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Close relationships as a contributor to chronic pain pathogenesis: Predicting pain etiology and persistence



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ARTICLE INFO

Keywords:

Acute pain
Chronic pain
Depression
Family relations
Family therapy
Friends
Middle aged adults
Systems theory

ABSTRACT

Rationale. Chronic stress contributes to the pathogenesis of chronic pain. Yet, the role of close relationship stress in these pathways to pain is not fully understood. **Objective.** To delineate specific psychosocial pathways associated with chronic pain, specifically emphasizing close relationships for midlife adults. We tested whether relationship strain, relationship support, social integration, depression, anxiety, and pain severity predict chronic pain etiology and persistence over 10 years, highlighting specific associations for acute versus chronic pain. **Method.** Using data from the National Survey of Midlife in the U.S. (MIDUS 2 and 3, collected in 2004–2006 and 2013–2014, respectively), we used logistic regression to test the etiology of new chronic pain ($n = 1591$) and persistence of pain for adults with acute ($n = 352$) and chronic pain ($n = 367$) conditions at baseline. **Results.** Of participants who reported they did not have chronic pain at baseline, the development of chronic pain 10 years later was significantly associated with baseline family strain ($OR = 1.38, p < .01$). For participants with acute pain at baseline, the transition of this pain to chronic a decade later was significantly associated with initial reports of pain interference ($OR = 1.24, p < .001$), family support ($OR = 0.60, p < .05$), and depression ($OR = 1.20, p < .05$). Persistent chronic pain was solely associated with baseline pain interference ($OR = 1.21, p < .01$). **Conclusions.** Family strain is an important part of the chronic stress profile associated with chronic pain etiology, whereas family support is associated with a reduced risk of acute pain transitioning to chronic pain over time. Prioritizing family relationships in treatment approaches to pain may be an indicated, innovative approach to preventing pain development and escalation and requires systems training in healthcare.

1. Introduction

Among growing rates of morbidity for midlife adults, are worsening reports of pain and declines in self-reported mental health. National Health Interview Survey (CDC, 2019) data in the U.S. demonstrate significant increases in each type of chronic pain assessed, as well as a significant rise in serious psychological distress, between 1999 and 2013 (Case and Deaton, 2015). These increases mirror global rates of pain, which suggest 20% of adults suffer from pain, and an additional 10% receive a new diagnosis of chronic pain, annually (Goldberg and McGee, 2011). International pain prevalence is a sizable threat to aging health, per the World Health Organization (Briggs et al., 2016). A large body of literature specifies that chronic distress contributes to chronic pain pathogenesis, including depression and anxiety (Edwards et al.,

2016; Meints and Edwards, 2018; Pincus et al., 2013). These mental health sequelae are further associated with a greater risk of being prescribed a higher dose of opioids for chronic pain (Scherrer et al., 2015). Yet, close relationship stress has infrequently been conceptualized as part of the stress-pain pathway.

1.1. Close relationships and chronic pain

Close relationships include immediate and extended family, intimate partnerships (including spousal relationships), and friends. Prior research demonstrates that better marital quality is associated with less pain (Reese et al., 2010) and lower pain-related disability (Robles et al., 2014), whereas patients whose partners are responsive to their pain experiences report improved physical functioning (Wilson et al., 2017).

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<https://doi.org/10.1016/j.socscimed.2019.112452>

Received 14 September 2018; Received in revised form 9 July 2019; Accepted 26 July 2019

Available online 30 July 2019

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In contrast, marital conflict is associated with increases in pain intensity (Burns et al., 2013). Moreover, the impact of intimate partner relationship quality on chronic pain appears to be moderated by depression (Campbell et al., 2012), suggesting a complex network of individual and relational links to pain.

Family relationships, other than intimate partnerships, are also associated with chronic pain. Recent research indicates that the amount of time spent with family, and the quality of family relationships, may be critical (Campbell et al., 2018). Specifically, adults with pain who have understanding, communicative families are more likely to sustain employment (McCluskey et al., 2015), and arthritis patients with greater family support from multiple sources (i.e., spouse, children, other family) report less depression (Hung et al., 2017).

Conversely, family dysfunction contributes to worse pain, including patients' disability (Akbari et al., 2016); this effect is mediated by loneliness and psychological distress (Stensland et al., 2014). Specific to the family-of-origin context, offspring of mothers or fathers who report chronic pain experience worse emotional wellbeing and social competence and have a greater likelihood of reporting chronic pain themselves (Higgins et al., 2015). The finding that parental chronic pain may be transmitted to adult offspring, such that adults who have parents with chronic pain are more likely to experience chronic pain themselves, is increasingly substantiated (e.g., Lier et al., 2015). This intergenerational transmission effect likely increases with time, as chronic pain may take years to develop and, thus, may be more observable in adult children (Lier et al., 2015). Moreover, in clinical studies of parents with chronic pain, participants report worse family functioning, although it remains unclear whether this, and other, offspring sequelae are directly related to the parental pain, itself, or common correlates (e.g., worse parental mental health; Higgins et al., 2015).

Overall, research is increasingly suggesting a close *relationship-stress-pain pathway* via cross-sectional associations in pain populations. These connections may be especially powerful for midlife adults (age 40–65) in the U.S., who are uniquely prone to the pain epidemic (Case and Deaton, 2015), and increasingly face shifting family relationships during their lifespan (Brewer et al., 2016). A cyclical relationship is likely whereby a lack of family or partner support and greater relational conflict contributes to individuals' experiencing greater stress; this increased distress increases pain severity and impairs pain recovery. Reciprocally, severe and chronic pain negatively impacts family functioning by causing stress, worry (De Souza and Frank, 2011), and interfering in family members' usual responsibilities (Strunin and Boden, 2004; Shaw et al., 2013). As further evidence, Jaremka et al. (2013, 2014) have found that associations between relationship support, social isolation, and pain are often clustered with depression and anxiety, reflecting a truly psychosomatic experience. The likely circular interactions between close relationships, emotional distress, and chronic pain suggests pain could best be understood using a systemic, biopsychosocial approach (Engel, 1980).

