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Family Strain, Depression, and Somatic Amplification in Adults with Chronic Pain

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Abstract

Background The associations between family strain, depression, and chronic pain interference vary across individuals, suggesting moderated relations, and one possible moderator is somatic amplification. The current study examined a moderated mediation model that investigated (a) whether depression mediated the relation between non-spouse family strain and chronic pain interference and (b) whether somatic amplification moderated the association between depression and chronic pain interference.

Methods Data came from 933 adults who participated in the National Survey of Midlife Development in the USA. Participants completed telephone interviews or self-report measures.

Results The relationship between non-spouse family strain and chronic pain interference was mediated by depression, and this mediation depended on the degree of somatic amplification. Specifically, individuals who experienced more non-spouse family strain were more likely to experience depression and higher levels of chronic pain interference. Somatic amplification significantly moderated the effect of depression on chronic pain, such that individuals with higher levels of somatic amplification and depression were likely to experience higher levels of chronic pain interference. The indirect effect of non-spouse family strain on chronic pain through depression was significant for low, middle, and high levels of somatic amplification.

Conclusions The presence of chronic pain has been associated with family dynamics changing, which may be linked with higher levels of non-spouse family strain. A negative family environment may be related to the development of depression, which may be associated with the severity and inability to cope with chronic pain. Somatic amplification may strengthen the association between depression and pain intensity.

Keywords Chronic pain · Somatic amplification · Depression · Family strain

Introduction

Social relationships have been demonstrated to be large contributors to the physical and psychological health of individuals with chronic pain [1–5]. In particular, the family context and how family members interact with each other can have a major influence on an individual's perception of chronic pain severity [2, 3]. Indeed, people experiencing greater disability associated with chronic pain tend to come from families characterized by poorer family cohesion [4], family conflict [1], and worse family functioning in non-spouse [3, 5]. Family systems theory posits that there are reciprocal influences between a person with chronic pain and their family [6, 7]. For example, family members may adjust how they interact with the person in pain to physically and emotionally support them [6, 7]. These changes in family dynamics may lead to a continuous cycle of increased family closeness, which may be followed by stress and strain because the individual may not be able to maintain employment or complete household activities [7]. Additionally, families of individuals with chronic pain may feel overwhelmed and isolated [8] due to the frequent care an individual with chronic pain may require. A family member may then experience strain due to taking on a new role as a caregiver and may experience guilt if they have to leave the individual with chronic pain alone [9]. Family members then might be more likely to experience negative interactions and resent the individual with pain, which could be related to more overall family strain.

Currently, although studies have examined the associations between spousal strain [10–13] and family interactions in general [2–5, 14] on chronic pain, scant research has been conducted on the connections between non-spouse family strain and chronic pain interference. For example, research has demonstrated that high levels of conflict and criticism from a

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spouse or partner are associated with greater pain intensity in individuals with chronic pain [10–13, 55]. Cano et al. (2004) indicated that negative spouse responses were significantly linked with higher levels of depression and anxiety in individuals with chronic pain [13]. Other studies have found that the support, attention, and sympathy spouses provide are significantly associated with greater impairment and greater self-reports of pain intensity [15, 16]. Currently, no studies have examined the relation between non-spouse family strain and chronic pain. Thus, non-spouse family strain will be examined to provide insight on how the relationships and interactions between individuals with chronic pain and other family members are affected. Additionally, studying non-spouse family strain will provide important information on how it is associated with pain interference. The results of the current study may inform how clinicians examine non-spouse family relationships in relation to chronic pain interventions.

