Americans

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ARTICLE INFO

Keywords:
Aging
Biobehavioral model
Black or African American
Chronic pain
Depressive symptoms
Family relations

ABSTRACT

Objective: Family relationship quality has been linked to pain outcomes, recently for aging Black adults, yet possible mechanisms of effect remain heretofore untested. Guided by the Biobehavioral Family Model, this study tested whether depressive symptoms mediate prospective associations between family emotional climate and pain prevalence, and whether associations are moderated by baseline chronic pain status.
Methods: We tested hypothesized associations among Black American participants of the nationally representative Midlife in the United States study (second [2004–2006] and third [2013–2015] waves) who reported on pain prevalence ($N = 471$; 65.2 % women; μ[age] = 50.45), using mediation and moderated mediation models.
Results: Greater family strain was linked to a greater likelihood of chronic pain 10 years later via greater depressive symptoms. Greater family support was associated with a decreased likelihood of chronic pain via decreased depressive symptoms. Baseline chronic pain status moderated this indirect effect via its moderation of the direct link between family support and depressive symptoms, such that this effect was significantly stronger for individuals reporting baseline chronic pain.
Conclusion: Depression is well-supported as a chronic pain antecedent. This study provides initial evidence it may also serve as a mutable mechanism linking family emotional climate to pain for aging Black Americans. Primary care-based assessments of family emotional climate to intervene on family strain while mitigating depressive symptoms which reflect chronic strain may be pain-protective. Interventions may also benefit from enhancing family support especially for patients experiencing chronic pain. Additional within-group, longitudinal research is needed to further support the tested mechanism.

Family relationship quality has been linked to chronic pain outcomes, including for Black midlife and older adults who often identify family as key for pain management [1–4] and are particularly likely to experience pain disparities [5–7]. One psychological mechanism of effect potentially linking families to pain includes depression. The Biobehavioral Family Model (BBFM; [8]) – a biopsychosocial, family systems theoretical model – posits a mediation pathway whereby family emotional climate (i.e., positive/supportive versus negative/strained relationship quality) is linked to disease activity (including pain) via biobehavioral reactivity (i.e., psychophysiological regulation/dysregulation). Biobehavioral reactivity is often operationalized as depression which is well-supported as a consequence of strained family emotional climate, an antecedent to pain, and empirically supported as a family—health mechanism in BBFM-guided research [8–12]. Identifying mutable mechanisms may inform the development of layered pain management interventions that prioritize family-based, positive, intergenerational strategies to disrupt the pain-depression-disability constellation for aging Black Americans. Thus, we leverage the BBFM to test hypotheses that greater family strain and decreased family support will be mediated by greater depressive symptoms and associated with subsequent (10 years later) greater likelihood of chronic pain for midlife and older Black Americans. We also test whether these associations differ for individuals with and without chronic pain at baseline due to the potential for pre-existing chronic pain to interact with family emotional climate and promote depressive symptoms [12,13], as well as the likelihood of chronic pain to persist over time particularly for Black

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https://doi.org/10.1016/j.jpsychores.2025.112509
Received 29 September 2025; Received in revised form 11 December 2025; Accepted 26 December 2025
Available online 31 December 2025

Americans [3,5,7]. This study builds on our prior research identifying direct links between family support/strain (but not intimate partner support/strain) and pain outcomes for aging Black Americans [2–4]. Combined with prioritizing the study of culturally-salient relationships [4,14,15], we operationalize family emotional climate as family support and strain in this study.

1. Method

This study was approved by UT Southwestern Medical Center's institutional review board as exempt due to the use of deidentified, publicly available data (STU-2022-0836).

1.1. Sample

We include participants in the second (2004–2006) and third (2013–2015) waves of the Midlife in the United States (MIDUS) study who self-identified as Black or African American and who reported on the presence of chronic pain at baseline (i.e., MIDUS 2/MIDUS 2 Milwaukee) and approximately 10 years later (i.e., MIDUS 3/MIDUS 3 Milwaukee; $N = 471$, 28.7 % reporting chronic pain at baseline) [16,17]. The MIDUS Milwaukee subproject was initiated at MIDUS 2 to expand study diversity via recruiting additional African Americans [18].

1.2. Measures

Family emotional climate and pain prevalence measures were completed via a self-administered questionnaire. Depression was assessed via telephone interview. Table 1 presents correlations among baseline variables.

1.2.1. Family emotional climate

Participants completed two valid, reliable, well-established measures assessing relationship quality with their "family members, not including your spouse or partner" [2,19,20]. Item responses were reverse coded then averaged to calculate scale scores.

