Research article

Depressive symptomatology after spinal cord injury: A multi-center investigation of multiple racial-ethnic groups

Yue Cao¹, Chao Li¹, Anne Gregory¹, Susan Charlifue², James S. Krause¹

¹Department of Health Sciences and Research, College of Health Professions, Medical University of South Carolina, Charleston, SC, USA, ²Craig Hospital, Englewood, CO, USA

Objective: To identify (1) racial-ethnic differences in depressive symptomatology after spinal cord injury (SCI) and (2) the relationship of multiple additional factors to depressive symptoms, including health behaviors, employment, fatigue, and pain interference.

Design: Cross-sectional

Setting: Data were collected at 3 specialty hospitals in different regions of the USA (Southeastern, Mountain, Western).

Participants: Participants (N = 1,063) were identified from outpatient records of the 3 hospitals with oversampling of racial-ethnic minority groups.

Interventions: N/A

Main Outcome Measure(s): The outcome, depressive symptomatology, was measured by the Older Adult Health and Mood Questionnaire (OAHMQ). Participant demographic and injury characteristics were measured as statistical controls, as well as other variables including health behavior factors, depression/stress relief medication usage, fatigue, and pain interference. The multivariate analyses were developed using OLS regression models and logistic regression models.

Results: Employment was protective for depressive symptomatology, whereas fatigue, pain interference, and binge drinking were risk factors for higher OAHMQ scores. Although there were no bivariate racial-ethnic differences in depressive symptoms, fatigue and pain interference had suppression effects on the relationship between race-ethnicity and depressive symptomatology. After controlling for fatigue and pain interference, Hispanic participants had significantly lower OAHMQ scores and lower odds of probable major depression (PMD) than non-Hispanic Whites and Blacks.

Conclusions: Fatigue and pain interference are associated with both race-ethnicity and depressive symptomatology. Assuming the same level of fatigue and pain interference, Hispanics will have a lower risk of depressive symptoms than non-Hispanic Whites and Blacks.

Keywords: Spinal cord injury, Depression, Pain, Fatigue, Health behavior, Disparities

Spinal cord injury (SCI) is a traumatic injury involving partial or total loss of movement and/or motor function of arms and/or legs. Because SCI drastically changes an individual's life, much research has been devoted to both physical and mental health outcomes after SCI. Numerous studies have tried to identify depression rates after SCI with estimates ranging rather widely from 10% to over 40%. ¹⁻⁶

Correspondence to: Yue Cao, Department of Health Sciences and Research, College of Health Professions, Medical University of South Carolina, 77 President St, C207, MSC 700, Charleston, SC 29425 USA. Email: caoyu@musc.edu.

Several factors have been identified as associated with depressive symptomatology after SCI, such as socioeconomic factors, health factors, and psychological factors. More specifically, low income, 100 education level, 100 and unemployment 100 are positively related to depressive symptoms. Depressive symptoms are more likely to be seen among those with worsening health problems, pressure sores, lack of effective treatment, 100 presence of health complications, 100 pain, 15,16 pain interference, 100 fatigue, 100 and undesirable health behaviors, such as smoking. 100 The following psychological factors are also positively associated with depressive symptomatology:

psychological disorders,²⁰ substance abuse disorders,^{14,21} pre-injury psychological issues,^{25,26} and inadequate coping abilities.²⁷ Meanwhile, problem solving^{22–24} and adjustment capabilities²⁸ are negatively associated with depressive symptomatology.

Although many studies have investigated depressive symptomatology after SCI, limited research exists on the racial-ethnic differences of depressive symptomatology. The results from existing literature are also conflicting. Some studies have found depressive symptoms are unrelated to race. 7,29,30 Myaskovsky et al. 30 only included Black and White participants, and Dunn et al.²⁹ found no race effect after accounting for economic factors. However, other research has indicated significant racial-ethnic differences in depressive symptoms. Using the Older Adult Health and Mood Questionnaire (OAHMQ), one study indicated Latino/ Hispanic individuals have significantly depression scores and greater prevalence of probable major depression (PMD), based on cutoff scores of 11 or higher, than all other racial-ethnic groups.³¹ Arango-Lasprilla et al.² investigated changes in major depressive disorder (MDD) between 1 and 5 years post-SCI with the Patient Health Questionnaire (PHQ-9). MDD was defined as at least 1 essential criterion, plus a total of 5 or more symptoms presented more than half the days in the past 2 weeks. They noted the odds of MDD for non-Hispanic Blacks significantly decreased during the 5 years, whereas other racialethnic groups did not change. Krause et al. found American-Indians reported higher depressive symptomatology and higher PMD rates than the general population. Different measures of depressive symptoms and differing cutoff scores or criteria for depressive disorders may lead to conflicting results with regards to race. For example, the PHQ-9 establishes depressive disorder diagnoses, which is consistent with the DSM-IV criteria, while the OAHMQ is designed to assess depressive symptomatology of older people, who are more likely to experience disability and ageing related symptoms. Krause et al. 32 compared the OAHMQ33 with the PHQ-9³⁴ and suggested the use of the OAHMQ results in higher reported rates of depression and race differences whereas no differences were identified when using the PHQ-9. Other possible reasons for conflicting results could be different sample sizes and controlling variables used in each study.

