Incident chronic spinal pain and depressive disorders: Data from the National Comorbidity Survey

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Highlights

- Chronic pain and depression frequently co-occur
- Chronic pain or depression increases the risk for developing the other condition
- Chronic depression is particularly linked to developing chronic spinal pain

Running head: Chronic pain and depression

Incident chronic spinal pain and depressive disorders: Data from the National Comorbidity Survey

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Abstract

This study examined pre-existing depression as a risk factor for the development of chronic spinal pain, and pre-existing chronic spinal pain as a risk factor for the development of depression. Data from the National Comorbidity Survey, a stratified sample of 5001 participants evaluated in 1990-1992 (NCS-1) and again in 2000-2001 (NCS-2) were used to address these associations. Cox regression was used to estimate hazard ratios and time-to-incidence after NCS-1. Participants with antecedent acute or chronic depressive disorders at NCS-1 were more likely to develop chronic spinal pain in the ensuing 10 years compared to participants without depressive disorders. Those with antecedent chronic spinal pain at NCS-1 were more likely to develop dysthymic disorder than subjects without chronic spinal pain at NCS-1, however, antecedent chronic spinal pain was not associated the subsequent development of major depressive disorder. These results suggest that both pain and depression are associated with the development of the other condition. In particular, chronic depression is more strongly linked to chronic spinal pain than is acute depression. The results are discussed in terms of the need to assess the presence of both disorders given the presence of one.

Perspective: Chronic spinal pain and depressive disorders, especially chronic depression, increase the likelihood for the subsequent development of the other condition. The results underscore the need to routinely assess for the presence of both disorders given the presence of one in order to mitigate the effects of developing comorbid conditions.

Keywords: chronic spinal pain; dysthymic disorder; incidence; major depressive disorder; persistent depressive disorder

Introduction

Chronic spinal pain and depression are common conditions associated with a good deal of morbidity and healthcare utilization. In the US, low back pain, neck pain, and major depressive disorder comprise three of the top four leading causes of years lived with disability.[51] The point-prevalence or past-year prevalence of chronic neck and back pain has been estimated at 8.1%-19% in large population-based studies.[15,19,25,49,52] The lifetime prevalence of major depressive disorder (MDD) was 17.1% and 16.2% and dysthymic disorder (DD) was 6.4% and 6.9% in the original National Comorbidity Survey (NCS) and its replication (NCS-R), respectively.[29,31] MDD requires two or more weeks of five or more symptoms; DD requires two or more years of three or more symptoms.[2] In the United States, the direct and indirect costs attributed to low back pain were estimated to be $84-$623B for 2008, with the majority of costs attributed to lost work productivity.[20] The economic burden attributed to depression was estimated to be $211B in 2010, which was largely due to costs related to comorbid conditions, especially pain-related conditions, and only 38% of the total costs was attributed to depression alone.[21]

A number of studies have assessed cross-sectional associations between chronic spinal pain and depression. Most studies have examined samples of subjects with chronic spinal pain and found that 4.6-45% had comorbid MDD and 2-23.3% had DD.[4,7,15,18,43,52] It is less common to characterize rates of chronic pain in those with depression:[5] in one study, 21% had chronic spinal pain.[41]

The temporal sequence of depression and chronic spinal pain has been of interest insofar as it may elucidate possible mechanisms underlying the association and facilitate treatment.[10,47] Studies that have examined the development of chronic spinal pain in those with depression have

found 1.8- and 4.0-fold increased likelihoods of new chronic pain 1-2 years later in those with antecedent depression symptoms,[12,14] but another study did not find that chronic depressive symptoms were associated with incident back pain over a 3 year period.[53] We found no longitudinal studies that solely examined the development of depression in those with and without antecedent chronic spinal pain.