Family support. Four items assessed how much family members care, understand the participant's feelings, can be relied upon for help, and can be opened up to. Item responses ranged from 1 (*a lot*) to 4 (*not at all*). The scale was reliable in this sample ($\alpha = 0.82$).

Family strain. Four items assessed how often family members are demanding, critical, unreliable, or get on participants' nerves on a scale from 1 (*often*) to 4 (*never*). This scale was reliable in this sample ($\alpha = 0.78$).

1.3. Depressive symptoms

Participants completed the reliable and valid Composite International Diagnostic Interview-short form, Major Depressive Disorder subscale (CIDI), a diagnostic interview based on DSM criteria [21]. Participants who reported feeling either depressed mood or anhedonia for (a) two weeks or more during the past 12 months and (b) whose symptom(s) lasted most of the day/all day long and (c) occurred almost every day/every day completed the full CIDI module assessing the incidence of seven depressive symptoms, total, such as fatigue and difficulty concentrating. Item responses were summed, for a range of scale scores from 0 (denied depression, denied anhedonia, completed no further items) to 7. Depressive symptoms are assessed at baseline to test the causal mechanism posited by the BBFM, and the long-term impact of biobehavioral reactivity on pain.

1.4. Pain prevalence

Participants responded to a dichotomous item at MIDUS 2 assessing whether they have chronic pain "that persists beyond the time of normal healing and that has lasted anywhere from a few months to many years" (0 = *no*, 1 = *yes*), a method supported for capturing pain prevalence [2,22]. Baseline chronic pain status was tested as a moderator.

This pain item was repeated at MIDUS 3. Our outcome variable, pain prevalence, thus reflects pain incidence (development of novel chronic pain) and pain persistence (chronic pain reported at both waves).

1.5. Covariates

We controlled for age, sex (0 = *male,* 1 = *female*), marital status (0 = *single*, 1 = *cohabiting/married*), and MIDUS subsample (0 = *core*, 1 = *Milwaukee*) to account for potential variations in pain prevalence due to these characteristics.

1.6. Analyses

Mediation and moderated mediation models were tested in SPSS (v29.02; [23]) using the PROCESS macro (Models 4 and 8 [24]) and logistic regression with listwise deletion and bootstrapping (5000 resamples) resulting in bias-corrected CIs. We first estimated the direct and indirect effects of family emotional climate (either family support or family strain) on chronic pain 10 years later through depressive symptoms. Second, we tested whether baseline pain status moderates the direct and indirect effects of family emotional climate on 10-year pain prevalence; we test whether baseline pain moderates the indirect effect through its moderation of the effect of family emotional climate on depression. An index of moderated mediation was estimated to determine the significance of variations in the indirect effect due to baseline pain [25]. Finally, we conducted sensitivity analyses via calculating *E*-values to assess the potential impact of unmeasured confounding on our mediation models' pathways [26,27].

2. Results

We observed a significant indirect effect of family support on pain

Table 1
Participant reports of family emotional climate variables, depressive symptoms, chronic pain, and covariates at baseline: correlations ($N = 471$).

Variables	1	2	3	4	5	6	7
1. Family Support	–	–	–	–	–	–	–
2. Family Strain	−0.345***	–	–	–	–	–	–
3. Depressive Symptoms	−0.278***	0.175***	–	–	–	–	–
4. Baseline pain status	−0.175***	0.102*	0.189***	–	–	–	–
5. Age	0.134**	−0.171***	−0.069	0.029	–	–	–
6. Sex	0.006	0.070***	0.103*	0.010	0.057	–	–
7. Subsample	−0.044	0.052	0.040	−0.073	−0.146**	−0.003	–
M	3.421	2.231	0.684	0.287	50.540	1.650	0.822
SD	0.658	0.769	1.859	0.453	10.675	0.477	0.383

Note: Baseline pain status coded as 1 for *chronic pain at baseline* and 0 for *pain-free/no chronic pain at* baseline. Sex coded as 2 for *female* and 1 for *male.* Subsample coded as 1 for *MIDUS Milwaukee subsample* and 0 for *MIDUS core subsample*. Family support and strain variables are each missing two participants' responses. ***$p < .001$, **$p < .01$., *$p < .05$.

prevalence 10 years later via depressive symptoms (Table 2). Effects were in an expected direction whereby greater family support was linked to decreased depressive symptoms, and worse depressive symptoms were linked to a greater likelihood of pain prevalence 10 years later. Our moderated mediation model identified that the link between family support and depressive symptoms was contingent on baseline pain ($\beta = -0.846$) such that the effect was significantly stronger for individuals living with pre-existing chronic pain (versus pain-free individuals; Table 3). The observed indirect effect via depressive symptoms was thus conditional on baseline pain status, such that it was significantly stronger for individuals with pre-existing chronic pain.

