

An examination of the mechanisms of exercise-induced change in psychological well-being among people with spinal cord injury

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Abstract—For individuals with a spinal cord injury (SCI), regular exercise training leads to significant improvements in pain, stress, and depression. The mechanisms by which exercise effects change in these variables are poorly understood. To address this issue, we examined the mediated effects of exercise on the psychological well-being of individuals with SCI according to the relationships described in the Chronic Pain Process Model. Twenty-one individuals with traumatic SCI either participated in a 9-month, twice-weekly exercise program ($n = 11$), or participated as nonexercising controls ($n = 10$). Measures of pain, stress, and depression were administered at the baseline and 3, 6, and 9 months into the intervention. Change in pain mediated exercise-induced change in stress, $F_{4,17} = 7.72$, $p < 0.01$. Change in stress mediated exercise-induced change in depression, $F_{4,17} = 7.68$, $p < 0.01$. With the identification of these factors as mediators of exercise-related changes in pain and well-being, exercise interventions can be designed that specifically target these mediators and possibly maximize intervention efficacy.

Key words: depression, exercise, pain, paraplegia, quadriplegia, spinal cord injury, stress, tetraplegia.

INTRODUCTION

A traumatic spinal cord injury (SCI) causes structural damage to the spinal cord, resulting in sensory and functional loss. In addition to the disabling effects of SCI, injured individuals must also contend with many secondary impairments (e.g., urinary tract infections, pressure ulcers, and chronic pain) [1]. However, because of the catastrophic, life-threatening nature of the injury, little

appreciation is given to the extent and nature of these secondary conditions. Consequently, the comorbidities associated with SCI are poorly understood, and effective management strategies for each comorbidity require further development [1].

Pain is one such comorbidity of SCI for which there is limited clinical appreciation [2]. Reports have shown that the majority of people with SCI suffer pain symptoms, many of whom report that the pain is severe and/or disabling [3]. Of further concern, pain has been reported to affect psychological well-being and handicap patients beyond levels attributable to the SCI per se. In particular, among people with SCI, pain has been found to be associated with elevated levels of stress, anxiety, and depression [4].

Abbreviations: ADL = activity of daily living, ASIA = American Spinal Injury Association, CES-D = Centre for Epidemiological Studies Depression, PSS = Perceived Stress Scale, SCI = spinal cord injury, SF-36 = 36-Item Short-Form Health Survey, VIF = variance inflation factor.

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According to the Chronic Pain Process Model (Figure 1) [5], the negative effects of pain on psychological outcomes are the result of a cyclical process whereby physical changes attributable to injury can alter the pain threshold, leading to an exacerbation of pain symptoms. The presence of prolonged pain can subsequently cause an increase in psychophysiological stress and tension, thus producing additional distress and emotional changes. Emotional changes, in turn, can lead to a disruption in activities of daily living (ADLs). Ultimately, in a cyclical fashion, reduced levels of daily activity negatively affect physical function. In addition, it has been suggested that the cycle can work in the reverse direction. This Chronic Pain Process Model makes good conceptual sense, but it has never been tested experimentally.

One way to test and validate the model's causal predictions is to test the mediated effects of a pain reduction intervention on the model's constructs. If the model is accurate, then treatment-induced changes in one construct (e.g., stress) should mediate changes in another construct (e.g., depression). Yet, according to Baron and Kenny's recommendations, mediated relationships can only be tested when the treatment is related to both the mediator and the outcome variables [6]. Exercise represents one type of intervention that fits this criterion and would be suitable for testing the validity of the Chronic Pain Process Model in the SCI population. Exercise has been shown to affect both pain and psychological well-being in people with SCI [7–9]. For example, our own

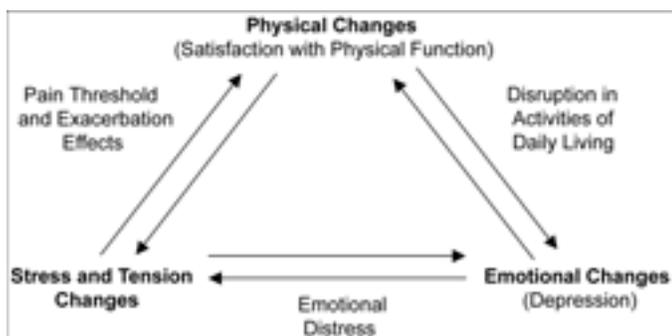


Figure 1.