1.2. Present study

Pain, and accompanying opioid addiction, are notoriously challenging public health issues, complicated by prescribing practices and sociodemographic disparities (Grol-Prokopczyk, 2018). It is of particular importance to identify risk factors for chronic pain that are amenable to change. Demonstrating specific relationship stress pathways to the development and persistence of pain could inform public health initiatives about where to effectively intervene. It is important to note that research in this area requires longitudinal analyses of large sample sizes in order to explore specific relational factors by which close relationships impact pain, for a population most at risk (Robles et al., 2014). Therefore, we test the following hypotheses using a large, longitudinal study of U.S. adults:

1.) **Etiology** - Greater relationship strain; less relationship support; less

social integration; depression; and, anxiety will be associated with an increased risk of the onset of chronic and persistent pain 10 years later.

2.) **Persistence** - Greater relationship strain; less relationship support; less social integration; depression; anxiety; and, greater pain severity, will be associated with an increased risk of the persistence of chronic pain over 10 years.

We will test the second hypothesis for persistence of pain related to acute pain conditions (i.e., transitioning to chronic pain), as well as for chronic pain conditions.

2. Method

2.1. Sample

Data for this study are from the National Survey of Midlife in the U.S. (MIDUS), a longitudinal, nationally representative study investigating biopsychosocial pathways to health spanning 20 years (Ryff et al., 2017a). MIDUS is uniquely suited to test the present hypotheses, as it includes a rich collection of measures surveying midlife adults regarding their social, psychological, and physical health. The original MIDUS data collection occurred in 1995–1996 and included over 7000 U.S. adults recruited via random digit-dialing. The present study used MIDUS 2 (the initial follow-up to the first MIDUS project, collected in 2004–2006; $N = 4963$) and MIDUS 3 (collected in 2014–2016; $N = 3,294$, or 66% of MIDUS 2 participants) data, which are freely available through the Inter-University Consortium for Political and Social Research (Ryff et al., 2017a, 2017b). Participant retention in MIDUS has been impacted by sociodemographics, such that White, married, more educated, and healthier MIDUS 1 participants were more likely to participate in MIDUS 2 (Radler and Ryff, 2010).

We determined our etiology and persistence samples using a pain screener item included solely at MIDUS 2 and 3 (this item was not included at MIDUS 1). This item asked participants, “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?” allowing for a dichotomous response (yes/no). Participants who answered “No” at MIDUS 2 (i.e., denying pain), and responded to this same item at MIDUS 3 (“No” or “Yes”), were included in the etiology sample. Participants who answered “Yes” to this item at MIDUS 2 (i.e., reported experiencing persistent pain), and responded again to this item at MIDUS 3 (“No” or “Yes”), were considered for inclusion in the persistence samples. A physician/health care professional provided all pain diagnoses, which were gathered from participants using open-ended responses and coded by MIDUS researchers (Ryff et al., 2017a, 2017b). We excluded participants who reported their pain was due to cancer, or who reported ongoing cancer treatment/therapy (i.e., as “cancer” was not a pain diagnosis category at MIDUS 3) and concurrent chronic pain (due to “other” or “undiagnosed” reasons). Table 1 provides a summary of the pain characteristics of the present samples.

2.1.1. Etiology sample

The etiology sample is comprised of MIDUS 2 participants who denied experiencing chronic and persistent pain ($n = 2484$) and who also answered this item at MIDUS 3 ($n = 1592$). While eight participants in the etiology sample reported having chronic pain as well as undergoing cancer treatment at MIDUS 3, one listed a nonspecific pain diagnosis (i.e., “other muscle problem,” as well as “other” for type of cancer) and was excluded. This process resulted in a sample size of 1591 participants who denied chronic pain at baseline (MIDUS 2) and are included in the present etiology analyses. Approximately 26% ($n = 406$) of this subsample reported experiencing chronic pain 10 years later (at MIDUS 3); 71% of these reported receiving a pain diagnosis from a physician ($n = 75$ reported greater than 1 diagnosis; Table 1).

Table 1
Pain characteristics of participants with chronic pain in etiology and persistence samples.

Variables	Etiology Sample MIDUS 3 n = 406 with pain	Acute Pain Sample MIDUS 2 n = 352	Chronic Pain Sample MIDUS 2 n = 367
	n (%)	n (%)	n (%)
<i>Primary location of pain^a</i>			
Back	181 (44.6)	159 (45.2)	202 (55.0)
Legs	159 (39.2)	116 (33.0)	170 (46.3)
Knees	121 (29.8)	76 (21.6)	182 (49.6)
Shoulders	89 (21.9)	95 (27.0)	119 (32.4)
Hips	81 (20.0)	59 (16.8)	139 (37.9)
Arms	80 (19.7)	66 (18.8)	147 (40.1)
Neck	70 (17.2)	79 (22.4)	124 (33.8)
Head	24 (5.9)	29 (8.2)	50 (13.6)
Other	36 (8.9)	79 (22.4)	45 (12.3)
<i>Saw physician about pain</i>	320 (78.8)	339 (96.3)	361 (98.4)
<i>Pain diagnosis by physician^a</i>	290 (71.4)	323 (91.8)	349 (95.1)
Arthritis/ osteoarthritis	94 (23.2)	–	264 (71.9)
Disk related issue	78 (19.2)	42 (11.9)	46 (12.5)
Neck/shoulder problem	27 (6.7)	–	–
Fibromyalgia	6 (1.5)	–	31 (8.4)
Accident/injury	9 (2.2)	32 (7.7)	–
Hip problem/sciatica	20 (4.9)	4 (1.1)	6 (1.6)
Foot/ankle problem	20 (4.9)	3 (0.9)	11 (3.0)
Nerve problem	23 (5.7)	11 (3.1)	6 (1.6)
Other ^b	117 (28.8)	238 (67.6)	149 (40.6)
<i>Prescription pain medication use</i>	176 (43.4)	140 (39.8)	175 (47.7)

MIDUS = National Survey of Midlife in the U.S.

