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ORIGINAL ARTICLE

Spinal pain is prospectively associated with cardiovascular risk factors in girls but not boys (CHAMPS study-DK)

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Abstract

Purpose To examine the prospective associations between spinal pain exposures and risk factors for cardiovascular disease in children and explore the mediating role of health-related physical activity.

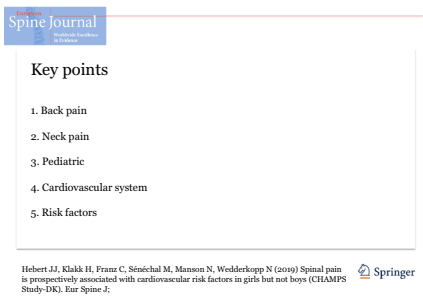
Methods Students were recruited from ten public primary schools. Each week from November 2008 to October 2010, parents reported spinal pain occurrences in their children via text messaging. Clustered cardiovascular risk was estimated with a composite score comprising fasting serum triglycerides, homeostasis assessment model-estimated insulin resistance (HOMA-IR), total to high-density lipoprotein cholesterol ratio, and systolic blood pressure. Additional outcomes were fasting serum insulin and glucose concentrations and body mass index categories. Associations were explored with multilevel mixed regression models and reported with beta coefficients (β) and percent difference scores. All models were adjusted for potential confounders.

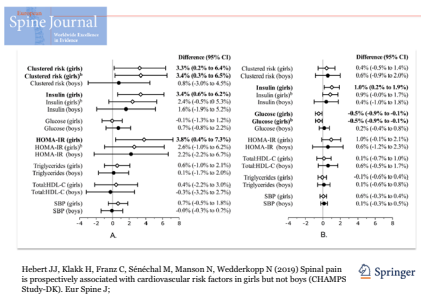
Results Data from 1022 children (53% female) with mean ± SD age of 8.4 ± 1.4 years were included. Girls with spinal pain had greater clustered cardiovascular risk (β [95% CI]; percent difference [95% CI] = .41 [.02–.80]; 3.3% [.2–6.4%]) than those without spinal pain. Similar outcomes were observed for log insulin (percent difference [95% CI] = 3.4% [.6–6.2%]) and log HOMA-IR = (percent difference [95% CI] = 3.8% [.4–7.3%]). Remaining associations between spinal pain and cardiovascular risk in girls were nonsignificant. There were no associations between spinal pain and cardiovascular risk in boys. Moderate-to-vigorous-intensity physical activity did not appear to mediate this relationship.

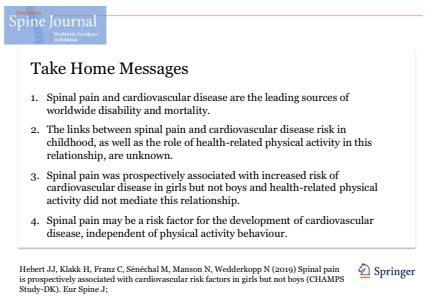
Conclusion These findings suggest a potentially important link between spinal pain and cardiovascular risk in girls that may be independent of health-related physical activity.

Graphic abstract

These slides can be retrieved under Electronic Supplementary Material.







Keywords Back pain · Neck pain · Pediatric · Cardiovascular system · Risk factors

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Introduction

Spinal pain, physical inactivity, and cardiovascular disease are important public health challenges. Spinal pain is a leading cause of disability in young people [1], and back pain tracks into adulthood [2], when it becomes the greatest contributor to years lived with disability [3]. In the USA, total indirect and direct healthcare costs attributable to back pain are greater than $100 billion per year [4].

Health-related physical activity is essential to health and normal development in youth [5, 6], while physical inactivity is a leading source of mortality and morbidity [7]. Cardiovascular disease accounts for nearly one-third of all deaths worldwide [8], while the costs associated with cardiovascular disease comprise 17% of overall national health expenditure in the USA [9]. The development of cardiovascular risk can be identified in youth [10, 11], marking childhood as an opportune time to reduce modifiable cardiovascular disease risk factors.

Adult studies demonstrate that spinal pain is associated with coronary heart disease, myocardial infarction, and death from ischemic heart disease [12–14]. In young people, physical inactivity is linked with cardiovascular disease risk [15] and spinal pain in children [16, 17]. Spinal pain has potential to reduce health-related physical activity and therefore may increase risk for cardiovascular disease. However, the links between spinal pain, physical activity, and cardiovascular disease risk in childhood are unknown. Given their socioeconomic burden, the relations between these health problems should be investigated.

