

Pain and Depression in Late Life: Mastery as Mediator and Moderator

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Objectives. This study examines how mastery mediates and moderates the relationship between pain and depression among older adults, as well as the extent to which these processes differ by the timing of pain in late life, while utilizing statistical methods that comprehensively control for time-stable confounds.

Methods. Data are derived from multiple observations of adults aged 65 years and older in the Washington, DC, metropolitan area over a 4-year period. Fixed effects models are used to control for time-stable influences.

Results. With all time-stable influences controlled, pain is positively related to symptoms of depression, although this relationship is substantially reduced in comparison with a model in which all time-stable confounds are not held constant. Mastery does not mediate this relationship because pain is not significantly related to mastery once time-stable factors are taken into account. Mastery buffers the relationship between pain and depression, but only for elders later in late life.

Discussion. This study suggests that a synthesis of stress process and life course perspectives is critical for understanding how pain influences depression in late life. However, research that does not comprehensively control for time-stable factors may overestimate the consequences of pain for older adults.

Key Words: Depression—Mastery—Mental health—Pain.

THIS paper examines the relationship between pain and symptoms of depression among older adults. Pain is of particular concern in the study of depression in late life because pain is prevalent among older adults (Bergh et al., 2003; Bonnewyn et al., 2009; Ciper & Clifford, 2004) and pain is a substantial source of personal distress (Gureje, 2007). Although there is some evidence that the pain–depression relationship is bidirectional, pain is associated with increases in depression over time and the development of depression among those who were not previously depressed (Chou, 2007; Chou & Chi, 2005; Gayman, Brown, & Cui, 2011; Geerlings, Twisk, Beckman, Deeg, & van Tilburg, 2002; Mäntyselkä, Lupsakko, Kautiainen, & Vanhala, 2010; Mavandadi, Sorkin, Rook, & Newsom, 2007). This research therefore underscores an “emerging consensus that persistent pain is more likely to lead to depression than vice versa” (Campbell, Clauw, & Keefe, 2003, p. 399).

Despite this emerging consensus, a recent review of the literature suggests that research often does not take into account the full breadth of factors that may simultaneously expose older adults to pain and depressive symptoms (Gagliese, 2009). In particular, there are a wide variety of time-stable factors that are associated with the etiology of pain (e.g., Gamsa, 1990), and these factors likely shape depression as well. Any attribute that does not change over time may be considered time stable, but three classes of factors are likely to be especially important in studying pain and depression among older adults; these are early life course experiences, personality characteristics, and genetic or physiological

factors. Life course factors may contribute to a spurious relationship between pain and depression in late life because early life course adversities such as a childhood maltreatment and family financial hardship are associated with an increased risk of chronic pain in adulthood (Brown, Berenson, & Cohen, 2005; Davis, Luecken, & Zautra, 2005; Jones, Power, & Macfarlane, 2009), and research examining similar adversities has shown that they may deleteriously influence adult mental health as well (Horwitz, Widom, McLaughlin, & White, 2001; Mensah & Hobcraft, 2008; Springer, Sherdian, Kuo, & Carnes, 2007). In addition, personality traits have been shown to have a substantial degree of stability in mid to late life (Caspi, Roberts, & Shiner, 2005), and personality characteristics have been shown to be related to both pain and depression (Applegate et al., 2005; Kotov, Gamez, Schmidt, & Watson, 2010; Ramirez-Maestre, Martinez, & Zarazaga, 2004). Furthermore, research suggests that experiences of pain and depression share common genetic factors and biological pathways (Bair, Robinson, Katon, & Kroenke, 2003; Gambassi, 2009), indicating that physiological or genetic factors may predispose individuals to pain and depression. What is therefore needed to better demonstrate a causal connection between pain and depression among older adults is research that more comprehensively takes the breadth of life course experiences and time-stable individual characteristics into account.