^a Participants noted all areas where pain is primarily located, and all pain diagnoses; therefore, percentages will equal > 100% of the samples.

^b Includes tendonitis, muscle related problems, rotator cuff problems, cartilage problems, surgery-related pain, migraines, etc.

2.1.2. Persistence samples

Of the full MIDUS 2 sample, 1461 participants self-reported experiencing chronic and persistent pain. Of the MIDUS 2 chronic pain sample, 61% ($n = 886$) completed the same pain identifier item at MIDUS 3 (a portion of the original sample was lost due to completing the computer-assisted telephone interview only [$n = 115$]). Three participants were excluded as they reported their pain diagnosis as “cancer” (at MIDUS 2), and two participants were excluded as they were currently undergoing cancer treatment and newly reported their pain diagnosis as “other” at MIDUS 3. This process resulted in a preliminary sample size of 881 participants who reported chronic pain at baseline (MIDUS 2). The majority of this sample (61%, $n = 540$) reported continuing to experience chronic and persistent pain at MIDUS 3 (i.e., pain duration equaling 10 years); 78% of these participants reported a specific pain diagnosis given by a physician.

2.1.2.1. Acute versus chronic pain at baseline. To test our persistence hypothesis, we separated the persistent chronic pain sample ($n = 881$) into two subsamples, by whether their pain was acute or chronic at MIDUS 2. This was achieved by reviewing physician-provided pain diagnoses or, when unavailable, examining health conditions participants reported receiving treatment for in the past year, current prescription medications (e.g., prescription arthritis medications used in past 30 days), and primary locations of pain. Acute pain diagnoses included, for example, accident or injury, herniated disk, surgery-related pain, cartilage problems, and inflammation/infection ($n = 352$). Chronic pain diagnoses included, for example, arthritis, degenerative disk disease, fibromyalgia, scoliosis, migraines, and stenosis ($n = 367$; Table 1). The remaining participants with pain at baseline ($n = 162$) were excluded from analyses due to the lack of

information regarding the cause or chronicity of their pain condition at MIDUS 2. Since we were unable to identify the diagnosis or cause of the chronic pain, we chose to remove them from the analysis in order to avoid confounding our findings.

2.1.2.2. Between group differences. Analysis of variance was used to compare the acute pain and chronic pain samples at baseline. The group of participants with chronic pain conditions at baseline were significantly older ($F = 11.46$, $p = .001$) and significantly more likely to be female ($F = 20.89$, $p < .001$) at baseline than participants with acute pain conditions. Total household income did not significantly differ between the two groups ($F = 1.46$, $p = .228$); nor did pain interference scores ($F = 1.80$, $p = .180$). Because these two groups differed significantly in their demographic makeup, and the characteristics of age and gender likely influence pain pathophysiology, they were analyzed separately to tease out distinct predictors of pain persistence among those with acute versus chronic pain conditions at baseline.

In sum, the present study examines three samples over 10 years: (1) those with no pain at MIDUS 2, to test our etiology hypothesis (i.e., whether participants remain pain-free or develop persistent pain); (2) those with acute pain at MIDUS 2, to test our persistence hypothesis (i.e., whether acute pain remits or transitions to chronic pain); and (3), those with chronic pain conditions at MIDUS 2, to test our persistence hypothesis (i.e., whether chronic pain remits or continues). As data are secondary and de-identified, human subjects review was not required for this project.

3. Measures

Each of the measures included were completed via the MIDUS 2 self-administered questionnaire. Descriptive statistics for each measure are summarized in Table 2, indicating each measure was reliable in the current samples. MIDUS researchers used mean imputation to replace missing data in each of the relationship strain, relationship support, and mental health measures. Each of the scale scores were calculated using a mean of item responses for all participants with a minimum of one valid item response (Ryff et al., 2017b). Following imputation, missing data were less than 5% in each of the relationship strain, relationship support, mental health, and pain interference measures.

3.1. Relationship strain

Three distinct measures of relationship strain were included, specific to relationships with family, friends, and intimate partners/spouses.

3.1.1. Family strain

The measure of family strain, developed by Walen and Lachman (2000), included four items assessing how often family members, “not including your spouse or partner,” make too many demands, criticize, let the participant down, or get on the participant’s nerves. Participants responded on a scale of 1 (*often*) to 4 (*never*); responses were reverse scored and averaged, such that higher scores indicate greater strain.

3.1.2. Friend strain

The measure of friend strain, also developed by Walen and Lachman (2000), included four items reflective of the family strain measure, above. Specifically, these items assessed how often a participant’s friends make too many demands, criticize them, let the participant down, and get on the participant’s nerves. Responses ranged from 1 (*often*) to 4 (*never*), and were reverse scored and averaged; higher scores indicate greater friend strain.

3.1.3. Intimate partner strain

The third relationship strain measure assessed intimate partner

Table 2
Independent variables: Descriptive statistics.

Variables	Etiology Sample n = 1591		Acute Pain Sample n = 352		Chronic Pain Sample n = 367	
	M (SD)	α	M (SD)	α	M (SD)	α
<i>Relationship strain</i>						
Family strain	2.00 (0.56)	.77	2.10 (0.59)	.77	2.13 (0.62)	.81
Friend strain	1.82 (0.48)	.78	1.88 (0.49)	.77	1.83 (0.51)	.81
Intimate partner strain	2.12 (0.58)	.86	2.22 (0.65)	.88	2.18 (0.62)	.88
<i>Relationship support</i>						
Family support	3.56 (0.53)	.82	3.53 (0.58)	.83	3.55 (0.62)	.88
Friend support	3.34 (0.62)	.87	3.24 (0.69)	.88	3.38 (0.62)	.89
Intimate partner support	3.66 (0.49)	.89	3.55 (0.64)	.93	3.64 (0.50)	.89
<i>Social integration</i>						
Family contact	2.97 (1.42)	–	3.11 (1.52)	–	2.84 (1.46)	–
Friend contact	3.34 (1.65)	–	3.45 (1.72)	–	3.23 (1.66)	–
<i>Mental health</i>						
Depression	.45 (1.49)	–	.96 (2.11)	–	.82 (1.95)	–
Anxiety	.07 (0.62)	–	.20 (1.05)	–	.20 (1.20)	–
<i>Pain severity</i>						
Pain interference	–	–	2.97 (2.43)	.91	3.22 (2.46)	.90

Note: M = Mean; SD = Standard deviation; α = Cronbach's alpha.

strain (for participants currently in an intimate partnership) and included six items asking how often the participant's spouse or partner makes too many demands, argues with the participant, makes the participant feel tense, criticizes, lets the participant down, and gets on the participant's nerves; participants responded on a scale of 1 (*often*) to 4 (*never*; Walen and Lachman, 2000). Participant responses were reverse coded then averaged; higher scores reflected greater strain (Ryff et al., 2017b).

