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ORIGINAL ARTICLE

A latent structural equation model of risk behaviors and pressure ulcer outcomes among people with spinal cord injury

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Study Design: Cross-sectional.
Objective: Our purpose was to develop a latent structural model to demonstrate the relationship between factor structures of risk health behaviors and pressure ulcer (PrU) outcomes among participants with spinal cord injury (SCI).
Setting: Data were collected at a large specialty hospital and analyzed at a medical university in the Southeastern USA.
Methods: In total, 1871 participants with traumatic SCI of at least 1-year duration were recruited. Four latent risk behavior indicators were developed and further linked with a higher dimension which is classified as the risk dimension. A latent PrU variable was created and measured by four observable PrU-related outcomes. Latent structural equation modeling was performed to assess the relationship between the latent risk behavior and the latent PrU outcome. Several exogenous variables were also included in the structural equation model.
Results: The risk behavior dimension had a significant direct effect on the latent PrU (direct effect = 0.323, $P<0.01$). All direct relationships between the risk behavior dimension and risk behaviors were also significant ($r_{\text{smoking}} = 0.436$, $r_{\text{prescription compliance}} = 0.351$ and $r_{\text{specific prescription misuse}} = 0.502$), except alcohol consumption ($r_{\text{alcohol consumption}} = 0.087$). Participants who were African American, had higher injury levels and longer time since SCI were more likely to have worse PrU outcomes.
Conclusions: The overall findings of this study suggest the need to reduce risk behaviors to prevent adverse PrU outcomes. The risk of PrU outcomes is especially high among people who are African American, have higher level of SCI and have longer time since SCI.
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INTRODUCTION

Pressure ulcers (PrU), also known as decubitus ulcers or pressure sores, are an injury to the skin or underlying tissue as a result of pressure and/or shear.[1] PrU are one of the most common and debilitating secondary health conditions following spinal cord injury (SCI). As a result of both motor and sensory impairments, as well as activity limitations, individuals with SCI are particularly susceptible to PrU development. Some estimates suggest that nearly half of the individuals with chronic SCI have had a PrU in the past year,[2–7] and approximately a quarter have had a PrU at any given time.[2–4,6,7] PrU are associated with numerous negative outcomes, including decreased daily activities and quality of life,[8–10] costly complications and hospitalizations,[11–13] and even mortality.[14–16] Owing to the negative effects of PrU, extensive research has centered on the study of risk factors[17–21] and prevention and treatment of PrU.[22–25] However, few studies have assessed behavioral risk factors, and they have primarily focused on single observable factors, rather than the impact of multiple risk factors on PrU outcomes.

Behavioral risk factors such as cigarette smoking, alcohol consumption and prescription medication use and abuse have been studied in association with PrU outcomes; however, the number of studies assessing these factors is limiting.[20] Cigarette smoking has long been recognized as having adverse effects on wound healing and PrU outcomes and is considered a risk factor for PrU development and severity.[3,26–29] Physiologically, smoking decreases cutaneous blood flow,[30] which leads to negative PrU outcomes, including more severe sores and sores that heal less quickly. Additionally, smoking may result in pulmonary disease, which has been associated with PrU outcomes after SCI.[27]

Few studies have examined the relationship between alcohol use and PrU outcomes after SCI. Salzburg *et al.*[27] found that alcohol use and cigarette smoking were highly correlated; however, they reported cigarette smoking was a more direct risk factor than drinking. A recent study found that individuals who drank more than 30 drinks per month had a greater risk of PrU compared with those who drank less than 30 drinks per month.[31] According to the limited research, no significant associations exist between alcohol use and PrU;[27,28,32] thus Gelis *et al.*[20] suggest alcohol use is not a PrU risk factor. Unlike smoking, where there is no type of use that may be beneficial, moderate alcohol consumption has been associated with beneficial effects.[33,34] However, heavy or problematic use is often associated with adverse effects, both indirect and direct, which offers a physiologic rationale for continued study of alcohol consumption in relation to PrU outcomes. For example, heavy drinking may cause anemia,[35] which is a risk factor for PrU outcomes after SCI.[27,36] Additionally, heavy alcohol consumption is linked to malnutrition,[35,37,38] which

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is a risk factor for PrU outcomes.[21] Furthermore, heavy and binge drinking has been linked to a greater risk of unintentional injury after SCI,[39,40] and such injuries may either lead to or exacerbate PrU.