The previous studies also found interactions between race-ethnicity and sex. Krause *et al.*¹³ found that non-White women were at a higher risk for PMD than White men. After accounting for education and income, the risk diminished but did not disappear

completely, with non-White women still significantly more likely to report PMD than White men. Another study showed Black and White females with SCI were more likely to have PMD than White males with SCI.³⁵

The above studies underscore the importance of identifying mediators and implementing statistical controls for other important variables that may be related to depressive symptomatology. Our purpose was to enhance our understanding of depressive symptomatology after SCI related to racial-ethnic differences, while also assessing the relationship of other risk and protective factors for depressive symptoms, including demographic, injury, and socioeconomic variables, health behaviors, and health factors. To accomplish this goal, we oversampled underserved racial-ethnic groups, including Hispanic and non-Hispanic Black participants. Because health behaviors, pain, and fatigue not only impact mental health but also are associated with race-ethnicity, 36,37 we included smoking, binge drinking, fatigue, and pain interference as important correlates in our study to obtain unbiased estimation of the relationship between independent variables and depressive symptoms.

Methods

Participants

Approval was obtained from the Institutional Review Boards at each of the 3 collaborating institutions from different regions of the USA (Southeastern, Mountain, Western). All participants met the following inclusion criteria: (1) traumatic SCI, not completely recovered, (2) 18 years or older, and (3) at least 1 year since injury. We collected data from 1,066 participants between 2011 and 2014. Among them, there were 1,063 participants with valid race-ethnicity information broken down as follows by region: Southeastern (n = 620, with 4% Hispanic, 45% non-Hispanic Black, 47% non-Hispanic White, and 4% others), Mountain (n = 93, with 35% Hispanic, 13% non-Hispanic Black, 37% non-Hispanic White, and 15% others), and Western (n = 350, with 63\% Hispanic, 20\% non-Hispanic Black, 13% non-Hispanic White, and 5% others). We excluded 3 participants with missing race-ethnicity information from our analysis.

Procedures

The data were housed at the lead site in the Southeastern United States. Participants identified at the lead site were from rosters from previous studies and were first identified from a specialty hospital in the Southeastern USA. Those from the Mountain and Western regions were also identified through previous studies or from

hospital databases at those institutions. At each site, a portion of non-Hispanic White participants was enrolled, along with oversampling of other racial-ethnic groups, consistent with the characteristics of the population in that region. This resulted in higher portions of non-Hispanic Blacks from the Southeastern region, Hispanics from the Mountain region, and Hispanics from the Western region.

At each of the 3 collaborating data collection sites, introductory letters were sent to potential participants. In the Southeastern region, all data were collected by mail. In the Mountain region, the same self-report data were collected primarily by telephone interview. In the Western region, data were collected in conjunction with a study of biomarkers, so participants either returned the materials by mail or dropped them off at the time of their appointment. Non-respondents received follow-up mailings, and all participants were offered \$50 in remuneration.

Measures

The OAHMQ was used to measure depressive symptomatology,³³ as it has been used in several earlier studies of race-ethnicity and depression after SCI.^{13,31,38,39} It is a 22-item measure created to focus on aspects of depression that limit the number of somatic symptomatology, as these symptoms can be indicative of symptoms of physical disability, older age, or medical treatment.³² The maximum score possible on the OAHMQ is 22, and scores of 11 or higher are considered indicative of PMD. The reliability and validity of the OAHMQ previously have been shown in studies of traumatic SCI.^{32,38}

Race-ethnicity was categorized into 4 groups: non-Hispanic White, non-Hispanic Black, Hispanic, and others. Other variables included sex (male vs. female), marital status (married vs. others), employment status (employed vs. others), age at assessment, years since injury, cause of injury (transportation, violence, and others), and injury severity, which was categorized into 4 groups consistent with previous research, 40,41 including: (a) C1–C4 level injury, non-ambulatory, (b) C5–C8 level injury, non-ambulatory, (c) non-cervical injury, non-ambulatory, and (d) ambulatory, regardless of injury level. Health behavior variables included selfreport of current smoking status (yes vs. no), and self-report binge drinking in the past month (yes vs. no for 5 or more drinks on one occasion). We also measured usage of depression or stress relief medication (ves vs. no).