Chronic Pain Process Model *Source:* Gatchel RJ. Psychological disorders and chronic pain: cause-and-effect relationship. In: Gatchel RJ, Turk DC, editors. Psychological approaches to pain management: a practitioner's handbook. New York: The Guilford Press; 1996. In our study, physical change was operationalized as satisfaction with physical function, and emotional change was operationalized as

research has shown significant improvements in pain and psychological well-being among people with SCI after only 3 months of exercise training [8]. However, the mechanisms by which exercise causes these changes are not well understood. The Chronic Pain Process Model may provide a framework for understanding the psychological mechanisms by which exercise elicits such changes. A better understanding of these mechanisms would be of value to both researchers and practitioners concerned with developing interventions to improve the psychological well-being of people with SCI.

Accordingly, our study examined the effects of exercise on psychological well-being as depicted in the Chronic Pain Process Model. In accordance with the model, we hypothesized that (1) exercise-induced changes in perceived pain would mediate the effects of exercise on stress and (2) exercise-induced changes in stress would mediate the effects of exercise on emotional well-being (operationalized as depression). In addition, when testing the second hypothesis, we also examined the concurrent, mediating role of pain. Although this relationship is not wholly consistent with the Chronic Pain Process Model, accumulating evidence indicates a strong relationship between pain and emotional well-being, which might suggest that pain and stress be examined as concurrent mediators [3,4].* However, given the exploratory nature of testing this doubly mediated relationship, we advanced no specific hypotheses.

Also notable is that, despite the model's suggestion that the relationships between variables operate bidirectionally, we elected to test the relationships in a single direction only. Lazarus' Cognitive-Motivational-Relational Theory of emotion indicates that stress mediates the effects of an individual's perceptions (e.g., perceptions of pain) on emotion [10,11]. Consistent with this suggestion that stress is an antecedent of emotion, we allowed the direction of the relationships examined in this study to be dictated by Lazarus' well-validated theory.

In addition to the relationships tested in the current study, the Chronic Pain Process Model describes other reciprocal interrelations between various constructs (Figure 1). However, when a model has not been rigorously tested, one should first establish the individual relationships between the proposed constructs rather than attempt to validate all the model's proposed

*We acknowledge an anonymous reviewer for suggesting this analysis.

relationships concurrently. Testing only specific aspects of a model is not uncommon. For example, tests of Bandura's seminal Social Cognitive Theory are traditionally conducted only on the component parts of the theory (e.g., self-efficacy, impediments) [12] and not on the theory in its entirety [13]. The advantages of testing isolated relationships in a model are that it requires a smaller sample size than testing an entire model [14], and that it takes a progressive, systematic "bottom-up" approach to validating the model. Once each relationship within the model has been empirically supported, the entire model can be examined. This approach creates a solid foundation for testing and explaining the entire model. In contrast, a "top-down" approach tests the entire model from the outset, rather than one relationship at a time. If the model is not supported in this initial test, researchers are forced to either dismiss the model, despite the value of certain relationships within the model, or backtrack and examine isolated relationships within the model (the initial step in a "bottom-up" approach) [15]. Thus, determining that the relationships described in the Chronic Pain Process Model hold up when tested individually provides a basis for future, large sample tests of the model in its entirety.