This study aimed to examine the prospective associations between spinal pain exposures and risk factors for cardiovascular disease in children and explore the mediating role of health-related physical activity in these relations. We hypothesized that spinal pain would be associated with increased cardiovascular risk and that health-related physical activity would mediate these relationships.

Methods

Study design and participants

This prospective cohort study was nested in the Childhood Health, Activity, and Motor Performance School Study Denmark (CHAMPS study-DK). The CHAMPS study-DK is a quasi-experimental trial designed to estimate the effects of physical education and other variables on cardiovascular risk factors, musculoskeletal health, and motor performance in children [18].

All public primary schools within the municipality of Svendborg, Denmark ($N = 19$), were invited to participate in the study. Ten schools elected to participate, with four schools continuing the usual physical education program (90 min/week) and six schools providing 270 min of physical education per week. All participating children from the ten schools were included in a common cohort for analysis in the current study.

Participating children were enrolled into the study on a rolling basis from November 2008 to August 2009. Spinal pain exposures were measured each week for the duration of the study. Cardiovascular risk outcomes were measured at baseline and repeated in October 2010.

Additional information on the study procedures and sample has been reported previously [18, 19]. The Regional Scientific Ethical Committee of Southern Denmark approved the study (ID S-20080047). All parents supplied provided written consent, and children gave verbal consent prior to study enrollment.

Spinal pain

Parents reported occurrences of spinal pain each week via mobile phone SMS messaging. We undertook surrogate reporting by parents owing to concerns over the validity of self-reporting by children [20, 21]. All SMS responses were automatically uploaded to a Web-based system developed to monitor recurring events (SMS-Track, Esbjerg, Denmark). This approach is reliable, acceptable to research participants, and valid compared to structured interviews [22].

Each Sunday, parents reported whether the child had experienced any pain in the "neck, mid back and/or lower back" during the preceding week. Pain reports triggered research staff to telephone parents within three days. If the child's pain was still present at the time of telephone follow-up, a trained primary healthcare provider performed a standardized clinical examination within 2 weeks. If necessary, children were also referred for paraclinical testing and/or consultation with an orthopedic surgeon.

We included two measures of spinal pain. First, we categorized the occurrences of spinal pain during the study period. Second, we measured the total number of weeks of spinal pain experienced by each child.

Pubertal development

We estimated pubertal development using the Tanner stages [23]. Tanner stage was self-assessed using explanatory text and visual representations of pubic hair development in boys and breast development in girls [24]. Pubertal development was reported on a scale from 1 to 5, with higher scores indicating later pubertal stages.

Physical activity

Physical activity was objectively measured with GT3X Actigraph accelerometers (Actigraph, Pensacola Florida) during a seven-day measurement period. Participants had to accumulate a minimum of 10 h of wear time on at least 4 of 7 days for their physical activity data to be included in the analysis. Non-wear time was defined as the absence of signals lasting 30 or more consecutive minutes. As reported previously, adherence to the measurement procedures among participants in the CHAMPS study-DK was high. The mean (SD) wear time and valid measurement days were 13.3 (1.3) hours and 6.1 (.9) days, respectively. Consequently, 87% of children attained four or more days of valid accelerometry data. Missing physical activity data were imputed using a combined multiple imputation approach [25].

Trained research staff fit each child with customized elastic belts securing the accelerometer to the child's right hip [26]. The children were instructed to wear the device each day from the time they awoke until they went to bed and to remove the device only when swimming or bathing.

We processed the accelerometry data with a customized software program (Propero, version 1.0.18, University of Southern Denmark, Odense, Denmark). The accelerometers recorded physical activity data every 2 s, and we subsequently collapsed these data into 10 s epochs [27]. Signals were digitized and passed through a filter with band limits of .25–2 2.5 HZ to eliminate extraneous accelerations not associated with human movement (e.g., vibration). We applied cut points to identify time (proportion of the day) in moderate-to-vigorous physical activity intensities (i.e., health-related physical activity) [28, 29].