We also observed a significant indirect effect of family strain on pain prevalence via depressive symptoms (Table 2). Greater family strain was linked to greater depressive symptoms which, in turn, were linked to a greater likelihood of reporting chronic pain 10 years later. However, the link between family strain and depressive symptoms was not contingent on baseline pain in our moderated mediation model ($\beta = -0.012$; Table 3). Though we observed a significant indirect effect of family strain on pain prevalence through depressive symptoms for pain-free individuals but not those living with chronic pain at baseline, the index of moderated mediation's CIs included zero indicating the size of the indirect effect did not significantly differ between the two groups (Table 3).

Sensitivity analyses (Supplementary Table 1) indicate it is possible, but less likely, that unmeasured confounders could explain away observed links between depressive symptoms and pain prevalence in both mediation models, above and beyond measured covariates. Such a confounder would need to be associated with both depression *and* pain prevalence by a risk ratio of 1.40–1.45-fold, each, and thus be a large effect. Observed links between family emotional climate and depressive symptoms, and family support and pain prevalence, are more robust. For example, an unmeasured confounder would need to be associated with both family support and depressive symptoms by a risk ratio of 2.28-fold, each, above and beyond our included covariates, to explain away our observed effect. Unmeasured confounders that would impact both at that magnitude are unlikely.

3. Discussion

Our findings support depressive symptoms as a possible mechanism of effect linking family emotional climate to pain prevalence for aging Black Americans. Depressive symptoms significantly mediated the link between greater family strain and a greater likelihood of 10-year pain prevalence, both for individuals developing novel chronic pain and those reporting chronic pain that may have persisted over the decade between study waves. Depressive symptoms also significantly mediated the link between family support and pain prevalence. However, this indirect effect is conditional on pre-existing chronic pain: the significantly stronger association between family support and depressive symptoms for those with pre-existing chronic pain, compared to pain-free individuals, drove a significantly stronger indirect effect for this group.

Identifying depressive symptoms as a mediator potentially provides two areas amenable to intervention: family relationships as well as individuals' stress reactivity or mental health. Assessments of family relationships and family member wellbeing are rare in primary care [28]. Assessing aging Black American patients' family emotional climate in order to provide targeted, early intervention for family strain may be a protective and pain-affirming approach to mitigating differential pain outcomes, and culturally-appropriate [1,29]. Conversely, identifying and engaging positive family relationships may protect against pain via improved perceptions of safety in the healthcare system plus the pain-protective benefits of enhancing supportive relationships [12,15]. Depression is an important target for holistic pain management, given the prevalence of undertreated, concurrent depressive symptoms for individuals living with chronic pain [30] and the potential for emotional distress to exacerbate worse pain outcomes [12]. Tailoring that care to mitigate stress reactivity and depressive symptoms that reflect chronic family strain may be a unique strategy, particularly if incorporated in family-based pain interventions which, to date, remain underdeveloped [2]. Moreover, interventions may benefit from incorporating a focus on enhancing the supportive nature of family relationship quality, particularly for patients already experiencing chronic pain, for whom both the direct and indirect effects of family support on pain prevalence 10 years later were especially potent. Prior research supports greater emotional support among families, as part of diverse social activity, may be linked

Table 2
Simple mediation models: direct and indirect links between family emotional climate (family support or family strain) and pain prevalence via depressive symptoms ($N = 469$).