Pain interference was measured by interference items from the Brief Pain Inventory. 42 We removed 1 item,

walking ability, from the pain interference scale because of our largely non-ambulatory sample. Fatigue was measured by the Modified Fatigue Impact Scale-5 item version. ^{43,44} All scales had good reliability in our study, with a standardized alpha coefficient of the OAHMQ scale 0.84, Pain Interference Scale 0.95, and Fatigue Impact Scale 0.90.

Data analysis

The racial-ethnic groups were compared on depressive symptomatology measures, demographics, characteristics, health behaviors, pain interference, and fatigue, using the χ^2 for categorical variables and t-tests and the ANOVA test for continuous variables (Table 1). We also conducted multivariate analyses, using Ordinary Least Squares (OLS) regression models for OAHMQ total scores and logistic models for PMD. To assess the impact of fatigue and pain interference on the relationship between race-ethnicity and depressive symptoms, we developed 2 OLS models and 2 logistic models. The first model did not include fatigue and pain interference, with both measures added to the second model. We then compared the 2 models with the F-test for the OLS models and the likelihood ratio test for the logistic models.

Results

Among 1,063 participants, 35% were non-Hispanic White (n = 369) and the remaining were 34% non-Hispanic Black (n = 361), 26% Hispanic (n = 277), and 5% others (n = 56). Those classified as others included: American Indian, Asian, Native Hawaiian, and those self-reported as being "more than one race." We did not find significant differences of PMD percentages and OAHMQ total scores among the 4 racialethnic groups (Table 1). When comparing the groups on other factors, non-Hispanic Whites included a significantly higher percentage of individuals who were female (38.5%), married (39%), employed (26.9%), using depression/stress relief medication (41%), and a lower percentage of those who engaged in smoking (20%) or binge drinking (15.7%), were ambulatory (10.6%), and had an SCI caused by violence (2.7%) compared to the other groups. The smoking prevalence for non-Hispanic Blacks (32.1%) was higher than all the others, while Hispanic participants reported significantly higher binge drinking (33.2%) than all the others. The prevalence of violent etiologies was higher for both Hispanics (48.9%) and non-Hispanic Blacks (33.8%) than for other groups. The average ages of Hispanics (44 years) and non-Hispanic Blacks (46 years) were younger than non-Hispanic Whites

Table 1 Comparing study participants' characteristics across 4 racial/ethnic groups

	Hispanic (n = 277)	Non-Hispanic Black (n = 361)	Non-Hispanic White (n = 369)	Other (n = 56)*	P-value**
OAHMQ total score: mean ± SD	7.2 ± 4.5	6.6 ± 4.5	6.6 ± 5.1	6.7 ± 4.7	0.374
PMD: n (%)					0.475
No	202 (76.5)	271 (80.2)	262 (76.2)	46 (82.1)	
Yes	62 (23.5)	67 (19.8)	82 (23.8)	10 (17.9)	
Sex: n (%)					< 0.001
Male	228 (82.6)	289 (80.1)	227 (61.5)	45 (76.3)	
Female	48 (17.4)	72 (19.9)	142 (38.5)	14 (23.7)	
Chronological age: mean ± SD	44.3 ± 12.6	46.0 ± 11.5	49.3 ± 13.2	49.4 ± 13.0	< 0.001
Marital status: n (%)					< 0.001
Married	65 (23.5)	72 (19.9)	144 (39.0)	19 (32.2)	
Other	212 (76.5)	289 (80.1)	225 (61.0)	40 (67.8)	
Employment status: n (%)	_:_(::::)	()	(*)	(3.15)	< 0.001
Employed	37 (13.7)	33 (9.6)	95 (26.9)	6 (10.9)	
Other	233 (86.3)	312 (90.4)	258 (73.1)	49 (89.1)	
Smoking: n (%)	(,	- (/	, ,	,	0.001
No	216 (78.0)	245 (67.9)	295 (80.0)	42 (71.2)	
Yes	61 (22.0)	116 (32.1)	74 (20.0)	17 (28.8)	
Binge drinking: n (%)	- (==:-)	(==,	(==)	(====)	< 0.001
No	185 (66.8)	274 (75.9)	311 (84.3)	49 (83.1)	
Yes	92 (33.2)	87 (24.1)	58 (15.7)	10 (16.9)	
Years since injury: mean ± SD	17.6 ± 18.5	15.3 ± 9.7	17.0 ± 15.4	16.6 ± 13.1	0.044
Injury Severity: n (%)	=	10.0 = 0	=	10.0 = 10.1	0.002
Non-ambulatory,C1–4 levels	16 (5.8)	41 (11.4)	42 (11.4)	9 (15.3)	
Non-ambulatory,C5–8 levels	54 (19.5)	90 (24.9)	113 (30.6)	12 (20.3)	
Non-ambulatory, other levels	164 (59.2)	173 (47.9)	175 (47.4)	28 (47.5)	
All ambulatory	43 (15.5)	57 (15.8)	39 (10.6)	10 (16.9)	
Cause of injury: n (%)	.0 (.0.0)	0. (10.0)	00 (10.0)	.0 (10.0)	< 0.001
Violence	134 (48.9)	121 (33.8)	10 (2.7)	6 (10.3)	10.001
Transportation	88 (32.1)	150 (41.9)	202 (54.9)	35 (60.4)	
Other	52 (19.0)	87 (24.3)	156 (42.4)	17 (29.3)	
Depression/stress relief medication usage: n (%)	02 (10.0)	01 (21.0)	100 (12.1)	17 (20.0)	< 0.001
No	196 (72.3)	268 (78.6)	213 (59.0)	40 (72.7)	10.001
Yes	75 (27.7)	73 (21.4)	148 (41.0)	15 (27.3)	
Fatigue impact score: mean ± SD	7.1 ± 4.9	4.1 ± 3.9	4.2 ± 4.1	4.9 ± 4.5	< 0.001
Pain interference score: mean ± SD	23.5 ± 20.2	19.2 ± 18.8	20.6 ± 18.8	18.8 ± 18.9	0.047

