Original article

Physical activity is indirectly associated with pain in college women through associations with somatization and panic disorder symptoms: a cross-sectional study

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ABSTRACT

Purpose: This study tested whether the severity of somatization, panic, generalized anxiety, and depression symptoms mediated relationships between physical activity and pain using structural equation modeling.
Methods: College women ($n = 1036$; mean = SD age of 19.7 ± 3.0 years) reported past week physical activity and the presence of persistent pain (≥ 1 month). The Psychiatric Diagnostic Screening Questionnaire assessed hypothesized mediators of physical activity effects on pain via symptoms of generalized anxiety, panic, major depressive disorder, and somatization disorder.
Results: The percentage of the sample screening positive was 15.7% for generalized anxiety, 5.8% for panic, 11.0% for major depressive, and 16.0% for somatization. The hypothesized model had good fit and accounted for a significant amount of variance in pain (9.1%). Direct paths from physical activity to somatization, generalized anxiety, major depressive disorder, and panic disorder were significant. Paths to pain were significant from somatization and panic disorder. The path from physical activity to pain was not direct but indirect and through significant paths to pain from somatization and panic disorder. Each standard deviation increase in physical activity decreased the probability of pain by 11.0% through panic disorder and 10.7% through somatization disorder.
Conclusions: Physical activity is associated with pain indirectly through associations with panic disorder and somatization disorder symptoms in college women.

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Introduction

Pain and psychiatric disorders are common. More than one in three adults in the United States are burdened by persistent pain [1] and the 12-month prevalence of generalized anxiety disorder (GAD), panic disorder (PD), and major depressive disorder (MDD) in adults in the United States has been estimated as 2.9%, 3.1%, and 7.7%, respectively [2]. GAD, PD, MDD, and somatization disorder (SD) often co-occur with pain [3–6]. For example, in a representative sample of adults in the United States and compared with those free of pain, a significantly higher percentage of those

reporting pain related to a chronic medical condition had a past-year diagnosis of MDD (20.2% vs. 9.3%), GAD (7.3% vs. 2.6%), and PD (6.5% vs. 2.6%) [7]. The severity of anxiety and depressive symptoms also is positively associated with the severity of pain symptoms in population-based samples [8].

Pain, anxiety, and depressive disorders are leading causes of disability [9], especially in young women [10]. Pain and psychiatric symptoms appear to be exacerbated by inadequate physical activity [11–13]. About 30% of women in the United States engage in no leisure-time moderate or vigorous physical activity, and inadequate physical activity contributes to increases in obesity [14]. Population-based studies show that physical inactivity is associated with chronic widespread pain [15] and elevated anxiety and depressive symptoms [16]. Adults in pain who adopt regular physical activity show moderate pain relief in randomized controlled trials [17]. Adults with anxiety or depressive disorders [18–20], and healthy adults without elevated psychiatric symptoms [21], show reductions in anxiety and

depressive symptoms in response to exercise training experiments. Clinical guidelines recommend mild-to-moderate physical activity as an evidence-based first- or second-step treatment for depressed adults [22] and as an adjunct for PD [23].

Whether associations between physical activity and pain are mediated by psychiatric symptoms is understudied. We are unaware of any studies examining relationships between physical activity and SD. One small randomized trial found that 6 week of resistance exercise training improved both pain intensity and anxiety symptoms in patients with GAD, but no mediation analysis was presented [24]. In one study of ~179,000 adults living in low- and middle-income countries, low physical activity among individuals with MDD was partially mediated by pain [25]. Separate literatures link physical activity to PD and PD to pain. Cross-sectional [26] and prospective cohort [27] studies support that physical activity is associated with a reduced risk of PD. Randomized controlled trials show that symptoms of PD are reduced after the adoption of a regular physical activity program [28] and a single bout of exercise [29]. Most of the research regarding PD and pain has focused on cardiac pain [30], but evidence also links PD symptoms to laboratory-induced pain [31] and chronic pain from noncardiac clinical conditions [32,33].