The relationship between medication use and PrU outcomes has been less frequently studied. In an early, smaller study of behavioral factors associated with PrU development, the use of prescription medications to treat pain or spasticity was associated with an increased risk of PrU.[28] Later, in the first stage of a longitudinal study, which is the forerunner for this work, Krause *et al.*[3] reported on the association between prescription medication use and patterns in PrU history. In this sample, only the use of sleep medication was significantly related to recurrent PrU.[3] Data taken from the second stage of the longitudinal study found that each increase in the number of prescription medications taken (maximum of four) resulted in a 24% increase in the odds of a PrU.[5] It is possible that misuse of prescription medications may result in altered mental awareness, which may inhibit individuals from taking the actions necessary to prevent or care for PrU. It may also be the case that prescription medication use is a consequence of health conditions that may either elevate the risk of PrU or that are themselves secondary consequences of PrU.

Other research has focused on protective behaviors, including fitness, exercise, diet, hours per week in home maintenance activities, volunteer work and recreational activities. In a prelude to the current analysis using similar methods and the same overall data set, we were unable to obtain convergence in a model that included both risk and protective behaviors, leading to the distinct analysis of risk behaviors.[41] The study of risk behaviors is important for future tailoring of prevention and/or intervention strategies, which may attenuate the risk associated with these behaviors and PrU outcomes. PrU risk cannot be explained by a single factor, as it is multifactorial; thus there is a need to examine multiple risk factors in the assessment of PrU outcomes. It is also important to consider multiple PrU outcomes, rather than a single outcome such as current PrU.

Our purpose was to model the relationship between latent PrU and latent risk behaviors using structural equation modeling (SEM), accounting for multiple indicators of PrU outcomes. This analysis explicitly builds upon previously reported analysis of protective behaviors, using similar methods, data and design, but with a different set of behavioral predictors (that is, risk rather than protective behaviors).

METHODS

Study design and participants

We conducted a cross-sectional study, which was nested within a prospective cohort study initiated in 1997–1998 in a large specialty hospital in the Southeastern USA. A detailed description of the prospective cohort study has been previously published.[41] To be eligible, all participants met the following criteria: (1) traumatic SCI, (2) at least 1 year since SCI onset, (3) 18 years of age or older and (4) some residual deficits from the SCI (not complete recovery, AIS A – D). A total of 1871 eligible participants were included in this analysis.

Procedures

The study procedures and general methodology were previously described in our recently published study of protective behaviors and PrU outcomes.[41] The current analysis uses a similar statistical approach; thus we highlight only the major methodological points. Approval from the Institutional Review Board was obtained before initiating data collection. All self-report assessments were obtained by mail, with up to three mailings conducted and a follow-up phone call. Participants were offered $50 upon completion and return of the self-report assessments.

Table 1 Demographic characteristics

	N	%
Sex		
Male	1394	74.5
Female	447	25.5
Race		
White	1386	74.4
Black	406	21.8
Others	71	3.8
Marital status		
Married	770	41.5
Single	1084	58.5
Chronological age		
<40	515	27.5
40–49	491	26.2
50–59	459	24.5
60–69	300	16.0
70+	106	5.7
Years post injury		
<20	1296	69.3
20–29	402	21.5
30–39	119	6.4
40–49	41	2.2
50+	11	0.6
Injury severity		
Non-ambulatory: C1–C4	183	9.9
Non-ambulatory: C5–C8	469	25.5
Non-ambulatory: non-cervical	643	34.9
Ambulatory	547	29.7

Measures

All information were measured via composite self-report assessments. Socio-demographic characteristics included age, sex (male and female), race (White, Black and other races) and marital status (married vs others). Injury-related variables included years since SCI and injury severity (C1–4, non-ambulatory; C5–8, non-ambulatory; non-cervical, non-ambulatory; and ambulatory, any level).

Smoking and alcohol consumption were measured by self-reported questions adapted from the Behavioral Risk Factor Surveillance System (BRFSS).[42] We used four BRFSS items to measure latent alcohol consumption: (1) 'During the past month, how many days did you drink any alcoholic beverage, such as beer, wine, wine coolers, or liquor?' (2) 'A drink is 1 can or bottle of beer, 1 glass of wine, 1 can or bottle of wine cooler, 1 cocktail, or 1 shot of liquor. On the days when you drank, about how many drinks did you drink on average?' (3) 'Considering all types of alcoholic beverages, how many times during the past month did you have 5 or more drinks on one occasion?' (4) 'During the past 30 days, what are the most drinks you had on any occasion?' Participants responded to three smoking-related questions: (1) 'Have you smoked at least 100 cigarettes in your entire life?' (2) 'On the average, about how many cigarettes a day do you now smoke?' (3) 'Did you ever smoke on a regular basis (every day)?'