3.2. Relationship support

Three measures of relationship support were included, each developed by Walen and Lachman (2000) as counterparts to the family, friend, and intimate partner strain measures described above.

3.2.1. Family support

The family support measure asked participants to describe their relationships with their family, “not including your spouse or partner” (Walen and Lachman, 2000). The measure included four items asking participants how much family members care about them, understand the way they feel, can be relied on for help in a serious problem, and can be opened up to when the participant needs to discuss their worries. Participants responded on a scale from 1 (*a lot*) to 4 (*not at all*); each of these responses was reverse coded and averaged, such that higher scores reflect greater support.

3.2.2. Friend support

Similar to family support, the friend support measure used four items assessing participants' perspectives regarding how much their friends care about them, understand how they feel, can be relied upon, and can be opened up to (Ryff et al., 2017b; Walen and Lachman, 2000). MIDUS respondents answered items on a scale of 1 (*a lot*) to 4 (*not at all*), each of which was reverse scored. Scale scores were computed using a mean of item responses.

3.2.3. Intimate partner support

The intimate partner support measure (for participants currently in an intimate partnership) included six items assessing the same four areas as the prior two support measures, along with asking how much a spouse/partner appreciates the participant, and how much the participant can relax and be themselves around their partner (Walen and Lachman, 2000). Participants rated their response on a scale from 1 (*a lot*) to 4 (*not at all*); responses were reverse coded and averaged, such that higher scores reflect greater intimate partner support.

3.3. Social integration

In addition to assessing *relationship strain* and *relationship support* domains, we included three single-item measures of *social integration*, including frequency of contact with family, frequency of contact with friends, and marital status. The *frequency of contact with family* item asked, “How often are you in contact with any members of your family, that is, any of your brothers, sisters, parents, or children who do not live with you, including visits, phone calls, letters or electronic mail messages?” The item assessing *frequency of contact with friends* asked, “How often are you in contact with any of your friends, including visits, phone calls, letters, or electronic mail messages?” Respondents answered both items using an 8-anchored scale ranging from 1 (*several times a day*) to 8 (*never or hardly ever*). These two social contact measures have been successfully used in prior MIDUS research, and linked to persistent major depression (Walker and Druss, 2015) and psychophysiological stress (Gruenewald et al., 2012). Lastly, participants self-reported their current marital status, which was dichotomized (1 = *married*, 0 = *not married*).

3.4. Mental health

We included two measures of mental health, assessing depression and anxiety, both originating from the World Health Organization's (WHO, 1990) Composite International Diagnostic Interview-Short Form (CIDI-SF). The CIDI-SF was developed to categorize respondents in accordance to the *Diagnostic and Statistical Manual of Mental Disorders* (American Psychiatric Association, 2000; Kessler et al., 1998). While the CIDI-SF major depression and generalized anxiety subscale scores are continuous and can be correlated to probabilities of meeting diagnostic thresholds, they are not in and of themselves diagnostic, in the short form. The psychometric properties of these CIDI-SF subscales have been widely substantiated and demonstrated with the present MIDUS dataset, including good reliability and specificity (Walker and Druss, 2015). The dichotomous responses of the CIDI-SF scale prevents calculating reliability estimates (Vittengl, 2017) for the present pain subsamples.

3.4.1. Depression

The first mental health measure included was the CIDI-SF major depression subscale (including depressed affect and anhedonia), which assessed the presence of seven symptoms during a two-week period in the past 12 months, including loss of interest, change in appetite, trouble concentrating, and feeling down or worthless (Wang et al., 2000). Major depressive episode scale scores are calculated as a sum of “Yes” responses to each symptom, ranging from 0 (*lowest depression*,

Table 3
Summary of univariate regression analysis for relationship quality, social integration, and mental health predicting the etiology of chronic pain.

Variable	OR (95% CI)
Family strain	1.34** (1.09, 1.63)
Friend strain	1.25 (0.98, 1.58)
Intimate partner strain	1.30* (1.05, 1.62)
Family support	1.14 (0.92, 1.42)
Friend support	0.95 (0.79, 1.14)
Intimate partner support	0.86 (0.67, 1.10)
Family contact	0.93 (0.85, 1.00)
Friend contact	0.99 (0.93, 1.06)
Marital status	1.05 (0.81, 1.35)
Depression	1.08* (1.01, 1.16)
Anxiety	1.00 (0.83, 1.20)
Age	1.00 (0.99, 1.01)
Sex	0.87 (0.70, 1.09)
Baseline income	1.06 (0.84, 1.33)

Note: OR = Odds ratio; CI = Confidence interval. Sex coded as 1 for male and 0 for female at MIDUS 2. Baseline income coded as 1 for below the median and 0 for median income or greater at MIDUS 2. *p < .05, **p < .01.

equating to participants denying the occurrence of two weeks of depressed affect as well as denying two weeks of anhedonia) to 7 (highest depression). Participants who score a 3 or greater are likely to meet full diagnostic criteria for a major depressive episode if assessed using the complete CIDI interview (Nelson et al., 2001).