The reasons for the relationship between pain and symptoms of depression are also not clear (Gayman et al., 2010). However, the predominant perspective in the sociology of mental health, a stress process perspective, suggests that

stress often influences mental health indirectly by depleting psychological resources (Pearlin, 1999), and research demonstrates that a sense of control is a key resource in this process (Avison & Cairney, 2003; Mirowsky & Ross, 2003). A sense of control is “a set of beliefs held by individuals regarding the amount of control they have over what happens in their lives” (Skaff, 2006, p. 188). Sense of control has been examined using a number of concepts, such as locus of control, mastery, and helplessness (Pearlin & Pioli, 2003). However, many constructs tend to overlap, “and they are not seen as very distinct” (Ross & Mirowsky, 2003, p. 424). Because mastery is a global construct of perceived control that is specifically embedded within the stress process perspective (Skaff, 2007), this research will concentrate on mastery, but there is likely to be a high degree of conceptual and empirical overlap between mastery and alternative conceptualizations.

Mastery is “the extent to which one regards one’s life chances as being under one’s control in contrast to being fatalistically ruled” (Pearlin & Schooler, 1978, p. 5). Loss of mastery can have substantial consequences for symptoms of depression because the impression that success is merely random and people have little influence on their own lives can be quite distressing (Mirowsky & Ross, 2003). Pain is likely to reduce mastery because “pain can be conceptualized as an aversive experience that is often inescapable or unable to be palliated” (Banks & Kerns, 1996, p. 100). Being exposed to a noxious circumstance that one is powerless to avoid or mitigate is likely to reduce perceptions of control. Pain can also influence the ability to participate in social, vocational, and family activities (Cannella, Lobel, Glass, Lokshina, & Graham, 2007; Rudy, Kerns, & Turk, 1988), which can in turn influence perceptions of control over life; as one individual dealing with recurrent pain describes, “I can’t manage my everyday activities, my daily life” (quoted in Heath, Saliba, Mahmassani, Major, & Khoury, 2008, p. 305).

The role of mastery as a mediator in the pain–depression relationship is supported by research showing that perceptions of control help explain this relationship (e.g., Mingo, McIlvane, & Baker, 2008; Rudy et al., 1988; Turk, Okifuji, & Scharff, 1995), but time-stable factors that influence pain and depression may also influence mastery. For example, early life course adversities, such as child maltreatment and economic hardship, are associated with lower perceived control in adulthood (Pitzer & Fingerman, 2010; Pudrovska, Schieman, Pearlin, & Nguyen, 2005). Furthermore, a number of personality traits are related to measures of perceptions of control (Hofer, Busch, & Kiessling, 2008; Lachman, Rosnick, & Röcke, 2009), although evidence indicates that the genetic influence on these perceptions is relatively modest (Bullers & Prescott, 2001; Johnson & Krueger, 2005). Research examining how mastery mediates the relationship between pain and depression while more thoroughly taking life course experiences and time-stable personality factors

into account would therefore provide enhanced support for the importance of perceptions of control in explaining this relationship.

A stress process perspective also suggests that psychological resources weaken the relationship between stress and mental health, a process known as “buffering” (Pearlin, 1999). A number of studies have shown that mastery buffers the mental health effects of stress (Thoits, 1995), although little research has examined whether mastery buffers the effects of pain. Mastery can be a powerful buffer because people with a strong sense of control over their lives “see the undesirable events and outcomes in their own lives as unusual for them, and probably as something they can correct now and avoid in the future” (Mirowsky & Ross, 2003, p. 195). Pain is therefore less likely to result in depression among individuals with a strong sense of mastery because such individuals are less likely to experience pain as an overwhelming experience with holistic life effects. Mastery may also be important as a buffer because a low sense of control is likely to decrease the motivation to solve problems, whereas a strong sense of control is likely to spur behavior to adapt to problematic health conditions (McIlvane, Schiaffino, & Paget, 2007; Mirowsky & Ross, 2003; Thoits, 2006). This argument is supported by research showing that higher levels of mastery are related to greater problem and accommodation-focused coping and less avoidance and disengagement (Ben-Zur, 2002). Thus, individuals with greater mastery are more likely to actively attempt to lessen the adverse effects of pain on their daily lives; conversely, those with a low level of mastery are more likely to allow the pain to fester and have a more pervasive detrimental influence on their lives.