Cardiovascular risk factors

Blood samples

Fasting blood samples were obtained at the participating primary schools by trained research staff between 8:00 and 10:30 a.m. The samples were immediately put on ice and transported to the local laboratory where they were centrifuged, pipetted (serum), and stored at − 80 °C. The analyses of all samples were conducted in a certified laboratory at the University of Vienna using a Roche/Hitachi cobas c system (Roche, Mannheim, Germany). The following serum markers were measured: total cholesterol, high-density lipoprotein cholesterol (HDL), total cholesterol:HDL ratio, triglycerides, glucose, and insulin. Total cholesterol, triglycerides, HDL, and glucose were quantitatively analyzed using enzymatic, colorimetric methods. Insulin was analyzed by way of solid-phase enzyme-labeled chemiluminescent immunometric assay. Homeostasis assessment model-estimated insulin resistance (HOMA-IR) scores were calculated as insulin (μU/mL) × glucose (mmol/L)/22.5 [30].

Systolic blood pressure

Systolic blood pressure was recorded using an automated blood pressure monitor [Welch Allyn® (New York, USA) vital signs monitor 300 series with FlexiPort™] applied to the left arm. Prior to measurement, participants rested in the sitting position for 5 min. Blood pressure measures were obtained at 1-min intervals until three stable measures or five total measures were obtained. The mean of the final three measures was used for analysis.

Anthropometric measures

Measures of height and weight were obtained with children barefoot and wearing light clothes. Height was measured with a portable stadiometer (SECA 214, Seca Corporation, Hanover, MD, USA) to the nearest .5 cm, and weight was measured to the nearest .1 kg using a calibrated Tanita BWB-800S digital scale (Tanita Corporation, Tokyo, Japan). Body mass index was classified as normal, overweight, or obese using age- and sex-specific norms from the International Obesity Task Force [31].

Clustered cardiovascular risk score

We created clustered cardiovascular risk scores by summing the standardized values of the total cholesterol:HDL ratio, log triglycerides, log HOMA-IR, and systolic blood pressure outcomes [32]. All scores were converted to positive values, with larger scores representing higher levels of cardiovascular disease risk. Clustered cardiovascular risk is preferred to single risk factors as measure of cardiovascular health [33], and recent research has supported the use of combined continuous risk factors to estimate the cardiometabolic risk in children [34].

Statistical analysis

All analyses were conducted with Stata v14.2 software (StataCorp, College Station, TX, USA). Given the clustered nature of the data, we examined the associations between spinal pain and cardiovascular disease risk using multilevel mixed-effects regression models with random intercepts. Separate models were constructed for clustered cardiovascular risk, as well as each of the individual risk factors. Variable and residual normality and the linearity of associations (when appropriate) were examined graphically. We log-transformed outcome variables with non-normal distributions.

Spinal pain predictors comprised two variables. First, we constructed a dichotomous spinal pain variable to identify children who experienced one or more episodes of spinal pain during the study period. Second, we calculated the duration of spinal pain experienced by study participants. Body mass index, pubertal status, school type (usual or increased physical education) and baseline outcome score were included as covariates in each model. Continuous model outcomes were reported with unstandardized beta coefficients (β) and 95% confidence intervals representing the change in outcome represented by 4 weeks of spinal pain. We chose a four-week duration as it approximated the average pain duration experienced by children.

To aid the interpretation of the parameter estimates, we also transformed beta coefficients to scores representing the percent difference (relative to the group baseline mean) in cardiovascular risk associated with 4 weeks of spinal pain. Log-transformed variables were converted to positive values for these calculations.

Similar multilevel mixed-effects Poisson regression models with robust standard errors were constructed for BMI categorical outcomes. These models controlled for baseline BMI score, pubertal status, and school type (usual or increased physical education). The strength of associations was reported with incidence rate ratios (IRR) and 95% confidence intervals.

To explore the potential mediating role of health-related physical activity in the relations between spinal pain and cardiovascular risk, we included the mean proportion of time in moderate-to-vigorous-intensity physical activity as an additional covariate in each model identifying a significant association between spinal pain and cardiovascular risk.

Preliminary analyses identified a moderating role of sex in the relations between spinal pain and cardiovascular risk. Therefore, the reporting of all parameter estimates was stratified by sex. As this study was exploratory, alpha was set to .05 for all analyses.

