



FIGURE 3. Lateral weight-bearing radiograph of the left ankle, with numerous, similarly sized chondral and osteochondral bodies (arrow) compatible with primary synovial osteochondromatosis. In a young patient, without prior trauma or significant arthrosis, this is most consistent with primary synovial osteochondromatosis.

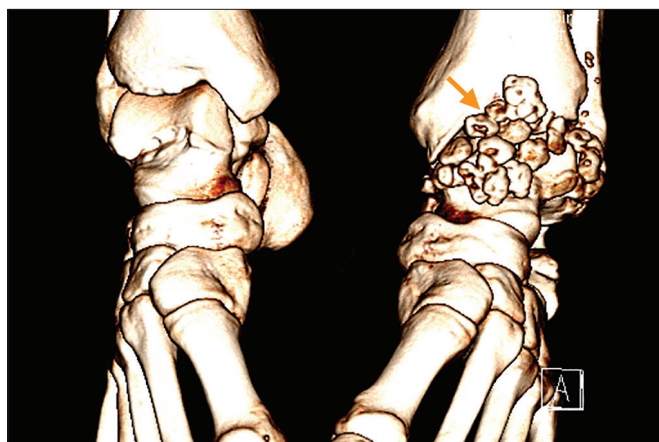


FIGURE 4. Anterior-to-posterior computed tomography scan, with numerous chondral and osteochondral bodies (arrow) in 3-D, compatible with primary synovial osteochondromatosis.

Primary Synovial Osteochondromatosis Discovered After Ankle Sprain

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A 23-YEAR-OLD MILITARY CADET PRESENTED to a direct-access physical therapy clinic for an ankle sprain 1 day after inverting his left ankle while dismounting a wall on an indoor obstacle course. He immediately resumed and completed the practice session in full weight bearing. Physical examination revealed anterior talofibular and calcaneofibular ligament laxity, no deformities, no proximal fibular pain, and minimal swelling. The cadet returned to physical therapy 48 hours later with ongoing pain in the lateral ankle and new pain in the proximal left fibula. Site-specific bone pain and tenderness to palpation in the cadet's proximal one third of the left fibula were reproduced during the tibia-fibula squeeze test and fibular shearing.

Lower-leg radiographs were ordered by the physical therapist to rule out a fracture of the proximal fibula, also known as a Maisonneuve fracture³ (FIGURES 1 and 2, available at www.jospt.org). The radiographs showed no fracture; however, small, rounded, partially calcified bodies were noted in the ankle. Additional ankle imaging (FIGURE 3) was ordered under radiologist guidance. Preoperative computed tomography of the cadet's left ankle, ordered by orthopaedics, demonstrated chondral and osteochondral bodies (FIGURE 4).

Osteochondromatosis, also known as Reichel's syndrome,² is a monoarticular condition that is present in males twice as often as in females, usually in the third to fifth decades of life and diagnosed predominantly in the knee.¹ Osteochon-

dromatosis stems from chondrocyte metaplasia within the synovium, forming pedunculated cartilage interarticularly and extra-articularly. Due to synovial fluid nourishment, the cartilaginous bodies may ossify, proliferate, and break free.¹

Primary synovial osteochondromatosis, while benign, increases the risk for osteoarthritis and malignant chondrosarcoma.¹ Due to these risks, the cadet was referred to an orthopaedic surgeon for definitive care. Thorough surgical resection of the chondrocyte-producing tissue is vital in decreasing osteochondromatosis recurrence.^{1,2} The cadet was commissioned into the US Army with normal ankle mobility and function 8 weeks after surgery. ●
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References

1. Isbell JA, Morris AC, Araoye I, Naranje S, Shah AB. Recurrent extra- and intra-articular synovial chondromatosis of the ankle with tarsal tunnel syndrome: a rare case report. *J Orthop Case Rep.* 2017;7:62-65.
2. Philip MC, Usman S. Synovial chondromatosis: a rare differential diagnosis of hip pain in a child. *J Orthop Case Rep.* 2017;7:37-39.
3. Stufkens SA, van den Bekerom MP, Doornberg JN, van Dijk CN, Kloen P. Evidence-based treatment of Maisonneuve fractures. *J Foot Ankle Surg.* 2011;50:62-67. <https://doi.org/10.1053/j.jfas.2010.08.017>

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The Association Between Changes in Weekly Running Distance and Running-Related Injury: Preparing for a Half Marathon

Recreational distance running is one of the most accessible and popular forms of physical activity worldwide.^{1,7,19,33} According to various sources, participation in the half marathon running distance (21.1 km) has steadily increased since 2000.^{7,9,35} The physical and psychological health-related benefits associated with distance running are well documented.^{11,21,30} However, a major barrier

to continued participation is the high risk of sustaining a running-related injury (RRI).³⁷ For example, the pooled time-loss RRI cumulative incidence proportions for runners who increased their distance from 10 km to a half marathon and a full marathon were reported to be 31.7% and 52.0%, respectively, during a 1-year follow-up period.²⁰ The establishment of effective RRI prevention intervention strategies is, therefore, an important area of future scientific research.

The ability to design and implement the most effective RRI prevention interventions at the population level initially requires an in-depth understanding of how and why RRI develops.^{2,10} The primary exposure of training load has long been recognized as a necessary causal component in any given mechanism of injury.^{3,23} To be precise, RRI is sustained when the load capacity of the runner's musculoskeletal system has been exceeded by the applied structure-specific training load.^{3,15,25,36} Given that structure-specific load (ie, the sum of step-specific loads that a certain musculoskeletal structure is exposed to during a running

● **BACKGROUND:** Sudden changes in training load may play a key role in the development of running-related injury (RRI). Because the injury mechanism depends on the runner's musculoskeletal load capacity, the running schedule followed prior to sudden change in training load may influence the amount of change that a runner can tolerate before the runner is at a higher risk of RRI.

● **OBJECTIVES:** To investigate the association between change in weekly running distance and RRI, and to examine whether the association may be modified by the running schedule the runner follows.

● **METHODS:** Two hundred sixty-one healthy (noninjured) runners were included in this prospective cohort study over a period of 14 weeks. Data on running activity were collected daily and objectively, using a global positioning system watch or smartphone. Instances of RRIs were collected using weekly e-mailed questionnaires. Primary exposure was defined as changes in weekly running distance. Data were analyzed with time-to-event models that produced cumulative risk difference as the measure of association.

● **RESULTS:** A total of 56 participants (21.5%) sustained an RRI during the 14-week study period. Twenty-one days into the study period, significantly more runners were injured when they increased their weekly running distance by 20% to 60% compared with those who increased their distance by less than 20% (risk difference, 22.6%; 95% confidence interval: 0.9%, 44.3%; $P = .041$). No significant difference was found after 56 and 98 days. No significant effect-measure modification by running schedule was found.

● **CONCLUSION:** Significantly more runners were injured 21 days into the study period when they increased their weekly running distances by 20% to 60% compared with those who increased their distances by less than 20%.

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● **KEY WORDS:** etiology, prospective cohort, sports, time-to-event analyses

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session³⁾ is difficult to measure in epidemiological studies, scientists have investigated the association between variables related to training load (eg, running distance) and RRI. Researchers are revisiting the concept of training load by focusing on how it changes in relation to RRI and sports injury development.^{5,26,28} Anecdotally, this is also known as the “too much, too soon” theory.³⁶ Therefore, to better understand the complex etiology of RRI, there is a need to further examine how changes in weekly running distance are associated with the risk of sustaining an RRI.

The current general assumption held by several sports injury researchers is that a sudden increase, no change, or a decrease in running distance should be investigated in future epidemiological sports injury studies.^{12,28,36} Because the injury mechanism of sudden increases in running distance also depends on the runner’s specific level of load capacity,^{3,36} the measurement of this variable also needs to be taken into account. As with structure-specific load, it is also not possible to directly measure musculoskeletal load capacity in large-scale epidemiological studies.³⁶ Therefore, proxy variables, such as the amount and type of running activity (eg, running long distances or running at fast paces) undertaken prior to a sudden increase in running distance, may be factors that influence the amount of change in running distance a given runner is able to tolerate before increasing the risk of RRI.

The association between changes in running distance and RRI could be modified by the type of running activity that runners perform and differ within strata of different running schedules. Specifically, sudden increases in running distance may be more injurious for runners with a high-volume running schedule (a distance-based schedule) compared to those who run fewer kilometers per week at a faster pace (a pace-based schedule). Runners following a distance-based schedule might be closer to exceeding their load capacity. In contrast, runners with a

pace-based schedule that includes a larger proportion of interval-based training sessions may have a greater tolerance for sudden increases in running distance, as they may not be operating at the upper limit of load capacity.¹⁸ To our knowledge, this association has not been reported.

The purpose of this study was to investigate the association between changes in weekly running distance and RRI, and to examine whether this association could be modified by the type of running schedule the runner followed. A dose-response relationship was hypothesized to be found between changes in training load and RRI. Further, it was expected that this association could be modified by the specific running schedule followed, with more RRIs in those who followed a distance-based schedule versus a pace-based schedule.

METHODS

Study Design

THIS STUDY WAS PART OF A BROADER research program called Project-Run21, designed as a nationwide, prospective epidemiological cohort study in Denmark with a 14-week follow-up. The follow-up period was a preparatory phase to prime participants for a half marathon road race. In this more specific study, the participants freely chose 1 of 2 different running schedules: a distance-based or a pace-based schedule. To reduce the number of dropouts from each running schedule and to mitigate the risk of selection bias, the lead investigator (C.D.) maintained ongoing communications with participants. A detailed description of the study design has been published elsewhere.⁸

This study was approved by the local institutional human research ethics committee (project number 187/2015). The Danish Data Protection Agency approved the study, including its data-collection procedures (journal number 2015-57-0002; Aarhus University journal number 62908, serial number 224). Informed consent of participation was

implied by returning the baseline questionnaire. Reporting of the study followed the STrengthening the Reporting of OBservational studies in Epidemiology (STROBE) statement,³⁸ except for the reporting of descriptive data regarding participant demographics (item 14), which is given for each effect modifier instead of by exposure.

Study Population

Healthy participants of all levels, with no weekly running restrictions, were eligible to participate. Participants were recruited during summer and fall 2016 via advertisements placed in sports shops, running clubs, universities, news media, and social media. Potential participants were screened for eligibility using an online questionnaire and included in the study if they (1) were between 18 and 65 years of age, (2) agreed to use a global positioning system (GPS) watch or an application for an Android or iOS smartphone to quantify their running training load, and (3) agreed to report running data and RRI status on a daily and a weekly basis, respectively.

Participants were excluded if they (1) reported an RRI in the lower musculoskeletal extremity or lower back, and/or any other injury that limited their running activity within the 6 months prior to baseline; and/or (2) had contraindications for vigorous physical activity, including symptoms of heart or chest pain, previous heart or chest surgery, lung diseases, dizziness or discomfort when physically active, pregnancy, or nonregulated diabetes, or were for any other reasons diagnosed by their general practitioner.