The purpose of this study was to examine, as a secondary data analysis, cross-sectional relations of physical activity, pain, and pain-related psychiatric symptoms in a cohort of college women. Compared with young men, college-aged women are at increased risk of reporting pain [34] as well as anxiety and depressive symptoms [10]. We hypothesized that the association between self-reported physical activity and the presence or absence of pain lasting more than one month would be partially or fully indirect, operating through direct associations with symptoms of MDD [3], GAD [35], PD [36], and/or SD [37], psychiatric conditions known to be associated with an increased risk of pain. Because obesity is related positively with anxiety, depression and somatic symptoms [38,39], and inversely with physical activity, we also examined the potential influence of obesity as a confounder of the relationship between physical activity and pain.

Methods

Participants

Recruitment efforts, made in English, occurred at one large public University in the southeastern United States using flyers placed on campus bulletin boards, solicitations sent via LISTSERV, and electronic or in-person announcements made to large lecture classes (e.g., art history, biology, psychology). The recruitment sought women aged 18–39 years who had at least one of the following GAD-related symptoms: fatigue, sleep problems, difficulty concentrating, irritability, restlessness, muscle tension, or worry. The recruitment indicated an incentive of up to $80 and that the study may involve 6 weeks of supervised exercise should the individual be eligible. Participants were 1036 female students enrolled at large university in the southeastern United States. The sample had a racial/ethnic distribution of approximately 78% white, 9% Asian, 8% black or African American, 3% Hispanic/Latino, and 2% multiracial/other which mirrored that of the 34,885 person student body (about 77%, 8%, 8%, 3%, and 4%, respectively). Descriptive statistics for other study variables are provided in Table 1.

Procedures

Female students were recruited to participate, and interested individuals were directed to a website, www.surveymonkey.com, which contained study details, the informed consent and surveys about height, weight, habitual physical activity, pain, psychiatric symptoms, and contraceptive and psychoactive medication use. A total of 1044 signed the informed consent and completed screening surveys between August 2009 and April 2010, when the exercise training enrollment target was reached. Eight screening respondents were excluded because of missing data or sex was reported as male. The exercise training study and cross-sectional associations from the screening data focused on self-esteem have been reported elsewhere [18,40]. This study is a cross-sectional secondary analysis of the screening data. All research procedures were conducted in accordance with the Code of Ethics of the World Medical Association and after obtaining approval from the institutional review board.

Measures

Physical activity

Physical activity was measured using the 7-day Physical Activity Recall (7dPAR). The 7dPAR provides an estimate of usual physical activity by assessing multiple days of physical activity in a single reporting session and expressing it in metabolic equivalents (METs). One MET is defined as 1 kcal·kg body weight·hour and is approximately equal to basal metabolic rate. Thus, physical activity

Table 1
Descriptives for participant characteristics, physical activity, and psychiatric screening data

Participant characteristics	Entire sample		Pain > 30 d (11.4% of sample)		Pain absent or ≤ 30 d (88.6% of sample)		Group difference significance
	Mean/%	SD	Mean/%	SD	Mean/%	SD	
Age (y)	19.7	3.0	19.8	3.1	19.6	3.0	NS
Height (cm)	165.1	7.3	165.0	7.6	165.1	7.1	NS
Weight (kg)	61.6	10.8	62.4	11.7	61.4	10.6	NS
BMI (kg/m²)	22.6	3.7	22.9	3.9	22.5	3.7	NS
Contraceptive med. use (%)	42.1	—	40.5	—	42.4	—	NS
Psychoactive med. use (%)	15.9	—	24.8	—	14.9	—	<.01
Exercise bouts (past month)	11.6	7.0	11.0	7.2	11.7	7.0	NS
Physical activity (kcal/wk)	270.4	39.1	268.9	36.7	270.5	39.3	NS
Physical activity (MET h/wk)	44.5	38.9	43.6	38.4	44.8	39.0	NS
Psychiatric diagnostic screening questionnaire (% screening positive)	% of entire sample		% of pain present sample		% of pain absent sample		
Somatization disorder	16.0		35.9		13.5		<.001
Generalized anxiety disorder	15.7		29.1		14.0		<.001
Major depressive disorder	11.0		22.2		9.7		<.001
Panic disorder	5.8		17.1		4.4		<.001

level is expressed in multiples of basal metabolic rate, where 10 METs is 10 × basal metabolic rate. Respondents estimated the number of hours spent during an average weekday and weekend day engaged in moderate, hard, and very hard activities, which were then converted into hours of total METs (MET·hours·week^{-1}) of moderate (estimated as 4 METs), hard (6 METs), and very hard (10 METs) types of physical activities. Those who reported 600 or more MET·minutes·week^{-1} were physically active enough to meet current health recommendations. Evidence supports the validity of the 7dPAR for use with college students [41].