Some studies have examined both paths of antecedent depression to subsequent incident chronic spinal pain and antecedent chronic pain to subsequent incident depression over periods ranging from 2 to 13 years. Antecedent depressive disorder,[16,36] depressive symptoms,[22,39] or mixed depression/anxiety symptoms[23] were associated with significantly increased odds of developing chronic spinal pain in most studies (range: 2.1- 2.9) ,[16,23,36,39] with one exception.[22] Fewer studies have found that antecedent chronic spinal pain was associated with significantly increased odds of subsequent depression (range: 1.1-2.9)[22,23,39] but two studies did not.[16,36]

The purpose of this study was to examine the development of chronic spinal pain and depressive disorders, and to determine the risk of incident chronic spinal pain given a history of depressive disorders, and of incident chronic depressive disorders given a history of chronic pain. NCS data were used: 5001 subjects were assessed in 1990-2[26] and 10 years later in 2000-02.[27] Based on the equivocal results in the literature, it was predicted that the presence of chronic spinal pain would increase the likelihood of developing subsequent depressive disorders, and that the presence of a depressive disorder would increase the likelihood of developing subsequent chronic pain, after controlling for sociodemographic variables.

Methods

Sample Recruitment and Procedure

The NCS-1 sample involved 8098 respondents aged 15-54 who comprised a “stratified,

multistage area probability sample of the noninstitutionalized (United States) civilian population…" [p. 9].[31] As described by Kessler and colleagues,[31] after obtaining informed consent (or parental consent for those ages 15-17), subjects received a structured interview that included questions about demographic characteristics, chronic health conditions, and mental health symptoms. The first wave of NCS-1 data were collected from 1990-1992; respondents were re-interviewed 10 years later for NCS-2 in 2001-2002. The NCS-2 traced 5463 of the NCS-1 respondents: 166 were deceased, and 5001 were subsequently interviewed.[30] Customized interview questions based upon responses in the index interview (NCS-1) were included in the NCS-2 survey.

The use of the NCS data was certified as exempt from human subjects review by the Office of Research Assurances, Washington State University.

Variables

Case weight

Cases were weighted to correct the sample for compositional biases: the distribution of participants across demographic groups in the NCS was compared with the distribution in the National Health Interview Survey (NHIS) to determine the case weights for participants.[37]

Sociodemographic variables

Respondents' age, gender, race/ethnicity, and years of education at NCS-1 were retained for use in analysis.

Chronic spinal pain

The NCS-1 interview did not query subjects about chronic back or neck pain, however, the NCS-2 interview contained a set of questions "have you ever had any of the following conditions since (NCS-1 year)" including "chronic back or neck problems." Subjects who

responded in the negative were categorized as not having chronic spinal pain. Subjects who responded positively were asked the age at which they first had chronic back or neck problems. This response was compared to their age at NCS-1 and resulted in categorizing subjects as having chronic spinal pain at NCS-1 if its onset was at the same age or earlier than the NCS-1 interview, or as having incident chronic spinal pain after NCS-1 if the age of onset was after their age at NCS-1 the interview. The number of years subsequent to NCS-1 to the development of incident chronic spinal pain was retained for analysis.

Although the interview did not assess the duration or severity of the chronic pain, its concurrent validity was supported by comparing those with and without chronic pain at NCS-2. Subjects were asked, "How often did you experience physical discomfort, such as pain, nausea, or dizziness in the past 30 days?" on a 1 (all days) to 5 (no days) scale. Those who reported chronic spinal pain at NCS-2 had significantly more frequent (M = 3.50) physical discomfort than did those who did not report chronic spinal pain (M = 4.36) ($t(2538.3) = -28.88$, $p < .001$). If any physical discomfort was reported, subjects were asked, "On average, how severe was your physical discomfort during the time you experienced it in the past 30 days?" on a 1 (mild) to 4 (very severe) scale. Those with chronic spinal pain had more severe discomfort (M = 1.90) than those who did not report chronic spinal pain (although they may have had other pain-related conditions such as headaches, M = 1.59, $t(3269) = 10.93$, $p < .001$). This sample's pain frequency and severity compares favorably to other studies,[4,10,25] but may be milder than another.[19]