Data Collection

Data on Running Activity Data on running activity were captured with a GPS watch or smartphone application and manually uploaded to an internet-based training diary (Help2run ApS, Hornslet, Denmark). To reduce the risk of potential information bias following missing or inaccurate uploading of running data, the uploading process occurred daily

through automatically generated e-mails sent out by the system. The data included information about whether the participant had run on the present day (yes/no) and, if yes, the distance run. When the participant did not reply to the daily training e-mails (or to the weekly injury e-mails described subsequently), a reminder e-mail was sent out the next day to enhance compliance. The time taken to complete the daily questionnaire was approximately 2 minutes. The research team had direct access to data for surveillance purposes.

Outcome Measures The primary outcome of interest was an RRI, based on the consensus definition³⁹: “running-related (training or competition) musculoskeletal pain in the lower limbs that causes a restriction on or stoppage of running (distance, speed, duration, or training) for at least 7 days or 3 consecutive scheduled training sessions, or that requires the runner to consult a physician or other health professional.” Information regarding RRI was gathered on a weekly basis by distribution of an additional automatically generated e-mail to all runners.

Exposure Measurements The primary exposure was change (increase or decrease) in weekly running distance, defined as the ratio between the cumulative running distance in the present week (current day plus the 6 days prior to that) divided by the cumulative running distance during the previous week (7 days), calculated following each running session. As the running distance differed between sessions and thus between weeks, change in the exposure ratio was time dependent.

Consistent with an existing epidemiological study, which investigated the association between time-dependent changes in training load (minutes per week) and sports injury development,²⁴ the weekly changes in kilometers were categorized a priori into the following 3 exposure states: (1) slight increase or decrease of less than 20%, (2) moderate increase between 20% and 60%, and (3) sudden excessive increase of greater than 60%.

Within the RRI research context, other cutoff values (less than 10% increase or decrease, increase between 10% and 30%, and greater than 30% increase) for time-dependent changes in running distance have been used to investigate their association with development of RRI.^{5,28} Both of those studies found no significant difference in RRI risk between the 3 exposure states but did note a trend toward a higher hazard ratio for runners increasing their distance by more than 30%.²⁸ Therefore, it was deemed more appropriate to apply the cutoff values used by Møller et al²⁴ to investigate whether an association between changes in weekly running distance and RRI exists.

Further, due to the definition of the primary exposure, it was not possible to calculate the exposure ratio in the first week of participation or in the event that a participant had a week without running. As such, data from those weeks were categorized in a fourth group as “not available.” Because this categorization and the exposure ratio were time dependent, the participants were able to move between exposure states (or remain in the same state) after each running session throughout the follow-up period.

Effect-Measure Modifiers The type of running schedule chosen by each participant (distance based or pace based) was included as an effect-measure modifier in order to investigate whether it modified the association between sudden changes in weekly running distance and RRI development.

Statistics

The association between changes in running activity and RRI was analyzed using time-to-event statistics, specifically by using a pseudo-observation method through a generalized linear regression model. Exposure status was modeled as time dependent (multistate model).³⁴ Runners were dropped from the study when their running schedules were discontinued (no running data upload) for any reason.

Cumulative risk difference (RD) at 7 days and 14 days was used as the measure of association²⁹ for time to first RRI, comparing the RRI risk between each exposure state using days as the time scale. Data were analyzed at 21 days, 56 days, and 98 days (last day). Due to the lack of scientific consensus regarding which time scale and time point to use in the analyses, we chose to use days as the time scale and to split the follow-up time into tertiles of days, with 21 (3 weeks), 56 (8 weeks), and 98 days (14 weeks) as time points for the analyses.

Those assumed to have the lowest RRI risk (less than 20% increase or decrease) were used as the reference group. Results were presented with estimated precision (95% confidence interval) and considered statistically significant at $P < .05$. All statistical analyses were conducted using Stata Version 12 (StataCorp LLC, College Station, TX).

RESULTS

A TOTAL OF 1000 PERSONS SIGNED UP to participate in ProjectRun21. After the eligibility assessment, 784 participants were included in the broader research program, of whom 261 were included in this study. Of these 261, 165 and 96 chose to follow the distance-based or the pace-based schedules, respectively. An overview of the inclusion and follow-up process is provided in the **FIGURE**. The demographics of the included participants are presented in **TABLE 1**.

The 261 runners participated for 9283 days (4875 days for the distance-based schedule and 4408 days for the pace-based schedule), covered 20 720 km (11 738 km for the distance-based schedule and 8982 km for the pace-based schedule), and completed 3205 running sessions (1596 sessions for the distance-based schedule and 1609 sessions for the pace-based schedule) until first RRI or being dropped from the study. The numbers of sessions run in each exposure state, and the number of RRI incidences for each exposure state for the 2 running

schedules, combined and separately, are presented in **TABLE 2**.

A total of 56 participants (21.5%) sustained an RRI during follow-up. The crude analyses revealed that significantly more runners were injured when increasing their weekly running distance by 20% to 60% compared with those increasing their distance by less than 20% (RD = 22.6%; 95% confidence interval: 0.9%, 44.3%; $P = .041$) after 21 days. This association was nonsignificant after 56 days and 98 days. No significant differences in RRI susceptibility at 21, 56, and 98 days were found when comparing the greater than 60% state with the less than 20% state. The results from the analyses, including running schedule as an effect-measure modifier on the association between changes in distance and RRI, revealed no significant differences (**TABLE 3**).

DISCUSSION

THE PURPOSE OF THIS STUDY WAS TO explore the association between changes in weekly running distance and RRI, and to examine whether this association was modified by the type of running schedule (distance based or pace based). A dose-response relationship was hypothesized to be found between changes in training load and RRI. Further, it was expected that this association would be modified by the specific type of running schedule followed, with more runners sustaining an RRI following the distance-based schedule versus the pace-based schedule.

Main Findings

The crude results show that significantly more runners were injured when increasing their weekly running distance by 20% to 60% compared with runners who increased their weekly distance by less than 20% after 21 days. This association became nonsignificant after 56 days and 98 days. As no significant differences in RRI susceptibility at 21, 56, and 98 days were found when comparing runners who increased their weekly running by 60% or

greater with those who increased it by less than 20%, a dose-response relationship between change in training load and RRI could not be confirmed.

These findings corroborate those of a previous study that found no dose-response relationship between training load changes of less than a 10% increase or decrease, a 10% to 30% increase, and an increase greater than 30%, although a higher hazard ratio was found for an increase in weekly distance of greater than 30% compared to one of less than 10%.²⁸

To our knowledge, there is limited empirical evidence to support the association between changes in training load and RRI. Future epidemiological studies are needed to further examine the relationship between sudden changes in training load and RRI on a weekly basis. Importantly, because the results of this study only apply to a single increase from one week to another, multiple weekly changes over several weeks should be examined.

Although the findings of our stratified analysis were not statistically significant,

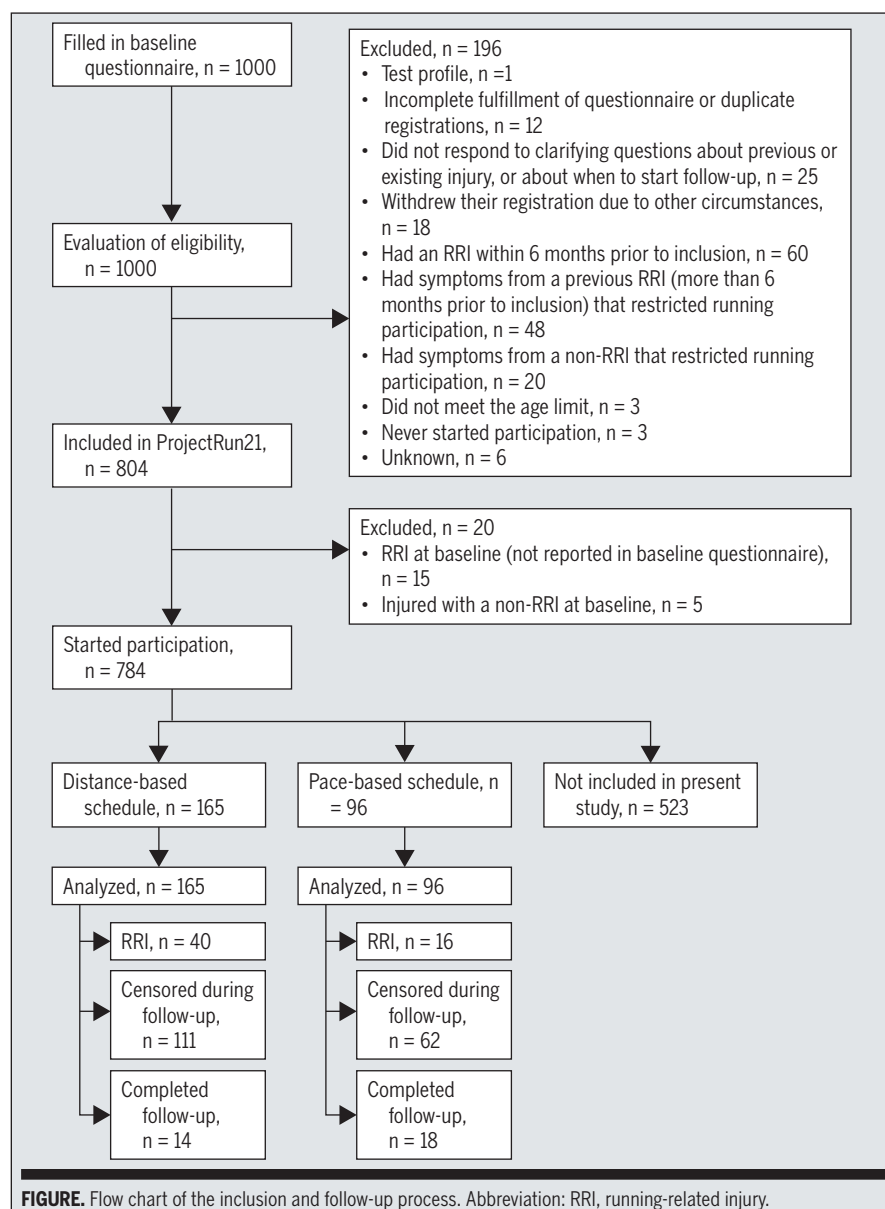


FIGURE. Flow chart of the inclusion and follow-up process. Abbreviation: RRI, running-related injury.

they indicate that approximately 30% more runners in the increased exposure state of 20% to 60% sustained an RRI after both 21 and 56 days when they followed a distance-based schedule compared to a pace-based schedule. As the same tendency applied to runners who increased their weekly running distance by greater than 60%, the results are consistent with our hypothesis that running schedule can modify the effect of changes in training load on RRI. However, given the relatively small sample size and low number of injuries in the present study, this modifying effect of running schedules should be further investigated.

Sudden Change Assessment

Changes in training load can be calculated in several ways. The present study calculated the weekly changes across time points. This approach is similar to those used in other studies that have examined the association between changes in training load and RRI.^{4,28} In the literature, other approaches, such as the acute-chronic workload ratio (ACWR),²² have been used in studies of handball, rugby, and other team sport injuries.^{16,17,24} The ACWR represents the ratio of the “acute load,” defined as the cumulative load in the most recent 7-day period, divided by the “chronic load,” defined as the average load in the prior 4 weeks. The association

between the ACWR and team sport injuries reveals that a ratio of 1.5 or greater is associated with an increased RRI risk compared to a ratio in the “sweet spot” (or low-risk RRI zone), which ranges from 0.8 to 1.3.¹⁷

Unlike the ACWR, the approach of weekly calculation of changes provides more specific information about any instantaneous changes in training load, given that it is based on relative weekly changes versus a rolling average of the absolute training load over the past month. Thus, a weekly training load calculation can examine the association between changes in the applied training load and RRI with greater precision. On the other hand, as the weekly calculation does not consider the scheduling of chronic training loads prior to the 14 days before RRI onset, it remains unknown whether, and to what extent, performances preceding this 2-week period may affect the development of RRI. However, as the training load in the present study was predetermined and thus well known throughout follow-up by the specific running schedule, it enabled us to take into account the longer-term scheduling of training load by performing stratified analyses with respect to running schedule.