We developed two latent prescription medication misuse (PMM) variables. The first measured general prescription compliance. Participants responded to various questions regarding general prescription compliance, measured on a 5-point scale (never, occasionally, sometimes, often and always), including: (1) 'I stop taking a medication when I start feeling better, rather than taking them until they are gone;' (2) 'I forget to take my medication on time;'

Table 2 Correlation matrix

	1	2	3	4	5	6	7	8	9	10	11	12	13	14
1. Days drank alcoholic beverages	–													
2. Average number of drinks per occasion	0.88**	–												
3. Number of five or more drinks on one occasion	0.54**	0.62**	–											
4. Most drinks on any occasion	0.92**	0.95**	0.65**	–										
5. Smoked at least 100 cigarettes in lifetime	0.06**	0.10**	0.12**	0.09**	–									
6. Ever smoked on a regular basis	0.02	0.06**	0.08**	0.05*	0.82**	–								
7. Number of cigarettes in a day now	0.08**	0.15**	0.21**	0.14**	0.49**	0.52**	–							
8. Stop taking prescription when feel better	0.10**	0.12**	0.14**	0.12**	0.03	0.01	0.10**	–						
9. Forget to take prescription on time	0.05*	0.07**	0.10**	0.07**	0.04	0.05	0.06**	0.27**	–					
10. Skip prescription doses	0.06**	0.07**	0.09**	0.07**	0.01	0.01	0.08**	0.36**	0.47**	–				
11. Prescription for pain	−0.14**	−0.10**	−0.05*	−0.13**	0.10**	0.10	0.10**	0.03	0.12**	0.11**	–			
12. Prescription for spasticity	−0.14**	−0.12**	−0.06**	−0.13**	0.03	0.05*	0.02	−0.07**	0.02	0.01	0.28**	–		
13. Prescription for sleep	0.05*	−0.05*	−0.01	−0.06**	0.07**	0.05*	0.09**	0.06**	0.05*	0.09**	0.34**	0.20**	–	
14. Prescription for depression/stress	0.06**	−0.06**	−0.04	−0.06**	0.09**	0.08**	0.09**	0.02	0.09**	0.08**	0.28**	0.20**	0.31**	–

*$P<0.05$; **$P<0.01$.

(3) 'I skip prescribed doses of one or more of my medications.' The second latent PMM variable was related to specific prescription medication usage patterns for pain, spasticity, sleep and stress. Participants responded to the frequency of prescription usage for each health condition, measured on a 4-point scale (never, sometimes, weekly and daily).

Our main outcome, latent PrU, is identical to that used in our study on protective factors and PrU.[41] The variable contained four separate items related to current PrU status, number of PrU in the past year, number of weeks that PrU resulted in reduced sitting time in the past year and the number of times hospitalized for a PrU in the past year.

Statistical analyses

We calculated means and s.d. for all continuous variables as well as frequencies and percentages for categorical variables. In accordance with previous studies,[43] Pearson and Spearman correlations were computed to investigate correlations among smoking, alcohol consumption and PMM.

SEM was used to examine the hypothesized model based on the bi-dimensional behavioral model[44] and a replication of an earlier research factor analysis.[43] Four latent risk behavior indicators were measured by corresponding observed variables and further liked with a higher dimension which is classified as the risk behavior dimension. The latent PrU was treated as the outcome in the modeling in relation to the risk behavior dimension and also several exogenous variables including sex, age, race, marital status, years since SCI and injury severity. We used multiple adequacy of fit criteria to evaluate the model fit of the hypothesized model: χ^2, comparative fit index (CFI), Tucker Lewis index (TLI) and root mean square of approximation (RMSEA). The CFI and TLI with values of >0.95 indicate a good match between the data and the hypothesized model. RMSEA of <0.08 suggests good model fit.

SAS (Version 9.4, SAS Institute Inc, Cary, NC, USA) was used to conduct all descriptive analyses. We used M-plus (Version 7.4, Muthen & Muthen, Los Angeles, CA, USA) for all correlations and SEM. The standard of significance was set at $P<0.05$.