^{*}We classified American Indian, Asian, Native Hawaiian, and those self-reported as being "more than one race" into this category because of their small sample sizes.

(49 years) at the time of survey. The average fatigue score for Hispanic participants was 7.1 and pain interference score was 23.5. Both scores were significantly higher than other groups. All these differences were statistically significant at the 0.05 level. The bivariate comparison also showed that violence was elevated among Hispanics and non-Hispanic Blacks. We built 2 OLS regression models for the OAHMQ total score (Table 2). The first model included demographics, characteristics, injury health behaviors, depression/stress relief medication as co-variables. There was no significant association between race-ethnicity and total OAHMQ score in this model, while smoking, binge drinking, medication usage, and ambulation were positively associated with the OAHMQ total score. A greater number of years post-injury and being employed were related to lower scores. The second model indicated fatigue and pain interference scores

were positively related to the OAHMQ score. After the addition of fatigue and pain interference, Hispanics had significantly lower OAHMQ total scores than non-Hispanic Whites, and the adjusted R² increased from 0.15 to 0.44. The F-test for the increment of adjusted R² indicated adding fatigue and pain interference significantly improved the model fit (P < 0.001).

The first logistic model found that employment was significantly associated with lower odds of PMD, and depression/stress relief medication usage was related to higher odds of PMD (Table 3). The second logistic model indicated both fatigue and pain interference were significantly associated with higher odds of PMD. After adding fatigue and pain interference into the second model, the odds of Hispanics having PMD were only 34% that of non-Hispanic Whites. The difference in odds between Hispanics and non-Hispanic Whites was statistically significant. We conducted the

 $^{^{*}\}chi^{2}$ test for categorical variables, and ANOVA test for continuous variables.

Table 2 OLS Regression Analysis of OAHMQ Total Score

	Model 1		Model 2	
	Coefficient (b)	Р	Coefficient (b)	Р
Race/ethnicity (ref = Non-Hispanic White)		0.551		0.014
Hispanic	0.05	0.906	-1.10	0.005
Non-Hispanic Black	-0.46	0.270	-0.08	0.836
Others	0.09	0.904	0.12	0.843
Male (ref = Female)	-0.64	0.082	-0.47	0.141
Chronological age	0.01	0.621	-0.01	0.657
Married (ref = No)	-0.31	0.399	-0.02	0.950
Employed (ref = Others)	-1.59	< 0.001	-0.81	0.027
Current smoking (ref = No)	0.99	0.010	0.53	0.115
Binge drinking (ref = No)	0.90	0.021	1.11	0.001
Years since injury	-0.06	0.002	-0.05	0.003
Injury level (ref = Non-C non-ambulatory)		0.017		0.812
C14:Non-ambulatory	-0.49	0.342	-0.25	0.601
C58: Non-ambulatory	-0.61	0.108	-0.26	0.434
Ambulatory	1.02	0.036	0.09	0.824
Cause of injury (ref = Transportation)		0.123		0.371
Violence	0.80	0.056	0.03	0.939
Others	-0.06	0.874	-0.45	0.181
Depression or stress medication (ref = No)	2.46	< 0.001	1.11	0.001
Fatigue	-	-	0.38	< 0.001
Pain interference	-	-	0.07	< 0.001
Adjusted R ²	0.15		0.44	

likelihood ratio test for the 2 logistic models. The test also suggested that, after adding fatigue and pain interference, the model fit was significantly increased (P < 0.001).