Although a stress process perspective is useful for understanding how stress influences mental health, it has also been argued that additional insights may be gained by integrating a stress process perspective with a second perspective that is particularly relevant to the study of aging, a life course perspective (Pearlin & Skaff, 1996). A life course perspective views development as a life-long process in which the influence of important experiences on people’s lives can vary depending on the timing of these experiences in the life course (Elder, Johnson, & Crosnoe, 2003; Giele & Elder, 1998). This suggests that the timing of pain in late life may be particularly important for conditioning its effects on depression. These timing-based contingencies may occur because, as people age, they are more likely to accept pain as a part of the aging process (Appelt, Burant, Siminoff, Kwoh, & Ibrahim, 2007; Gibson, 2005), and pain may therefore be more distressing when it occurs for individuals earlier in late life. Similarly, negative stereotypes of aging may lead individuals later in late life to be less distressed by pain because of “a sufficiently dreary picture of old age” against which they are comparing their experiences of pain (Bultena & Powers, 1978, p. 753; Idler, 1993). Although research has produced conflicting results regarding timing-related differences

in the consequences of pain (e.g., Currie & Wang, 2004; Geerlings et al., 2002; Miller & Cano, 2009; Turk et al., 1995), such conflicting results underscore a need to better control for the time-stable influences that contribute to these relationships.

Social and personal expectations may also create timing-based contingencies in the extent to which mastery mediates and buffers the effects of pain on depression. Younger adults are likely to face greater expectations that they fulfill work and family-related obligations, which will increase the degree to which pain interferes with daily activities, as suggested by research showing that pain is more weakly related to interference as age increases (LaChapelle & Hadjistavropoulos, 2005; Molton, Jensen, Ehde, & Smith, 2007). The greater interference that pain may cause earlier in the life course is in turn likely to lead to a stronger detrimental influence of pain on mastery for younger elders (LaChapelle & Hadjistavropoulos, 2005). Furthermore, although mastery buffers stress in part by facilitating effective coping strategies (Ben-Zur, 2002), the greater demands that individuals tend to face earlier in the life course may blunt these coping efforts, which will reduce the potency of mastery to buffer the effects of pain. Conversely, individuals later in the life course may have greater experience with pain and in turn be more adept at developing effective coping strategies (Molton et al., 2008), thereby accentuating the benefits of mastery for spurring effective coping.

In sum, this research has several related aims. The first is to offer more robust evidence of the association between pain and symptoms of depression in late life by more fully controlling for life course and time-stable factors that may confound this relationship. The second is to examine the extent to which mastery mediates and buffers the association between pain and depressive symptoms in late life. The third is to examine the extent to which these processes are in turn contingent on timing.

METHOD

Sample

Data for this study come from the Aging, Stress, and Health study, which is a longitudinal study of people aged 65 years and older residing in the District of Columbia and two adjoining Maryland counties, Prince George's and Montgomery. Consistent with the purpose of the project to investigate status inequality and health disparities, a socially and economically diverse sample was sought. The three locales subsume this diversity. Sample selection and recruitment began with the Medicare Beneficiary files for the three areas. In addition to the names of all people aged 65 years and older who are entitled to Medicare, the files provided information about the race and gender of each beneficiary. To maximize the social and economic diversity within the sample, a total of 4,800 names were randomly