Statement of ethics

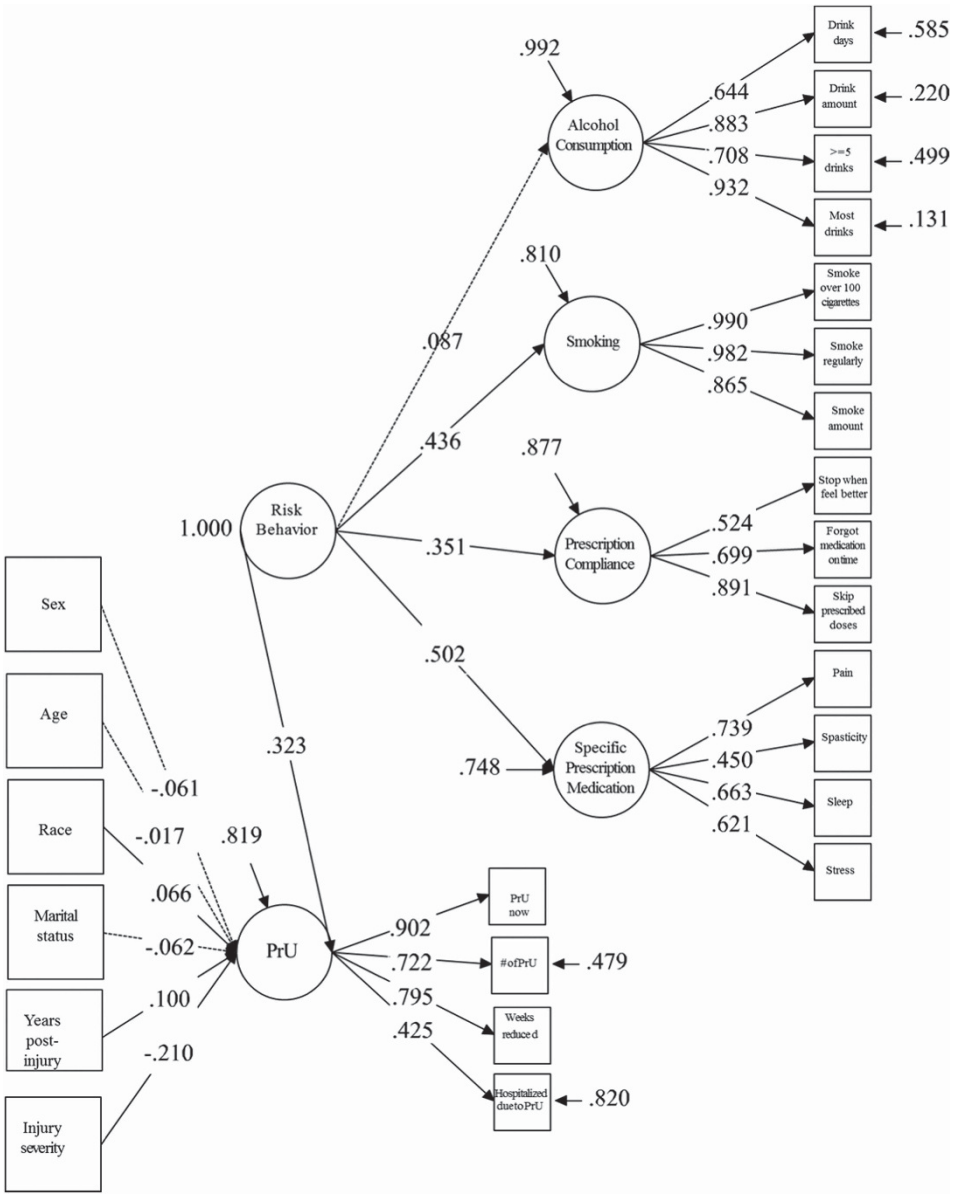
We certify that all applicable institutional and governmental regulations concerning the ethical use of human volunteer were followed during the course of this research.

RESULTS

Study participants were primarily White (74.4%) and male (74.5%); 41.5% of participants were married. The mean age was 48.3 (s.d. = 13.3) years. The average time since injury was 15.9 years (s.d. = 10.1). Thirty-five percent of participants had a non-cervical non-ambulatory injury, followed by ambulatory injury (29.7%), C5–8 non-ambulatory injury (25.5%) and C1–4 non-ambulatory injury (9.9%; Table 1). Correlations among smoking, alcohol consumption and PMM are provided in Table 2.