We developed additional OLS regression models and logistic models by using non-Hispanic Blacks as the reference group and found Hispanic participants had significantly lower OAHMQ scores and lower odds of PMD than non-Hispanic Blacks while controlling for pain and fatigue (tables available upon request). No other significant differences between racial-ethnic groups were observed.

Discussion

This study was designed to identify the relationship between depressive symptomatology and race-ethnicity,

Table 3 Logistic Regression Analysis of probable major depression (PMD)

	Model 1		Model 2	
	OR (95% CI)	Р	OR (95% CI)	Р
Race-ethnicity (ref = Non-Hispanic White)		0.457		0.006
Hispanic	0.78 (0.48-1.27)		0.34 (0.18-0.64)	
Non-Hispanic Black	0.69 (0.43–1.09)		0.74 (0.41–1.32)	
Others	0.78 (0.34–1.77)		0.52 (0.19–1.43)	
Male (ref = Female)	0.72 (0.49–1.07)	0.105	0.74 (0.45–1.18)	0.204
Chronological age	0.99 (0.97–1.01)	0.284	0.98 (0.96–1.00)	0.058
Married (ref = No)	0.75 (0.50–1.14)	0.183	0.92 (0.55–1.54)	0.742
Employed (ref = Others)	0.50 (0.29–0.87)	0.013	0.59 (0.30–1.15)	0.121
Current smoking (ref = No)	1.21 (0.81–1.80)	0.349	0.88 (0.53–1.45)	0.608
Binge drinking (ref = No)	1.28 (0.85–1.93)	0.230	1.62 (0.99–2.67)	0.057
Years since injury	0.98 (0.96–1.00)	0.116	0.98 (0.96–1.00)	0.123
Injury Level (ref = non-C non-ambulatory)	,	0.326	,	0.979
C14:Non-ambulatory	0.70 (0.39-1.26)		0.91 (0.41-2.02)	
C58: Non-ambulatory	0.75 (0.49–1.15)		1.09 (0.64–1.84)	
Ambulatory	1.13 (0.68–1.91)		1.01 (0.58–1.73)	
Cause of injury (ref = Transportation)	,	0.215	,	0.862
Violence	1.48 (0.94–2.33)		1.17 (0.66–2.05)	
Others	1.25 (0.81–1.92)		1.00 (0.58–1.73)	
Depression or stress relief medication (ref = No)	2.76 (1.94–3.93)	< 0.001	2.03 (1.30–3.17)	0.002
Fatigue	-	-	1.24 (1.17–1.32)	< 0.001
Pain interference	_	-	1.03 (1.02–1.04)	< 0.001
−2 Log L	863.696		582.641	

NO. 1

health behaviors, employment, fatigue, and pain interference. We found significantly less depressive symptomatology among Hispanic participants but only after adjusting for fatigue and pain interference. Such phenomena indicate fatigue and pain interference have suppression effects on the relation between race-ethnicity and depressive symptoms. 45 Suppression effects happen when the direct relationship between an independent variable and a dependent variable is suppressed by the indirect relationship through a third variable. Our study found that pain interference and fatigue reported by Hispanic participants were significantly higher than for other groups, while pain interference and fatigue were related to higher depression scores and greater PMD odds. After the statistical adjustment of the pain interference and fatigue, the suppressed direct relationship between Hispanics and depressive symptoms became significant. One previous study found Hispanic individuals had significantly higher depression scores and a greater prevalence of PMD,³¹ but the study reported only bivariate analyses of a smaller participant sample without accounting for the indirect effects through pain interference and fatigue.

The substantially higher fatigue and pain interference scores among Hispanic participants are noteworthy. Other studies with the general population also suggest racial-ethnic minorities are more likely to experience pain⁴⁶ and fatigue.³⁷ Taken together, the findings regarding race-ethnicity present a complicated picture. Hispanic participants presenting with depressive symptoms may have other significant issues with fatigue and pain interference, and these may be overlooked or misdiagnosed as depressive disorders. We cannot rule out a cultural component affecting the reporting or interpretation of symptoms. In the general population, non-Hispanic Whites are more likely to use antidepressants and psychotherapy to treat depressive symptoms than other groups. 47-49 Our study also found a similar pattern, which might suggest racial-ethnic disparities in mental health care access among people with SCI. A study based on a nationally representative sample found that Whites had a greater prevalence of MDD than Mexican Americans and African Americans.⁵⁰ However, our SCI sample shows a different pattern that Hispanics had the most severe depressive symptomology compared to other groups, which suggests fatigue and pain might play an important role in the SCI sample. Employed participants had significantly lower depression scores than other participants. This protective effect is consistent with what has been found in the literature. ^{2,10,11,13} We also found binge drinking was associated with higher depression scores, but neither employment nor binge drinking were significantly related to the severe depressive symptomatology as measured by PMD. However, we still need to be aware that unemployment and alcohol abuse issues may develop from a transient experience to a clinical diagnosis of MDD. Our study implies that further research should put emphasis on reducing depressive symptomatology by promoting positive health behaviors, encouraging active employment, expanding mental health care coverage, and treating fatigue and pain in the SCI population.