selected, equally divided among the three locales, Blacks and Whites, women and men, creating 12 groups, each containing 400 names. However, the goal was to enlist a sample of 1,200 people living independently (with approximately 100 people in each of the 12 groups). Of the 4,800, a subset met the study criteria, and approximately 65% of all eligible respondents who were contacted agreed to participate, yielding 1,167 cases. The age distribution within the four gender-race groups was similar to the population from the 2000 Census (Schieman & Plickert, 2007). Interviews for Wave 1 occurred during 2001–2002. Wave 2 was a year later and comprised 1,000 respondents (an 85.7% retention rate), Wave 3 was 2 years after Wave 1 and comprised 925 of the original respondents (a 79.3% retention rate), Wave 4 was 4 years after the first wave and comprised 789 respondents (a 67.6% retention rate), and Wave 5 was 5 years after the first wave and comprised 716 respondents (a 61.4% retention rate). Although this is a fairly strong retention rate given the age of the sample, analytic techniques described later are used to address sample attrition. Unfortunately, the pain scale used in this study was not on the first wave of the survey, so this study utilizes Waves 2 through 5. Attrition was relatively light between the first and second wave, suggesting little bias from not using the first wave of the study. In addition, preliminary analyses indicated that neither mastery nor depression significantly predicted attrition between Waves 1 and 2, further suggesting that study results would not be biased by beginning with the second wave of the study.

Measures

Pain.—Respondents were asked how often in the previous month they had “back pain,” painful knees or other joints,” and “muscle aches or soreness.” Response choices ranged from *never* (1) to *more than 5 times* (5). Although brief, this measure was useful in that it indicated experiences of pain across multiple sites in the body and in some of the most frequent sites of pain among older adults. Principal components analyses described below also supported using these items as one overall measure of pain, and responses were averaged to create a pain scale (Cronbach's alpha was between .63 and .65 across the four waves).

Depression.—Similar to previous research using these data, symptoms of depression were measured using four items taken from the Hopkins Symptoms Checklist (Derogatis, Lipman, Rickels, Uhlenhuth, & Covi, 1974). Consistent with common practice in measuring depression, depression was measured using a combination of symptoms of mood and malaise: “feel downhearted or blue,” “lack enthusiasm for doing anything,” “feel bored or have little interest in things,” and “cry easily or feel like crying.” Respondents indicated how often they had experienced

Table 1. Descriptive Statistics of Study Variables and Core Sociodemographic Characteristics

	Time 2		Time 3		Time 4		Time 5	
	<i>M</i>	<i>SD</i>	<i>M</i>	<i>SD</i>	<i>M</i>	<i>SD</i>	<i>M</i>	<i>SD</i>
Depression	1.357	0.539	1.389	0.533	1.363	0.544	1.348	0.538
Pain	2.341	1.225	2.426	1.249	2.388	1.257	2.357	1.221
Economic hardship	1.133	0.301	1.135	0.305	1.158	0.323	1.140	0.328
Functional limitations	0.207	0.406	0.234	0.423	0.253	0.435	0.213	0.409
Fair or poor self-rated health	1.146	0.374	1.173	0.409	1.193	0.443	1.181	0.401
Married	0.524	0.500	0.518	0.500	0.496	0.500	0.480	0.500
Mastery	2.857	0.575	2.862	0.560	2.984	0.547	2.802	0.576
Age	75.196	6.346	76.013	6.216	77.441	5.877	78.134	5.645
Black	0.491	0.500	0.485	0.500	0.479	0.500	0.469	0.499
Women	0.492	0.500	0.492	0.500	0.498	0.500	0.508	0.500
College graduate or more	0.446	0.497	0.446	0.497	0.457	0.498	0.471	0.500

Notes: *N* at Time 2 is 1,000. Although the sample size is reduced in subsequent waves due to sample attrition, full information maximum likelihood techniques account for missing data in the multivariate analyses.

each symptom in the previous week from *no days* (1) to *5 or more days* (4). This also raised a question of whether the pain items were additional indicators of malaise. However, principal components analyses of the pain and depression items at each wave indicated two components with eigenvalues greater than 1; with varimax rotation, the pain and depression items loaded at .6 or above on separate components, with all cross-component loadings less than .2. These analyses therefore supported analyzing pain and depression as distinct measures; these analyses also supported combining questions examining malaise and mood into one measure of depression. Responses were averaged to create a scale of depression (Cronbach's alpha .71–.75).