3.4.2. Anxiety

The second CIDI-SF measure used was the generalized anxiety disorder (GAD) subscale, which included 10 items assessing the frequency of anxiety symptoms over the past 12 months. These items were asked of participants who responded positively to the pre-condition GAD items that they worry “a lot more” than most people, worry “every day, just about every day, or most days,” and worry about either “more than one thing” or different worries “at the same time.” Example symptoms include restlessness, trouble falling asleep, and trouble remembering things because of worry. Respondents answered on a scale from 1 (most days) to 4 (never). Scale scores were calculated using the total number of “most days” responses, and ranged from 0 (lowest anxiety, assigned to participants who did not meet the pre-condition GAD items or whose individual symptoms occurred half the days or fewer) to 10 (highest anxiety).

3.5. Pain severity

We utilized two measures of pain severity: an assessment of the interference of pain in participants' lives and use of prescription pain medications. These baseline measures of pain severity were administered only to participants who responded “Yes” to the MIDUS 2 chronic pain screener item and, therefore, are used only in pain persistence analyses.

3.5.1. Pain interference

Five items from the Brief Pain Inventory Short Form (BPI) were used to assess pain interference in participants' daily activities (Cleeland, 2009). Participants were asked, “How much, during the past week, your pain interfered with your ...,” followed by: general activity, mood, relations with other people, sleep, and enjoyment of life. Participants responded using a 10-point Likert scale, ranging from 0 (did not interfere) to 10 (completely interfered). Scale scores were calculated using mean responses; higher scores reflect greater pain interference. Multiple versions of the BPI have been demonstrated as valid and reliable (Williams et al., 2006), and scores are significantly related to past week pain intensity (Raichle et al., 2006). The five-item version has been

successfully used in prior pain research examining MIDUS participants, and found to be reliable ($\alpha = 0.95$; Brown et al., 2018), as it was with the present samples (Table 2). Mean imputation was used in this study for participants with a minimum of two valid responses.

3.5.2. Prescription pain medication use

Participants were asked, “During the past 30 days, have you taken prescription medicine for any of the following conditions?” and were specifically asked about “pain” as a condition, allowing for a dichotomous response (yes/no). The MIDUS self-administered questionnaire did not further specify type of pain medication (Ryff et al., 2017b).

3.6. Confounding variables

Literature supports that age (Briggs et al., 2016, Brown et al., 2018), sex (Kennedy et al., 2014), and income (Goldberg and McGee, 2011) are each associated with chronic and persistent pain. Thus, we include each variable in our full models. Both sex (coded as 1 for male and 0 for female at MIDUS 2) and baseline income (coded as 1 for below the median and 0 for median income or greater at MIDUS 2) are entered as dichotomous covariates.

4. Analyses

We first conducted univariate logistic regression analyses to identify significant independent predictors of MIDUS 3 chronic pain (Tables 1 and 3). Second, we conducted full model testing, using multivariate logistic regression and entering sequentially (1) potential confounding variables (i.e., age, sex, income), and (2) each of the independent variables found to be significant predictors of pain in the univariate analyses (Tables 2 and 4). Model fit was assessed by examining the significance of model χ^2 statistics (i.e., $p < .05$). Odds ratios (OR) and 95% confidence intervals (CI) for each independent variable are reported.

Table 4

Summary of logistic regression full model analysis for relationship strain and mental health predicting the etiology of chronic pain ($n = 1247$).

	OR	95% CI	R ²	χ^2 (df)
<i>Baseline Model:</i>				
Age	1.00	0.99, 1.01	.001	0.68 (3)
Sex	1.11	0.86, 1.44		
Income	1.00	0.76, 1.31		
<i>Model 1:</i>				
Age	1.00	0.99, 1.02	.01	7.07** (1)
Sex	1.08	0.83, 1.40		
Income	.97	0.74, 1.28		
Family strain	1.38**	1.09, 1.74		
<i>Model 2:</i>				
Age	1.00	0.99, 1.02	.01	2.88 (1)
Sex	1.08	0.83, 1.40		
Income	.98	0.74, 1.28		
Family strain	1.29*	1.01, 1.66		
Intimate partner strain	1.22	0.97, 1.53		
<i>Model 3:</i>				
Age	1.01	0.99, 1.02	.02	2.14 (1)
Sex	1.06	0.81, 1.38		
Income	.99	0.75, 1.29		
Family strain	1.27	.995, 1.63		
Intimate partner strain	1.21	0.96, 1.52		
Depression	1.07	0.98, 1.17		

Note: OR = Odds ratio (exponentiated β); CI = confidence interval. Sex coded as 1 for male and 0 for female at MIDUS 2. Baseline income coded as 1 for below the median and 0 for median income or greater at MIDUS 2. Etiology of pain coded as 1 for chronic pain and 0 for no chronic pain at MIDUS 3. *p < .05, **p < .01.

5. Results

5.1. Etiology

The average age of the etiology sample was 54.75 ($SD = 11.28$), and the majority identified as female (54%), White (92.9%), non-Hispanic (97.2%), and married (74%), with a median household income of \$65,000 at MIDUS 2 (including total wages, pension, SSI, and any other government assistance; $M = \$79,351$, $SD = \$62,118$).

Univariate regressions indicated that family strain and intimate partner strain were each significantly associated with the likelihood of developing chronic pain (see Table 3). None of the remaining relational variables were significantly associated with later chronic pain. Depression was minimally significant, associated with increased odds of developing chronic pain at MIDUS 3 by 8%, whereas anxiety was nonsignificant. The full model incorporated each of these variables; however, intimate partner strain and depression were rendered nonsignificant. Solely family strain was significantly associated with new chronic pain at MIDUS 3; specifically, each one-unit increase in family strain was associated with a 38% increased risk in the likelihood of developing chronic pain over the 10-year span. Neither age, gender, nor income were significant predictors of pain etiology; thus, their inclusion rendered the full model nonsignificant (Model 1, $\chi^2 = 7.753$, $p = .101$, Nagelkerke $R^2 = 0.01$; see Table 4).

5.2. Persistence - acute

The average age of the acute pain sample was 56.05 ($SD = 11.36$) at MIDUS 2, and the majority identified as female (53.7%), White (93.5%), non-Hispanic (98%) and married (71.9%), with a median household income of \$58,000 ($M = \$70,474.41$, $SD = \$58,367.86$).