Limitations

There are several important limitations that must be considered when interpreting the findings. First, our results are based on a sample selected from 3 clinical sites, all participants were at least 1 year post-injury, and 80% of the sample had lived with traumatic SCI for 5 years or more. Therefore, it is most accurate to interpret our findings as the relationship between raceethnicity and other factors with depressive symptomatology for those with chronic SCI and receiving treatment in specialty hospitals. Second, race-ethnicity distribution varied at different data collection sites. Therefore, it is best to interpret the racial-ethnic differences of depressive symptomatology, specifically the lower levels observed among those who were Hispanic, as reflecting both the racial-ethnic and geographic differences in which they are embedded. Third, the mode of data collection (mail, drop-off, and interview) varied by the study sites. It is possible that a part of the observed racial differences in the outcomes measured were due to the variation in the mode of data collection as well as racial-ethnic distribution across the 3 study sites. Fourth, this is a cross-sectional study, which precludes determination of causality. Fifth, although the OAHMQ is a clinically validated scale to assess depressive symptomatology, like all screening measures, it is not the equivalent of a thorough interview-based mental status examination, and PMD cannot be used as a diagnostic tool for depressive disorder. Other measures are more widely in use currently, although this measure has been used in previous studies of SCI and race-ethnicity. 7,32,38 Confirmatory assessment would be helpful in further diagnosing any depressive disorders. Lastly, although we have controlled for depression/stress relief medication usage in our model, it is a general dichotomous measure, and we need more detailed information on antidepressant medication and/or psychotherapy services in a future study.

NO 1

Conclusion

Our study revealed complexities of the relationship between race-ethnicity and depressive symptomatology. We identified a suppression effect of fatigue and pain interference on the relationship between race-ethnicity and depressive symptomatology. Hispanic participants had higher fatigue and pain interference levels, which increased the possibility of depressed mood and PMD. However, assuming the same level of fatigue and pain interference, Hispanic participants would have been less likely to report significant depressive symptomatology than non-Hispanic Whites and Blacks. Unemployment and binge drinking were also associated with depressive symptoms.

Disclaimer statements

Contributors None.

Funding This work was supported by the US Department of Health and Human Services, Administration for Community Living, NIDILRR grant numbers 90DP0004 and 90DP0050, and the South Carolina Spinal Cord Injury Research Fund grant number SCIRF 11-006. However, those contents do not necessarily represent the policy of the Department of Health and Human Services or the SCSCIRF, and you should not assume endorsement by the Federal Government or the state of South Carolina.

Conflict of interest None.

Ethics approval None.

References

- 1 Bombardier CH, Richards JS, Krause JS, Tulsky D, Tate DG. Symptoms of major depression in people with spinal cord injury: implications for screening. Arch Phys Med Rehabil 2004;85(11): 1749–56.
- 2 Arango-Lasprilla JC, Ketchum JM, Starkweather A, Nicholls E, Wilk AR. Factors predicting depression among persons with spinal cord injury 1 to 5 years post injury. NeuroRehabil 2011;29 (1):9–21.
- 3 Fann JR, Bombardier CH, Richards JS, Tate DG, Wilson CS, Temkin N. Depression after spinal cord injury: comorbidities, mental health service use, and adequacy of treatment. Arch Phys Med Rehabil 2011;92(3):352–60.
- 4 Hoffman JM, Bombardier CH, Graves DE, Kalpakjian CZ, Krause JS. A longitudinal study of depression from 1 to 5 years after spinal cord injury. Arch Phys Med Rehabil 2011;92(3):411–8.
- 5 Fuhrer MJ, Rintala DH, Hart KA, Clearman R, Young ME. Depressive symptomatology in persons with spinal cord injury who reside in the community. Arch Phys Med Rehabil 1993;74: 255–60.
- 6 Frank RG, Chaney JM, Clay DL, Shutty MS, Beck NC, Kay DR, et al. Dysphoria: a major symptom factor in persons with disability or chronic illness. Psychiatry Res 1992;43(3):231–41.
- 7 Brown SA, Saunders LL, Krause JS. Racial disparities in depression and life satisfaction after spinal cord injury: a mediational model. Top Spinal Cord Inj Rehabil 2012;18(3):232–40.
- 8 Krause JS, Coker J, Charlifue S, Whiteneck G. Depression and subjective well-being among 97 American Indians with