Mastery.—Mastery was measured using four items from Pearlman and Schooler's (1978) mastery scale: "You have little control over the things that happen to you," "There is really no way you can solve some of the problems you have," "You often feel helpless in dealing with problems of life," and "Sometimes you feel that you are being pushed around in life." Response choices ranged from *strongly agree* (1) to *strongly disagree* (4), and responses were averaged to create a scale of mastery (Cronbach's alpha was between .72–.75).

Age.—Age was measured as the respondent's age in years and was derived from a respondent's reported date of birth.

Covariates.—Since all time-stable characteristics were controlled using statistical methods described below, only time-variant measures needed to be included as controls. Because health could covary with the experience of pain and also influences depression, overall health status was measured with a commonly used indicator of self-rated health (e.g., Idler & Benyamini, 1997) in which a score of 1 indicated poor or fair health and 0 indicated good, very good, or excellent health. In addition, functional limitations have been shown to be antecedent to both pain and depression

(e.g., Gayman, Turner, & Cui, 2008), so these limitations were controlled using a five-item measure adapted from previous research (Bierman & Statland, 2010). This scale asked about bathing, dressing, toileting, transferring, and ambulation (walking balance), with responses measured along gradients of independence from *without difficulty* (1) to *unable to do this without complete help from someone or special equipment* (4; Cronbach's alpha .74–.79). Socioeconomic status (SES) is a fundamental influence on physical and mental well-being (Link & Phelan, 2000; Pearlin, 1999) and may therefore create a spurious relationship between pain and depression. Because commonly measured aspects of SES such as education and income are relatively stable in late life, time-variant aspects of SES were controlled using a scale of economic hardship in which respondents indicated difficulty in paying for five aspects of daily life, including housing, food, transportation, medical expenses, and clothing. Responses ranged from *not at all difficult* (1) to *very difficult* (3). Responses were averaged to create a scale of economic hardship (Cronbach's alpha was between .82–.87). Similarly, marital status is a primary predictor of mental health (Umberson & Williams, 1999) and has been shown to be related to the experience of pain (e.g., Currie & Wang, 2004). Because transitions out of marriage may increase in late life, marital status at each wave was controlled using a dichotomous indicator in which 1 indicated married and 0 indicated non-married.

Descriptive statistics for study variables and core demographic characteristics are presented in Table 1.

Statistical Analyses

This research utilizes fixed effects models for analyzing longitudinal data. The advantage of this modeling strategy is that all time-stable individual sources of variation are held constant (Wooldridge, 2002), thereby ruling out confounding influences of life course experiences and individual differences. However, an important drawback to fixed effects modeling is that because all time-stable influences

Table 2. Association Between Pain and Symptoms of Depression

	Model 1		Model 2		Model 3	
	<i>b</i>	<i>SE</i>	<i>b</i>	<i>SE</i>	<i>b</i>	<i>SE</i>
Pain	0.039***	0.010	0.035***	0.010	0.035***	0.010
Economic hardship			0.155***	0.039	0.151***	0.039
Functional limitations			0.144***	0.033	0.140***	0.033
Fair or poor self-rated health			0.113***	0.026	0.110***	0.026
Married			-0.259***	0.054	-0.263***	0.053
Mastery					-0.091***	0.020
χ^2	19.245		120.384***		135.622***	
<i>df</i>	18		62		73	
CFI	0.999		0.959		0.959	
RMSEA	0.008		0.031		0.029	
SRMR	0.017		0.016		0.014	

Notes: CFI = comparative fit index; RMSEA = root mean square error of approximation; SRMR = standardized root mean square residual. Metric coefficients are presented; *SE* indicates the standard errors of the coefficients.

* $p < .05$; ** $p < .01$; *** $p < .001$ (two-tailed tests).

are held constant, separate time-stable predictors cannot be included in the models; as a result, the specific time-stable influences that most contribute to spuriousness cannot be identified. Ultimately, though, fixed effects modeling is a conservative strategy because it does not require the assumption that measures of all important time-stable confounds are included in the models.