METHODS

Participants

This investigation included data collected from participants who were involved in a 9-month randomized controlled trial assessing the physiological and psychological benefits of exercise for individuals with SCI [16]. Contraindications for participation and recruitment strategies are outlined elsewhere [16]. In general, 34 sedentary

individuals with traumatic SCI at least 1 yr postinjury were enrolled in the study. The retention rate was 52 percent in the exercise group and 92 percent in the control group. Additionally, two participants in the control group had incomplete data sets and were excluded from this study. A multivariate analysis of variance revealed no difference in baseline scores on the outcome variables between individuals who adhered to their assigned group condition and individuals who dropped out ($p > 0.05$; refer to Hicks et al. [16] for details of program dropout). In sum, for the current investigation, complete data sets were available for 21 participants (aged 19–65 yr) with traumatic SCI for longer than 1 yr (1–24 yr postinjury). Characteristics of participants who completed the study are summarized in **Tables 1** and **2**.

Participant Description Measures

Degree of impairment was determined with the American Spinal Injury Association (ASIA) Neurologic and Functional Classification of SCI Standards [17]. Based on the results of an extensive neurological assessment, individuals' level of impairment was graded on a 5-point scale, A to E. Classification indicated completeness of the injury (e.g., A = complete, B to D = incomplete, E = normal) and motor and sensory function below the level of the lesion (A = no motor or sensory function is preserved in the sacral segments; B = sensory function but no motor function is preserved; C = motor function is preserved with the majority of key muscles capable of active movement with gravity eliminated, at most; D = motor function preserved with majority of key muscles capable of active movement against gravity, at minimum). The scale is widely used as a standard measure of impairment [17].

Table 1.
Participant demographic data.

Group	ASIA Classification				Level		Gender		Age	Yr Postinjury
	A	B	C	D	Cervical	Thoracic	Male	Female	(Mean \pm SD)	(Mean \pm SD)
Exercise ($n = 11$)	5	2	3	1	7	4	3	8	38.27 \pm 10.04	10.54 \pm 7.49
Control ($n = 10$)	3	1	3	3	3	7	2	8	43.08 \pm 11.04	14.58 \pm 14.31

Note: A multivariate analysis of variance revealed no between-group differences in either age or years postinjury. Fisher's exact tests revealed no between-group differences in either gender or level of injury (all $p > 0.05$). Between-group differences in ASIA impairment could not be assessed with (1) Fisher's exact test because a 2 (group) \times 4 (impairment) design was used or with (2) chi-square analysis because of the small sample size (<5) in many of the data cells.

ASIA = American Spinal Injury Association

SD = standard deviation

Table 2.

Between-group comparison of baseline scores for mediating and outcome variables.

Group	Pain Score (Mean \pm SD)	Stress Score (Mean \pm SD)	Depression Score (Mean \pm SD)
Exercise ($n = 11$)	5.09 \pm 2.02	36.73 \pm 12.53	10.45 \pm 8.30
Control ($n = 10$)	5.92 \pm 2.38	41.42 \pm 11.02	12.42 \pm 8.04

Note: No significant between-group differences were observed. SD = standard deviation

Intervention Outcomes

Using the two-item pain subscale from the 36-Item Short-Form Health Survey (SF-36) [18], participants rated how much pain they experienced and how much pain interfered with normal work in the last 4 weeks on a 6-point scale, (1 = none/not at all, 6 = very severe/extremely). The SF-36 pain subscale has been shown to be a reliable and valid measure of pain in a variety of patient populations (e.g., people with neuropathic pain [19] and individuals with spinal disorders [20]). Although more comprehensive measures of pain are available, the SF-36 pain subscale has been shown to be sensitive to exercise-related change in other clinical populations (e.g., patients with arterial claudication [21] and people with clinical back pain [22]) and so we considered it appropriate for use in our study.

Psychological Well-Being

Stress

Using the 14-item Perceived Stress Scale (PSS) [23], participants indicated how frequently they encountered stressful life experiences over the past 4 weeks. Items were rated on a 6-point frequency scale ranging from 1 (all the time) to 6 (none of the time). The reliability and validity of the PSS has been shown [23]. The internal consistency of the PSS was adequate at all measurement points, as indicated by Cronbach's alpha coefficients (>0.70) [24].