Pain

Participants responded (yes or no) to the question "Do you have any pain you have been experiencing for more than one month?" Single-item pain measures are widely used and those with a recall time frame of one month are appropriate for epidemiological studies of pain [42,43]. Single-item pain scales are well established as a valid approach for measuring pain, including the presence of pain [44]. A body of research supports the reliability and validity of the general approach to pain measurement used in the present study (i.e., self-reported absence or presence of pain) [45]. There is no consensus optimal time interval for assessing self-reported outcomes [46], and there are advantages and disadvantages to shorter and longer recall intervals about pain. For example, the one-month time period used here may be less biased than widely used longer time periods (e.g., 6 months) but less effective at differentiating people with recurrent pain that periodically abates from those with more enduring chronic pain [47]. A single item was used to minimize participant burden and attrition, resulting from a failure to complete a long screening survey.

Psychiatric symptoms

Symptoms were measured using the Psychiatric Diagnostic Screening Questionnaire (PDSQ). The PDSQ asks binary yes or no questions. Affirmative answers are summed, and cut scores can be used to screen for the most common Diagnostic and Statistical Manual (DSM-IV) axis psychiatric disorders. Evidence primarily from psychiatric outpatients supports the reliability and validity of the PDSQ scales [48]. Our analysis focused on the items used to screen for the probable presence of four psychiatric conditions that have been associated with pain and were common enough in our sample to permit an adequate analysis. The conditions are MDD (21 items, past 2-week time frame, example item—"did you feel sad or depressed?"), GAD (10 items, past 6-month time frame, example item—"were you a nervous person on most days?"), PD (8 items, past 2-week time frame, example item—"did you worry a lot about having unexpected anxiety attacks?"), and SD (5 items, past 6-month time frame, example item—"are your doctors usually not able to find a physical cause for your physical symptoms?"). The PDSQ has not been widely used among college students and norms for young adults are unavailable. Here, we tested the factor validity of the scales using confirmatory factor analysis of the four correlated, single-factor scales for MDD (using 21 items), GAD (10 items), PD (8 items), and SD (5 items).

Covariates

Self-reported height and weight were used to calculate body mass index (BMI), a measure of excess weight for height (kg/m^2) commonly used as an estimate of body fatness. Self-reported current use or nonuse of contraceptives was included as a covariate because the use of these medications has been associated with weight gain [49]. Use of psychoactive medications was used as a covariate because these medications have been associated with reductions in pain, psychiatric symptom severity, and physical activity [50–52]. In response to questions about medication use, the participants reported yes they used or no they did not use, and in the analysis reported nonuse was coded as 0 and use was coded as 1.

Statistical analysis

Confirmatory factor analysis of PDSQ scales and structural equation models were tested in M*plus* 8.0 [53], using weighted least squares means and variance adjusted estimation, which gives imputed parameter estimates with standard errors and a χ^2 test statistic that are robust to non-normality of observations using categorical variables. To simplify complexity (5–21 binary item-indicators per scale) in the structural model, PDSQ-summated scores were modeled as observed variables after adjustment for measurement error, using standard latent procedures [54]. The factor loading representing the association from the latent variable to the observed score was fixed at the square root of the measure's reliability (i.e., internal consistency) and the error/uniqueness term was fixed at 1 minus the reliability times the variance of the observed score. Effects were tested as fully standardized probit regression coefficients with asymmetrical confidence intervals for indirect effects estimated by bias corrected bootstrapping (2000 samples) [55] and also by Bayesian estimation (10,000 iterations) [56].