- spinal cord injury: a descriptive study. Rehabil Psychol 1999; 44:354–72.
- 9 Scivoletto G PA, Di Lucente L, Castellano V. Pyschological investigation of spinal cord injury patients. Spinal Cord 1997;35(8): 516–20.
- 10 Kalpakjian CZ, Albright KJ. An examination of depression through the lens of spinal cord injury: comparative prevalence rates and severity in women and men. Womens Health Issues 2006;16(6):380–8.
- 11 Hwang M, Zebracki K, Chlan KM, Vogel LC. Longitudinal employment outcomes in adults with pediatric-onset spinal cord injury. Spinal Cord 2014;52(6):477–82.
- 12 Arango-Lasprilla JC, Ketchum JM, Francis K, Lewis A, Premuda P, Wehman P, *et al.* Race, ethnicity, and employment outcomes 1, 5, and 10 years after spinal cord injury: a longitudinal analysis. PM R 2010:2(10):901–10.
- 13 Krause JS, Kemp B, Coker J. Depression after spinal cord injury: relation to gender, ethnicity, aging and socioeconomic indicators. Arch Phys Med Rehabil 2000;81(8):1099–109.
- 14 Tate D, Forchheimer M, Maynard F, Dijkers M. Predicting depression and psychological distress in persons with spinal cord injury based on indicators of handicap. Am J Phys Med Rehabil 1994;73(3):175–83.
- 15 Cairns D, Adkins R, Scott M. Pain and depression in acute traumatic spinal cord injury: origins of chronic problematic pain. Arch Phys Med Rehabil 1996;77(4):329–35.
- 16 Craig AR, Hancock K, Dickson H. Spinal cord injury: a search for determinants of depression two years after the event. Br J Clin Psychol 1994;33(2):221–30.
- 17 Krause JS, Brotherton S, Morrisette D, Newman S, Karakostas T. Does pain interference mediate the relationship of independence in ambulation with depressive symptoms after spinal cord injury? Rehabil Psychol 2007;52(2):162–9.
- 18 Craig A, Tran Y, Siddall P, Wijesuriya N, Lovas J, Bartrop R, *et al.* Developing a model of associations between chronic pain, depressive mood, chronic fatigue, and self-efficacy in people with spinal cord injury. J Pain 2013;14(9):911–20.
- 19 Weaver FM, Smith B, LaVela SL, Evans CT, Ullrich P, Miskevics S, *et al.* Smoking behavior and delivery of evidence-based care for veterans with spinal cord injuries and disorders. J Spinal Cord Med 2011;34(1):35–45.
- 20 Judd FK, Stone J, Webber JE, Brown DJ, Burrows GD. Depression following spinal cord injury. A prospective in-patient study. Br J Psychiatry 1989;154:668–71.
- 21 Dryden DM, Saunders LD, Rowe BH, May LA, Yiannakoulias N, Svenson LW, et al. Depression following traumatic spinal cord injury. Neuroepidemiol 2005;25(2):55–61.
- 22 Heppner PP. The problem solving inventory: Manual. Palo Alto, CA: Consulting Psychologists Press; 1988.
- 23 Elliott TR, Godshall FJ, Herrick SM, Witty TE, Spruell M. Problem-solving appraisal and psychological adjustment following spinal cord injury. Cog Ther Res 1991;15(5):387–98.
- 24 Elliott TR, Marmarosh C. Social-Cognitive Processes in Behavioral Health Implications for Counseling. Counsel Psychol 1995;23(4):666–81.
- 25 Johnson RL, Gerhart KA, McCray J, Menconi JC, Whiteneck GG. Secondary conditions following spinal cord injury in a population-based sample. Spinal Cord 1998;36(1):45–50.
- 26 Kishi Y, Robinson RG, Forrester AW. Prospective longitudinal stiudy of depression following spinal cord injury. J Neuropsychiatry Clin Neurosci 1994;6(3):237–44.
- 27 Pollard C, Kennedy P. A longitudinal analysis of emotional impact, coping strategies and post-traumatic psychological growth following spinal cord injury: a 10-year review. Br J Health Psychol 2007;12(Pt 3):347–62.
- 28 Dorsett P, Geraghty T. Depression and adjustment after spinal cord injury: a three-year longitudinal study. Top Spinal Cord Inj Rehabil 2004;9(4):43–56.
- 29 Dunn M, Love, L, Ravesloot, C. Subjective health in spinal cord injury after outpatient healthcare follow-up. Spinal Cord 2000;38 (2):84–91.
- 30 Myaskovsky L, Burkitt KH, Lichy AM, Ljungberg IH, Fyffe DC, Ozawa H, *et al.* The association of race, cultural factors, and health-related quality of life in persons with spinal cord injury. Arch Phys Med Rehabil 2011;92(3):441–8.