Strengths

The prospective nature of the Project-Run21 study has allowed us to account for the temporal association between the primary exposure (training load changes) and main outcome (RRI).¹⁴ Eliminating ambiguities about the direction of causal mechanisms has recently been encouraged in RRI research, and is important from both a theoretical and a practical standpoint to understand how and why RRI develops.³ Furthermore, data were collected on a daily basis, utilizing advanced data-collection devices based on the latest smart technologies. Not only are individual GPS devices more reliable and objective compared to traditional paper-based surveys, but, in the present case of daily data collection, they also enable the use of an advanced statistical

TABLE 1

BASILINE CHARACTERISTICS FOR ALL PARTICIPANTS BY RUNNING SCHEDULE*

	Distance-Based Schedule (n = 165)	Pace-Based Schedule (n = 96)	All (n = 261)
Demographics			
Age, y	37 (27-46)	35 (28-44)	36 (27-45)
Sex, n (%)			
Female	103 (62.4)	54 (56.3)	157 (60.2)
Male	62 (37.6)	42 (43.7)	104 (39.8)
Body mass, kg [†]	76.3 ± 16.0	74.1 ± 13.0	75.5 ± 15.0
Height, cm [†]	173.3 ± 9.6	174.5 ± 8.1	173.8 ± 9.1
Running			
Experience, mo	36 (15-84)	60 (24-120)	48 (20-96)
Average weekly distance, km	5 (3-12)	8 (5-15)	7 (4-14)
Completed half marathon previously, n (%)			
Yes	65 (39.4)	55 (57.3)	120 (46.0)
No	100 (60.6)	41 (42.7)	141 (54.0)
Injury, n (%)			
Previous RRI [‡]			
Yes	49 (29.7)	32 (33.3)	81 (31.0)
No	116 (70.3)	64 (66.7)	180 (69.0)
Previous other sports injuries [‡]			
Yes	32 (19.4)	19 (19.8)	51 (19.5)
No	133 (80.6)	77 (80.2)	210 (80.5)
Participation in other sports, n (%) [§]			
Yes	84 (50.9)	67 (69.8)	151 (57.8)
No	81 (49.1)	29 (30.2)	110 (42.2)

Abbreviation: RRI, running-related injury.

*Values are median (interquartile range) unless otherwise indicated.

[†]Values are mean ± SD.

[‡]More than 6 months prior to inclusion.

[§]At baseline.

approach involving analyses of time-dependent exposures on RRI risk.

Limitations

Although the present study included more than 700 runners, the number of RRIs that occurred restricted the number of variables available to be examined in the stratified analyses. Additionally, due to the potential for sparse data bias (fewer than 5 injuries per exposure group), the results must be carefully interpreted.¹³

Another concern is possible selection bias, due to runners being allowed to self-select their running schedule: if runners' preference for a selected schedule is governed (to some extent) by their ability (perceived or experienced) to follow that schedule without getting injured, then self-selection may have resulted in underestimation of injury risk in each of the schedule groups. However, as the number of runners in each group having selected a schedule based on injury risk is likely comparable, underestimation is assumed to be the same for each group.

There is also the question of whether running distance, as used in the present study, is the most appropriate variable for

representing training load. A recent article promoting the theoretical, causal framework underpinning RRI development³ highlighted that the number of steps taken is a superior exposure measurement compared to distance as a means of quantifying training load. A reason for this has been suggested in biomechanical studies observing that the number of steps

taken over a given running distance (eg, 10 000 m) differed considerably between runners.^{31,32} As each step produces a series of forces through the musculoskeletal structures of the body, the number of steps has been promoted as an important measure in large-scale epidemiological studies.³ Unfortunately, it was not possible to quantify number of steps in this study, as

TABLE 2

RUNNING-RELATED INJURY INCIDENCE AND TRAINING INFORMATION FOR EACH RUNNING SCHEDULE BY EXPOSURE STATE

	<20%	20%-60%	>60%	NA
Both schedules				
RRI incidence (n = 56), n	23	11	11	11
Sessions, n	1328	500	757	620
Distance run, km	8810	3385	5283	3242
Distance-based schedule				
RRI incidence (n = 40), n	14	7	9	10
Sessions, n	670	191	380	355
Distance run, km	5222	1450	3092	1974
Pace-based schedule				
RRI incidence (n = 16), n	9	4	2	1
Sessions, n	658	309	377	265
Distance run, km	3588	1935	2191	1268

Abbreviations: NA, not available; RRI, running-related injury.

TABLE 3

CUMULATIVE RISK-DIFFERENCE ESTIMATES AFTER 21, 56, AND 98 DAYS OF FOLLOW-UP*

Days/Exposure State [†]	All Participants (n = 261)	P Value	Distance-Based Schedule (n = 165)	P Value	Pace-Based Schedule (n = 96)	P Value	Effect-Measure Modification	P Value
21 d								
<20% (reference)	0		0		0		0	
20%-60%	22.6 (0.9, 44.3)	.041	38.3 (-4.9, 81.4)	.082	9.0 (-13.2, 31.1)	.427	29.3 (-19.2, 77.8)	.236
>60%	5.8 (-8.4, 20.1)	.422	9.6 (-10.4, 29.5)	.347	0.9 (-19.2, 20.9)	.933	8.7 (-19.6, 37.0)	.547
56 d								
<20% (reference)	0		0		0		0	
20%-60%	19.0 (-11.9, 50.0)	.228	37.9 (-16.1, 91.9)	.169	7.6 (-30.3, 45.5)	.695	30.3 (-35.7, 96.3)	.368
>60%	7.8 (-14.0, 29.6)	.482	19.7 (-13.1, 52.5)	.239	-12.3 (-35.3, 10.8)	.296	32.0 (-8.1, 72.1)	.118
98 d								
<20% (reference)	0		0		0		0	
20%-60%	4.2 (-26.2, 34.7)	.785	11.6 (-38.7, 62.0)	.650	-1.7 (-39.8, 36.5)	.931	13.3 (-49.9, 76.5)	.680
>60%	-4.3 (-27.9, 19.3)	.722	0.4 (-35.4, 36.3)	.980	-22.3 (-48.6, 4.0)	.097	22.7 (-21.7, 67.1)	.317

*Values in parentheses are 95% confidence interval. The cumulative running-related injury incidence proportions for the crude analyses were 8.7 (5.3, 12.1), 27.5 (15.8, 39.3), and 41.8 (24.6, 58.9) after 21, 56, and 98 days of follow-up, respectively. For the distance-based schedule, the cumulative running-related injury incidence proportions were 9.3 (8.3, 10.3), 32.5 (15.2, 49.8), and 52.3 (25.1, 80.0) after 21, 56, and 98 days of follow-up, respectively. For the pace-based schedule, the cumulative running-related injury incidence proportions were 7.7 (0.2, 15.7), 21.8 (5.8, 37.8), and 32.5 (11.5, 53.6) after 21, 56, and 98 days of follow-up, respectively.

[†]Weekly changes in distance (kilometers).

current technologies are limited in this regard.

It has been suggested^{27,28} that certain injuries develop due to application of different structure-specific loads by different running activities, such as distance-based versus pace-based running regimes. Unfortunately, the number of injuries did not allow for analysis in a competing-risk setting.

Last, it is possible that the results represented in the current article might have differed if other cutoff values had been used,⁶ and it should be noted that the results only apply to a single increase from one week to another, whereas a weekly increase over several weeks was not investigated. This could lead runners to assume that just keeping the weekly increase to less than 20% would keep the injury risk low, no matter how the runners schedule their running. For instance, the injury risk may be low when increasing the weekly distance by, for example, 5%, then by 7%, and finally by 3%, but may be too high to tolerate for most runners when progressing by 18%, then by 18%, and again by 18% over a 4-week period, even though each progression did not exceed 20%.

Perspective

Seen from a clinical and a practical standpoint, studies shedding light on the “too much, too soon” theory among runners with different characteristics must be given careful attention. Importantly, weekly changes in training load alone are unable to cause RRI, as other variables influence the amount of change in training load a runner can tolerate before RRI occurs.³ Effectively, it is important to examine how runners of different shapes and sizes and with different running histories can tolerate changes to their running distance (or, even better, number of steps) in a noninjurious manner. In ProjectRun21, the influence of changes in running distance within strata of a capacity-related variable (type of running schedule) was analyzed. This analysis of different running schedules can help runners and

coaches better understand the trade-offs required in the application of training loads in the form of distance-based running and pace-based running, and thus how to structure their running schedules to optimize running performance.

In terms of generalizability of the study results, the broad population of runners included (eg, running experience ranging from 20 to 96 months) increases the external validity of the study. However, as the study population was heterogeneous in several other characteristics (eg, body mass and age), the results can only be generalized to a well-defined, specific subpopulation of runners. In the future, even more effect-measure modifiers, such as but not limited to body mass, previous RRI, and age, should be included. To answer the research question of how much change in training load is injurious among runners with different characteristics, epidemiological studies with very large sample sizes (exceeding 50 000 runners) are needed.

CONCLUSION

COUNTERINTUITIVELY, NO DOSE-RESPONSE relationship was found between changes in training load and RRI, although more runners were injured after 21 days when weekly running distance was increased by 20% to 60% compared to less than 20%. Although the results are not statistically significant, they further indicate that injury susceptibility varies with increased running distance, depending on the type of running schedule followed. ●

KEY POINTS

FINDINGS: Significantly more runners were injured after 21 days of follow-up when increasing their weekly running distance by between 20% and 60% compared with those who increased their running distance by less than 20%. Although not statistically significant, this association may be modified by the type of running schedule followed prior to a sudden increase in running distance.

IMPLICATIONS: Different running schedules have the potential to help runners and coaches better understand how to minimize injury risk.

CAUTION: The results should be interpreted with caution, as they only apply to a single increase from one week to another and not to a weekly increase over several weeks. This could lead runners to assume that just keeping the weekly increase to less than 20% would keep their injury risk low, no matter how the runners schedule their running. In addition, these results should only be generalized with caution to a well-defined, specific subpopulation of runners.