Non-spouse family strain was examined as a driver of chronic pain because there is more empirical evidence supporting this direction as opposed to chronic pain driving family strain [3, 5, 14, 17]. For instance, one study compared two different models examining the directions between pain catastrophizing and family functioning variables. Akbari, Denhghani, Khatibi, and Vervoort (2016) found stronger support for the model examining family functioning variables predicting pain catastrophizing compared with the model examining pain catastrophizing predicting family functioning variables [5]. Similarly, one study found that high levels of family depressive symptoms predicted patient's pain scores over 1 year [14]. Additionally, family strain and negative family interactions have been associated with severity of pain [17] as well as disability associated with pain in general [3]. However, the literature on the relation between family strain and chronic pain has been inconsistent. For example, Kashikar-Zuck et al. (2008) found that family functioning variables did not predict pain-related disability. Additionally, Ross et al. (1993) concluded that greater family cohesion was associated with higher levels of pain in a sample of youth. These mixed results imply that sometimes family strain is linked with more severe chronic pain, but other times, it is not. Although these associations have been frequently examined in pediatric populations, less is known how family interactions affect an individual's perception of chronic pain in middle-aged and elderly adults. Additionally, these studies examined family functioning variables in general and did not examine non-spouse family strain exclusively. Thus, a comprehensive model with mediators and moderators examining the relations between non-spouse family strain and chronic pain is needed.

Depression is likely to be a mediator between non-spouse family strain and chronic pain interference because negative family interactions may be associated with an individual experiencing depression, which may be related to someone perceiving pain as being more severe in adult populations [18, 19]. If a family is characterized by constant strain, this may be correlated with an individual with pain experiencing a lower threshold for coping with these family stressors, thus increasing their vulnerability to developing depression. Depression in turn may be a causal risk factor for developing chronic pain [20, 21], such that experiencing depression may limit one's ability to effectively cope with the experience of chronic pain [20]. Studies have indicated that there is a bidirectional relationship between depression and chronic pain, such that chronic pain may be a causal risk factor for depression, and depression may be a causal risk factor for developing chronic pain [20, 21]. However, the current study is examining depression as a driver for pain, as studies have found that depression is predictive of more intense pain [22], more persistent pain [23], increased follow-up physician visits for pain [24], and more pain medication refills [24]. Palermo, Valrie, and Karlson (2007) investigated the associations between poor family functioning, depression, and chronic pain among adolescents and found that poorer family functioning was associated with greater depression and greater overall pain disability. Similarly, Driscoll et al. (2013) concluded that, among women, higher levels of family conflict and depression were linked with more chronic pain. Of note, one study, Kaczynski, Gambhir, Caruso, and Lebel (2016), which examined depression as a mediator of the relation between family functioning and functional disability in a sample of adolescents with chronic pain found that depression was not a significant mediator, but found that family functioning was significantly associated with depression [2]. These mixed results indicate that some people who have family strain may experience chronic pain but others may not. Thus, it is important to examine moderators of this relationship to better understand how chronic pain interference may be experienced and for whom these pain interferences are experienced to the greatest degree.

Studies suggest that somatic amplification is associated with higher levels of depression and chronic pain [25–28]. Somatic amplification refers to how individuals perceive normal body sensations as pathologically abnormal [25, 29, 30]. Somatic amplification is conceptualized differently from somatization, which refers to a more general physical process of how individuals respond to stress [31], whereas somatic amplification specifically refers to people who perceive bodily sensations as being unusual [25, 29, 30]. Somatic amplification is being examined as a moderator between depression and chronic pain because the degree to which an individual perceives bodily sensations as abnormal may play a role in the development of depression and also may be related to these individuals being more susceptible to the development of chronic pain [25–27]. For example, Lavigne, Saps, and Bryant (2014) concluded that somatization mediates the relation between depression and chronic pain and other studies have concluded that individuals who experience somatic

amplification in conjunction with depression may experience more intense chronic pain [25–27].

Thus, the current study examined a moderated mediation model investigating the associations between non-spouse family strain, depression, somatic amplification, and chronic pain interference, as both depression and somatic amplification have been significantly linked with higher levels of chronic pain in adult populations [2, 20, 21, 25–27]. Data were analyzed from a community sample of participants with chronic pain from the third wave of the National Survey of Midlife Development in the United States (MIDUS). In the present study, it was expected that somatic amplification would be a psychophysiological variable that moderates the association between depression and chronic pain interference. Specifically, it was hypothesized that the relation between depression and chronic pain interference would be more strongly positive at increasing levels of pain amplification. Overall, somatic amplification is important to examine as a moderator between depression and chronic pain interference because higher levels of somatic amplification may prompt those with higher depression to be more sensitive to their pain symptoms, thereby experiencing more severe chronic pain [26, 27]. Thus, targeting somatic amplification in interventions that aim to manage chronic pain may reduce individuals' depression as well. Additionally, examining how somatic amplification and depression are associated with chronic pain interference in the family context may inform the development of family-based interventions targeting depression and chronic pain. The identification of how somatic amplification is related to chronic pain may also inform the development of future longitudinal studies examining how somatic amplification affects depression and chronic pain over time.