- 31 Kemp BJ, Krause JS. Depression and life satisfaction among people aging with post-polio and spinal cord injury. Disabil Rehabil 1999;21:241–9.
- 32 Krause JS, Saunders LL, Reed KS, Coker J, Zhai Y, Johnson E. Comparison of the Patient Health Questionnaire and the Older Adult Health and Mood Questionnaire for self-reported depressive symptoms after spinal cord injury. Rehabil Psychol 2009;54(4): 440–8.
- 33 Kemp BJ, Adams BM. The Older Adult Health and Mood Questionnaire: a measure of geriatric depressive disorder. J Ger Psychiatry Neurol 1995;8(3):162–7.
- 34 Kroenke K, Spitzer RL, Williams JB. The PHQ-9: validity of a brief depression severity measure. J Gen Int Med 2001;16(9): 606–13.
- 35 Saunders LL, Krause JS, Focht KL. A longitudinal study of depression in survivors of spinal cord injury. Spinal Cord 2012; 50(1):72–7.
- 36 Jackson JS, Knight KM, Rafferty JA. Race and unhealthy behaviors: chronic stress, the HPA axis, and physical and mental health disparities over the life course. Am J Public Health 2010; 100(5):933–9.
- 37 Dinos S, Khoshaba B, Ashby D, White PD, Nazroo J, Wessely S, *et al.* A systematic review of chronic fatigue, its syndromes and ethnicity: prevalence, severity, co-morbidity and coping. Int J Epidemiol 2009;38(6):1554–70.
- 38 Krause JS, Kemp B, Coker JL. Depression after spinal cord injury: relation to gender, ethnicity, aging, and socioeconomic indicators. Arch Phys Med Rehabil 2000;81(8):1099–109.
- 39 Kemp B, Krause J, Adkins R. Depression among African Americans, Latinos, and Caucasians with spinal cord injury: a exploratory study. Rehabil Psychol 1999;44(3):235–47.
- 40 Krause JS, Saunders LL, Acuna J. Gainful employment and risk of mortality after spinal cord injury: effects beyond that of

- demographic, injury and socioeconomic factors. Spinal Cord 2012;50(10):784-8.
- 41 Saunders LL, Krause JS. Behavioral factors related to fatigue among persons with spinal cord injury. Arch Phys Med Rehabil 2012;93(2):313–8.
- 42 Cleeland CS, Ryan KM. Pain assessment: global use of the brief pain inventory. Ann Acad Med Singapore 1994;23(2):129–38.
- 43 Fisk JD, Ritvo PG, Ross L, Haase DA, Marrie TJ, Schlech WF. Measuring the functional impact of fatigue: initial validation of the fatigue impact scale. Clin Infect Dis 1994;18(Suppl 1): S79–S83.
- 44 Multiple Sclerosis Council for Clinical Practice Guidelines. Fatigue and multiple sclerosis: evidence-based management of strategies for fatigue in multiple sclerosis. Washington, DC: Paralyzed Veterans of America; 1998.
- 45 MacKinnon DP, Krull JL, Lockwood CM. Equivalence of the mediation, confounding and suppression effect. Prev Sci 2000;1 (4):173–81.
- 46 Shavers VL, Bakos A, Sheppard VB. Race, ethnicity, and pain among the U.S. adult population. J Health Care Poor Underserved 2010;21(1):177–220.
- 47 Pratt LA, Brody DJ, Gu Q. Antidepressant use in persons aged 12 and over: United States, 2005–2008. NCHS Data Brief 2011; (76):1–8.
- 48 Blazer DG, Hybels CF, Simonsick EM, Hanlon JT. Marked differences in antidepressant use by race in an elderly community sample: 1986–1996. Am J Psychiatry 2000;157(7):1089–94.
- 49 Olfson M, Marcus SC. National patterns in antidepressant medication treatment. Arch Gen Psychiatry 2009;66(8):848–56.
- 50 Riolo SA, Nguyen TA, Greden JF, King CA. Prevalence of depression by race/ethnicity: findings from the National Health and Nutrition Examination Survey III. Am J Public Health 2005;95(6):998–1000.

92