There are multiple ways of estimating fixed effects models that produce similar results (Allison, 2009), and within this research, a structural equation modeling (SEM) approach is used. In an SEM approach, a latent variable that represents the influence of all time-stable factors on the outcomes are estimated. Specifically, the repeated outcome is used as the manifest indicators, and each loading is set to 1, so that the latent variable represents the consistent influence of unchanging conditions on the outcome. By allowing the latent variable to covary with pain or the other time-varying predictors of the outcome, the estimates for the relationship between pain and the outcome is independent of all time-stable factors. Essentially, in allowing the latent variable to covary with the predictors, all time-stable factors are controlled, although no time-stable factors are ever directly measured.

An SEM approach to fixed effects modeling is particularly advantageous because this approach provides multiple indicators of model fit and allows models to be estimated using full information maximum likelihood (FIML) estimation. FIML is capable of providing unbiased parameter estimates in the presence of missing data (Enders, 2006) and, in the presence of sample attrition, provides less biased estimates compared with more conventional missing data methods (such as list-wise deletion or mean imputation; Wothke, 2000). FIML does assume that data are “missing at random” (MAR), but even when data are not MAR, methods assuming MAR often present results better than those produced using more conventional estimation procedures (Allison, 2003). All models were estimated using Mplus 5.21, and preliminary analyses of serial correlation suggested adjusting for correlated errors between Waves

4 and 5 in models predicting depression as well as between Waves 2 and 3 in models predicting mastery. Furthermore, because sampling was not based on the dependent variables and the models adjust for the potential disproportionality of race and gender created through the sampling procedures, these analyses do not utilize sampling weights (Winship & Radbill, 1994).

RESULTS

Model 1 of Table 2 presents a fixed effects model for the bivariate relationship between pain and depression. Although the significant model chi-square indicates poor model fit, the chi-square statistic may be inflated in the case of large sample sizes (Little, Preacher, Selig, & Card, 2007), and alternative approximate fit indices for this and subsequent models indicate acceptable fit (Kline, 2005). Substantively, the relationship between pain and symptoms of depression is positive and significant, indicating that even when all time-stable factors are taken into account, greater pain is associated with greater levels of depression. Furthermore, Model 2 shows that when relevant time-varying covariates are included in the model, the coefficient for pain is reduced marginally, by about 10%, demonstrating that these time-varying influences play a minor role in contributing to the relationship between pain and depression. In additional analyses not shown here, a model interacting age and pain was not significant, suggesting that the timing of pain in late life does not influence the relationship between pain and depression. However, analyses described below indicate that there may be a more complex influence of timing on this relationship.

To further demonstrate the utility of a fixed effects model in understanding the pain–depression relationship, an ancillary test compared Model 2 with a model in which multiple social statuses (e.g., race, gender, education) were included as predictors along with the time-varying controls in Model 2, but unobserved time-stable factors were not held constant (i.e., a “random effects” model). This

Table 3. Interaction Models for Association Between Pain and Symptoms of Depression

	Model 1		Model 2		Model 3: age $\leq 75^a$		Model 4: age $> 75^b$	
	<i>b</i>	<i>SE</i>	<i>b</i>	<i>SE</i>	<i>b</i>	<i>SE</i>	<i>b</i>	<i>SE</i>
Pain	0.087*	0.039	0.100*	0.039	0.008	0.063	0.236***	0.050
Mastery	-0.047	0.037	-0.040	0.037	-0.094	0.058	0.043	0.048
Pain \times Mastery	-0.018	0.013	-0.023	0.013	0.008	0.022	-0.072**	0.016
Pain \times Age			0.013*	0.006				
Mastery \times Age			0.006	0.006				
Pain \times Mastery \times Age			-0.005*	0.002				
χ^2	311.271***		214.547***		159.883***		132.748***	
<i>df</i>	180		117		84		84	
CFI	0.919		0.939		0.916		0.933	
RMSEA	0.038		0.029		0.040		0.037	
SRMR	0.018		0.010		0.017		0.019	

Notes: CFI = comparative fit index; RMSEA = root mean square error of approximation; SRMR = standardized root mean square residual. Metric coefficients are presented; *SE* indicates the standard errors of the coefficients. All models include controls for self-rated health, functional limitations, economic hardship, and marital status.