Overall, 1.6% of the data were missing (913 of 56,980 total responses). The missing responses were 1.5% for 7dPAR, 0.5% for PDSQ, and 0.5% for BMI. Covariances could be computed for at least 98% of all observed variables for the measurement model and at least 89% of variables in the structural models. Descriptive statistics were computed using IBM SPSS 23.0.

Model fit

The χ^2 statistic, comparative fit index (CFI), Tucker-Lewis Index (TLI), root mean square error of approximation (RMSEA), and the weighted root mean square residual (WRMR) were used to evaluate model fit. The χ^2 statistic assesses absolute fit of the model and usually suggests rejection of the hypothesized model in samples with 400 cases or more. Hence, it is not used alone to draw conclusions about model fit. The CFI and TLI test proportionate improvement in fit by comparing the target model to a baseline model (values of .90 and .95 are acceptable and good, respectively). Unlike the CFI, the TLI is affected by model parsimony (more complex models are penalized). The RMSEA represents closeness of fit of population data to the model and is one of the most informative fit criteria. Values ≤ 0.06 represent close fit. The WRMR represents the weighted average error between the observed and specified covariances and is particularly sensitive to mis-specified factor loadings. Values less than 0.90 indicate good fit [57].

The structural model tested the mediating effects of psychiatric symptoms on the relationship between physical activity and pain. Physical activity is reported here by a fully saturated latent factor indicated by the three intensity components of total MET hours/week: moderate (β = 0.292, SE = 0.046), hard (β = 0.523, SE = 0.056), and very hard (β = 0.560, SE = 0.062), which was inversely correlated with BMI (β = −0.112, SE = 0.043). Thus, the hard and very hard components of physical activity were more influential in the latent physical activity scores than the moderate physical activity component. Seven percent of the participants reported no leisure-time physical activity; so, square root transformations of moderate, hard, and very hard scores were used for analysis. Physical activity was also expressed as a categorical variable, according to whether public health recommendations [58] for sufficient weekly physical activity were met (600 met-minutes or more; 83%) or not met (17%).
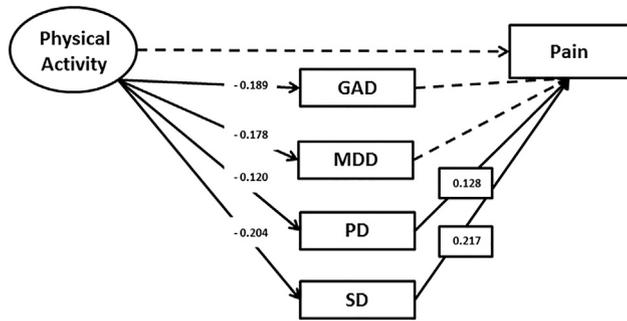
Fig. 1. Structural model of hypothesized associations among latent physical activity, pain of more than one month, and symptoms of generalized anxiety disorder (GAD), major depressive disorder (MDD), panic disorder (PD), and somatization disorder (SD). Numerical values represent fully standardized regression coefficients. Broken lines indicate structural paths with critical values (i.e., β/SE = z-statistic) less than 1.96 ($P > .05$).

The structural model (Fig. 1) illustrates relationships between the exogenous variables (physical activity, MDD, GAD, PD, and SD) and the endogenous variable (pain). The model includes a direct path from physical activity to pain and indirect paths to pain from physical activity through the four psychiatric symptom variables. The hypothesized mediation effects were also tested in an alternative model that specified a reciprocal causal direction from pain through psychiatric symptoms (as exogenous variables) to physical activity (as the endogenous variable).

To examine whether body fatness or medication use confound the relationships of physical activity with psychiatric symptoms or pain, the structural model also was tested after adjusting for BMI and psychoactive or contraceptive medication use as exogenous covariates. Invariance of the direct effects parameters between participants who did or did not report taking psychoactive or contraceptive medications was tested by the Wald statistic.

Results

Pain duration of more than one month was reported by 11.4% of the sample. Bivariate correlations among pain, psychiatric symptom scores, BMI, and latent physical activity scores are presented in Table 2.