The model fit indices of the hypothesized SEM model were excellent: $\chi^2 = 1238.712$, DF = 232, $\chi^2/\mathrm{DF} = 5.34$, $P<0.0001$, RMSEA = 0.049, CFI = 0.975, and TLI = 0.972. The risk behavior dimension had a significant direct effect on the latent PrU (direct effect = 0.323, $P<0.01$). All direct relationships between the risk behavior dimension and risk behaviors were also significant ($r_{\text{smoking}} = 0.436$, $r_{\text{general prescription compliance}} = 0.351$ and $r_{\text{specific prescription misuse}} = 0.502$), except alcohol consumption ($r_{\text{alcohol consumption}} = 0.087$). The risk behavior dimension mediated the relationships between the latent PrU and smoking (indirect effect = 0.323*0.436 = 0.141), alcohol consumption (indirect effect = 0.323*0.087 = 0.0281), general prescription compliance (indirect effect = 0.323*0.351 = 0.113) and specific prescription use (indirect effect = 0.323*0.502 = 0.162; Figure 1).

In regard to the exogenous variables, race, years since SCI and injury severity were significantly related to latent PrU. We created dummy variables for categorical exogenous variables, which had more than two categories (Ref_{race} = White and $\text{Ref}_{\text{injury severity}}$ = ambulatory), and re-ran the SEM analyses to examine the within-group differences. The results are summarized in Table 3. According to the findings, adverse effects of PrU are more likely among non-ambulatory participants, compared with those who are ambulatory. The more severe the SCI, the worse the PrU outcomes ($r_{\text{non-ambulatory:C1–C4 vs ambulatory}} = 0.450$, $r_{\text{non-ambulatory:C5–C8 vs ambulatory}} = 0.361$, $r_{\text{non-ambulatory: non-cervical vs ambulatory}} = 0.232$). Years since SCI showed a marginal significant positive association with PrU, controlling for chronologic age (that is, greater time since SCI predicted more consequences of



Note: A solid line indicates a significant effect and a dashed line indicates a non-significant effect.

Figure 1 Structural equation model relating risk factors to the risk dimension and exogenous variables with the latent pressure ulcer dimension.

PrU). Race was significantly associated with latent PrU, with Blacks scoring higher on latent PrU compared with Whites. There were no significant relationships between the latent PrU and sex, marital status or chronologic age.

DISCUSSION

We examined the association between latent risk behaviors and latent PrU outcomes and found that latent risk behaviors had a significant adverse effect on latent PrU outcomes including the number of PrU in the past year, the number of weeks in the past year that a PrU resulted in reduced sitting time, the number of times hospitalized for a PrU and current PrU. Our findings also indicate that African Americans and individuals with a higher level of injury or longer time since SCI are more vulnerable to worse PrU outcomes. Taken together, the findings indicate the need for addressing risk behaviors, including smoking, prescription compliance and prescription medication use for pain, spasticity, sleep or depression/anxiety.

Consistent with previous studies,[3,5,26–28] this study confirmed that latent smoking and PMM were risk factors for PrU outcomes. We had the opportunity to create latent dimensions of both smoking and PMM and PrU outcomes, which allows representation of broader constructs not limited to single observable health behaviors and PrU outcomes. It has been reported that smoking could impact the endothelial vasorelaxation function and cause vasoconstriction in the vascular system, which could extend the healing process of PrU.[45–47] One possible explanation for the adverse effect of PMM on PrU outcomes could be that medication misuse may

Table 3 Path coefficients from the hypothesized structural model

Direct Effects	*Estimates*[a]	*s.e.*[b]	*Est/s.e.*[c]	*StdYX*[d]
Risk behaviors → PrU	0.701	0.340	2.059*	0.312
Sex (female) → PrU	−0.132	0.076	−1.748	−0.058
Race (Black) → PrU	0.231	0.069	3.353***	0.096
Race (other) → PrU	0.018	0.154	0.116	0.003
Marital status (married) → PrU	−0.073	0.062	−1.183	−0.036
Chronological age → PrU	−0.001	0.003	−0.337	−0.012
Years post injury → PrU	0.006	0.003	1.937	0.061
Injury severity (Non-ambulatory: C1–C4) → PrU	0.940	0.097	9.700***	0.450
Injury severity (Non-ambulatory: C5–C8) → PrU	0.823	0.101	8.154***	0.361
Injury severity (Non-ambulatory: non-cervical) → PrU	0.773	0.123	6.300***	0.232