Evaluation of depressive disorders

The NCS utilized a modified version of the Composite International Diagnostic Interview (CIDI),[32,46] a structured psychiatric diagnostic interview, which was administered by trained

interviewers.[31] Diagnoses resulting from the pattern of CIDI responses were evaluated according to the DSM-III-R[1] and DSM-IV,[2] for the NCS-1 and NCS-2 interviews, respectively. All diagnoses were considered in their most conservative form as meeting all of the criteria, including diagnostic hierarchy. For example, a diagnosis of schizophrenia would take precedence over the diagnosis of dysthymic disorder.

Major depressive disorder

Participants who met criteria for lifetime major depressive disorder (MDD) with hierarchy at NCS-2 were asked their age of onset. This response was compared to their age at NCS-1 and resulted in categorizing subjects as having current or historic MDD at NCS-1 if the MDD onset was at the same age or earlier than the NCS-1 interview, or as having incident MDD after NCS-1 if the age of MDD onset was after their age at NCS-1 the interview. The number of years subsequent to NCS-1 to the development of incident MDD was retained for analysis.

Dysthymic disorder

Participants who met criteria for lifetime dysthymic disorder (DD) at NCS-2 were asked at what age their DD started. This response was compared to their age at NCS-1 and resulted in categorizing subjects as having a current or historic DD at NCS-1 if the dysthymia onset was at the same age or earlier than NCS-1, or as having incident DD after NCS-1 if their age of DD onset was after their age at the NCS-1 interview. The number of years subsequent to NCS-1 to the development of incident DD was retained for analysis.

Data analysis

Variables from NCS-1 and NCS-2 were merged by the unique participant code. All analyses were performed by weighting the cases according to the case weighting variable described above.

Descriptive statistics were calculated to describe the sociodemographic characteristics of the total sample, and the prevalence of individual and comorbid pain and depressive disorders. Risk ratios were calculated for the development of incident MDD, DD, or chronic spinal pain after NCS-1, given antecedent chronic pain or depressive disorders, respectively, at NCS-1. These ratios were compared by Z tests for differences in independent samples. For example, the risk of developing incident cases of MDD after NCS-1 was compared for those with and without chronic spinal pain at NCS-1 to determine if the development of MDD was more likely among those with antecedent chronic spinal pain.

Four Cox regression analyses were used to predict: (1 and 2) among those without chronic pain at NCS-1, time-to-incident chronic pain after NCS-1 (i.e., comparing censored cases that did not develop chronic pain versus cases that did develop chronic pain) by first entering sociodemographic variables in a block (age, gender, race/ethnicity coded as White or non-White, and years of education coded as 0-15 years or 16 or more years), followed by antecedent MDD or DD at NCS-1 as a binary variables (absent or present at NCS-1); (3) among those without MDD at NCS-1, time-to-incident MDD after NCS-1, by first entering sociodemographic variables in a block, followed by antecedent chronic pain at NCS-1; and (4) among those without DD at NCS-1, time-to-incident DD after NCS-1, entering sociodemographic variables first, then antecedent chronic pain at NCS-1. Estimates and significance tests were bootstrapped using 1000 replications. The Cox regression estimation method interprets case weights as the number of replicated cases, which precludes the use of non-integer case weights. Therefore, the case weights were rounded to integer values, and cases with weights less than 0.50 were recoded to 1.00, so that these cases were not excluded because of being rounded to zero weights.

Results

Participant characteristics and descriptive statistics

A total of 5001 participants were re-evaluated on average 10 years after their initial evaluation. The weighting procedure resulted in equal proportions of respondents of each gender, who averaged 33.13 years of age (*SD* = 10.71), and had 12.80 years of formal education (*SD* = 2.46). The majority of respondents were white (75.5%), followed by smaller proportions of participants of Black, Hispanic, or other race/ethnicity (11.6%, 9.4%, and 3.5%, respectively).