^a*N* = 571.

^b*N* = 429.

p* < .05; *p* < .01; ****p* < .001 (two-tailed tests).

alternative model fit significantly worse than Model 2 ($p < .001$), and the coefficient for the relationship between pain and depression was approximately 40% larger in the random effects model, suggesting that the relationship between pain and depression may be substantially inflated if time-stable factors are not comprehensively taken into account.

Model 3 controls for mastery to test whether mastery mediates the relationship between pain and depression. Higher levels of mastery are significantly related to lower levels of depression, but the coefficient for pain is not reduced between Models 2 and 3, indicating that mastery does not explain the effects of pain on depression (Baron & Kenny, 1986). Ancillary analyses examining the relationship between pain and mastery demonstrated why mastery failed to evidence a mediating effect. Although the cross-sectional correlation between pain and mastery was significant at each wave, in the fixed effects this relationship was not significant. These additional analyses therefore suggest that previous research has likely overstated a relationship between pain and perceptions of control because of the difficulty in comprehensively accounting for time-stable covariates. The ancillary analyses also indicated that the relationship between pain and mastery did not differ by age, suggesting that the influence of pain on mastery did not differ by the timing of pain in late life.

Even though mastery does not mediate the relationship between pain and depression, it is still possible that mastery buffers this relationship. This question is addressed in Table 3. Model 1 includes an interaction between pain and mastery. This interaction is not significant, suggesting that mastery does not moderate the effects of pain on depression. However, Model 2 includes a three-term interaction between pain, mastery, and age, and this interaction is significant, suggesting that the extent to which mastery moderates the effects of pain varies by the timing of pain in late life.

Models 3 and 4 explicate the meaning of this three-term interaction by dividing the sample into two age groups—those older than age 75 years at Wave 2 and those aged 75 years and younger—and testing the interaction between pain and mastery. The interaction between mastery and pain is significant for the older group but not for the younger group. The direction of the coefficient for the significant interaction is negative, indicating that the relationship between pain and symptoms of depression weakens as mastery increases. Mastery therefore buffered the effects of pain on depression, but only for the older group. Additional analyses indicated that when the nonsignificant interaction was eliminated for the younger group, pain remained significantly related to depression ($b = .033$, $p < .01$). Further analyses indicated that the buffering effect progressively strengthened as the minimum age for determining the older sample was increased, and the interaction progressively weakened as the minimum age for determining the older sample was decreased. Thus, although the split-sample interactions help provide an understanding of the three-term interaction, these additional analyses demonstrate that the split-sample interactions are best thought of as illustrating timing-related contingencies across the continuum of late life rather than showing a discrete difference in moderation between age groups. Overall, though, these analyses suggest that pain is related to symptoms of depression in late life even when time-stable factors are comprehensively held constant. As pain occurs later in late life, mastery helps reduce the influence of pain on depressive symptoms. For individuals earlier in late life, pain is related to depression regardless of mastery.

DISCUSSION

A stress process perspective suggests that psychological resources such as mastery will buffer the effects of pain on depression, whereas a life course perspective suggests that

the influence of pain on depression will vary depending on when pain occurs in late life. The results of this research illustrate how a synthesis of these perspectives is essential for understanding the relationship between pain and depression among older adults. An interaction between mastery and pain indicated little buffering by mastery, and the influence of pain on depression did not differ in and of itself by the timing of pain in late life. However, when timing-based contingencies in buffering were taken into account, mastery weakened the influence of pain on depression for older elders, but pain was related to depression for younger elders regardless of mastery. The timing of pain in late life does create contingencies in the effects of pain, but in terms of the extent to which psychological resources help prevent these effects.