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REFERENCES

1. Australian Bureau of Statistics. Participation in Sport and Physical Recreation, Australia, 2011-12. Canberra, Australia: Australian Bureau of Statistics; 2013.
2. Bahr R, Krosshaug T. Understanding injury mechanisms: a key component of preventing injuries in sport. *Br J Sports Med*. 2005;39:324-329. <https://doi.org/10.1136/bjsm.2005.018341>
3. Bertelsen ML, Hulme A, Petersen J, et al. A framework for the etiology of running-related injuries. *Scand J Med Sci Sports*. 2017;27:1170-1180. <https://doi.org/10.1111/sms.12883>
4. Buist I, Bredeweg SW, Lemmink KA, van Mechelen W, Diercks RL. Predictors of running-related injuries in novice runners enrolled in a systematic training program: a prospective cohort study. *Am J Sports Med*. 2010;38:273-280. <https://doi.org/10.1177/0363546509347985>
5. Buist I, Bredeweg SW, van Mechelen W, Lemmink KA, Pepping GJ, Diercks RL. No effect of a graded training program on the number of running-related injuries in novice runners: a randomized controlled trial. *Am J Sports Med*. 2008;36:33-39. <https://doi.org/10.1177/0363546507307505>
6. Carey DL, Crossley KM, Whiteley R, et al. Modeling training loads and injuries: the dangers of discretization. *Med Sci Sports Exerc*. 2018;50:2267-2276. <https://doi.org/10.1249/MSS.0000000000001685>
7. Cave A, Miller A. Marathon runners sign up in record numbers. *The Telegraph*. March 24, 2016. <http://www.telegraph.co.uk/investing/business-of-sport/marathon-running>
8. Damsted C, Parner ET, Sørensen H, Malisoux L, Nielsen RO. Design of ProjectRun21: a 14-week prospective cohort study of the influence of running experience and running pace on running-related injury in half-marathoners. *Inj Epidemiol*. 2017;4:30. <https://doi.org/10.1186/s40621-017-0124-9>
9. Dansk Atletik Forbund/MotionDANMARK. Løb i Danmark - En rapport om løbsmarkedet. Brøndby, Denmark: Dansk Atletik Forbund/ MotionDANMARK; 2014.
10. Drew MK, Cook J, Finch CF. Sports-related workload and injury risk: simply knowing the risks will not prevent injuries: narrative review. *Br J Sports Med*. 2016;50:1306-1308. <https://doi.org/10.1136/bjsports-2015-095871>
11. Evenson KR, Wen F, Herrington AH. Associations of accelerometry-assessed and self-reported physical activity and sedentary behavior with all-cause and cardiovascular mortality among US adults. *Am J Epidemiol*. 2016;184:621-632. <https://doi.org/10.1093/aje/kww070>
12. Gabbett TJ. The training-injury prevention paradox: should athletes be training smarter and harder? *Br J Sports Med*. 2016;50:273-280. <https://doi.org/10.1136/bjsports-2015-095788>
13. Greenland S, Mansournia MA, Altman DG. Sparse data bias: a problem hiding in plain sight. *BMJ*. 2016;352:i1981. <https://doi.org/10.1136/bmj.i1981>
14. Hill AB. The environment and disease: association or causation? *Proc R Soc Med*. 1965;58:295-300.
15. Hreljac A. Etiology, prevention, and early intervention of overuse injuries in runners: a biomechanical perspective. *Phys Med Rehabil Clin N Am*. 2005;16:651-667. <https://doi.org/10.1016/j.pmr.2005.02.002>
16. Hulin BT, Gabbett TJ, Blanch P, Chapman P, Bailey D, Orchard JW. Spikes in acute workload are associated with increased injury risk in elite cricket fast bowlers. *Br J Sports Med*. 2014;48:708-712. <https://doi.org/10.1136/bjsports-2013-092524>
17. Hulin BT, Gabbett TJ, Lawson DW, Caputi P, Sampson JA. The acute:chronic workload ratio predicts injury: high chronic workload may decrease injury risk in elite rugby league players. *Br J Sports Med*. 2016;50:231-236. <https://doi.org/10.1136/bjsports-2015-094817>
18. Hulme A, Thompson J, Nielsen RO, Read GJM, Salmon PM. Towards a complex systems approach in sports injury research: simulating running-related injury development with agent-based modelling. *Br J Sports Med*. In press. <https://doi.org/10.1136/bjsports-2017-098871>
19. Hulteen RM, Smith JJ, Morgan PJ, et al. Global participation in sport and leisure-time physical activities: a systematic review and meta-analysis. *Prev Med*. 2017;95:14-25. <https://doi.org/10.1016/j.ypmed.2016.11.027>
20. Kluitenberg B, van Middelkoop M, Diercks R, van der Worp H. What are the differences in injury proportions between different populations of runners? A systematic review and meta-analysis. *Sports Med*. 2015;45:1143-1161. <https://doi.org/10.1007/s40279-015-0331-x>
21. Lee DC, Pate RR, Lavie CJ, Sui X, Church TS, Blair SN. Leisure-time running reduces all-cause and cardiovascular mortality risk. *J Am Coll Cardiol*. 2014;64:472-481. <https://doi.org/10.1016/j.jacc.2014.04.058>
22. Malisoux L, Frisch A, Urhausen A, Seil R, Theisen D. Monitoring of sport participation and injury risk in young athletes. *J Sci Med Sport*. 2013;16:504-508. <https://doi.org/10.1016/j.jsams.2013.01.008>
23. Malisoux L, Nielsen RO, Urhausen A, Theisen D. A step towards understanding the mechanisms of running-related injuries. *J Sci Med Sport*. 2015;18:523-528. <https://doi.org/10.1016/j.jsams.2014.07.014>
24. Møller M, Nielsen RO, Attermann J, et al. Handball load and shoulder injury rate: a 31-week cohort study of 679 elite youth handball players. *Br J Sports Med*. 2017;51:231-237. <https://doi.org/10.1136/bjsports-2016-096927>
25. Nielsen RO, Bertelsen ML, Møller M, et al. Training load and structure-specific load: applications for sport injury causality and data analyses. *Br J Sports Med*. 2018;52:1016-1017. <https://doi.org/10.1136/bjsports-2017-097838>
26. Nielsen RØ, Malisoux L, Møller M, Theisen D, Parner ET. Shedding light on the etiology of sports injuries: a look behind the scenes of time-to-event analyses. *J Orthop Sports Phys Ther*. 2016;46:300-311. <https://doi.org/10.2519/jospt.2016.6510>
27. Nielsen RO, Nohr EA, Rasmussen S, Sørensen H. Classifying running-related injuries based upon etiology, with emphasis on volume and pace. *Int J Sports Phys Ther*. 2013;8:172-179.
28. Nielsen RØ, Parner ET, Nohr EA, Sørensen H, Lind M, Rasmussen S. Excessive progression in weekly running distance and risk of running-related injuries: an association which varies according to type of injury. *J Orthop Sports Phys Ther*. 2014;44:739-747. <https://doi.org/10.2519/jospt.2014.5164>
29. Parner ET, Andersen PK. Regression analysis of censored data using pseudo-observations. *Stata J*. 2010;10:408-422. <https://doi.org/10.1177/1536867X1001000308>
30. Pedersen BK, Saltin B. Exercise as medicine - evidence for prescribing exercise as therapy in 26 different chronic diseases. *Scand J Med Sci Sports*. 2015;25 suppl 3:1-72. <https://doi.org/10.1111/sms.12581>
31. Petersen J, Nielsen RO, Rasmussen S, Sørensen H. Comparisons of increases in knee and ankle joint moments following an increase in running speed from 8 to 12 to 16 km·h⁻¹. *Clin Biomech (Bristol, Avon)*. 2014;29:959-964. <https://doi.org/10.1016/j.clinbiomech.2014.09.003>
32. Petersen J, Sørensen H, Nielsen RO. Cumulative loads increase at the knee joint with slow-speed running compared to faster running: a biomechanical study. *J Orthop Sports Phys Ther*. 2015;45:316-322. <https://doi.org/10.2519/jospt.2015.5469>
33. Pilgaard M, Rask S. Danskernes motions- og sportsvaner 2016. Aarhus, Denmark: Idrættens Analyseinstitut; 2016.
34. Putter H, Fiocco M, Geskus RB. Tutorial in biostatistics: competing risks and multi-state models. *Stat Med*. 2007;26:2389-2430. <https://doi.org/10.1002/sim.2712>
35. Running USA. 2014 Annual Half Marathon Report. Troy, MI: Running USA; 2014.
36. Soligard T, Schwellnus M, Alonso JM, et al. How much is too much? (Part 1) International Olympic Committee consensus statement on load in sport and risk of injury. *Br J Sports Med*. 2016;50:1030-1041. <https://doi.org/10.1136/bjsports-2016-096581>
37. Videbaek S, Bueno AM, Nielsen RO, Rasmussen S. Incidence of running-related injuries per 1000 h of running in different types of runners: a systematic review and meta-analysis. *Sports Med*. 2015;45:1017-1026. <https://doi.org/10.1007/s40279-015-0333-8>
38. von Elm E, Altman DG, Egger M, Pocock SJ, Gøtzsche PC, Vandenbroucke JP. The Strengthening of Reporting of Observational Studies in Epidemiology (STROBE) statement: guidelines for reporting observational studies. *J Clin Epidemiol*. 2008;61:344-349. <https://doi.org/10.1016/j.jclinepi.2007.08.011>

org/10.1016/j.jclinepi.2007.11.008

39. Yamato TP, Saragiotto BT, Lopes AD. A consensus definition of running-related injury in recreational

runners: a modified Delphi approach. *J Orthop Sports Phys Ther.* 2015;45:375-380. <https://doi.org/10.2519/jospt.2015.5741>



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Rethinking Dynamic Knee Valgus and Its Relation to Knee Injury: Normal Movement Requiring Control, Not Avoidance

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The risk of knee injury in sport may be related to deviations in lower-limb alignment.^{3,9} Dynamic knee valgus, which occurs across 3 planes of movement and consists of internal rotation and adduction of the femur and concomitant contralateral pelvic drop,⁵ is an example of biomechanical deviation. Although dynamic knee valgus is considered by many to be one of the most important predictors of serious knee injury,⁵ the predictive validity of commonly used screening tests for dynamic knee valgus, such as the vertical drop jump, has recently been questioned.¹ In a prospective study of 710 athletes, there was a poor association between vertical drop jump and anterior cruciate ligament injury risk.⁸ One reason the vertical drop jump continues to be advocated as a screening test, despite limited evidence, may be that dynamic knee valgus has traditionally been examined through a reductionist lens, that is, understanding the nature of a complex thing by reduc-

ing it to the interactions of its parts or to simpler, more fundamental things.¹

With regard to dynamic knee valgus, such a reductionist approach would be the equivalent of trying to simplify the complexity of triplanar movement into component movements and to define a single critical cut point for safe versus unsafe knee range of motion. In this Viewpoint, we argue that assessing the risk of knee injury is complex,² and is affected by determinants such as triplanar lumbopelvic position (we use the term *pelvic pronation*), muscle system tension, and the interaction between these 2 variables.

We endeavor to present pelvic pronation and system tension as a 3-D construct to consider during physical assessments and exercise design, and to recognize dynamic knee valgus as a normal and necessary response to ground reaction forces.

Movement Coupling: Pronation Occurs in 3 Planes

Many physical assessments overlook the movement coupling that occurs throughout the global kinetic chain during sporting activities, specifically the interplay between the lumbopelvic region and the femur. During single-limb loading, contemporary methods involve labeling the position of the femur at the knee; however, the movement of the acetabulum over a fixed femur may be an underappreciated component of dynamic knee valgus. In single-leg stance, the pelvis moving over a fixed femur is normal biomechanics. This

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“socket-over-ball” movement occurring in 3 planes is needed to facilitate the absorption of ground reaction forces and is a necessary component of human movement.