The current study builds on the findings of Kaczynski et al. (2016) and examines depression as a mediator of the relation between non-spouse family strain and chronic pain interference, in addition to examining somatic amplification as a moderator of the relation between depression and chronic pain interference. Overall, there are mixed results regarding the relation between family strain and chronic pain. Specifically, some studies have found that negative family interactions and family strain are associated with more chronic pain [3, 17] and other studies did not support this association [32, 33]. In addition, there is no available research examining how non-spouse family strain is associated with depression and chronic pain. Although prior research has found that poorer family functioning was associated with greater depression and greater chronic pain [2, 18, 19], only one study [2] has examined these variables under a mediation framework. As shown in Fig. 1, it was predicted that depression would mediate the relation between non-spouse family strain and chronic pain interference because family strain and conflict may be proximally associated with depression, and comorbid depression in the context of chronic pain may lead to more severe pain. It was also hypothesized that the magnitude of indirect effects of non-spouse family strain on chronic pain through depression would depend on the level of an individual's somatic amplification, such that individuals with higher levels of depression will report higher levels of chronic pain interference if they have higher levels of somatic amplification.

The current study fills in several gaps in the literature regarding chronic pain and negative psychosocial outcomes. The study seeks to clarify the relation between non-spouse family strain and chronic pain interference, as the existing evidence supporting this association is inconclusive. This study is the first to examine the relations between non-spouse family strain and chronic pain interference. Additionally, this study is the first to examine somatic amplification as a moderator of depression and chronic pain interference within the context of family dynamics. The results of the current study will also generalize to older and middle-aged adults because a nationally representative sample was used, which is important as a high percentage of older adults experience comorbid depression and chronic pain [34, 35]. Most studies examining these associations have focused exclusively on children and adolescents [2–4] or middle-aged adults [5]. Overall, the current study will extend the literature regarding how the family environment is an important aspect to consider in relation to middle-aged and older adults experiencing negative psychosocial issues related to chronic pain.

Participants and Procedure

Data were analyzed from a community sample of 933 participants with chronic pain from the third wave of the National Survey of Midlife Development in the United States (MIDUS), a 2013 national survey of non-institutionalized English-speaking adults who resided in the USA ([36, 37], for a detailed description of the original study). Participants ranged in age from 39 to 93 years (mean age = 64.4 years, SD = 10.9) and included middle-aged adults and elderly adults. In terms of gender, 41.4% identified as male and 58.6% identified as female. Approximately 89.6% identified as White, 3.2% identified as Black and/or African American, 1.0% identified as Native American or Alaskan Islander, 0.2% identified as Asian, 0.1% identified as Native Hawaiian or Pacific Islander, and 5.0% identified as other races/ethnicities. All participants used in the study experienced some form of chronic pain. Specifically, all participants responded "yes" to the question, "Do you have chronic pain, that is do you have pain that persists beyond the time of normal healing and has lasted from anywhere from a few months to many years?" Participants completed questionnaires regarding depression and somatic amplification by a telephone interview and completed questionnaires regarding family strain and chronic pain interference through self-administered questionnaires.