Depression

Using the Centre for Epidemiological Studies Depression (CES-D) Scale [25], participants indicated how often, over the past week, they experienced each of the 20 symptoms described in the CES-D Scale. Responses were made on a 4-point scale ranging from 0 (rarely or none of the time) to 3 (most of or all the time). Scores on the scale can range from 0 to 60. Scores of 16 or higher are generally considered to indicate an increased risk of experiencing clinical depression [26].

The CES-D has demonstrated a high degree of reliability and validity [25]. For the current study, adequate internal consistency was demonstrated ($\alpha > 0.70$) [24].

Procedure

The human research review board at the participating institution approved the study, and all participants gave written informed consent to be in the study. Before randomization, all participants completed the experimental measures described. A research assistant presented the questionnaire items verbally to each participant and recorded the responses. An interview format was used because not all the participants were able to use pen and paper independently. Additionally, during this initial testing session, physiological parameters were assessed for a concurrent study. These physiological variables are reported elsewhere [16].

All pretest measures were repeated at approximately the 3-, 6-, and 9-month points of the intervention. To ensure that all exercisers had the same amount of training, we used an absolute time measure to schedule testing sessions. That is, each participant completed 22 to 24 training sessions between test points.

Intervention

In the control condition, participants were instructed to continue with their normal activities and were requested to refrain from starting a regular exercise routine for the duration of the study. To provide control participants with the social opportunities and attention afforded to participants in the exercise condition, we invited them to attend monthly hour-long education sessions in which health issues relevant to managing the symptoms of SCI were discussed (e.g., proper nutrition). However, after three education sessions, this service was discontinued as a result of poor attendance by the control participants. Upon completion of the study, these participants were invited to join a twice-weekly exercise

training program for people with SCI that was virtually identical to the experimental program.

In the exercise condition, participants trained twice weekly for 9 months at the McMaster Centre for Health Promotion and Rehabilitation. Throughout the training period, participants exercised in small groups (i.e., 3 to 5 people). At each exercise session, able-bodied volunteers provided exercise assistance and safety tips and supervised the participants. Training sessions included a 5 min stretching phase, 15 to 30 min of aerobic arm ergometry exercise, and 45 to 60 min of resistance exercise. The aerobic and resistance training workloads were progressively increased throughout the program. Further details of the training protocol are outlined elsewhere [16].

Statistical Analyses

All analyses were evaluated with a priori contrasts employing per comparison error rates ($p < 0.05$) [27]. Per Baron and Kenny's recommendations [6], to test for mediation, we established the four conditions represented in **Figure 2** (Paths A, B, C, and D) through prospective hierarchical linear regression analyses (i.e., the 0- to 6-month percent change score for the mediator variable was used to predict the 0- to 9-month percent change in the outcome variable). If the first three conditions were met (Paths A to C), we conducted a fourth regression in which group was

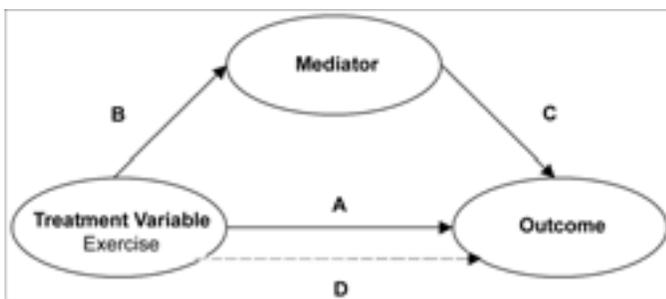


Figure 2.