At the time of the initial evaluation (NCS-1), 13.4% of the sample (*n* = 664) had chronic spinal pain; 15.0% (*n* = 751) and 3.0% (*n* = 149) of the sample had MDD and DD, respectively. Of interest were participants who did not meet criteria for MDD or DD at NCS-1 but did so subsequent to NCS-1; and participants who did not have chronic spinal pain at NCS-1, but did subsequently. At NCS-2, 12.0%, 10.2%, and 2.6% had developed incident chronic spinal pain, MDD, and DD, respectively.

At NCS-1, the comorbidity rates were as follows: among those with chronic spinal pain, 22.5% also met criteria for MDD, and 3.3% met criteria for DD. Among those with MDD or DD at NCS-1, 20.2% and 14.8% also had chronic spinal pain, respectively.

Risk ratios for incident chronic spinal pain and depressive disorders

Table 1 shows the number and percentage of participants with and without chronic pain at NCS-1 by the number and percentage of participants who did and did not meet criteria for incident cases at NCS-2 of MDD or DD.

Participants with antecedent chronic spinal pain at NCS-1 were significantly more likely to subsequently develop incident DD at NCS-2 than were participants without antecedent chronic spinal pain. There was no difference in the likelihood of the development of incident MDD at NCS-2 among those with and without antecedent chronic spinal pain.

Participants with antecedent MDD or DD at NCS-1 were significantly more likely to subsequently develop incident chronic spinal pain than were participants without antecedent MDD or DD.

Predictors of the development of chronic spinal pain

Table 2 shows the results for the Cox regression analysis to predict incident cases of chronic spinal pain following NCS-1 among those without chronic pain at NCS-1 (n = 4187 with case weights). Two analyses were conducted entering sociodemographic variables followed by the presence or absence of each antecedent depressive disorder as predictors. For antecedent MDD, the sociodemographic variables were significantly associated with the development of incident chronic spinal pain after NCS-1 (χ^2 (4) = 27.09, p < .01), specifically female gender and having less than 16 years of education. The addition of antecedent MDD was significant (χ^2 (1) = 20.91, p < .001 for the change from the sociodemographic variables). Figure 1 shows the hazard functions for the development of chronic pain following NCS-1: both those with and without antecedent MDD had similarly low rates of incident chronic pain in the first year following NCS-1. However, the rate of developing chronic pain rose more quickly among those with antecedent MDD culminating with nearly a 7% greater rate in the cumulative incidence of chronic pain among those with antecedent MDD than among those without antecedent MDD by NCS-2.

This analysis was repeated using antecedent DD: the sociodemographic variables were significant predictors of incident chronic spinal pain after NCS-1 (see above), and the addition of antecedent DD was significantly associated with the development of incident chronic spinal pain (χ^2 (1) = 6.22, p < .05 for the change from the sociodemographic variables). (The pattern of the hazard functions with antecedent DD is quite similar to MDD, see Figure 1 in the supplemental

materials.)

Predictors of the development of depressive disorders

Table 3 shows the results for the Cox regression analyses to predict new cases of depressive disorders after NCS-1 among subjects without those disorders at NCS-1.

In the first analysis, only participants without antecedent MDD at NCS-1 and with complete sociodemographic data were included (n = 3592 with case weights). The sociodemographic variables were significant predictors of incident cases of MDD after NCS-1 (χ^2 (4) = 22.49, $p < .001$); females and younger subjects were more likely to develop incident cases of MDD. Antecedent chronic pain was not associated with the development of incident MDD (χ^2 (1) = 0.90, ns).

The second analysis included participants without DD at NCS-1 and with complete sociodemographic data were included (n = 4687 with case weights). The sociodemographic variables did not predict incident cases of DD (χ^2 (4) = 3.46, ns), but antecedent chronic spinal pain was significantly associated with incident DD (χ^2 (1) = 4.42, $p < .05$). (The pattern of the hazard functions is quite similar to Figure 1 but with lower cumulative hazard given the lower rate of DD, see Figure 2 in the supplemental materials.)