Confirmatory factor analysis indicated that the correlated, four-factor model of the PDSQ scales had acceptable fit to the data (X^2 [896] = 1717.05, $P < .001$, CFI = 0.965, TLI = 0.963, RMSEA = 0.03, WRMR = 1.338). Composite factor reliabilities were high for MDD (0.955), GAD (0.964), panic (0.953), and somatization (0.874). Factor intercorrelations ranged from 0.647 (PD with SD) to 0.749 (MDD with GAD).

The hypothesized structural model depicted in Figure 1 had good fit to the data (X^2 [10] = 18.07, $P = .054$, CFI = 0.991, TLI = 0.976, RMSEA = 0.028, WRMR = 0.550). The model accounted for a significant ($P = .001$) amount of variance (R^2, SE) in pain (9.1%,

2.7%). There were significant (P-values $\leq .015$) direct paths (β, SE) from physical activity to: SD (−0.204, 0.057), GAD (−0.189, 0.049), MDD (−0.178, 0.051), and PD (−0.120, 0.049). Paths to pain were significant (P-values $\leq .008$) for SD (0.217, 0.057) and PD (0.128, 0.048). Other paths were nonsignificant (P-values $\geq .657$). Indirect effects (β, 95% credibility interval [CI]) of physical activity were MDD (−0.005, −0.031 to 0.018), GAD (0.003, −0.024 to 0.034), PD (−0.015, −0.037 to −0.003), and SD (−0.044, −0.085 to −0.024). Each standard deviation increase in physical activity decreased the probability of pain by 11.0% through the indirect PD path and 10.7% through the indirect SD path. Bayes estimates of indirect effects (β, 95% CI) of physical activity were MDD (0.000, −0.023 to 0.024), GAD (0.007, −0.017 to 0.035), PD (−0.012, −0.034 to −0.001), SD (−0.040, −0.077 to −0.014). There was no direct effect (beta, SE) of physical activity on pain (0.028, 0.073, $P = 0.704$). Direct and indirect effects are summarized in Table 3.

Indirect effects (β, 95% CI) were similar when physical activity was expressed categorically as meeting or not meeting recommended guidelines: MDD (−0.005, −0.037 to 0.017), GAD (0.006, −0.033 to 0.057), PD (−0.020, −0.052 to −0.003), and SD (−0.065, −0.126 to −0.027). Bayes estimates of indirect effects (β, 95% CI) of physical activity were MDD (0.001, −0.021 to 0.026), GAD (0.010, −0.037 to 0.053), PD (−0.018, −0.048 to −0.002), and SD (−0.071, −0.127 to −0.026). There was no direct effect (beta, SE) of meeting physical activity guidelines on pain (0.034, 0.107, $P = .747$).

Adjustment for BMI and medication retained adequate model fit (X^2 [14] = 29.53, $P = .009$, CFI = 0.985, TLI = 0.953, WRMR = 0.582) and did not alter parameter estimates. Wald tests indicated that parameter estimates did not differ between participants taking or not taking psychoactive medications (P-values $\geq .122$) or contraceptives (P-values $\geq .253$).

Because one SD item asked if respondents had been "bothered by aches and pains in many different parts of your body?", the models were tested with this item excluded, and the results were not materially changed. Model fit data were (X^2 [10] = 18.09, $P = .053$, CFI = 0.991, TLI = 0.974, RMSEA = 0.028, WRMR = 0.548), and the amount of variance in pain accounted for was 8.3% (SE = 2.6%), $P = .001$. Indirect effects (β, 95% CI) of physical activity were MDD (−0.007, −0.034 to 0.014), GAD (−0.003, −0.031 to 0.026), PD (−0.018, −0.041 to −0.005), and SD (−0.031, −0.062 to −0.012). Bayes estimates of indirect effects (β, 95% CI) of physical activity were MDD (−0.002, −0.029 to 0.022), GAD (0.002, −0.025 to 0.028), PD (−0.015, −0.038 to 0.000), and SD (−0.028, −0.061 to −0.008).