Movement at the lumbopelvic hip complex during ground impact is a combination of 3-D shock-absorbing movements driving the pelvis into an anterior tilt, forward rotation, contralateral drop, and concomitant spinal extension/rotation and relative sacral nutation. Triplanar pelvic motion is analogous to pronation at the foot—an important mechanism for absorbing ground reaction forces⁶ and a key antecedent for effective propulsion during gait.¹⁰ Therefore, a term we define as *pelvic pronation*, like foot pronation, is part of an integrated and complex system, rather than an isolated movement that can predict injury.¹¹ We propose a paradigm shift to viewing pelvic pronation as triplanar, coupling with the lower extremity. Viewing pelvic pronation in this manner will help clinicians employ a 3-D approach in assessing and designing therapeutic exercises. There are also global musculoskeletal consequences to this 3-D pelvic pronation, because as the body moves across 3 planes, the overall tension placed on the muscular system changes.

Considering System Tension and a Global Systems Approach

When attempting to describe how system tension may affect athletic performance, clinicians commonly use a baseball pitcher as an example to illustrate how global positioning of the body can turn stored energy into kinetic energy via a whip-like mechanism throughout the kinetic chain.⁴ The key to efficient pitching mechanics is to connect the global kinetic chain as it spirals from the ground up toward the hand in a form of kinetic linking. Tension increases as energy moves up the kinetic chain. However, when there is a loss of system tension (eg, with a lax anterior shoulder capsule or poor scapular stability), adequate transfer of force to the ball is lost (loss of stored energy), and the velocity of the ball is decreased.⁷

Tak¹² provides an excellent example of how system tension can affect performance in the lower extremity: end range of motion at the hip joint is not a fixed value but is moderated by the relative positions of the pelvis and trunk. Inherent in this finding is the idea that counterrotation of the trunk on the pelvis (rotation about the vertical z -axis) increases tension in the entire musculoskeletal system, analogous to wringing out a wet towel.¹⁴ Although Tak’s research¹² was performed using passive trunk rotation, his data provide further evidence of a shifting paradigm where “kinetic chains” are not only conceptual pathways, but also physical entities dictating viscoelastic tension in the body.¹³

Extrapolating from the hip,¹² triplanar pronation of the trunk and pelvis over a fixed femur may play a key role in moderating levels of musculotendinous tension during athletic tasks. The golf swing is an example of how system tension could affect the lower extremity. Consider a golfer preparing to strike a ball. As the backswing is completed, the left hip and knee maintain alignment with trunk positioning and stability (arrows in panel A of the **FIGURE**), which in turn maintains system tension (represented here by an imaginary rubber band wrapped around the golfer). The rubber band represents the interconnected muscular pathways coursing through the anterior and posterior kinetic chains.^{4,7} Panel B of the **FIGURE** depicts the

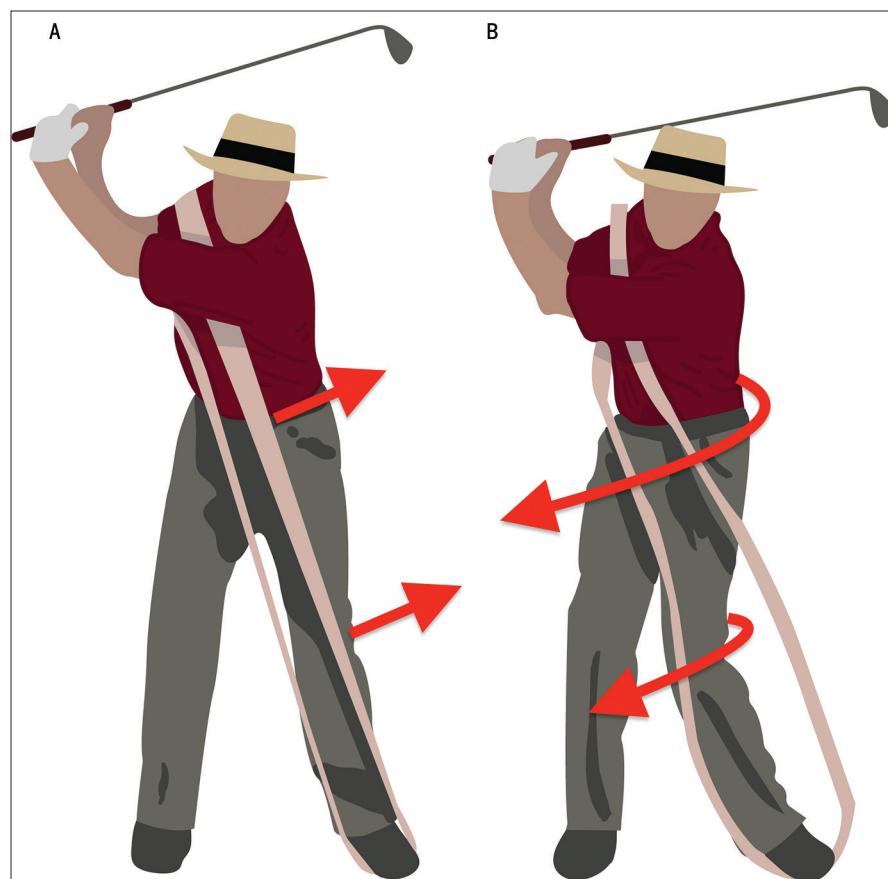


FIGURE. How triplanar pelvic position affects overall system tension. When pelvic pronation occurs, the trunk and pelvis spiral across 3 planes, moving the left side of the pelvis forward and downward toward the stable right lower extremity. The pelvic drop, coupled with this forward and downward motion, forces the right hip into adduction and internal rotation. As the left knee moves from a position of stability (A) to knee valgus (B), the musculoskeletal tension throughout the entire system is now compromised.

left lower extremity undergoing medial collapse, demonstrating dynamic knee valgus. These coupled motions have now resulted in the loss of system tension that was maintained when the golfer could keep the left hemi-pelvis stable.

The rubber band around the golfer may make it easier to perceive the elastic properties of an interconnected muscular system and how efficient propulsion is dependent on the body segments' ability to maintain stability. The possible consequence of a kinetic chain that loses system tension is decreased performance—the pitcher losing velocity or the golfer being unable to hit the ball as far. The 3-D position of the entire musculoskeletal system dictates how much tension can be maintained throughout the body. This is a construct that is separate from determinants like strength, because an individual muscle is only as strong as the position that it is in and the tension it is under. An athlete's ability to maintain global system tension during functional movement may offer a theory as to why some athletes have dynamic knee valgus yet do not sustain a knee injury.

During athletic performance, there are instances when the knee must move beyond a range that a clinician or researcher would regard as “safe” or “correct” alignment. A critical or universal threshold for knee valgus may not exist. Rather, the requisite amount of movement in each plane may be athlete or task dependent. When knee valgus is observed at a single time point or during a specific task, the first question should be, “How will it impact the tension within the global kinetic chain?” rather than, “Is this movement unsafe?”

Clinical Assessment and Exercise Design: Managing Complexity

Clinicians are challenged to appreciate human movement as a whole-system,

3-D construct. The complexity of the human movement system cannot be overstated. Determinants such as strength, balance, endurance, psychological readiness, and self-efficacy are also important. We hypothesize that a triplanar and tension-dependent construct might inform our understanding of knee injury etiology, risk factor modeling, and clinical management.

Key Points

- Dynamic knee valgus is a triplanar movement used to manage ground reaction forces and should be perceived as a normal motion requiring control rather than prevention.
- Three-dimensional positioning of the body affects the global tension in the musculoskeletal system, ultimately affecting performance.
- Appreciating how the trunk and pelvis rotate over a fixed femur (pelvic pronation) may inform new assessment strategies and therapeutic exercise design options for the lower extremity. ●

REFERENCES

1. Beckerman LP. Application of complex systems science to systems engineering. *Systems Eng*. 2000;3:96-102. [https://doi.org/10.1002/1520-6858\(2000\)3:2<96::AID-SYS4>3.0.CO;2-7](https://doi.org/10.1002/1520-6858(2000)3:2<96::AID-SYS4>3.0.CO;2-7)
2. Bittencourt NFN, Meeuwisse WH, Mendonça LD, Nettel-Aguirre A, Ocarino JM, Fonseca ST. Complex systems approach for sports injuries: moving from risk factor identification to injury pattern recognition—narrative review and new concept. *Br J Sports Med*. 2016;50:1309-1314. <https://doi.org/10.1136/bjsports-2015-095850>
3. Boling MC, Padua DA, Marshall SW, Guskiewicz K, Pyne S, Beutler A. A prospective investigation of biomechanical risk factors for patellofemoral pain syndrome: the Joint Undertaking to Monitor and Prevent ACL Injury (JUMP-ACL) cohort. *Am J Sports Med*. 2009;37:2108-2116. <https://doi.org/10.1177/0363546509337934>
4. Chu SK, Jayabalan P, Kibler WB, Press J. The kinetic chain revisited: new concepts on throwing

- mechanics and injury. *PM R*. 2016;8:S69-S77.
5. Hewett TE, Myer GD, Ford KR, et al. Biomechanical measures of neuromuscular control and valgus loading of the knee predict anterior cruciate ligament injury risk in female athletes: a prospective study. *Am J Sports Med*. 2005;33:492-501. <https://doi.org/10.1177/0363546504269591>
6. Horwood AM, Chockalingam N. Defining excessive, over, or hyper-pronation: a quandary. *Foot (Edinb)*. 2017;31:49-55. <https://doi.org/10.1016/j.foot.2017.03.001>
7. Kibler WB, Kuhn JE, Wilk K, et al. The disabled throwing shoulder: spectrum of pathology—10-year update. *Arthroscopy*. 2013;29:141-161. <https://doi.org/10.1016/j.arthro.2012.10.009>
8. Krosshaug T, Steffen K, Kristianslund E, et al. The vertical drop jump is a poor screening test for ACL injuries in female elite soccer and handball players: a prospective cohort study of 710 athletes. *Am J Sports Med*. 2016;44:874-883. <https://doi.org/10.1177/0363546515625048>
9. Lankhorst NE, Bierma-Zeinstra SM, van Middelkoop M. Factors associated with patellofemoral pain syndrome: a systematic review. *Br J Sports Med*. 2013;47:193-206. <https://doi.org/10.1136/bjsports-2011-090369>
10. McDonald KA, Stearns SM, Alderson JA, North I, Pires NJ, Rubenson J. The role of arch compression and metatarsophalangeal joint dynamics in modulating plantar fascia strain in running. *PLoS One*. 2016;11:e0152602. <https://doi.org/10.1371/journal.pone.0152602>
11. Nielsen RO, Buist I, Parner ET, et al. Foot pronation is not associated with increased injury risk in novice runners wearing a neutral shoe: a 1-year prospective cohort study. *Br J Sports Med*. 2014;48:440-447. <https://doi.org/10.1136/bjsports-2013-092202>
12. Tak IJR. Hip and groin pain in athletes: morphology, function and injury from a clinical perspective. *Br J Sports Med*. 2018;52:1024-1025. <https://doi.org/10.1136/bjsports-2017-098618>
13. Wilke J, Krause F, Vogt L, Banzer W. What is evidence-based about myofascial chains: a systematic review. *Arch Phys Med Rehabil*. 2016;97:454-461. <https://doi.org/10.1016/j.apmr.2015.07.023>
14. Zügel M, Maganaris CN, Wilke J, et al. Fascial tissue research in sports medicine: from molecules to tissue adaptation, injury and diagnostics: consensus statement. *Br J Sports Med*. 2018;52:1497. <https://doi.org/10.1136/bjsports-2018-099308>



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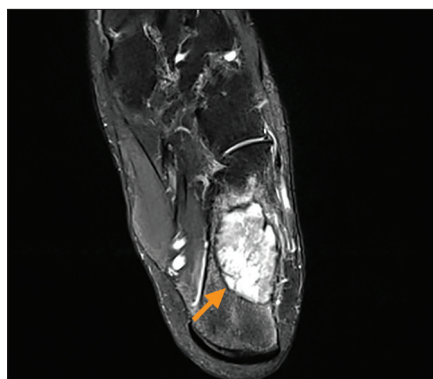


FIGURE 1. Axial, T2-weighted magnetic resonance image demonstrating a hyperintense mass (arrow) within the calcaneus, measuring $3.0 \times 4.6 \times 2.4$ cm and diagnosed as a giant cell-rich calcaneal osteosarcoma.