Discussion

This study analyzed data from a large stratified population-based sample to examine the development of chronic spinal pain and depression over a 10 year period. At the index evaluation, the proportions with chronic spinal pain, MDD, and DD (13.4%, 15.0%, and 3.0%) were similar to those reported in previous research.[4,15,25,29,31,49,52] The proportions of those who developed incident chronic spinal pain, MDD, and DD in the 10 years following the initial evaluation (12.0%, 10.2%, and 2.6%) may be similar to those reported in other studies, although

methodological differences limit comparability. For example, one study found that 24% and 23% of participants developed incident cases of chronic pain and depression over a 12 year follow-up, but chronic pain was not limited to spinal pain and depression was assessed by questionnaire[22] rather than a structured diagnostic interview as was done in the NCS, which may account for the higher rates of incidence compared to the present study. The comorbidities of chronic spinal pain and depressive disorders in the present student were within the ranges reported in previous studies.[4,5,8,15,18,43,52]

Over time, those with antecedent MDD or DD were about 1.5 times more likely to develop chronic spinal pain in the ensuing 10 years than subjects without antecedent MDD or DD. Inspection of the hazard function plots show small difference in the rates of incident chronic pain in the first year subsequent to NCS-1 among those with and without antecedent depressive disorders, but the rate of developing incident chronic pain rises more quickly among those with antecedent depressive disorders in nearly all subsequent years than among those without antecedent depressive disorders. These results are supportive of our hypothesis and similar to several previous studies that that showed antecedent depression to be associated with an increased risk for the subsequent development of chronic spinal pain.[12,14,16,23,36,39] Our results are inconsistent with the minority of previous studies that did not find antecedent depression to raise the risk of the subsequent development of chronic spinal pain.[22,53]

Our hypothesis regarding antecedent chronic spinal pain increasing the risk for the subsequent development of depressive disorders was partially supported: those with antecedent chronic spinal pain were more likely to develop DD than subjects without antecedent chronic pain. However, antecedent chronic spinal pain did not predict the subsequent development of MDD. As was discussed in the introduction, fewer studies have examined the development of

depression in the context of chronic pain, and the results have been equivocal, as ours were, with three previous studies finding antecedent chronic pain to be associated with the onset of depression,[22,23,39] and two not.[16,36]

The results suggest that individuals afflicted with depressive disorders or chronic spinal pain should be evaluated and monitored for the complementary condition to mitigate the development of additional symptoms and functional limitation. Among individuals with depressive disorders, those with comorbid chronic pain have been found to be more likely to receive any mental health care than those without – approximately 13% versus 7%, respectively – but overall, these are low rates of mental health care, which suggest that many individuals may be suffering needlessly.[8] The significant economic impact of chronic pain and depression also underscores the necessity for monitoring and preventative measures, specifically targeting at-risk populations to limit the impact these disorders have on the health and daily functioning of individuals.[20,21] A direction for future research would be to determine if interventions that prevent the onset of depression or relapse among those with recurrent depression, such as physical activity, maintenance antidepressant pharmacotherapy, or mindfulness-based cognitive therapy (MBCT),[34,40,48] might also mitigate the development of chronic pain. One study's suggestive results found that MBCT decreased pain catastrophizing among those with comorbid chronic pain and depression.[17]

This study makes unique contributions to the literature in at least two areas, in addition to its use of a large population-based sample. First, previous studies have examined depression as an antecedent to pain more often than pain as an antecedent to depression. Few studies have examined both paths;[16,22,23,36,39] the present study adds to this small literature that facilitates comparisons of both paths. Second, the majority of studies of chronic pain and depression have

been focused on major depressive disorder; dysthymic disorder has received little attention. Dysthymic disorder -- which was renamed persistent depressive disorder in the DSM-5[3] -- while less prevalent than major depressive disorder, has been associated with more disability than major depressive disorder.[7] The results of this study found stronger associations of DD with chronic spinal pain than of MDD with chronic spinal pain, so future research focused on DD is warranted. If possible, future studies should be directed towards examining these relationships in other population-based studies with multiple assessments over time to better ascertain disorder onset and offset.