Univariate regression analyses indicated that family support, depression, pain interference, and having used prescription pain medications were each significantly associated with the likelihood of acute pain transitioning to chronic pain over 10 years (see Table 5). Neither age, sex, nor income were significantly associated with the persistence of acute pain, though each were retained in full model testing as covariates. The full model results indicate family support, depression, and

Table 5

Summary of univariate regression analyses for relationship quality, social integration, and mental health predicting pain persistence.

Variable	Acute Pain Sample	Chronic Pain Sample
	$n = 352$	$n = 367$
	OR	OR
	(95% CI)	(95% CI)
Family strain	1.23 (0.86, 1.77)	1.37 (0.95, 1.98)
Friend strain	1.33 (0.86, 2.06)	1.28 (0.83, 1.99)
Intimate partner strain	0.80 (0.55, 1.16)	0.98 (0.65, 1.49)
Family support	0.60* (0.41, .90)	0.72 (0.48, 1.07)
Friend support	0.77 (0.56, 1.05)	0.78 (0.54, 1.14)
Intimate partner support	0.97 (0.67, 1.41)	1.09 (0.66, 1.80)
Family contact	1.05 (0.91, 1.21)	1.12 (0.95, 1.31)
Friend contact	0.96 (0.85, 1.09)	1.07 (0.93, 1.23)
Marital status	1.17 (0.73, 1.87)	1.15 (0.71, 1.87)
Depression	1.29*** (1.14, 1.46)	1.15* (1.01, 1.32)
Anxiety	1.08 (0.87, 1.34)	1.10 (0.88, 1.38)
Pain interference	1.29*** (1.17, 1.43)	1.23*** (1.10, 1.36)
Pain Rx use	2.25*** (1.41, 3.58)	1.69* (1.04, 2.74)
Age	1.00 (0.98, 1.02)	1.00 (0.97, 1.02)
Sex	1.03 (0.68, 1.59)	0.97 (0.60, 1.58)
Income	1.11 (0.73, 1.70)	1.93** (1.21, 3.08)

Note: OR = Odds ratio; CI = confidence interval; Rx = prescription medication. Sex coded as 1 for male and 0 for female at MIDUS 2. Baseline income coded as 1 for below the median and 0 for median income or greater at MIDUS 2. * $p < .05$, ** $p < .01$, *** $p < .001$.

pain interference remain significant predictors, while use of pain medications was rendered nonsignificant (Model 4, $\chi^2 = 46.88$, $p < .001$, Nagelkerke $R^2 = 0.19$; see Table 6). Specifically, baseline pain interference and depression were significantly associated with an increased risk of acute pain transitioning to chronic pain at MIDUS 3. A one-unit increase in pain interference at MIDUS 2 was associated with a 24% increased risk of continuing to report chronic pain at MIDUS 3, while a one-unit increase in depression was associated with a 20% risk increase. Family support was the sole significant relational predictor: A one-unit increase in family support at MIDUS 2 was associated with a 40% decreased risk of acute pain persisting, and transitioning into chronic pain, over the next 10 years.

5.3. Persistence - chronic

The average age of the chronic pain sample was 58.75 ($SD = 10.04$), and the majority identified as female (70%), White (93.5%); 2.5% Black or African American), non-Hispanic (94.3%) and married (68.9%), with a median household income of \$51,500 ($M = \$65,089.79$, $SD = \$58,502.38$).

Univariate regressions found that depression, pain interference, and having used prescription pain medications were significantly associated with the likelihood of chronic pain persisting over 10 years (see Table 5). Solely baseline pain interference remained significantly associated with the risk of chronic pain persisting at MIDUS 3 in the full model (Model 1, $\chi^2 = 22.69$, $p < .001$, Nagelkerke $R^2 = 0.10$; see Table 6). Similar to the acute pain condition sample, a one-unit increase in pain interference at MIDUS 2 was associated with a 21% increased risk of continuing to report chronic pain at MIDUS 3. Neither relational strain nor support were significantly associated with persistent chronic pain, contrary to our hypotheses.

6. Discussion

6.1. Etiology

Despite testing a comprehensive range of close relationship measures, participants' odds of developing chronic pain over 10 years was only associated with their baseline family strain. In other words, neither relationship support, nor alternate measures of relational strain (i.e., friend strain, intimate partner strain) were significantly associated with the later development of pain. Though participants experiencing greater family strain at baseline were significantly more likely to develop chronic pain 10 years later, intimate partner strain failed to remain significant in the full model. As neither marital status nor intimate partner measures were associated with the development of pain, it may be that other family relationships reflect different and unique processes that influence physiology.

Also notable are the nonsignificant contributions of social integration (i.e., family and friend contact) to the onset of chronic and persistent pain. While this result reflects recent research that finds a minimal contribution of social connectivity to physical health risk (Yang et al., 2016), social support research frequently evaluates variability in integration and embeddedness in social systems, as relevant for adult health (Shor and Roelfs, 2015). Intimate partner and friend relationship quality were also nonsignificant. Therefore, a potential explanation for the significant impact of family strain may be the longitudinal and emotionally impactful nature of family-of-origin relationships (Weihls et al., 2002). It may be that relationships with parents and siblings, especially those that are strained, have a greater capability to influence individual family members' stress reactivity and, through psychophysiological reactivity processes, impact disease activity. Further, given the psychosomatic nature of chronic pain, health risk in this area may be especially impacted by family relationship distress over time. While it may be intuitive to consider promoting family or friend support or social integration to improve health outcomes,

Table 6
Summary of logistic regression full model analyses for relationship quality, mental health, and pain severity predicting pain persistence.