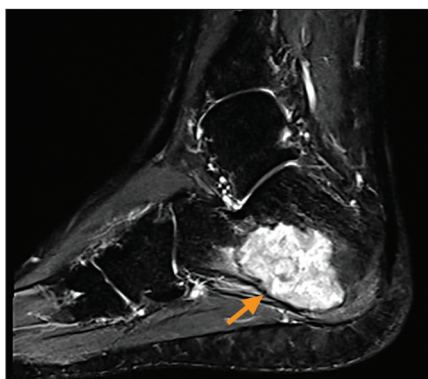


FIGURE 2. Sagittal, short-tau inversion recovery magnetic resonance image demonstrating a hyperintense mass (arrow) within the calcaneus, diagnosed as a giant cell-rich calcaneal osteosarcoma.

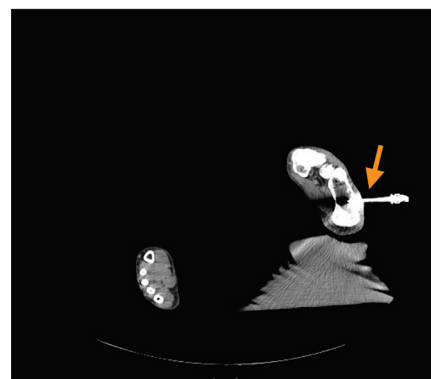


FIGURE 3. Axial, computed tomography-guided percutaneous biopsy of the osteolytic lesion in the left calcaneus, accessed from the lateral plantar foot (arrow).

Calcaneal Osteosarcoma in a Patient Referred for Plantar Heel Pain

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A 28-YEAR-OLD MAN WAS REFERRED TO physical therapy from his primary care provider with variable and intermittent left lateral plantar heel pain that ranged from 0 to 10 on an 11-point numeric pain-rating scale. His pain began insidiously about 10 months prior and was exacerbated by an inversion ankle sprain 7 months prior. Over 6 months before the physical therapy consultation, the patient was evaluated first in an emergency room, twice by his primary care provider, and once by a podiatrist. Four 3-view radiographs of the foot ($n = 3$) and ankle ($n = 1$), completed prior to the physical therapy evaluation, were interpreted as noncontributory.

Red flag screening questions regarding general health, weight changes, or

atypical night pain were negative. Physical examination revealed an antalgic gait with pain at heel strike, sharp pain during deep soft tissue palpation at the proximal cuboid from the plantar aspect, painful plantar flexion in weight bearing, and full and pain-free ankle active/passive range of motion and muscle strength. Initial hypotheses included a midfoot/rearfoot tendon or ligamentous injury.

Because his pain and function were minimally improved after 4 weeks of weight bearing as tolerated, ambulation with axillary crutches, and conservative care, the patient was referred for magnetic resonance imaging (FIGURES 1 and 2). An abnormal mass in the calcaneus prompted an urgent referral to an orthopaedic on-

cologist. Percutaneous biopsy (FIGURE 3) confirmed a diagnosis of calcaneal osteogenic sarcoma. He elected to have a below-knee amputation to maximize functional capacity.

Less than 1% of osteosarcomas occur in small bones, and early detection can lead to increased survival rates.² Most cases are misdiagnosed as soft tissue disorders and subsequently detected at advanced stages, when they become evident on radiographs. Clinicians should consider progression to more sensitive imaging when radiographs are noncontributory and a lack of response to treatment suggests that additional screening is necessary.¹⁻³ *J Orthop Sports Phys Ther* 2019;49(4):285. doi:10.2519/jospt.2019.8430

References

1. American College of Radiology. ACR Appropriateness Criteria: chronic foot pain. Available at: <https://acsearch.acr.org/docs/69424/Narrative/>. Accessed June 25, 2018.
2. Anninga JK, Picci P, Fiocco M, et al. Osteosarcoma of the hands and feet: a distinct clinico-pathological subgroup. *Virchows Arch*. 2013;462:109-120. <https://doi.org/10.1007/s00428-012-1339-3>
3. Young PS, Bell SW, MacDuff EM, Mahendra A. Primary osseous tumors of the hindfoot: why the delay in diagnosis and should we be concerned? *Clin Orthop Relat Res*. 2013;471:871-877. <https://doi.org/10.1007/s11999-012-2570-6>

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Treatment Effect Sizes of Mechanical Diagnosis and Therapy for Pain and Disability in Patients With Low Back Pain: A Systematic Review

Mechanical Diagnosis and Therapy (MDT), commonly referred to as McKenzie exercises, is a specific-subgroup, treatment-based approach used widely by physical therapists for the management of low back pain (LBP). Many randomized controlled trials (RCTs) have investigated the effectiveness of MDT in people with LBP. However, the results have varied, with some finding MDT to be effective^{19,28,42} and others finding no effect.^{21,37,44} Similarly,

the results of existing systematic reviews draw different conclusions regarding the effectiveness of MDT.^{8,27,29} There are a number of possible reasons for the mixed findings from previous trials and systematic reviews. One important difference between studies is that MDT was delivered differently: some trials closely followed the original descriptions and principles upon which MDT was developed and others did not.

Within the framework of MDT, mechanical LBP is classified into 1 of 3 diagnostic syndromes: derangement, dysfunction, and postural.^{35,36} People in the derangement syndrome are treated with repeated or sustained end-range positions according to their directional preference, based on symptom response to end-range loading strategies, which include change in pain location (centralization) and pain intensity and postural correction.^{35,36} In comparison, dysfunction syndrome is managed with repeated end-range movements according to movement loss. The aim is to gradually increase range and reduce the intensity of the pain over time, while the location of central LBP is unchanged.^{35,36} Postural syndrome is managed solely with postural correction techniques.^{35,36}

● **BACKGROUND:** Mechanical Diagnosis and Therapy (MDT) is a treatment-based classification system founded on 3 core principles: classification into diagnostic syndromes, classification-based intervention, and appropriate application of force. Many randomized controlled trials have investigated the efficacy of MDT for low back pain; however, results have varied. The inconsistent delivery of MDT across trials may explain the different findings.

● **OBJECTIVES:** To compare treatment effect sizes for pain or disability between trials that delivered MDT consistent with the core principles of the approach and trials that met some or none of these principles.

● **METHODS:** In this systematic review, databases were searched from inception to June 2018 for studies that delivered MDT compared to nonpharmacological, conservative control interventions in patients with low back pain and reported outcomes of pain or disability. Studies were classified as "adherent" (meeting the core principles of MDT) or "nonadherent" (using some or none of the

principles of MDT). Data were extracted by 2 independent reviewers. Meta-regression procedures were used to analyze the effect of delivery mode on clinical outcomes, adjusting for covariates of symptom duration (less than or greater than 3 months) and control intervention (minimal or active).

● **RESULTS:** Studies classified as adherent to the MDT approach showed greater reductions in pain and disability of 15.0 (95% confidence interval: 7.3, 22.7) and 11.7 (95% confidence interval: 5.4, 18.0) points, respectively, on a 100-point scale compared to nonadherent trials.

● **CONCLUSION:** This review provides preliminary evidence that treatment effects of MDT are greater when the core principles are followed.

● **LEVEL OF EVIDENCE:** Therapy, level 1a.
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● **KEY WORDS:** centralization, directional preference, extension exercises, lumbar spine, MDT

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The existing literature suggests that the core principles of MDT are (1) classification of LBP into diagnostic syndromes, (2) classification-based exercise consisting of repeated or sustained end-range loading strategies and positioning, and (3) appropriate application of force according to syndrome classification, guided by symptom response.^{10,11,32,35,36,48} However, adherence to these core principles, based on the original descriptions, is not consistently tested across clinical trials.

While some trials that report delivering MDT (or the McKenzie method) clearly follow the 3 core principles of MDT, others do not. For example, trials conducted by Hosseinifar et al²³ and Shah and Kage⁴⁷ described a treatment protocol that used “McKenzie’s repeated movement exercises” but did not describe delivery of MDT according to the core principles as we have defined them.^{23,47} Both of these trials reported using only repeated McKenzie’s extension exercises in prone-lying postures for all participants randomized to the MDT exercise group, regardless of the examination findings, which is a key deviation from the core MDT principles as operationally defined for the purpose of this analysis. These trials found no difference between the MDT group and the control group, but it is unclear whether following the core principles contributed to this result. Conversely, Long et al²⁸ reported close adherence to the core MDT principles as we have defined them and reported significantly greater benefit for those receiving MDT compared to unmatched exercises or general exercises.

We are unaware of any trial that has directly compared the delivery of MDT adherent to the core principles as we have defined them to MDT nonadherent to all the core principles. In the absence of any direct comparison, a systematic review using meta-regression could potentially provide preliminary evidence for whether the effects of MDT are different when the core principles are followed compared to a nonadherent approach that lacks some core principles, while controlling for other

study characteristics. No previous systematic review has investigated whether the treatment effects in studies that follow the core principles of MDT differ from those in studies that do not follow the core principles. If the effects of MDT are greater when core principles are followed, then this may assist clinicians in optimizing patient outcomes. Conversely, if the effects are not different, then attention to these principles is less important. This systematic review used regression analysis to compare the treatment effects for pain and disability in trials that used MDT adherent to the 3 core principles (classification, classification-based exercises and positioning, and appropriate application of force according to symptom response) to those in trials that used some or none of the MDT core principles in patients who reported LBP of any duration.

METHODS

Design and Registration

THIS REVIEW IS COMPLIANT WITH THE Preferred Reporting Items for Systematic Reviews and Meta-Analyses (PRISMA) checklist for systematic reviews.³⁸ The review protocol was registered prospectively with PROSPERO (registration number CRD42017054980). We reported prospectively in PROSPERO that we would include RCTs that reported on outcomes for pain, disability, function, and perceived recovery. Our search strategy returned few trials that reported outcomes for function and perceived recovery. Therefore, due to inadequate data, these outcomes were not included in a separate analysis. As disability and function are closely related constructs, trials that only reported on function were included in the analysis for disability.