Abbreviation: PrU, pressure ulcers.
[a]Model estimated value for each parameter.
[b]s.e. of the parameter estimates.
[c]Value of the parameter estimate divided by the s.e. (*t*-value). Values > 1.96 are statistically significant at $*P<0.05$, > 2.58 are significant at $**P<0.01$ and > 3.29 are significant at $***P<0.001$.
[d]Uses the variances of the continuous latent variables and of the background and outcome variables for standardization (standardized regression coefficient).
Note: each of the race and injury severity categories were compared with the last category.

result in altered mental awareness, which may inhibit individuals from taking the actions necessary to prevent or care for PrU. It may also be the case that PMM is a consequence of health conditions that may either elevate the risk of PrU or that are themselves secondary consequences of PrU. Although alcohol consumption has been reported to be associated with PrU by several previous studies,[27,31,35,36] our findings do not support such a relationship within the overall model.

The relationship between the latent PrU and exogenous variables was consistent with our earlier analysis of protective behaviors and PrU outcomes,[41] as well as other research. Chen *et al.*[8] reported that African Americans were at greater risk for PrU, possibly due to limited financial and educational resources.[6] Findings suggest that individuals with more severe SCI are more likely to have worse PrU outcomes, likely due to limited mobility and impaired motor and sensory function. The marginal effect of years since SCI on PrU outcomes was also reported by several studies,[3,5] showing that the skin would be more vulnerable to sustained pressure, friction or shear as a result of deterioration of skin composition and function over time.

Clinical considerations

This study supports earlier findings that smoking is a risk factor for PrU. In addition to current recommendations, we need to further promote and engage individuals in smoking cessation programs, in an effort to decrease smoking exposure and thus risk of adverse PrU outcomes. We believe it is essential to monitor the effectiveness of such programs after implementation. With regard to PMM, we believe programs to monitor prescription usage may be useful for assisting and ensuring that individuals with SCI are following the prescribed dosage and frequency. Moreover, we believe that clinicians should be mindful of the practice of prescribing medication to treat pain, spasticity, sleep and stress, and acknowledge the potential unintended effects of medication misuse.

Design considerations: strengths and limitations

There are a number of notable strengths of this study. The latent approach allowed us to aggregate multiple observable indicators to a single latent variable, which was more predictive of risk behaviors and PrU outcomes. In addition, the large sample size with extensive time since SCI allowed us to have sufficient statistical power to test our hypothesis. Several exogenous variables, such as sex, age, race, marital status, years since SCI and injury severity, were linked with the latent PrU outcomes to evaluate demographic and injury-related differences.

For limitations, first, the generalizability of our findings was limited because all study participants were recruited from one specialty hospital. Second, we used a self-report assessments to collect data from our participants, thus our findings are subject to recall bias and misreporting. However, we have no data to indicate that this necessarily occurred in this study. Third, the cross-sectional design does not allow us to draw causal conclusions, even though SEM is designed to confirm conceptual models with cross-sectional data. Fourth, our findings are restricted to risk behaviors. Protective behaviors were addressed separately in an earlier study.[41,43] Ultimately, our goal is to build larger data sets over time that are better suited for a larger number of variables. This will allow us to incorporate protective as well as risk behaviors in future studies. Using our current data and variables, we were unable to successfully develop a model that integrated both risk and protective behaviors in relation to PrU, so the development of separate models was necessary. Finally, specific locations, etiology and types of PrU are important aspects of PrU outcomes that may be related with risk behaviors. Due to data collection constraints, such relationships cannot be confirmed in this study.

Future research

Developing a risk and protective health behavior dimension, consistent with the bi-dimensional behavioral model,[24] will lead to a more comprehensive understanding of risk of PrU. Future studies are needed to expand the scope of risk and protective behaviors and PrU outcomes using a similar structural approach. We then will have a better understanding of the influence of health behaviors (both risk and protective) on PrU outcomes in terms of the theoretical risk and prevention model.[24,25]

CONCLUSION

Our study proposed a valid latent structural model of risk behaviors and PrU outcomes. This study suggests the need to reduce risk behaviors to prevent adverse PrU outcomes, particularly smoking, prescription medication use practices, and use of prescription medication to treat sleep, pain, spasticity and depression/anxiety. The risk of PrU outcomes is especially high among people who are African American, have higher level of SCI, and have a longer time since SCI.

DATA ARCHIVING

There were no data to deposit.

CONFLICT OF INTEREST

The authors declare no conflict of interest.

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