Acute Pain Sample <i>n</i> = 299					Chronic Pain Sample <i>n</i> = 299				
	OR	95% CI	R ²	χ ² (df)		OR	95% CI	R ²	χ ² (df)
<i>Baseline Model:</i>			.001	0.123 (3)	<i>Baseline Model:</i>			.06	12.35** (3)
Age	1.00	0.98, 1.02			Age	0.99	0.97, 1.02		
Sex	1.09	0.69, 1.72			Sex	0.79	0.45, 1.40		
Income	1.00	0.63, 1.60			Income	0.40**	0.24, .68		
<i>Model 1:</i>			.13	31.41*** (1)	<i>Model 1:</i>			.10	10.34** (1)
Age	1.01	0.99, 1.03			Age	1.00	0.97, 1.03		
Sex	0.92	0.57, 1.50			Sex	0.73	0.41, 1.31		
Income	1.18	0.72, 1.93			Income	.48**	0.28, .84		
Pain interference	1.34***	1.20, 1.49			Pain interference	1.21**	1.08, 1.36		
<i>Model 2:</i>			.15	4.90* (1)	<i>Model 2:</i>			.10	0.18 (1)
Age	1.01	0.99, 1.03			Age	1.00	0.97, 1.03		
Sex	1.01	0.61, 1.65			Sex	0.73	0.41, 1.30		
Income	1.20	1.19, 1.49			Income	.49*	0.28, .86		
Pain interference	1.28***	1.14, 1.44			Pain interference	1.19**	1.05, 1.35		
Family support	0.59*	0.37, .96			Depression	1.04	0.68, 1.22		
<i>Model 3:</i>			.18	6.71* (1)	<i>Model 3:</i>			.11	0.04 (1)
Age	1.01	0.99, 1.04			Age	1.00	0.97, 1.03		
Sex	0.94	0.57, 1.56			Sex	0.73	0.41, 1.30		
Income	1.22	0.74, 2.03			Income	0.50*	0.28, .87		
Pain interference	1.28***	1.14, 1.44			Pain interference	1.19*	1.04, 1.36		
Family support	0.62	0.38, 1.02			Depression	1.03	0.88, 1.22		
Depression	1.20*	1.04, 1.40			Pain Rx use	1.06	0.60, 1.87		
<i>Model 4:</i>			.19	3.73 (1)					
Age	1.01	0.99, 1.03							
Sex	0.88	0.53, 1.47							
Income	1.25	0.75, 2.08							
Pain interference	1.24***	1.10, 1.40							
Family support	0.60*	0.36, .99							
Depression	1.20*	1.04, 1.39							
Pain Rx use	1.69	0.99, 2.89							

Note: OR = Odds ratio (exponentiated β); CI = Confidence interval; Rx = Prescription medication. Sex coded as 1 for *male* and 0 for *female* at MIDUS 2. Baseline income coded as 1 for *below the median* and 0 for *median income or greater* at MIDUS 2. Prescription pain medication use coded as 1 for *yes* and 0 for *no* at MIDUS 2. Pain persistence coded as 1 for *chronic pain* and 0 for *no chronic pain* at MIDUS 3. **p* < .05, ***p* < .01, ****p* < .001.

our findings suggest ameliorating family strain may have more meaningful impacts on preventing the development of chronic pain.

Lastly, anxiety at baseline was not associated with later reports of chronic pain, and depression demonstrated a minimal effect that was nonsignificant in the full model. This finding may reflect alternate research in the literature that is beginning to highlight the role of pain treatment in the etiology of depression and anxiety symptoms, rather than the reverse (e.g., Scherrer et al., 2016).

6.2. Persistence

While more strained family relationships were associated with new pain, supportive family relationships were associated with a decreased risk of acute pain transitioning into chronic pain. This same finding, though, was not true for participants with chronic pain conditions at baseline. One interpretation is the potential for family support to buffer against worse outcomes from acute pain conditions such as injury, surgery, and infection, whereas receiving this benefit may not be possible for midlife adults with more serious pain-related conditions, such as arthritis and fibromyalgia. Moreover, depression at MIDUS 2 was also associated with whether acute pain participants, but not those with chronic pain conditions, continued to report pain at MIDUS 3. Therefore, though both groups of participants who reported experiencing pain at baseline demonstrated associations between initial pain severity and chronicity of pain over time, family support and depression were associated with the persistence of pain over time solely for those with acute pain conditions.

Lastly, though our univariate findings suggest having ever used prescription pain medications is associated with persistent pain, the effects did not remain significant when modeled alongside pain interference for either sample. This variable may therefore reflect the intensity and severity of baseline pain, rather than meaningful

contributions of prescription pain medications to the physiology of pain persistence over time, as others have found (Scherrer et al., 2016). Additional research is needed to test the combined effects of these psychosocial variables, and how family relationships, especially, may be leveraged to promote acute pain recovery.

6.3. Practice implications

Stressful, conflictual family relationships alone were associated with the risk of developing chronic and persistent pain; caring, consistent, supportive family relationships were associated with acute pain healing. Once pain developed, its maintenance was associated with the severity of the pain at baseline and its interference in multiple areas of life, as well as depression for those with acute pain. These associations potentially speak to two areas of public health importance: pain prevention and pain intervention.

6.3.1. Pain prevention

Efforts to prevent chronic pain (or promote targeted early treatment of acute pain, preventing escalation) remain elusive (Friction, 2015), which is, in part, due to the prevalence of chronic pain across health conditions, such that no one large group of researchers or providers is dedicated to developing, testing, and implementing preventive efforts (Institute of Medicine, 2011). Despite the critical importance of comprehensive, biopsychosocial pain prevention, approaches are generally limited to methods aimed at the individual patient. Self-care, health education, and shaping patient expectations are examples; these efforts are often located in primary care, which is not supported by adequate training to do this preventive work thoroughly (Institute of Medicine, 2011).