Conceptual model used for mediational analyses. We established four conditions represented (Paths A, B, C, and D) through prospective hierarchical linear regression analyses (i.e., 0- to 6-month percent change score for mediator variable was used to predict 0- to 9-month percent change in outcome variable). If first three conditions were met (Paths A to C), we conducted fourth regression in which group was entered after controlling for mediator. Perfect mediation occurred if, once mediated variable was controlled, exercise no longer had effect on outcome variable (i.e., Path D was not significant). However, if, in final regression model, exercise had effect on outcome but effect was smaller than when mediator was unaccounted for, partial mediation occurred. Dashed line indicates nonsignificant relationship after controlling for Path C.

entered after controlling for the mediator. Perfect mediation occurred if, once the mediated variable was controlled, exercise no longer had an effect on the outcome variable (i.e., Path D was not significant). However, if, in the final regression model, exercise had an effect on the outcome but the effect was smaller than when the mediator was unaccounted for, partial mediation occurred.

Before conducting these regression analyses, we calculated the variance inflation factor (VIF) for each independent variable and the equivalent statistic for the model ($VIF_{\text{model}} = 1/[1 - R^2 \text{ model}]$) to determine whether multicollinearity adversely influenced the stability of the R^2 estimates. Per the guidelines of Freund and Wilson [28], we found no evidence of strong multicollinearity because none of the VIF values ($VIF < 1.9$) exceeded the equivalent statistic for the corresponding model ($VIF_{\text{model}} > 2.8$).

RESULTS

Perceived Pain as a Mediator of Stress

As indicated in **Table 3**, the three preliminary steps for testing for mediation were satisfied (Paths A, $\Delta R^2 = 0.21$, $p < 0.05$; Path B, $\Delta R^2 = 0.28$, $p < 0.01$; Path C, $\Delta R^2 = 0.11$, $p < 0.01$). As can be seen in **Table 3**, after controlling for pain, we found that group no longer had an effect on stress ($\Delta R^2 = 0.08$, $p = 0.06$). Thus, all four conditions for mediation held, indicating that, as postulated in hypothesis 1, the effects of exercise on stress were mediated by exercise-induced change in perceived pain.

Stress and Perceived Pain as a Mediator of Depression

Separate hierarchical regression analyses controlling for baseline values revealed that Path A ($\Delta R^2 = 0.19$, $p < 0.05$) and Path B for perceived pain ($\Delta R^2 = 0.28$, $p < 0.01$) and stress ($\Delta R^2 = 0.23$, $p < 0.01$) were significant. Preliminary analyses of Path C revealed that depression was predicted by stress ($\beta = 0.49$, $p = 0.02$) but not by perceived pain ($\beta = 0.29$, $p = 0.25$). Thus, perceived pain was removed from the model, and only stress was retested as a mediator. As described in **Table 4**, adjusted stress scores explained a significant amount of variance in depression, thus satisfying the conditions for Path C ($\Delta R^2 = 0.43$, $p < 0.05$). In the last step required to test the model, the final variable (group) contributed no additional variance to the regression model ($\Delta R^2 = 0.00$, $p = 0.66$). Thus, Path D was not significant, and the conditions for mediation were

Table 3.

Linear models testing 0- to 6-month percent change in perceived pain as mediator of 0- to 9-month percent change in stress.

Predictor Variables	Outcome Variables	ΔR^2 Change	Adjusted ΔR^2	β	<i>t</i> for Parameter for β
Path A	0- to 9-month stress				
Baseline stress		0.32*	—	-0.67	-4.25*
Group		0.21*	0.49*	0.47	3.01†
Path B	0- to 6-month pain				
Baseline pain		0.15	—	-0.52	-3.03†
Group		0.28*	0.37*	0.54	3.04*
Path C	0- to 9-month stress				
Baseline stress		0.32*	—	-0.83	-4.52*
Baseline pain		0.13†	—	0.59	2.99†
0- to 6-month pain		0.11†	0.49*	0.36	2.12†
Path D	0- to 9-month stress				
Baseline stress		0.32*	—	-0.85	-4.99*
Baseline pain		0.13†	—	0.43	2.13†
0- to 6-month pain		0.11†	—	0.15	0.76
Group		0.08	0.56*	0.36	1.98

Note: Four separate regression models (Paths A–D) were calculated in accordance with Baron and Kenny's steps for testing for mediation. *Source:* Baron RM, Kenny DA. The moderator-mediator variable distinction in social psychological research. *J Pers Soc Psychol.* 1986;51:1173–82. * $p < 0.01$, † $p < 0.05$.