Results

The study sample comprised 1022 participants (53% female) with mean (SD) age of 8.4 (1.4) years at baseline. The mean (SD) duration of study participation was 73 (13) weeks. Descriptive statistics for demographic, spinal pain, and cardiovascular risk variables are presented in Tables 1 and 2. Table 3 and Fig. 1 report the associations between spinal pain and each of the cardiovascular risk outcomes.

Spinal pain and cardiovascular risk

Girls who experienced spinal pain had greater clustered cardiovascular risk (β [95% CI] = .41 [.02, .80]) than girls without spinal pain. This corresponds to a 3.3% (95% CI = .2% to 6.4%) increase in clustered risk. Spinal pain was also associated with increased log insulin (β [95% CI] = .10 [.02, .17]) and log HOMA-IR (β [95% CI] = .09 [.01, .17]) concentrations, equating to 3.4% (95% CI = .6–6.2%) and 3.8% (95% CI = .4–7.3%) increases, respectively. Spinal pain was not associated with serum glucose, triglycerides, total:HDL cholesterol ratio, systolic blood pressure, or overweight/obesity in girls. There were no associations between spinal pain and any cardiovascular risk factor in boys.

Spinal pain duration and cardiovascular risk

Spinal pain duration was associated with increased log insulin (β [95% CI] = .03 [.00, .05]) and decreased glucose (β [95% CI] = − .02 [− .04, − .01]) concentrations in girls. These associations equate to a 1.0% (95% CI = .2–1.9%) increase in insulin and a .5% (95% CI = .9–.1%) decrease in glucose concentration per 4 weeks of spinal pain. Spinal pain was not associated with clustered cardiovascular risk, log HOMA-IR, total:HDL cholesterol ratio, triglycerides, systolic blood pressure, or overweight/obesity in female participants. There were no associations between spinal pain and any cardiovascular risk factors in boys.

Spinal pain, physical activity, and cardiovascular risk

Controlling for moderate-to-vigorous-intensity physical activity had modest impacts on the association estimates between spinal pain and cardiovascular risk in girls (Table 4, Fig. 1a). The associations between spinal pain and clustered cardiovascular risk were nearly unchanged and remained significant (β [95% CI]; % difference [95% CI] = .43 [.04–.82]; 3.4% [.3–6.5%]). The associations between spinal pain and log insulin (β [95% CI]; % difference [95% CI] = .07 [− .01–.15]; 2.4% [− .5–5.3%]) as well as log HOMA-IR (β [95% CI]; % difference [95% CI] = .06 [− .02–.15]; 2.6% [− 1.0–6.2%]) decreased slightly and became nonsignificant.

Similarly, controlling for moderate-to-vigorous-intensity physical activity had little impact on the relations between spinal pain duration and cardiovascular risk in girls (Table 4, Fig. 1b). The association between spinal pain duration and log insulin decreased by .1% and became nonsignificant (β [95% CI]; % difference [95% CI] = .02 [− .00 to .05]; .9% [− .0 to 1.7%]), while the association between spinal pain duration and serum glucose concentration remained unchanged (β [95% CI]; % difference [95% CI] = − .01 [− .04 to − .00]; − .5% [− .1 to − .9%]).

Table 1 Descriptive baseline and follow-up demographic and cardiovascular risk data stratified by sex[a]

Variables		Baseline	Follow-up
Age (y)	Girls ($n=541$)	8.3 (1.5)	10.3 (1.5)
	Boys ($n=481$)	8.4 (1.4)	10.5 (1.4)
Body mass index	Girls ($n=541$)	465 (86.0%)	466 (86.1%)
Normal (n, %)		65 (12.0%)	66 (12.2%)
Overweight (n, %)		11 (2.0%)	9 (1.7%)
Obese (n, %)			
	Boys ($n=481$)	438 (91.1%)	433 (90.0%)
		38 (7.9%)	42 (8.7%)
		5 (1.0%)	6 (1.3%)
Clustered CV risk	Girls ($n=381$)	12.5 (2.5)	16.1 (2.6)
	Boys ($n=364$)	11.7 (2.5)	15.5 (2.8)
Insulin (μU/mL)	Girls ($n=392$)	3.9 (3.0)	5.2 (3.0)
	Boys ($n=376$)	3.4 (2.1)	5.0 (6.8)
Glucose (mmol/L)	Girls ($n=392$)	4.5 (.4)	4.8 (.3)
	Boys ($n=376$)	4.7 (.9)	4.9 (.9)
HOMA-IR	Girls ($n=392$)	.8 (.6)	1.1 (.7)
	Boys ($n=376$)	.7 (.6)	1.3 (4.8)
Triglycerides (mg/dl)	Girls ($n=392$)	59.5 (22.1)	58.2 (25.4)
	Boys ($n=376$)	52.9 (22.3)	54.5 (28.1)
Total:HDL (mg/dl)	Girls ($n=392$)	2.9 (.7)	2.8 (.7)
	Boys ($n=376$)	2.6 (.6)	2.6 (.6)
Systolic blood pressure (mmHg)	Girls ($n=524$)	100.9 (8.0)	102.0 (8.0)
	Boys ($n=468$)	101.5 (8.9)	102.3 (8.4)
MVPA (% of day)	Girls ($n=480$)	7.5 (2.3)	7.2 (2.2)
	Boys ($n=389$)	9.1 (2.4)	9.6 (3.0)