This study had several potential limitations. One set of potential limitations involved the measures. For example, chronic spinal pain was not assessed in NCS-1 and was assessed by a single item in the NCS-2, with age of onset being used to determine the timing of its onset compared to NCS-1. Pain is a private experience that is appropriate to assess by self-report,[24] but pain duration or intensity were not assessed in the NCS. Although those who reported chronic pain at NCS-2 had significantly more frequent and severe physical discomfort in the 30 days prior to the interview than did those who did not report chronic pain, other conditions in addition to chronic spinal pain could have contributed to participant reports of the frequency and severity of physical discomfort. Subjects' retrospective reports were used to determine disorders' onsets, the validity of which could be affected by recall or reporting biases, although patients' retrospective reports of chronic pain have been found to be valid when compared to prospective reports.[35] In addition, missing data could affect the generalizability of the results in unknown ways. For example, the data from individuals who endorsed chronic back or neck pain but did not give an age of onset could not be used. Although the contributions of several sociodemographic variables were analyzed in the association of chronic pain and depression,

other potential moderators or mediators were not examined. For example, the development of depression after pain may be mediated by sleep problems secondary to the pain.[11] Others have emphasized the association of anxiety disorders to chronic arthritis pain.[41,42] An examination of these problems were beyond the scope of the present study, but would be valuable directions for future research.

Taken together, these results suggest that both pain and depression increase the risk for the development of the other condition, but do not clearly support one temporal sequence over the other – those with depression are more likely to develop pain than those without depression, and those with pain are more likely to develop chronic depression than those without pain. However, it appears that overall, chronic depression is more strongly linked to chronic spinal pain than is major depressive disorder. The lack of a clear temporal sequence raises the question of common processes involved in chronic pain and depression. As pain becomes chronic, pain perception shifts from somatosensory to affective brain regions, in concert with increasing behavioral withdrawal and avoidance.[6,9,44] These features, as well as catastrophizing cognitions, are also characteristic of depression.[38] Pharmacologic and cognitive-behavioral interventions that increase opportunities for positive reinforcement and that decease behavioral avoidance have been recommended separately for chronic pain and depression.[13,45] Further research is warranted that examines common processes and treatments among those with both conditions.[33]