Data Sources and Searches

We searched the electronic databases of MEDLINE, Embase, Physiotherapy Evidence Database (PEDro), Cochrane Central Register of Controlled Trials, CINAHL, LILACS, and Web of Sci-

ence from their inception to June 2018. We also conducted a hand search of the McKenzie core reference list maintained by the McKenzie Institute International.³⁴ We directly contacted experts in the McKenzie method (members of the McKenzie Institute International) to help identify unpublished trials.

The search strategy included a range of terms for back pain and the McKenzie approach (**APPENDIX**, available at www.jospt.org), using both English and American spellings where differences existed. No language restrictions were imposed on the search strategy.

Study Selection

For the purpose of this review, we only included RCTs that met the following criteria: participants of both sexes, over 18 years of age, who reported nonspecific mechanical LBP with or without leg pain, of any duration. We included trials that broadly tested MDT methods, defined as repeated or sustained end-range movements of the lumbar spine or exercises labeled as McKenzie exercises that were delivered individually for the management of LBP. Trials were evaluated as delivering McKenzie exercises based on their report of the intervention. Comparator interventions included alternative nonpharmacological, conservative treatment, such as exercise, education, placebo, or no treatment. Included trials had to report outcomes for pain or disability. Pain scales included tools such as the numeric pain-rating scale or visual analog scale.^{6,25} Disability and function were measured with the Oswestry Disability Index, Roland-Morris Disability Questionnaire, and Patient-Specific Functional Scale.^{13,43,50}

Two reviewers (A.A. and M.H.H.) independently screened the titles and abstracts to identify potentially eligible articles. The complete manuscripts were then independently reviewed to identify studies that met all the inclusion criteria. Disagreements were resolved by discussion and consensus. When a consensus could not be reached, a third independent reviewer (P.F.) was consulted.

Data Extraction and Quality Assessment

Predictor Variable Following trial screening and prior to collecting trial characteristics and outcome data, trials were categorized into adherent or nonadherent delivery of MDT. A predetermined protocol was used to classify the MDT intervention. To be classified as adherent to MDT, the manuscript had to explicitly describe the following criteria:

1. Classification: the trial specified that an MDT-based assessment was conducted to classify participants into 1 of the MDT diagnostic syndromes. Trials in which a directional preference classification was identified using formal MDT assessment procedures were also considered to have met this criterion. The assessment procedure had to consist of repeated or sustained end-range movements of the spine according to symptom response prior to treatment.
2. Classification-based treatment: the trial described treatment that consisted of repeated or sustained end-range movements of the spine and/or postural education according to the participants' classification or directional preference.
3. Appropriate application of force: the amount of force or load used when applying repeated or sustained end-range movements had to be guided by symptom response.

Any trial that failed to clearly describe and meet 1 or more of these 3 criteria was classified as nonadherent delivery of MDT. Level of McKenzie training was not considered when classifying trials. The classification of manuscripts as adherent or nonadherent was conducted by 2 independent reviewers (M.H.H. and G.M.), who were credentialed as therapists by the McKenzie Institute International. Disagreements regarding classification were resolved by consensus. If consensus was not possible, then a third reviewer (P.F.) with McKenzie training to the level of credentialed therapist resolved the disagreement.

Confounders We extracted data on 2 important potential confounders, symptom

duration and control group. Symptom duration was categorized as either less than or greater than 3 months. When the trial included a population with symptoms of mixed duration, we attempted to contact authors to request subsets of data for outcomes according to symptom duration (less than or greater than 3 months). When we were unable to get these data, we classified trials according to whether the mean pain duration was above or below the 3-month cutoff. When a trial failed to report symptom duration, we used the inclusion criteria of that trial to determine a trial classification of less than or greater than 3 months for symptom duration.

Control interventions were classified as either a minimal or active control. Trials were classified as having an active control when the control intervention included primarily manual therapy, whole-body exercises, or specific exercises. Trials reporting control interventions such as ice, heat, electrophysical agents, massage, education, placebo, or no treatment were classified as having minimal interventions. When a trial reported a multimodal control intervention, including an active and a minimal intervention delivered concurrently, the trial was classified as having an active control. Data were extracted for demographic and clinical characteristics from each trial, which included sample size, age, sex, symptom duration, number of treatments, treatment duration, and level of MDT training for therapists delivering MDT. When the mean and SD values were not available for demographic data, we used the statistical method published by Hozo et al²⁴ to calculate the mean and SD values. We extracted values for mean, SD, and 95% confidence interval (CI), where available, for each of the clinical outcomes for pain and disability, reported at baseline and the closest data point to the 3-month follow-up. All data were extracted by 2 independent reviewers (A.A. and A.G.) and entered into a standard data-collection sheet. Following data extraction, reviewers discussed any differences and reached consensus.

Study quality of each included trial was assessed with the PEDro scale, a reliable tool designed for assessing the methodological quality of RCTs.³¹ The PEDro scale scores range from 0 to 10, calculated from the total number of criteria met for items 2 through 11, with higher scores indicating better methodological quality. Trained raters are used to calculate PEDro scale scores. When a score was not available from PEDro, it was assessed by 2 independent reviewers and discussed if disagreement occurred (A.A. and A.G.).³¹

Data Synthesis

For each included trial, pain and disability outcomes were expressed as mean and 95% CI between-group difference in pain and/or disability at the follow-up time point closest to 3 months. We initially carried out a meta-analysis with a random-effects model of all included studies to investigate the differences between the MDT and control groups, regardless of the trial classification as adherent or nonadherent. We then carried out a random-effects meta-regression to explore the difference in effects between adherent and nonadherent trials. We first fitted crude models, with only the outcome and predictor variable for the hypothesis (adherent or nonadherent MDT). We conducted separate analyses for each outcome (eg, pain or disability). We then ran the primary analysis using adjusted models that included the 2 potential confounders of symptom duration (less than or greater than 3 months) and control intervention (minimal or active). For studies where data were presented separately for participants with pain duration less than or greater than 3 months, we entered the data for each subset as a separate study into the meta-regression. Similarly, when a trial had differently classified control groups that received active and minimal interventions, we entered the data into the meta-analysis as a separate study for each control group. In these cases, the number of participants in the MDT arm was divided by the number of control groups, as recommended in the *Cochrane Handbook*

for *Systematic Reviews of Interventions*.²² All analyses were performed using Comprehensive Meta-Analysis Version 3 Pro (Biostat, Englewood, NJ).

RESULTS

OUR SEARCH STRATEGY RETURNED A total of 649 citations. Following removal of duplicate titles, a total of 305 citations remained. Screening of citations, using titles and abstracts, resulted in a total of 69 potentially relevant trials, for which the complete manuscripts were retrieved. Following full-text review, 24 trials were included (FIGURE 1). However, 1 trial that reported pain as an outcome variable was not included in the analysis, as no data were presented⁴⁹ and data could not be acquired.

Characteristics of Included Studies

The remaining 23 trials included in the analysis had a total sample size of 2712 participants. Sample size for individual trials ranged from 25⁴⁵ to 350⁴² participants. The mean age was 42.9 years, and 49% of participants were male. Symptom duration ranged from 2.3 weeks to 200 weeks.^{3,4,44} The number and duration of MDT treatments ranged from 3 treatments over 1 week to 24 treatments over 8 weeks.^{2,3,18,26} Formal MDT training was reported by 11 of 23 trials; 8 described training to the credentialed level of training, 1 reported training at the diploma level of training, and 2 reported delivery of MDT by a certified therapist but failed to specify the level of training (TABLE 1).

Screening of trials for separation into adherent or nonadherent delivery of MDT resulted in 12 trials being classified as adherent and 11 as nonadherent to MDT (TABLE 1). For trials classified as nonadherent, 1 met 2 out of 3 selection criteria for adherent classification.⁴² Two met only 1 of the selection criteria.^{4,33} The remaining nonadherent trials failed to meet any of the MDT core principles (TABLE 1). The researchers responsible for classifying trials (M.H.H. and G.M.)

agreed on all but 2 trials,^{2,42} which were resolved by consensus.

Comparative data between the adherent and nonadherent trials for sample size, age, sex, symptom duration, and level of MDT training are described in TABLE 1. Comparative data for adherent and nonadherent trials were similar for most study-level characteristics. However, the total sample size for adherent trials was 1805 participants, compared to 907 for nonadherent trials. Delivery of MDT by therapists with certified training in the McKenzie method was reported in 8 adherent trials^{5,19,28,30,37,40,41,44} and 3 nonadherent trials^{18,42,45} (TABLE 1).

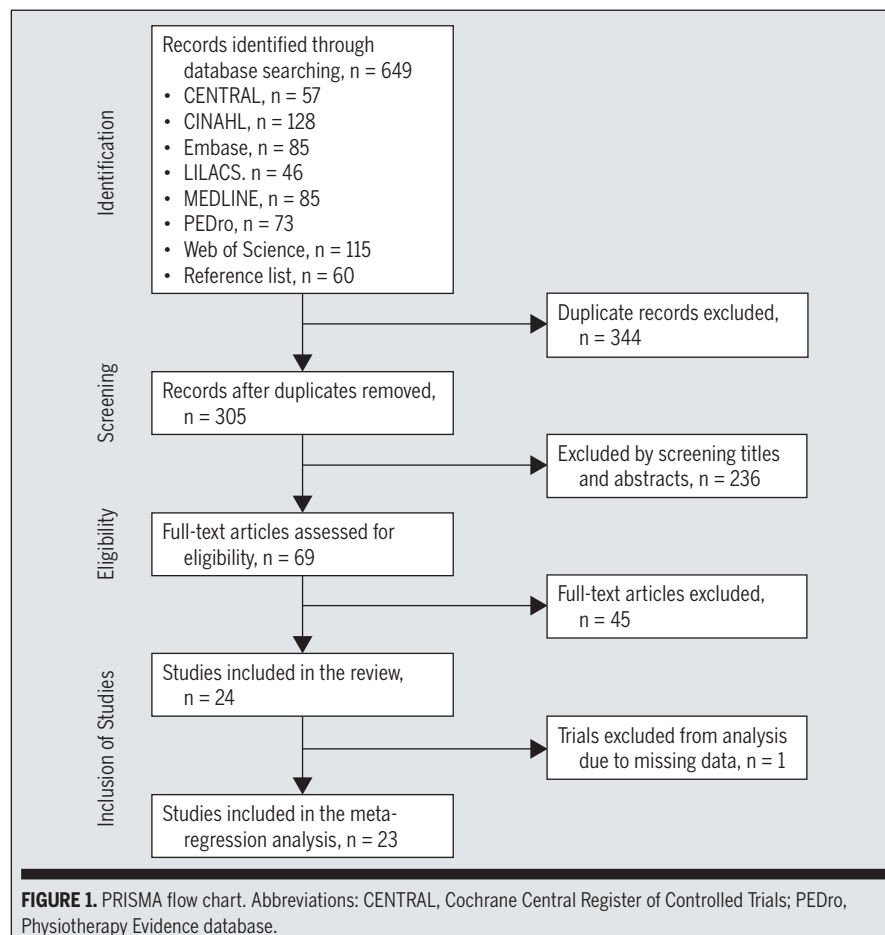
Quality Assessment

Quality scores were available for all of the trials from the PEDro public registry, except for Bid et al,² which was assessed manually. The PEDro scale scores for ad-

herent trials had a median of 6.5, with an interquartile range (IQR) of 5 to 8, while nonadherent trials had a median of 6 and an IQR of 4 to 7 (TABLE 2).