[a]Values are mean (SD) unless otherwise indicated

y years, *HOMA-IR* homeostasis assessment model-estimated insulin resistance, *HDL* high-density lipoprotein cholesterol, *CV* cardiovascular, *MVPA* moderate-to-vigorous-intensity physical activity

Table 2 Spinal pain prevalence and duration[a]

Variable	Spinal pain prevalence (n, %)[b]	Spinal pain duration (weeks)[c]
Girls ($n=541$)	230 (42.5)	4.6 (8.6)
Boys ($n=481$)	184 (38.3)	4.3 (10.2)

[a]Values are mean (SD) unless otherwise indicated

[b]Mean (SD) duration of study participation = 73 (13) weeks

[c]Among children with prevalent spinal pain during study period

Discussion

The findings of the current study provide important insights regarding the relationship between spinal pain and cardiovascular disease risk in children. First, spinal pain was associated with increased risk of cardiovascular disease in girls but not boys. Girls who experienced spinal pain had greater clustered cardiovascular risk, as well as higher levels of serum insulin and insulin resistance. Second, longer duration spinal pain was associated with higher serum insulin (1% per 4 weeks of pain), but there was no apparent dose–response relationship between spinal pain duration and other cardiovascular risk factors. Finally, controlling for moderate-to-vigorous-intensity physical activity had little impact on these relations, and contrary to our hypothesis, health-related physical activity did not demonstrate an obvious mediating effect on cardiovascular risk. Spinal pain may therefore be a risk factor for the development of cardiovascular disease, independent of physical activity behavior. Although the magnitudes of increased cardiovascular risk associated with spinal pain in girls were modest (~ 3%), they are similar to the protective associations related to beneficial activities such as intensive physical education programs [35] and organized sports participation [36]. These findings highlight a potentially important link between spinal pain and cardiovascular risk in girls and represent an early advance in understanding the relationship between these health problems.

The mechanisms responsible for sex-specific differences in the relations between spinal pain and cardiovascular risk in children are unclear. Our results were similar to those

Table 3 Associations between spinal pain and cardiovascular risk factors[a]

Outcome		Any spinal pain	Spinal pain duration[b]
Clustered CV risk	Girls ($n=381$)	**.41 (.02 to .80)**	.05 (−.07 to .18)
	Boys ($n=364$)	.09 (−.35 to .52)	.07 (−.10 to .24)
Log insulin (μU/mL)	Girls ($n=392$)	**.10 (.02 to .17)**	**.03 (.00** to **.05)**
	Boys ($n=376$)	.04 (**-**.05 to .14)	.01 (−.03 to .05)
Glucose (mmol/L)	Girls ($n=392$)	−.00 (−.06 to .05)	**−.02 (−.04** to **−.01)**
	Boys ($n=376$)	.03 (−.04 to .10)	.01 (−.02 to .04)
Log HOMA-IR	Girls ($n=392$)	**.09 (.01 to .17)**	.02 (−.00 to .05)
	Boys ($n=376$)	.05 (−.05 to .16)	.01 (−.03 to .05)
Log triglycerides (mg/dl)	Girls ($n=392$)	.02 (−.04 to .08)	−.00 (−.02 to .02)
	Boys ($n=376$)	.01 (−.07 to .08)	.00 (−.02 to .03)
Total: HDL (mg/dl)	Girls ($n=392$)	.01 (−.06 to .09)	.00 (−.32 to .44)
	Boys ($n=376$)	−01 (−.08 to .07)	.14 (−.25 to .53)
Systolic blood pressure (mmHg)	Girls ($n=524$)	.66 (−.52 to 1.85)	.06 (−.32 to .44)
	Boys ($n=468$)	−.09 (−1.41 to 1.24)	.14 (−.25 to .53)
Overweight or obese	Girls ($n=541$)	IRR = 1.11 (.66 to 1.85)	IRR = 1.00 (.88 to 1.15)
	Boys ($n=481$)	IRR = .67 (.43 to 1.06)	IRR = .85 (.68 to 1.05)