	Family Support Model			Family Strain Model		
Parameter Estimate	Unstandardized (SE)	*p*	95 % CI	Unstandardized (SE)	*p*	95 % CI
Mediator: Depressive symptoms	F [5] = 9.194, $R^2 = 0.090$, $p < .001$			F [5] = 4.014, $R^2 = 0.042$, $p = .001$		
Constant	2.798 (0.677)	0.000***	1.468, 4.127	−0.514 (0.655)	0.433	−0.1800, 0.773
Family emotional climate	−0.776 (0.127)	0.000***	−1.026, −0.527	0.387 (0.113)	0.001***	0.166, 0.608
Age	−0.006 (0.008)	0.445	−0.022, 0.010	−0.001 (0.008)	0.350	−0.024, 0.008
Sex	0.430 (0.178)	0.016*	0.079, 0.780	0.367 (0.183)	0.046*	0.007, 0.728
Marital status	0.077 (0.175)	0.660	−0.267, 0.421	0.018 (0.179)	0.563	−0.313, 0.575
Subsample	0.116 (0.217)	0.595	−0.311, 0.542	0.131 (0.226)	0.563	−0.313, 0.567
Outcome: Pain prevalence	Model LL = 29.899, Nagelkerke $R^2 = 0.085$, $p < .001$			Model LL = 21.187, Nagelkerke $R^2 = 0.061$, $p = .002$		
Constant	1.217 (0.826)	0.141	−0.402, 2.837	−0.972 (0.765)	0.204	−2.470, 0.527
Depressive symptoms	0.171 (0.054)	0.002**	0.065, 0.278	0.202 (0.053)	0.000***	0.098, 0.306
Family emotional climate	−0.535 (0.158)	0.001**	−0.845, −0.226	0.229 (0.133)	0.085	−0.031, 0.489
Age	0.001 (0.010)	0.924	−0.018, 0.020	0.000 (0.010)	0.976	−0.019, 0.019
Sex	−0.046 (0.217)	0.832	−0.472, 0.379	−0.097 (0.215)	0.651	−0.518, 0.324
Marital status	0.057 (0.212)	0.788	−0.358, 0.472	0.013 (0.209)	0.951	−0.397, 0.423
Subsample	−0.148 (0.263)	0.573	−0.664, 0.367	−0.152 (0.261)	0.560	−0.664, 0.359
Indirect effect of family emotional climate on pain prevalence via *depressive symptoms*						
Indirect effect	−0.133 (0.053)	–	−0.256, −0.049	0.078 (0.035)	–	0.026, 0.165

Note: Sex coded as 2 for *female* and 1 for *male*. Marital status coded as 1 for *cohabiting/married* and 0 for *single*. Subsample coded as 1 for *MIDUS Milwaukee subsample* and 0 for *MIDUS core subsample*. Standard errors and 95 % bias-corrected bootstrap CI for indirect effects are calculated using bootstrapping (5000 resamples). ***$p < .001$, **$p < .01$., *$p < .05$.

Table 3
moderated mediation models: direct and indirect links between family emotional climate (family support or family strain) and pain prevalence via depressive symptoms conditional on baseline pain prevalence ($N = 469$).

	Family Support Model			Family Strain Model		
Parameter Estimate	Unstandardized (SE)	*p*	95 % CI	Unstandardized (SE)	*p*	95 % CI
Mediator: Depressive symptoms	F [7] = 10.035, $R^2 = 0.132$, $p < .001$			F [7] = 5.153, $R^2 = 0.073$, $p < .001$		
Constant	1.191 (0.762)	0.119	−0.306, 2.688	−0.652 (0.665)	0.327	−1.960, 0.655
Family emotional climate	−0.361 (0.163)	0.027*	−0.681, −0.042	0.343 (0.135)	0.011*	0.078, 0.609
Baseline pain status	3.434 (0.872)	0.000***	1.721, 5.147	0.757 (0.567)	0.182	−0.356, 1.871
Family emotional climate x Baseline pain status	−0.846 (0.255)	0.001**	−1.348, −0.344	−0.012 (0.233)	0.960	−0.469, 0.446
Age	−0.007 (0.008)	0.387	−0.022, 0.009	−0.009 (0.008)	0.282	−0.025, 0.007
Sex	0.437 (0.175)	0.013*	0.091, 0.778	0.375 (0.181)	0.039*	0.020, 0.731
Marital status	0.052 (0.172)	0.763	−0.286, 0.390	0.044 (0.177)	0.806	−0.304, 0.638
Subsample	0.160 (0.216)	0.459	−0.265, 0.586	0.199 (0.224)	0.375	−0.241, 0.638
Outcome: Pain prevalence	Model LL = 69.13, Nagelkerke $R^2 = 0.188$, $p < .001$			Model LL = 63.88, Nagelkerke $R^2 = 0.175$, $p < .001$		
Constant	0.366 (0.970)	0.706	−1.536, 2.267	−1.468 (0.852)	0.081	−3.112, 0.182
Depressive symptoms	0.133 (0.058)	0.023*	0.019, 0.248	0.156 (0.056)	0.006**	0.046, 0.266
Family emotional climate	−0.418 (0.207)	0.043*	−0.824, −0.013	0.245 (0.175)	0.162	−0.098, 0.587
Baseline pain status	1.649 (1.175)	0.161	−0.645, 3.952	1.865 (0.681)	0.006**	0.530, 3.200
Family emotional climate x Baseline pain status	−0.078 (0.342)	0.820	−0.747, 0.592	−0.191 (0.279)	0.492	−0.738, 0.355
Age	−0.002 (0.010)	0.873	−0.021, 0.018	−0.003 (0.010)	0.738	−0.023, 0.017
Sex	−0.021 (0.227)	0.926	−0.467, 0.424	−0.067 (0.226)	0.768	−0.508, 0.375
Marital status	0.101 (0.223)	0.625	−0.336, 0.537	0.077 (0.220)	0.727	−0.355, 0.508
Subsample	−0.010 (0.277)	0.971	−0.553, 0.533	−0.005 (0.276)	0.986	−0.546, 0.537
Conditional direct effect of family emotional climate on pain prevalence						
Pain-free at baseline	−0.418 (0.207)	0.044*	−0.824, −0.013	0.245 (0.175)	0.163	−0.098, 0.587
Chronic pain at baseline	−0.496 (0.278)	0.074	−1.040, 0.0477	0.053 (0.222)	0.811	−0.382, 0.488
Conditional indirect effect of family emotional climate on pain prevalence via *depressive symptoms*						
Pain-free at baseline	−0.048 (0.033)	–	−0.142, −0.003	0.054 (0.031)	–	0.008, 0.130
Chronic pain at baseline	−0.161 (0.086)	–	−0.362, −0.018	0.052 (0.047)	–	−0.019, 0.173
Index of moderated mediation	−0.113 (0.073)	–	−0.306, −0.010	−0.002 (0.050)	–	−0.110 0.095