In contrast, the present findings highlight the potential adjunctive value of targeting family relationships in preventing the onset of

chronic pain, and the escalation of acute pain to chronic pain. This method would require specific family-based treatments targeting patients and their families, with the goal of lessening criticism, unreliability, and hostility, while promoting warmth and understanding, as would occur in systemic family therapy. In the context of acute pain, the minimal research investigating relational treatments has predominantly investigated spousal support (e.g., Abassi et al., 2012; Martire et al., 2010). Exceptions focus on family-level psychoeducation, promoting and adjusting patient-family communication (Deek et al., 2016), offering support to family members (Swift et al., 2014), addressing both patients' and family members' beliefs regarding pain, and exploring social stigma related to invisible health conditions (Mawdsley et al., 2016). Further, for individuals presenting with acute pain (e.g., injuries, surgery-related pain, new diagnoses of a herniated disk), clinically involving families early on may prevent the transition of the new, acute pain into chronic, unremitting pain. Evidence demonstrates patients and their family members want family to be involved in clinical care to ensure each are on the same page and that they can collaborate in treatment (Swift et al., 2014). Overall, a medical family therapist may be especially beneficial in caring for these patients (Paries et al., 2018; McDaniel et al., 2014).

Utilizing family-based interventions is likely to be effective (Gilbert et al., 2005), with cost offset effects (Crane and Payne, 2011). Implementation would require regular assessment of the quality of family relationships in health care, which could be done with ease (Woods et al., 2015) and should be done early on for those with acute pain, especially given a likely period of "watch and wait" prior to engaging in pain treatment (Shaw et al., 2013). Early intervention is especially important, as medical providers treating pain patients with greater psychosocial risk for pain-related disability are already likely to expand their assessment. Yet, these physicians tend to focus their more detailed questioning specifically on biomedical explanations for pain and medication management, rather than on psychosocial factors (Shaw et al., 2009).

6.3.2. Pain intervention

Current standards for pain treatment focus on engaging individual patients in their care and self-management, analgesics, exercise therapy, and cognitive behavioral therapy (Dowell et al., 2016; McGreevy et al., 2011). The present findings may lend support for this approach, for individuals with chronic pain conditions whose pain has persisted a decade or more. Interventions targeting pain's interference in patients' lives (including their close relationships) may be especially key. It may also be advantageous to consider family-based interventions, given the impact of families on pain, and the straining effects of chronic pain on close relationships. Current pain treatment paradigms may also exacerbate the negative impact of pain on family functioning, as these fail to consider the relational ramifications of pain; families may experience uncertainty and frustration with unclear treatment plans (Shaw et al., 2013). Future research should tease out how the impact of family strain on the etiology of chronic pain may continue, or worsen, in the presence of persistent pain.

6.3.3. Healthcare training

The above indications, for a focus on family, emphasize a need for multilevel, systemic, biopsychosocial assessment and treatment (Engel, 1980), which necessitates a systems approach to training healthcare providers, especially in regard to pain. In order for physicians, nurses, mental health providers, and other clinicians to be able to adapt their practice to evaluate and intervene in relational contributions to pain, their training must broaden beyond pain-specific biological (e.g., brain structure alterations) or psychological (e.g., negative affect) variables to include interpersonal and family-level aspects of health (Meints and Edwards, 2018). This inclusion is especially true of primary care, in which providers serve a key function of pain treatment coordination (Langford et al., 2018) and may be especially adept at developing a

family-oriented approach to healthcare (McDaniel et al., 2005). Training should also include the value and skills of interprofessional collaboration, promoting active integration of systemically-oriented behavioral health providers (e.g., marriage and family therapists) into pain treatment plans and healthcare settings. Application of the biopsychosocial model to pain has been infrequent in training and practice (Pincus et al., 2013; Woods, 2019); in order to improve the latter, clinicians must be prepared, up front, to consider multifactorial, systemic impacts on pain and intervene within families.

6.4. Limitations and future research

Though this study adds to the existing literature, it also presents opportunities for next research steps. First, as we were limited to two time points, we were also limited in our analytic plan. In other words, although we tested the hypotheses of pain etiology and persistence over 10 years, it is probable that trajectories of pain are nonlinear; future research should utilize more frequent sampling to ascertain changing pathways to pain over time. Second, although MIDUS is a cutting-edge project with a rich collection of biopsychosocial assessments, it also brackets the current project in regards to generalizability (Roberson et al., 2018). Our sample was mostly White, married, and had a moderately high average income. These characteristics may not adequately reflect the true population of U.S. adults with persistent pain or pain treatment (Grol-Prokopczyk, 2018). Therefore, additional research replicating the present tests with alternate samples is needed. We were also limited in how we assessed the quality of pain conditions at baseline, relying on participant report, including physician-made pain diagnoses. Similarly, though we are unable to ascertain whether the physicians rendering the present pain condition diagnoses were specifically pain specialists, the nature of the participants' diagnoses is reflective of current medical practice, as the majority of patients with chronic pain are assessed and treated in primary care, and pain is a highly prevalent presenting problem in primary care (Smith et al., 2019). Subsequent studies in this area should utilize medical samples (i.e., primary care or pain patients), with inclusion of medical record data. Finally, though we postulate family-pain associations, our analyses were conducted at the individual family member level. Studies of family-level processes contributing to pain experiences (and, vice versa) would be especially advantageous for informing family-based interventions for pain.

7. Conclusions

Our study highlights important differences between factors associated with the development of chronic and persistent pain and the factors that are associated with the risk of that pain continuing. Our results indicate that specific aspects of social support systems, namely the negative quality of family relationships, may be especially related to the risk of developing new chronic pain over the adult lifespan. Conversely, supportive family relationships are associated with a decreased risk of acute pain progressing, whereas depression is linked to an increased risk. Additionally, once pain develops, its persistence is associated with how interfering the pain was 10 years prior. Therefore, it may be that the most critical public health goal is to prevent chronic pain, rather than treat it, with a focus on ameliorating family stress and promoting family support.

It will be important to replicate the current findings, potentially with medical populations, before formalizing the implications of the present study. Yet, given the nature of the large, national sample, and the varied measures of relationship quality and social integration, the present results implicate the critical nature of including family relationships in the conceptualization of chronic pain pathogenesis. Future policy development should incorporate considerations of close family relationships in emphasizing non-opioid pain strategies.

Acknowledgements

The MIDUS study is funded by the National Institute on Aging (P01-AG020166).

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