Bolded values indicate statistically significant associations

[a]Values are unstandardized beta coefficients (95% confidence intervals) unless otherwise indicated. All models adjusted for body mass index, pubertal status, school type, and baseline outcome score

[b]Per 4-week period

HOMA-IR homeostasis assessment model-estimated insulin resistance, *HDL* high-density lipoprotein cholesterol, *IRR* incidence rate ratios, *CV* cardiovascular

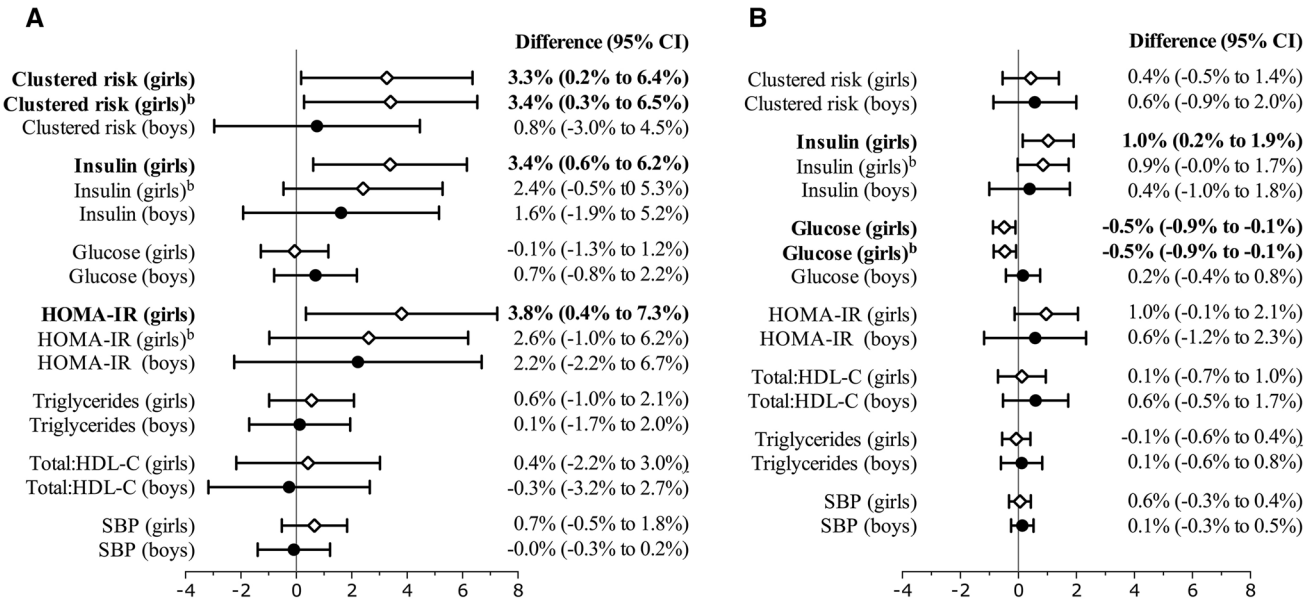

Fig. 1 Differences in cardiovascular risk associated with **a** the occurrence of spinal pain during the study period and **b** 4 weeks of spinal pain. [a]Values represent percent differences relative to the baseline mean (95% CI). All models adjusted for body mass index, pubertal status, school type, and baseline outcome score. [b]Additionally adjusted for time in moderate-to-vigorous-intensity physical activity

from a recent adult study reporting that sedentary behavior was cross-sectionally associated with low back pain in adult women but not men [37]. However, the authors cautioned that this relationship could be confounded by genetic or early environmental exposures and will be a constructive area for future research. A somewhat counterintuitive finding in the