Note: Baseline pain status coded as 1 for *chronic pain at baseline* and 0 for *pain-free/no chronic pain at* baseline. Sex coded as 2 for *female* and 1 for *male.* Marital status coded as 1 for *cohabiting/married* and 0 for *single*. Subsample coded as 1 for *MIDUS Milwaukee subsample* and 0 for *MIDUS core subsample*. Standard errors and 95 % bias-corrected bootstrap CI for conditional indirect effects are calculated using bootstrapping (5000 resamples). ***$p < .001$, **$p < .01$., *$p < .05$.

better pain outcomes for aging adults via decreased loneliness [31]. Further within-group research of the biobehavioral reactivity mechanism tested presently is needed to fully inform the development of culturally-responsive, family-based pain management interventions for midlife and older Black Americans.

4. Limitations

Our results are promising, though our study design is limited. The present study is longitudinal, but is limited by the contemporaneous measurement of independent and mediating variables. Studies testing temporal mediation pathways are needed, as well as testing reciprocal effects of pain on family emotional climate via worse depression. Though some research supports the strength of effect of non-intimate partner family relationships for health versus intimate partner relationships [2,32], further research testing variations in pain pathways by richer conceptualizations of longitudinal relationship biographies [33,34] would advance knowledge needed for family-based pain intervention. Finally, our sample was epidemiological; greater specificity and variation of depressive symptoms in a clinical sample of older Black Americans may provide greater opportunities to identify the size of this mediation effect. Testing variation in the biobehavioral reactivity mechanism due to contextual stressors (e.g., discrimination, neighborhood quality) may also be warranted in order to maximize our knowledge of how best to intervene and support aging Black Americans at risk of, or living with, chronic pain.

Author note

This work was supported by the National Institute on Aging of the National Institutes of Health under Award Number R21AG082344. The content is solely the responsibility of the authors and does not necessarily represent the official views of the National Institutes of Health. Data used for this study are publicly available and distributed via the Institute for Social Research at the University of Michigan. Meta-data for this study are available via the NIH HEAL Data Platform. There is no additional public preregistration affiliated with this study.

CRediT authorship contribution statement

Sarah B. Woods: Writing – review & editing, Writing – original draft, Visualization, Supervision, Methodology, Funding acquisition, Formal analysis, Data curation, Conceptualization. **Bhaskar Thakur:** Writing – review & editing, Validation, Methodology. **Staja Booker:** Writing – review & editing, Methodology, Conceptualization. **Beatrice Wood:** Writing – review & editing, Methodology, Conceptualization. **Patricia N.E. Roberson:** Writing – review & editing, Validation, Methodology, Data curation, Conceptualization.

Declaration of competing interest

The authors declare the following financial interests/personal relationships which may be considered as potential competing interests:

Sarah B. Woods reports financial support was provided by National Institute on Aging. If there are other authors, they declare that they have no known competing financial interests or personal relationships that could have appeared to influence the work reported in this paper.

Appendix A. Supplementary data

Supplementary data to this article can be found online at https://doi.org/10.1016/j.jpsychores.2025.112509.

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