Timing is likely important for creating contingencies in the extent to which mastery buffers the effects of pain because individuals are more likely to face greater expectations to meet work and family demands earlier in the life course (LaChapelle & Hadjistavropoulos, 2005). Although mastery buffers stress in part by impelling problem-focused coping (Ben-Zur, 2002), ameliorative efforts may be less effective when met with a social context that is highly demanding or resistant to lessening obligations, leading mastery to more weakly buffer effects of pain for younger elders. Moreover, a life course perspective's emphasis on life-long development underscores how individuals can become more adept at effective coping with pain over time, and this increasing proficiency may enhance the extent to which mastery spurs effective coping for individuals later in the life course (Molton et al., 2008). Additional research should therefore examine how changes in social expectations with age, as well as the extent of experience with pain, pattern opportunities for and effectiveness of attempts to cope with pain.

This study also suggests that research that does not account for a wide variety of time-stable characteristics may overestimate the consequences of pain, as the relationship between pain and both depression and mastery was reduced with the use of fixed effects models. A broad range of time-stable factors may inflate estimates of the consequences of pain in late life—such as life course experiences, personality characteristics, and biological or genetic factors—and it is unlikely that researchers will be able to comprehensively account for all these sources of spuriousness using measured variables. Researchers studying the effects of pain, as well as additional stressors, should therefore consider techniques such as fixed effects models that comprehensively account for time-stable confounds.

It should also be noted that the measure of pain in this study was relatively brief and limited to the frequency of pain. Although this was useful as a general measure of pain, future studies should examine whether these results can be replicated with more standardized measures of pain or measures that take into account a wider variety of sites of the

body. More specific measures of perceived control may also show different patterns, as it is still possible that domain-specific aspects of control, such as perceived mastery of health, help to explain the relationship between pain and depression. Research that examines the way in which pain is experienced may also find a more refined set of relationships. For example, untreatable pain may have substantial consequences for both mastery and depression, whereas pain that is more easily managed less so. Similarly, pain that is particularly acute may have much greater consequences than low-intensity experiences of pain. These additional contingencies particularly suggest that future research should consider how the utilization and effectiveness of pain medication intersects with timing and mastery to form an additional context for the consequences of pain. At the same time, though, research that examines these additional issues should continue to take the breadth of potential time-stable confounds into account.

Limitations to the data should also be considered. First, the inability to use the first wave could introduce bias. However, attrition between Waves 1 and 2 was relatively light, and ancillary analyses suggest little substantial evidence of bias. Second, the sample on which this study is based is not nationally representative. The diversity with which it was purposely gathered suggests that these results are likely to generalize to a broader population, though, and previous research on pain and depression has in fact been criticized for not including minority populations (Campbell et al., 2003). Third, these data did not include measures of substance abuse, and future research should give greater attention to pain and substance abuse in older adults due to the potential for self-medication, especially because research suggests that contingent relationships between stress and mental health can vary depending on the outcome being considered (Aneshensel, Rutter, & Lachenbruch, 1991).

One final caution should be made in regards to the interpretation of the three-term interaction involving age. Baseline age is used as a focal moderator in this research because it indicates differences between individuals in placement in the life course, whereas time-varying age within fixed effects models indicates the intra-individual experience of time. However, research using growth curve modeling has recently examined the influence of time-stable and time-varying characteristics of age in terms of cohort and increasing age, respectively (e.g., Yang & Lee, 2009). An important area for future research on the effects of pain is therefore to use methods that make more fine-grained distinctions between age and cohort and examine the extent to which aging and cohort contribute to the moderating effects of baseline age observed here.

Overall, this research demonstrates that pain is related to depression in late life, even when time-stable factors are comprehensively controlled. However, research that does not comprehensively take time-stable factors into account may overestimate the consequences of pain. In addition,

psychological resources and the timing of pain intersect to shape the relationship between pain and depression in late life. This study therefore bolsters the argument that a synthesis of life course and stress process perspectives is advantageous for understanding influences on mental health (Pearlin & Skaff, 1996).

FUNDING

This work was supported by an NIA grant award AG17461 (Leonard I. Pearlin, P.I.).

ACKNOWLEDGMENT

A special thanks to the editor for his suggestions on the statistical methodology in this research and to Danielle Robinson for her comments on an earlier draft of this manuscript.

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