The reciprocal causal model from pain to psychiatric symptoms to physical activity had good fit (X^2 [10] = 13.57, $P = .194$), but there were nonsignificant direct ($P = .774$) and indirect (P-values $\geq .073$) effects from pain to physical activity.

Discussion

In college women, physical activity was associated with pain indirectly through its negative associations with symptoms of PD and SD but not through symptoms of MDD or GAD. The significant

Table 2
Bivariate correlations among pain, raw psychiatric symptom scores, body mass index, and latent physical activity

Variable	Pain	MDD	GAD	PD	SD	Body mass index
Major depressive disorder	.185***	—	—	—	—	—
Generalized anxiety disorder	.181***	.635***	—	—	—	—
Panic disorder	.223***	.461***	.492***	—	—	—
Somatization disorder	.258***	.474***	.503***	.407***	—	—
Body mass index	.046	.052	.064*	.010	.001	—
Physical activity	.004	−.132**	−.128**	−.079*	−.138**	−.112**

*$P < .05$, **$P < .01$, ***$P < .001$ two-tailed.

Table 3
Direct and indirect effects of the hypothesized model

Direct effects	Physical activity as a latent variable		
	β	SE	P
Activity to pain	0.028	0.073	.704
Activity to MDD	−0.178	0.051	.001
Activity to GAD	−0.189	0.049	<.001
Activity to PD	−0.120	0.049	.015
Activity to SD	−0.204	0.057	<.001
MDD to pain	0.028	0.063	.657
GAD to pain	−0.014	0.066	.839
PD to pain	0.128	0.048	.008
SD to pain	0.217	0.057	<.001
Indirect effects	β	95% CI	
Bootstrapped			
MDD	−0.005	−0.031, .018	—
GAD	0.003	−0.024, .034	—
PD	−0.015	−0.037, −.003	—
SD	−0.044	−0.085, −.020	—
Bayesian			
MDD	0.000	−0.023, .024	—
GAD	0.007	−0.017, .035	—
PD	−0.012	−0.034, −.001	—
SD	−0.040	−0.077, −.014	—

negative paths between physical activity and symptoms of MDD, GAD, and PD are consistent with prior epidemiological evidence showing negative associations of physical activity with these symptoms [13,19,25,26,59]. However, much less is known about relations between physical activity and SD.

The present findings are generally consistent with an analysis of a nationally representative sample of 3032 middle-aged U.S. adults (in which physical activity was not reported), which found that the associations between pain and panic attack symptoms were larger than for pain and depressive symptoms [60].

The significant negative path between physical activity and SD appears to be a novel finding, which cannot be directly compared with prior research. The present findings regarding physical activity and SD do add to the scant literature regarding relationships between physical activity and somatoform disorders. In the DSM-IV taxonomy, somatoform disorders included SD and several other disorders (e.g., conversion disorder, hypochondriasis), whereas DSM-5 reconceptualized SD as Somatic Symptom Disorder.

One study of randomly selected adolescents and young adults living in metropolitan Munich examined cross-sectional ($n = 172$ with a somatoform disorder) and longitudinal ($n = 602$ with a somatoform disorder) relationships between physical activity and somatoform disorders [61]. The 12-month prevalence of somatoform disorder was not significantly different between regularly active, nonregularly active, and inactive groups. Frequent participation in sports and exercise at baseline, however, was associated with a 31.0% reduction in the 4-year risk of developing a somatoform disorder and the population attributable risk fraction was 6.2% and 19.0% for females and males, respectively. In addition, one uncontrolled study of young women found that 12 weeks of yoga was associated with reduced somatization symptoms assessed using the SCL-90-R, a measure that includes muscle pain symptoms [62].

Yoga, which incorporates mindfulness, might be an attractive exercise mode for reducing symptoms of somatization because there is evidence that mindfulness alone, or as part of cognitive behavioral therapy, can be therapeutic for both chronic pain and somatic symptom disorder [63,64]. Currently, there is an absence of evidence from randomized controlled trials providing empirical support any type of physical activity as therapeutic for SD [65].