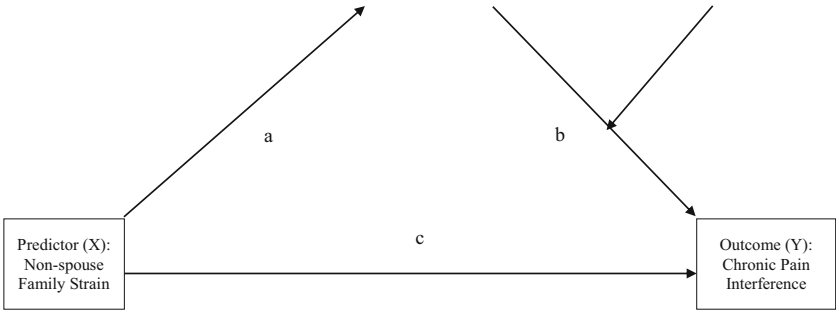

Fig. 1 This depicts the moderated mediation model examining depression as a mediator of the relation between non-spouse family strain and chronic pain interference. Somatic amplification is shown as a moderator of the relation between depression and chronic pain interference

Measures

Chronic Pain Interference To create an indicator of chronic pain, participants' answers to the MIDUS screening question, "Do you have chronic pain, that is, do you have pain that persists beyond the time of normal healing and has lasted anywhere from a few months to many years" were examined. For those who responded "yes," their score on the Brief Pain Inventory (BPI) interference scale was assessed [38]. The BPI is a valid and reliable measure for examining the extent to which chronic pain has interfered with general activity, walking, work, mood, relationships with other people, sleep, and enjoyment of life. The BPI interference scale has demonstrated strong validity through its strong association with pain severity across many samples [39] and good test-retest reliability and construct validity (all Cronbach's alphas > .90 [38]. Shortened interference scales are commonly used, as some items may not be applicable for all patients [40]. Responses on each pain interference question were measured using a 10-point numerical rating scale (0 = not at all, 10 = completely).

Non-spouse Family Strain In order to measure non-spouse family strain, questions measuring non-spouse family strain asked questions such as "Not including your spouse or partner, how often do members of your family make too many demands on you?" "How often do they criticize you?" and "How often do they let you down when you are counting on them?" Responses were measured using a 4-point numerical rating scale (4 = never, 1 = often). Based on the MIDUS sample, the non-spouse family strain scale has been validated [41]. The reliability of the non-spouse family strain scale was reported to be .80 [41].

Depression Depression was measured using a structured clinical interview that was developed from the World Health Organization's Composite International Diagnostic Interview (CIDI) [42]. The interview was given to each participant by a trained telephone interviewer. Although this version of the CIDI was based on the Diagnostic and Statistical Manual of Mental Disorders-3rd edition (DSM-III-R) [43], the criteria for major depressive disorder are the same as specified in the DSM-5 [44]. Participants were determined to have had a major depression episode if they had a period of two or more weeks in the past 12 months during which they experienced at least five of the following symptoms: depressed mood or loss of interest in most activities (for most of the day, nearly every day) as well as decreased or increased appetite, insomnia, fatigue or loss of energy, feelings of worthlessness, concentration problems, and recurrent thoughts of death. The final variable ranged from 0 to 7, where 0 represented participants being diagnosed as negative for major depression, and scores between one and seven represented the range of the symptom severity [45]. The scale has shown satisfactory validity and reliability [42]. The scale's interrater reliability was reported to be .95. Regarding validity, good overall diagnostic concordance was found between a clinical checklist and CIDI diagnoses ($K = .84$ for depressive disorders) [42]. Using MIDUS data, [46] found that depression was significantly associated with migraine pain (odds ratio = 1.70, confidence interval = 1.28–2.26, $p < .001$) as well as back pain (odds ratio = 1.40, confidence interval = 1.08–1.82, $p < .01$). Additionally, [47] used MIDUS data and concluded that depression was significantly linked with physical morbidity (e.g., an aggregation of chronic medical conditions) at two different time points ($r = .16$, $p < .001$).