Table 4.

Linear models testing 0- to 6-month percent change in stress as mediator of 0- to 9-month percent change in depression.

Predictor Variables	Outcome Variables	ΔR^2 Change	Adjusted ΔR^2	β	<i>t</i> for Parameter for β
Path A	0-to 9-month depression				
Baseline depression		0.15	—	-0.44	-2.43*
Group		0.19*	0.28*	0.44	2.41*
Path B	0- to 6-month stress				
Baseline stress		0.25*	—	-0.62	-3.76†
Group		0.23†	0.43†	0.50	3.03*
Path C	0-to 9-month depression				
Baseline depression		0.15	—	-0.63	-3.28†
Baseline stress		0.06	—	0.72	3.45†
0- to 6-month stress		0.43†	0.58†	0.75	4.62†
Path D	0-to 9-month depression				
Baseline depression		0.15	—	-0.63	-3.21*
Baseline stress		0.06	—	0.68	2.90*
0- to 6-month stress		0.43†	—	0.70	3.50†
Group		<0.01	0.56†	0.08	0.46

Note: Four separate regression models (Paths A–D) were calculated in accordance with Baron and Kenny's steps for testing for mediation. *Source:* Baron RM, Kenny DA. The moderator-mediator variable distinction in social psychological research. *J Pers Soc Psychol.* 1986;51:1173–82. * $p < 0.05$, † $p < 0.01$.

satisfied. This finding supported our second hypothesis—that change in stress would mediate the effects of exercise on depression. Perceived pain, however, did not mediate the effects of exercise on depression, which suggests that exercise exerts its influence on depression through stress, independent of pain.

DISCUSSION

Our study examined the effects of exercise on the psychological well-being of people with SCI according to the relationships depicted in the Chronic Pain Process Model. We examined the effects of exercise prospectively in a series of hierarchical linear regression analyses such that 0- to 6-month change in the mediator variable was used to predict 0- to 9-month change in the outcome variable. Consistent with the model, change in perceived pain mediated change in stress, and change in stress mediated change in depression. Furthermore, exploratory analyses revealed that perceived pain did not mediate the effects of exercise on depression. Each of these findings will be discussed in turn.

Congruent with the Chronic Pain Process Model and findings in other chronic pain populations (e.g., knee osteoarthritis) [29], the first set of regressions indicated that change in pain mediated the effects of exercise on stress. This mediated relationship might be the result of the prophylactic effects of exercise on pain. In the current study, the exercises were designed to train muscle groups in the upper body, which are frequently subject to overuse injuries and pain among individuals with SCI [30]. For participants in the intervention group, perhaps progressively training these injury-prone muscle groups prevented the exacerbation of pain symptoms, which, in turn, reduced a major source of stress [4]. For participants in the control group, failure to train these muscle groups may have increased the likelihood of incurring strain and injury over the course of the 9-month trial that ultimately enhanced stress. Further research examining site-specific sources of pain is needed to test this hypothesis.

In accordance with the Chronic Pain Process Model and Lazarus' Cognitive-Motivational-Relational Theory of emotion [10,11], the second set of regressions revealed that change in stress mediated exercise-induced change in depression. This mediated relationship might be due to changes in individuals' appraisal of stressful events resulting from changes in their physical ability to per-

form ADLs. For individuals with SCI, many ADLs are physical in nature (e.g., transferring, wheeling, eating) [31]. As a result, individuals who lack the physical capacity to perform basic ADLs may appraise these tasks as stressful because of feelings of helplessness and an inability to cope with the demands of daily living. Such feelings can bring about depressive symptoms [10,11]. As reported by Hicks and colleagues [16], the participants in the intervention condition exhibited significant increases in physical strength and endurance that may have increased perceptions of ability to manage ADLs, thus reducing stress and depressive symptoms. Further research is needed to examine the effects of exercise participation on perceptions of physical helplessness and ability to manage stressors of daily living.