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Figure 1

Hazard function for the development of chronic pain after NCS-1

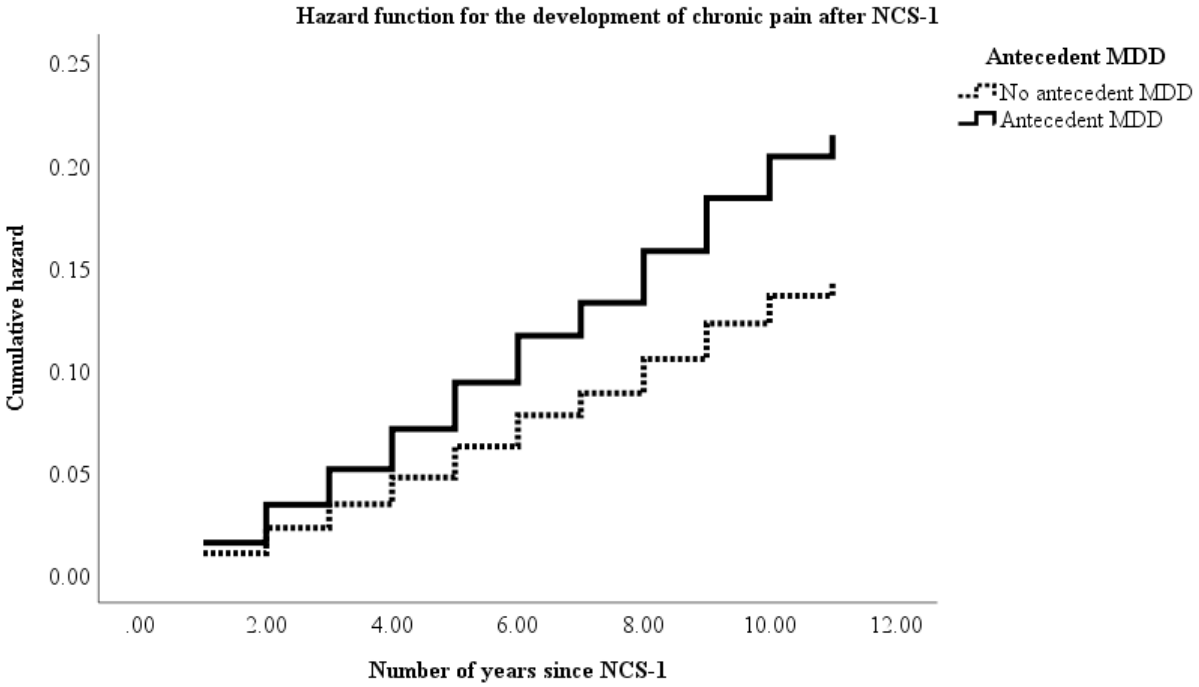

Table 1

Risk ratios for the development of incident depressive disorders or chronic spinal pain given antecedent chronic spinal pain or depressive disorders at NCS-1

	NCS-1 Chronic spinal pain			
	Absent (*n* = 4283)	Present (*n* = 664)	Risk Ratio	Z
NCS-2 incident MDD	10.27% (440)	10.69% (71)	1.04	0.33
NCS-2 incident DD	2.33% (100)	4.22% (28)	1.81	2.82*
	NCS-1 Major Depressive Disorder			
	Absent (*n* = 4209)	Present (*n* = 737)		
NCS-2 incident CPS	11.50% (484)	14.65% (108)	1.27	2.46*
	NCS-1 Dysthymic Disorder			
	Absent (*n* = 4800)	Present (*n* = 147)		
NCS-2 incident CPS	11.81% (567)	17.69% (26)	1.50	2.22*

Note: *$p < .05$; MDD = major depressive disorder; DD = dysthymic disorder; CPS = chronic spinal pain. *n*s are in parentheses.

Table 2

Cox regression analysis for the development of chronic spinal pain after NCS-1

	Incident Chronic Spinal Pain	
	Hazard Ratio (95% CI)	Hazard Ratio (95% CI)
Age	1.00 (0.99 – 1.00)	1.00 (0.99 – 1.00)
Sex	1.24** (1.08 – 1.43)	1.28** (1.11 – 1.47)
Education	0.73** (0.61 – 0.88)	0.74** (0.61 – 0.89)
Race	0.98 (0.83 – 1.15)	0.96 (0.81 – 1.12)
Antecedent MDD	1.50** (1.27 – 1.77)	
Antecedent DD		1.53** (1.12 – 2.11)

Note: * $p < .05$, ** $p < .01$

Table 3

Cox regression analysis for the development of depressive disorders after NCS-1

	Incident Major Depressive Disorder	Incident Dysthymic Disorder
	Hazard Ratio (95% CI)	Hazard Ratio (95% CI)
Age	0.98** (0.97 – 0.99)	1.00 (0.98 – 1.02)
Sex	1.34* (1.06 – 1.69)	1.33 (0.92 – 1.94)
Education	0.74 (0.53 – 1.03)	1.28 (0.83 – 1.97)
Race	0.79 (0.59 – 1.05)	0.94 (0.60 – 1.49)
Antecedent chronic spinal pain	0.83 (0.55 – 1.24)	1.68* (1.06 – 2.66)

Note: * $p < .05$, ** $p < .01$