Somatic Amplification Information regarding somatic amplification was collected using the Somatic Amplification Scale through telephone interviews and consisted of five items to assess each participant's awareness of bodily symptoms. The questions were as follows: (1) "I am often aware of various things happening in my body," (2) "I hate to be too hot or too cold," (3) "Sudden loud noises really bother me," (4) I am quick to sense hunger contractions in my stomach," and (5) "I have a low tolerance for pain." Participants rated their level of somatic amplification using a four-point numerical rating scale (1 = not at all true, 2 = a little true, 3 = moderately true, 4 = extremely true). The total somatic amplification score is obtained by averaging scores on the five items. Barsky and colleagues (1988) validated this scale and found the Cronbach's alpha to be .54 [29]. In a previous study using a sample of patients with upper respiratory tract infections, the scale's internal consistency was reported to be .72 with a test-retest reliability of .85 [29]. In the MIDUS sample as a whole,

the reliability estimate for the Somatic Amplification Scale was .53 (MIDUS, 2010).

Statistical Analysis

Preliminary analyses were first conducted in SPSS. Correlations were conducted to determine whether there were associations between the variables of interest (i.e., chronic pain interference, depression, non-spouse family strain, and somatic amplification (see Table 1). To examine the proposed hypotheses, Hayes's PROCESS macro was used [48]. To test whether the indirect path between the non-spouse family strain, depression, and chronic pain interference is contingent on somatic amplification, PROCESS Model 14 was used. This macro automatically produces the index of moderated mediation and utilizes bootstrapped confidence intervals to estimate conditional indirect relationships in which the indirect effect of the independent variable (e.g., non-spouse family strain) on the dependent variable (e.g., chronic pain interference) through the mediating variable (e.g., depression) is contingent on the moderator variable (e.g., somatic amplification). A total of 5000 bootstrap samples and a 95% CI were selected for these estimations. PROCESS also automatically produces conditional indirect effects at the mean and ± 1 SD from the mean and their bias-corrected bootstrap CI.

Results

Preliminary Findings

There were no missing data. The means, standard deviations, and intercorrelations of the variables used in our main analysis are presented in Table 1. Cohen's (1988) guidelines for the magnitude of effect sizes were used ($d = .20$ is a small effect size, $d = .50$ is a medium effect size, and $d = .80$ is a large effect size) [49]. Based on these guidelines, non-spouse family strain was positively associated with depression with a small effect size and depression was positively associated with chronic pain interference with a small effect size. Additionally, somatic amplification was positively associated with depression with a small effect size as well as chronic pain interference with a small effect size.

Table 1 Means, standard deviations, and Pearson correlations among chronic pain interference, depression, non-spouse family strain, and somatic amplification ($N = 933$)

Variable	*M*	*SD*	1	2	3	4
1. Chronic pain	3.22	2.62	–			
2. Depression	0.94	2.09	.29**	–		
3. Non-spouse family strain	2.01	0.66	.15**	.15**	–	
4. Somatic amplification	2.45	0.55	.22**	.15**	.19**	–

*Correlation is significant at the 0.05 level (2-tailed)

**Correlation is significant at the 0.01 level (2-tailed)

Moderated Mediation Analyses

PROCESS [48] was used to examine whether somatic amplification would moderate (a) the effect of depression on chronic pain interference as well as (b) the indirect effects of non-spouse family strain on chronic pain interference through depression. The first hypothesis predicted that depression would mediate the relation between non-spouse family strain and chronic pain interference. To test this hypothesis, PROCESS macro Model 14 [48] was used, which utilizes bootstrapping to calculate the indirect mediation effect. The results of the relations between non-spouse family strain, depression, and chronic pain are displayed in Table 2. Results demonstrated that non-spouse family strain was significantly associated with depression ($b = .371$, SE = .041, $p < .05$), and depression was significantly related to chronic pain ($b = .271$, SE = .188, $p < .05$), controlling for participants' age, gender, education, and ethnicity. These demographic variables were included as control variables to account for their relations with pain levels [50]. Specifically, being of minority status and being of lower socioeconomic status have been associated with higher pain scores and the association between gender and pain scores is inconclusive [50–52]. Further mediation analyses based on the bootstrapping method indicated that the effect of non-spouse family strain on chronic pain is mediated by depression at the mean of the moderator. However, the direct path between family strain and chronic pain given depression was significant ($b = .269$, SE = .128, $p < .05$), which indicates that partial mediation occurred. Thus, the first hypothesis was supported.