Additionally, this second set of analyses indicated that pain was not a significant independent predictor of depression and thus did not satisfy the criteria for mediation. This null finding further supports the Chronic Pain Process Model in suggesting that pain does not directly affect emotional well-being, but only has an indirect effect on depression via its impact on stress. Unfortunately, the statistical procedures used in this investigation were selected to accommodate the small sample size, which precluded the examination of the exercise-pain-stress-depression relationship. In the future, a path analysis with a large sample of individuals with SCI should be conducted to examine whether exercise leads to a change in pain that in turn leads to a change in stress and ultimately leads to a change in depression.

Collectively, these findings have important therapeutic and theoretical implications. From a therapeutic perspective, through the identification of the particular aspects of an exercise intervention that cause change in pain and psychological well-being (i.e., mediators), health professionals are provided with an indication of the essential intervention components necessary to maximize intervention effectiveness. Further, with the development of interventions that target change in mediating variables, a shorter intervention length is needed to effect change [32].

The findings from this intervention study also contribute initial empirical support for the relationships between psychosocial variables as described in the Chronic Pain Process Model and further our understanding of the effects of exercise on these relationships. This very preliminary support can be viewed as a gateway for further empirical examinations of the model. For example, we

recommend that these two relationships among psychosocial variables be tested (1) in other chronic pain populations and (2) with other forms of exercise different from those used in our study. Such tests would further validate the relationships described in the model and increase its generalizability.

Furthermore, an examination should be undertaken to test the additional relationships specified by the Chronic Pain Process Model that were not tested in our study. For example, it remains to be determined whether exercise-induced changes in pain are mediated by physical changes (e.g., change in muscle strength and endurance, aerobic fitness) associated with exercise participation. Establishing the additional relationships among Chronic Pain Process Model variables will serve as a basis for testing the model in its entirety with large-sample, confirmatory statistical procedures (e.g., structural equation modeling).

As one of the first randomized controlled trials to examine the effects of exercise on psychological well-being in individuals with SCI, our study results highlight many of the benefits of exercise and provide guidance for the development of exercise interventions. However, given the small sample of adherent participants remaining at the conclusion of the intervention, we caution readers in generalizing these findings to the larger SCI population. A similar study with a larger sample should be conducted to ensure that the findings are reproducible in a larger group. Conducting such a large-scale, long-term study would be truly challenging because program dropout due to SCI-related illness and lack of time are frequent concerns in this population [16]. Researchers should consider this susceptibility to nonadherence and ensure that an ample size sample is recruited at the onset of any study. As well, we recommend the use of intention to treat analysis (collecting data from all participants including dropouts at all assessment points) to provide strong evidence of the effectiveness of the exercise intervention.

A second limitation of this study was the length of the interval between assessment periods. Evidently, from the results of our study, the time-lagged design was an effective method for examining the effect of change in the mediator variable for predicting change in the outcome variable. However, the effects might have been stronger had the time between assessment intervals been less than 3 months. With evidence indicating that exercise can induce changes in psychological well-being in less than 3 months [33], perhaps changes were occurring

that were not captured by the 3-month assessments. Shortening the interval between assessment periods might allow for more accurate tracking of change and, in turn, more accurately indicate the relationships between variables.

CONCLUSION

In conclusion, our test use of the Chronic Pain Process Model has furthered our knowledge of the effects of exercise on the relationships among psychosocial variables. From a therapeutic perspective, these findings provide evidence as to why individuals with SCI should adopt a regular exercise regime and provide health professionals with direction for the development of efficacious, exercise-centered pain management programs. Additionally, in providing preliminary validation of the relationships within the Chronic Pain Process Model, these findings give direction for future theory-driven research in the exercise domain. With such promising evidence of the many psychological benefits of exercise for individuals with SCI, we hope that this study is only the first of many exercise interventions used to relieve the symptoms of a disabling comorbidity of SCI—pain.

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