Table 4 Associations between spinal pain and cardiovascular risk factors controlling for time in moderate-to-vigorous-intensity physical activity[a]

Outcome		Any spinal pain	Spinal pain duration[b]
Clustered CV risk	Girls ($n=381$)	**.43 (.04 to .82)**	–
Log insulin (μU/mL)	Girls ($n=392$)	.07 (−.01 to .15)	.02 (−.00 to .05)
Glucose (mmol/L)	Girls ($n=392$)	–	**−.02 (−.04 to −.00)**
Log HOMA-IR	Girls ($n=392$)	.06 (−.02 to .15)	–

[a]Values are unstandardized beta coefficients (95% confidence intervals). All models adjusted for time in moderate-to-vigorous-intensity physical activity, body mass index, pubertal status, school type, and baseline outcome score

[b]Per 4-week period

Bolded values indicate statistically significant associations

HOMA-IR homeostasis assessment model-estimated insulin resistance

present study was the association between increasing spinal pain duration and lower serum glucose concentrations in girls. The magnitude of this association (−.5%) is unlikely to represent a meaningful effect and may have resulted from pubertal advancement not captured by our self-reported pubertal estimates, or from the effect of increased insulin resistance in this healthy cohort of children approaching adolescence [38, 39].

Musculoskeletal pain, including spinal pain, is common in young people and a leading cause of disability [1, 40]. Compared to adult studies, relatively few studies have investigated potential risk factors for the development of spinal pain or the effectiveness of therapies in pediatric patients. Spinal pain in young people has been associated with several potential risk factors including poor general well-being, increased stress, screen time, poor sleep, familial history, scoliosis, and pubertal development and growth [41–44]. Some of these factors may also relate to cardiovascular risk factors, and this will be an important area for future research. A recent systematic review highlighted the lack of evidence to guide clinical decision making in young people, having identified only four randomized trials examining the effectiveness of conservative interventions for low back pain in children and adolescents [45]. The review identified moderate-quality evidence suggesting that exercise interventions improve pain intensity in children and adolescents compared to no treatment.

Exercise contributes to overall physical activity levels. Compared to boys, girls are less physically active [19, 46] and more likely to experience spinal pain [47–49]. Health-related physical activity behavior is a modifiable risk factor for cardiovascular disease [7], and moderate-intensity physical activity may also protect against spinal pain in children [50]. Therefore, despite our finding that moderate-to-vigorous physical activity does not have an obvious mediating role, previous studies demonstrate that health-related physical activity is independently associated with spinal pain and cardiovascular risk. Consequently, the current study results should not be viewed as contrary to guideline recommendations aimed at ensuring children participate in at least 60 min of moderate-to-vigorous-intensity physical activity per day [51]. Existing health promotion strategies include the regular monitoring of physical activity levels and appropriate physical activity counseling [52, 53], and specific guidance for pediatric patients is available [54]. Additional approaches for increasing health-related physical activity in young people may also include participation in organized sport [46, 55] or after-school programs [56].

The primary strengths of the current study include the prospective design, representative sample, and robust measures of spinal pain, physical activity, and cardiovascular risk. Although we accounted for several sources of potential confounding, we cannot rule out all factors with potential for influencing the study outcomes. Our measures of spinal pain did not account for pain intensity, and this may be relevant when seeking to understand the relations between spinal pain, physical activity, and cardiovascular risk. While accelerometry is preferable to self-reported physical activity estimates, it is unable to capture some modes of activity (e.g., cycling, swimming). These limitations represent potential sources of residual confounding in our analysis. The lack of a strong dose–response between pain duration and the clustered risk outcome may question the causal nature of the relationship between these variables. Understanding the role of pain characteristics such as frequency, intensity, and duration will be important for future research aimed at elucidating the relations of spinal pain and cardiovascular risk. Consistent with the exploratory nature of this study, we undertook multiple statistical tests without correcting the type I error rate. Consequently, these results require replication as well as further exploration of the causal pathway prior to confident clinical or policy implementation.

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Compliance with ethical standards

Conflict of interest Dr. Hebert receives salary support from the Canadian Chiropractic Research Foundation and the New Brunswick Health Research Foundation. The authors declare no relevant conflicts of interest.

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