The second hypothesis stated that the indirect effect of non-spouse family strain on chronic pain interference through depression would be contingent on individuals' somatic amplification, such that individuals with higher levels of depression would report higher levels of chronic pain interference if they have higher levels of somatic amplification. The bootstrapping method with the PROCESS macro [48] was utilized to investigate this moderated mediation effect. This study utilized the PROCESS Model 14, in which the effect of depression on chronic is moderated by somatic amplification, controlling for age, gender, education, and ethnicity. As seen in Table 2, the interaction effect of depression and somatic amplification on chronic pain was significant ($b = .147$, SE = .070, $p < .05$).

The conditional indirect effects further demonstrated that the indirect effect of non-spouse family strain on chronic pain interference through depression was contingent on individuals' somatic amplification. Therefore, a graph was plotted to illustrate the nature of this interaction (see Fig. 2). The results demonstrate that the indirect effect of non-spouse

Table 2 Moderated mediation analyses for chronic pain interference, depression, non-spouse family strain, and somatic amplification

Variable	*B*	SE *B*	*t*	95% CI
Mediator: depression				
Constant	− .24	.55	− .44	[− 1.325, .837]
Predictor: Family Strain	.37**	.11	3.52	[.164, .577]
Outcome: Chronic Pain				
Constant	3.1**	.66	4.69	[1.808, 4.410]
Mediator: depression	.27**	.04	6.62	[.191, .352]
Predictor: family strain	.27*	.13	2.21	[.018, .519]
Moderator: somatic amplification	.74**	.15	4.82	[.436, 1.037]
Interaction: somatic amplification × depression	.15*	.07	2.10	[.009, .284]

N = 933. The betas presented in the table are from the regression analyses

**$p < .05$. ***$p < .01$

family strain on chronic pain interference via depression becomes stronger as the level of somatic amplification increases. The indirect effect of non-spouse family strain on chronic pain interference through depression was significant for low ($b = .071$, SE = .036, 95% CI = .016–.163), middle ($b = .101$, SE = .038, 95% CI = .037–.191), and high levels of somatic amplification ($b = .130$, SE = .047, 95% CI = .048–.237). Low somatic amplification was defined as a value one standard deviation below the mean, middle somatic amplification was defined as the mean value, and high SA was defined as a value 1 SD above the mean. Additionally, the index of moderated mediation was significant ($b = .054$, SE = .032, 95% CI = .007–.139), providing evidence for a significant indirect effect of non-spouse family strain on chronic pain interference through depression moderated by somatic amplification. Therefore, the proposed moderated mediation hypothesis (H2) was supported. Specifically, the moderated mediation analysis suggests that individuals with higher levels of depression report higher levels of chronic pain interference if they have higher levels of somatic amplification.

Discussion

Social relationships have been shown to largely contribute to how one perceives chronic pain [1, 2]. Family strain and poor family functioning could be linked with an individual experiencing greater pain [4, 5] because that individual may require more frequent care, which might be associated with increased strain in the family. However, the literature is mixed regarding whether poor family functioning and family strain are correlated with more severe chronic pain [3, 17, 32, 33]. It is also unclear whether non-spouse family strain would also be associated with more severe chronic pain. Depression might mediate between non-spouse family strain and chronic pain interference because negative family interactions may correspond with an individual experiencing depression, which then may be linked with someone to perceive pain as being more severe [18, 19]. Studies also indicate that somatic amplification is linked with higher levels of depression and chronic pain [25, 27, 28]. Thus, research in this area calls for a model to be developed that includes mediators and moderators examining the relations between family strain and chronic pain.