The absence of a significant direct path in the structural model between physical activity and pain appears to be inconsistent with some studies of physical activity and pain. One population-based study of adults that measured 7-days of physical activity using accelerometers found chronic pain associated with less physical activity; however, the association between chronic pain and activity counts per minute was stronger for men than for women [15]. One clinical study of middle-aged women using both accelerometers and self-report to assess physical activity found that patients with fibromyalgia compared with healthy controls had lower physical activity measured either objectively or subjectively [66].

In contrast to the young women tested in the present study, most prior experimental and epidemiological studies of physical activity and pain have tested male and female adults between the ages of 25 and 75 years, usually with a specific pain condition and often conditions that are rare in young adults but prevalent in older adults (e.g., osteoarthritis) [67]. The greater functional reserve of younger adults is one factor that plausibly could allow them to remain physically active despite experiencing concomitant pain compared with older adults. Older adults in pain who reduced their physical activity may be more likely to show attenuated muscular strength and cardiorespiratory fitness, leading to less physical functional reserve [68].

High-intensity pain, or pain present in multiple locations, is more likely to be associated with reduced physical activity [69]. In the present study, the severity, type, and location(s) of pain were not measured. Prior studies often focused on samples that likely were in more widespread or severe pain than the present sample; for example, some studies required pain to be experienced for at least 3 months and present both above and below the waist as well as in contralateral body quadrants [70].

Musculoskeletal (e.g., low back), headache, and dysmenorrhea pain are likely key contributors to the pain burden in the present sample because of the relatively high prevalence of these types of pain in college-aged women [71–73]. Evidence shows that physical activity is associated with lower pain in people with these types of pain, but whether psychiatric symptoms play a role is unknown [74,75].

The present results should be interpreted in light of potential limitations. The potential role of expectations among the relations of physical activity, pain, and psychiatric symptoms was not considered here. Expectations may influence these relations, given that approximately half the anxiety and depression symptom improvement after exercise training may be the result of placebo responses and misinterpretation of symptoms may play a role in both PD and SD [76,77]. Thus, regularly active individuals may have previously experienced concomitant pain relief and PD and/or SD symptom improvement, and these experiences may have contributed to the present findings. One structural equation modeling study involving the PDSQ found that the relationship between anxiety symptoms and marijuana use was indirectly mediated through expectations for tension reduction in young adult women [78].

Other potential limitations were that the study focused on predominately white, normal-weight female students attending a single University in the southeastern United States. Thus, the generalizability of the findings to people of different races, ethnicities, cultures and to men, older adults, or the overweight is unknown. The nonsignificant direct and indirect effects from pain to physical activity found here may be stronger if, in the future, they were to be examined among other samples such as those suffering from a specific type of pain condition (e.g., migraine). However, the relationships observed here were not meaningfully changed when statistical adjustments were made for BMI, contraceptive use, or psychoactive medication use. No adjustments were made for the

type of psychoactive medication used which may have influenced the results because some medications have analgesic effects while others do not; moreover, these potential effects may depend on the type of pain (back pain vs. neuropathic pain) and the severity of psychiatric symptoms [50,79]. Pain was assessed with a single item, consequently no information about pain severity or location(s) was obtained, yet these aspects of pain could be important mediators of relationships between physical activity and pain. The PDSQ was developed based on samples of psychiatric outpatients and its psychometric properties for samples of college women are less well understood. Nevertheless, there is a substantial body of evidence to support construct validity of the PDSQ [80] and our confirmatory factor analysis found that a four-factor model (MDD, PD, GAD, and SD scales) acceptably reproduced our data from college women. The PDSQ assessed psychiatric symptoms as outlined in the DSM-IV; thus, the relationships described here may not generalize to the current diagnostic scheme outlined in DSM-5. The results also are delimited to the assessment of physical activity as measured by a well-validated, yet self-reported, measure of physical activity.

Recognizing these potential limitations, we conclude that in a sample of college women, physical activity was associated with pain indirectly through its associations with symptoms of PD and SD. The results suggest that a fuller understanding of relationships between pain and physical activity may require a concomitant consideration of the influence of physical activity on psychiatric symptoms associated with pain. The findings also imply clients suffering from pain and comorbid panic or SDs may benefit from clinicians who recommend regular physical activity.

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