Therefore, the purpose of this study was to examine depression as a mediator of the relation between non-spouse family strain and chronic pain interference. Additionally, somatic amplification was examined as a moderator of the relation between depression and chronic pain interference. This moderated mediation model was designed to understand the effect of non-spouse family strain on individuals with chronic pain. First, the results of the current study supported the hypothesis that depression mediates the association between non-spouse family strain and chronic pain interference. As informed by family systems theory, this suggests that family strain is correlated with an individual experiencing depression. Possibly as a result of these negative family interactions, which may be connected with the perception of more severe chronic pain. This finding demonstrates that interactions with family members besides spouses affect the perception of chronic pain. Second, our results supported the hypothesis that somatic amplification would moderate the relation between depression and chronic pain interference. In other words, individuals who experienced higher levels of somatic amplification together with higher levels of depression were likely to also experience higher levels of chronic pain interference. Perhaps individuals who pay increased attention to unpleasant bodily sensations and are also depressed are more likely to attend to their depressive symptoms, which might contribute to more severe chronic pain.

The results illustrated that depression was a significant mediator between non-spouse family strain and chronic pain interference. This finding is consistent with previous research suggesting that a negative family environment may partly explain why individuals develop comorbid depressive

symptoms, which may influence the severity of chronic pain [18, 19]. While previous studies have found a significant link between negative family functioning and chronic pain [2, 3], very few studies have examined the mediating role of depression on this relationship. As posited by family systems theory, when a family member is experiencing chronic pain, it is likely that family dynamics change and it is possible that more family tension and strain will occur [6, 7]. Thus, it is possible that the present results occurred because an individual may have developed depression from experiencing family tension among family members besides spouses, which may have negatively affected their ability to cope with their chronic pain [20, 21]. The results of the current study are similar to those of other studies, which suggest that family-based interventions may provide an important additional focus in treatment for individuals in chronic pain [2, 20, 21]. Additionally, it is essential that healthcare providers understand the important connection between chronic pain and non-spouse family strain and educate families about how taking care of an individual with chronic pain may affect the dynamic of the family.

In addition, somatic amplification was found to be a significant moderator of the relation between chronic pain interference and depression. Specifically, individuals with higher levels of depression were more likely to report higher levels of chronic pain interference if they have higher levels of somatic amplification. These findings were consistent with the results of [25–28] who found that higher levels of somatic amplification were significantly linked to higher levels of depression and chronic pain, but these findings were not examined in relation to family strain. It is possible that individuals who had higher levels of somatic amplification and who also experienced elevated levels of depression paid increased attention to their mood symptoms, which contributed to more severe chronic pain. These results demonstrate that it may be beneficial for healthcare providers to assess for somatic amplification and other negative psychosocial outcomes when treating patients with chronic pain. The findings of this study also indicate that individual differences in the perception of physical symptoms may make some individuals more susceptible than others to subclinical levels of chronic pain. If clinicians find that their patients are experiencing somatic amplification, it may be helpful for patients to be referred to cognitive-behavioral interventions that help patients cope with somatization in addition to their pain.

This study is not without limitations. One limitation is that the measures utilized are from a secondary dataset and in this dataset, somatic amplification was measured with questions asked in a clinical interview, but chronic pain interference and non-spouse family strain were assessed by self-report questionnaires. Therefore, a replication of this study using a clinician-administered clinical interview examining family strain and chronic pain measures is required. Lastly, the analysis for depression was based on data using the DSM-III for depression criteria rather than newly released DSM-5 criteria. Future studies should replicate the present findings using DSM-5 chronic pain and depression criteria, preferably from a heterogeneous population. Additionally, using CITI's structured clinical interview for depression, it is not completely clear if participants' depression reflects their current depression, a depressive episode during the past year, or both, as this measure asks questions about depressive symptoms in the past 12 months. This study also utilized parent self-report measures. Self-report measures are not always accurate, as the participant may over-report or under-report symptoms due to social desirability. Additionally, the design of this study is cross-sectional, which prevents the specific direction of influence among variables to be ascertained. Future studies should utilize a longitudinal design and examine the relations among

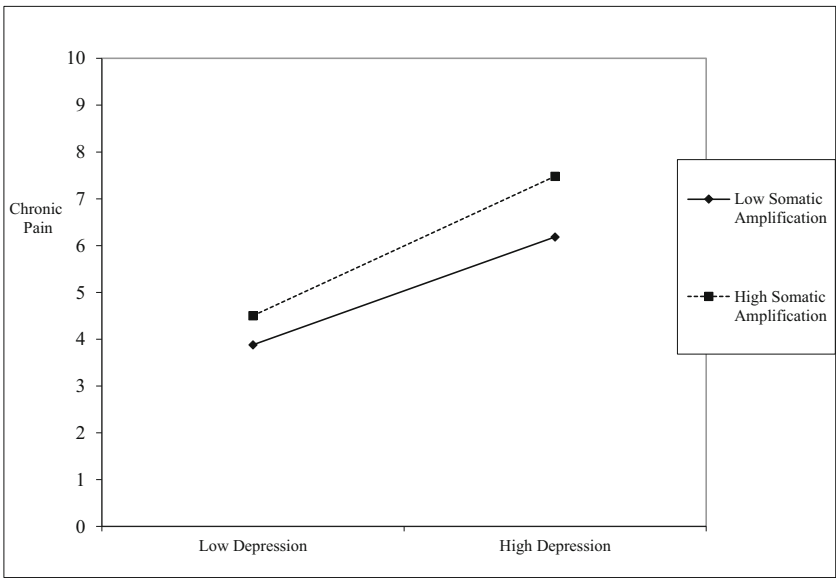

Fig. 2 This depicts somatic amplification as moderating the relation between depression and chronic pain (scores ranged from 0 to 10), such that individuals with higher levels of depression report higher levels of chronic pain interference if they have higher levels of somatic amplification. High and low depression and high and low amplification scores were created for the purposes of the figure using standard deviation values of 1 SD above the mean for high groups and 1 SD below the mean for low groups

family strain, depression, somatic amplification, and chronic pain over time. Another limitation is that the sample was primarily Caucasian and these results may not generalize to more diverse populations. Future studies should incorporate a more diverse sample in regard to ethnicity. The current study was also limited to one mediator (depression) and one moderator (somatic amplification). Future studies can be expanded to include other negative psychosocial outcomes that may be mediators including anxiety. Other moderators that may be relevant in explaining the relation between depression and chronic pain include health locus of control, self-esteem, and other psychological constructs. Additionally, although there were found to be significant associations between non-spouse family strain, somatic amplification, and chronic pain interference; the effects found in the model are generally small in magnitude. The magnitude of effects in prior studies was also small [2. 4. 49]; thus, it was expected that the effects of the current study would also be in the small to moderate range.

Despite these limitations, a major strength of this study is that the data utilized was from a large, nationally representative longitudinal study of middle-aged and elderly adults and there are important ways that the findings of the current study add to the current literature. The results demonstrate that it may be important for clinicians to assess family strain and family relationship dynamics among individuals with chronic pain [3, 17]. This study was also the first to examine whether non-spouse family strain was associated with depression and more chronic pain. Additionally, studies have indicated that chronic pain interventions incorporating the family have resulted in statistically significant reductions in perception of pain compared with patient-only interventions [53, 54]. Previous studies have found inconsistent results regarding the relation between family strain and chronic pain [3, 17, 32, 33]. The current study adds to the literature by providing a mechanism to explain this inconsistent relationship, such that depression mediates non-spouse family strain and chronic pain interference. Additionally, there is limited research examining how depression and somatic amplification affect family strain and chronic pain interference among elderly individuals with chronic pain. This study adds to the literature by providing empirical support that non-spouse family strain is linked with depression among middle-aged adults and elderly individuals, and that depression and somatic amplification may be contributors to the perception of more severe chronic pain. This moderated mediation model provides an excellent starting point in focusing on variables related to risk factors for the perception of more severe chronic pain among middle-aged and elderly individuals with chronic pain.

Compliance with Ethical Standards

Ethical Approval All procedures performed in studies involving human participants were in accordance with the ethical standards of the institutional and/or national research committee and with the 1964 Helsinki declaration and its later amendments or comparable ethical standards. This article does not contain any studies with animals performed by any of the authors.

Informed Consent Informed consent was obtained from all individual participants included in the study.

Conflict of Interest The authors declare that they have no conflict of interest.

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