Meta-regression

Two trials provided separate data sets for people with pain duration of less than or greater than 3 months.^{28,40} Seven trials reported multiple controls.^{4,5,21,26,28,33,40} As a result, we reported a total of 31 comparisons for pain from 21 trials (19 comparisons from 12 adherent trials and 12 from 9 nonadherent trials) (FIGURE 2). For disability, there were 31 comparisons from 21 trials (19 comparisons from 12 adherent trials and 12 from 9 nonadherent trials) (FIGURE 3). For example, Paatelma et al⁴⁰ provided subsets of data for participants with baseline symptom duration of less than or greater than 3 months and active or minimal control interventions.



The unadjusted meta-regression found significantly greater effect sizes in trials classified as adherent to MDT compared to those nonadherent to MDT for both pain (15.0 points on a 100-point scale; 95% CI: 7.3, 22.7 points; $P < .001$) and disability (11.7 points on a 100-point scale; 95% CI: 5.4, 18.0 points; $P = .001$) at 3-month follow-up. When we adjusted for the covariates of symptom duration (less than or greater than 3 months) and control intervention (minimal or active), the

TABLE 1

DEMOGRAPHIC AND INDIVIDUAL TRIAL CHARACTERISTICS BY TRIAL CLASSIFICATION (ADHERENT OR NONADHERENT)

Study	Sample Size, n	Age, y*	Sex (Male), %	Mean Pain Duration, wk	Criteria Met, n	Number of Treatments and Duration, wk	Level of MDT Training [†]	Control Intervention
Adherent MDT trials								
Bid et al ²	128	41.2 ± 7.5	50	175 ± 129	3	8 wk	Not certified	Motor control exercises
Cherkin et al ⁵	321	40.7 ± 10.1	52	NR	3	9 sessions over 4 wk	Certified	(1) Manipulation, (2) education booklet
Garcia et al ¹⁶	148	53.9 ± 1.6	55	94.5 ± 233.1	3	4 sessions over 4 wk	Not certified	Back school method
Garcia et al ¹⁷	148	56.5 ± 12.9	24	176.4 ± 415.8	3	10 sessions over 5 wk	Not certified	Placebo
Halliday et al ¹⁹	70	48.6 ± 13.2	20	32.1 ± 26.1	3	12 sessions over 8 wk	Certified	Motor control exercises
Long et al ²⁸	120	42.4 ± 10.6	56	3.5 ± 2.9	3	3-6 sessions over 2 wk	Certified	(1) Opposite-direction exercises, (2) multidirectional exercises
Machado et al ³⁰	146	46.7 ± 14.7	50	<6	3	6 sessions over 3 wk	Certified	First-line care
Miller et al ³⁷	29	49 ± 15.5	51	26 ± 44	3	6 wk	Certified	Stabilization exercises
Murtezani et al ³⁹	271	48.2 ± 8.9	57	>12	3	7 sessions over 4 wk	Not certified	Electrophysical agents
Paatelma et al ⁴⁰	133	44 ± 11.3	64.9	Any duration	3	3-7 sessions	Certified	(1) Orthopaedic manual therapy, (2) advice
Petersen et al ⁴¹	260	34.7 (23-52)	54	185 ± 308	3	15 sessions over 8 wk	Certified	Strengthening exercises
Schenk et al ⁴⁴	31	42 (18-65)	38	2.3	3	3 sessions over 2 wk	Certified	Manipulation
Pooled "original" studies	1805	45.6 ± 6.2	47.5	2.3-185	12 met 3 criteria	3 sessions over 2 wk to 15 sessions over 8 wk	8 certified	Active, 9; minimal, 6 [‡]
Nonadherent MDT trials								
Bonnet et al ³	54	47.4 ± 4.9	53	200	0	3 sessions over 1 wk	None	Mobilization, proprioception, massage, and TENS [§]
Brennan et al ⁴	123	37.7 ± 10.7	55	2.3 ± 1.5	1	8 sessions over 4 wk	None	(1) Motor control exercises, (2) manipulation
Elnaggar et al ¹²	56	39.1 ± 7.2	62	63.0 ± 37.0	0	14 sessions over 2 wk (6 sessions delivered by the therapist and 8 home exercises)	None	Flexion exercises
Gillan et al ¹⁸	40	26-58	48	<12	0	3 sessions over 1 wk	Certified	Massage and education
Hasanpour-Dehkordi et al ²¹	36	40-55	100	>12	0	18 sessions over 6 wk	None	(1) Pilates, (2) no treatment
Hosseinifar et al ²³	30	38.3 ± 9.5	NR	>12	0	18 sessions over 6 wk	None	Motor control exercises
Johnson et al ²⁶	53	45.3 ± 8.1	40	>26	0	24 sessions over 8 wk	None	(1) Education, (2) endurance exercises
Mayer et al ³³	100	31.2 ± 10.6	32	<12	1	5 sessions over 1 wk	None	(1) Heat wrap, (2) education
Petersen et al ⁴²	350	37.5 ± 9.9	44	95.5 ± 204.4	2	15 sessions over 12 wk	Certified	Manipulation
Schenk et al ⁴⁵	25	43 (21-76)	40	NR	0	3 sessions	Certified	Mobilization
Shah and Kage ⁴⁷	40	18-45	41	NR	0	7 sessions over 1 wk	None	Mobilization
Pooled "generic" studies	907	40.2 ± 5.7	56.7	2.3-200	1 met 2 criteria, 2 met 1 criterion	3 sessions over 1 wk to 24 sessions over 8 wk	3 certified	Active, 10; minimal, 5 [‡]

Abbreviations: MDT, Mechanical Diagnosis and Therapy; NR, not reported; TENS, transcutaneous electrical nerve stimulation.

*Values are mean ± SD or range or mean (range).

[†]Certified, credentialed or diplomaed; none, no training; not certified, partial training.

[‡]Active: exercise and/or manual therapy; minimal: heat, electrophysical agents, education, placebo, no treatment.

[§]Control was multimodal (active and minimal).

[RESEARCH REPORT]

effect in adherent MDT trials remained greater than in the nonadherent trials for both pain (15.2 points on a 100-point scale; 95% CI: 7.6, 22.7; $P = .001$) and disability (11.9 points on a 100-point scale; 95% CI: 5.4, 18.4; $P = .001$).

DISCUSSION

Principal Findings

THE KEY FINDING OF THIS STUDY IS that trials that followed all 3 core MDT principles (classification, classification-based exercise and positioning, and application of appropriate force ac-

cording to symptom response) reported larger treatment effects than studies that did not follow all of these principles. This was the case for both outcomes of pain and disability, and the effect remained after adjusting for covariates of symptom duration and control intervention. Our results suggest that strictly following the core principles of MDT may optimize the effect of the intervention.

Interpretation of Findings

The results of our study are somewhat consistent with the findings reported by previous systematic reviews.^{8,27} These pre-

vious reviews only included trials that followed the principles of MDT, although the exact inclusion criteria are a little different from our criteria for adherent trials. In particular, these previous reviews considered trials delivered by certified McKenzie therapists as being adherent to MDT principles. However, we did not consider these as adherent trials unless they met the core principles we described. In comparison, another previous systematic review²⁹ reported limited benefit of MDT for acute LBP of less than 1 week's duration, and moderate benefit of advice to remain active compared to MDT at 3 months. This

TABLE 2

PEDro SCALE

	Item*											
Study	1	2	3	4	5	6	7	8	9	10	11	Score [†]
Adherent MDT trials												
Bid et al ²¹	Yes	Yes	Yes	Yes	No	No	No	No	No	Yes	Yes	5
Cherkin et al ⁵	Yes	Yes	Yes	Yes	No	No	Yes	Yes	Yes	Yes	Yes	8
Garcia et al ¹⁶	Yes	Yes	Yes	Yes	No	No	Yes	Yes	Yes	Yes	Yes	8
Garcia et al ¹⁷	Yes	Yes	Yes	Yes	No	No	Yes	Yes	Yes	Yes	Yes	8
Halliday et al ¹⁹	Yes	Yes	Yes	Yes	No	No	Yes	Yes	No	Yes	Yes	7
Long et al ²⁸	Yes	Yes	Yes	Yes	No	No	Yes	Yes	Yes	Yes	Yes	8
Machado et al ³⁰	Yes	Yes	Yes	Yes	No	No	Yes	Yes	Yes	Yes	Yes	8
Miller et al ³⁷	Yes	Yes	No	Yes	No	No	No	Yes	No	Yes	Yes	5
Murtezani et al ³⁹	Yes	Yes	Yes	Yes	No	No	Yes	Yes	Yes	Yes	Yes	8
Paatelma et al ⁴⁰	Yes	Yes	Yes	Yes	No	No	No	Yes	Yes	Yes	Yes	7
Petersen et al ⁴¹	Yes	Yes	Yes	Yes	No	No	No	Yes	Yes	Yes	Yes	7
Schenk et al ⁴⁴	Yes	Yes	No	Yes	No	No	Yes	No	No	Yes	Yes	5
Nonadherent MDT trials												
Bonnet et al ³	Yes	Yes	Yes	Yes	No	No	Yes	Yes	No	Yes	Yes	7
Brennan et al ⁴	Yes	Yes	Yes	No	No	No	Yes	Yes	Yes	Yes	Yes	7
Elnaggar et al ¹²	No	Yes	No	Yes	No	No	Yes	Yes	No	Yes	Yes	6
Gillan et al ¹⁸	Yes	Yes	No	Yes	No	No	Yes	No	No	Yes	No	4
Hasanpour-Dehkordi et al ²¹	Yes	Yes	No	No	No	No	Yes	No	No	Yes	Yes	4
Hosseiniifar et al ²³	Yes	Yes	No	Yes	No	No	Yes	No	No	Yes	Yes	5
Johnson et al ²⁶	No	Yes	No	Yes	No	No	No	No	No	Yes	Yes	4
Mayer et al ³³	Yes	Yes	No	Yes	No	No	No	Yes	Yes	Yes	Yes	6
Petersen et al ⁴²	Yes	Yes	Yes	Yes	No	No	No	Yes	Yes	Yes	Yes	7
Schenk et al ⁴⁵	Yes	Yes	No	Yes	No	No	No	Yes	No	Yes	Yes	5
Shah and Kage ⁴⁷	Yes	Yes	Yes	Yes	No	No	No	Yes	Yes	Yes	Yes	7

Abbreviations: MDT, Mechanical Diagnosis and Therapy; PEDro, Physiotherapy Evidence database.

*Items: 1, eligibility criteria; 2, random allocation; 3, concealed allocation; 4, baseline comparability; 5, blinded subjects; 6, blinded therapists; 7, blinded assessors; 8, adequate follow-up; 9, intention-to-treat analysis; 10, between-group comparisons; 11, point estimates and variability.

[†]Scores are based on items 2 through 11.

[‡]Scored by study authors; not available in the PEDro database.

review used broader criteria, to include trials that either named the intervention as McKenzie exercises or a derivative or described the use of repeated or sustained

postures. This description fits with our classification of nonadherent trials.

Our results need to be interpreted cautiously, as other differences between

studies may have contributed to the differences we identified. Though we adjusted for pain duration and characteristics of the control group, we did not

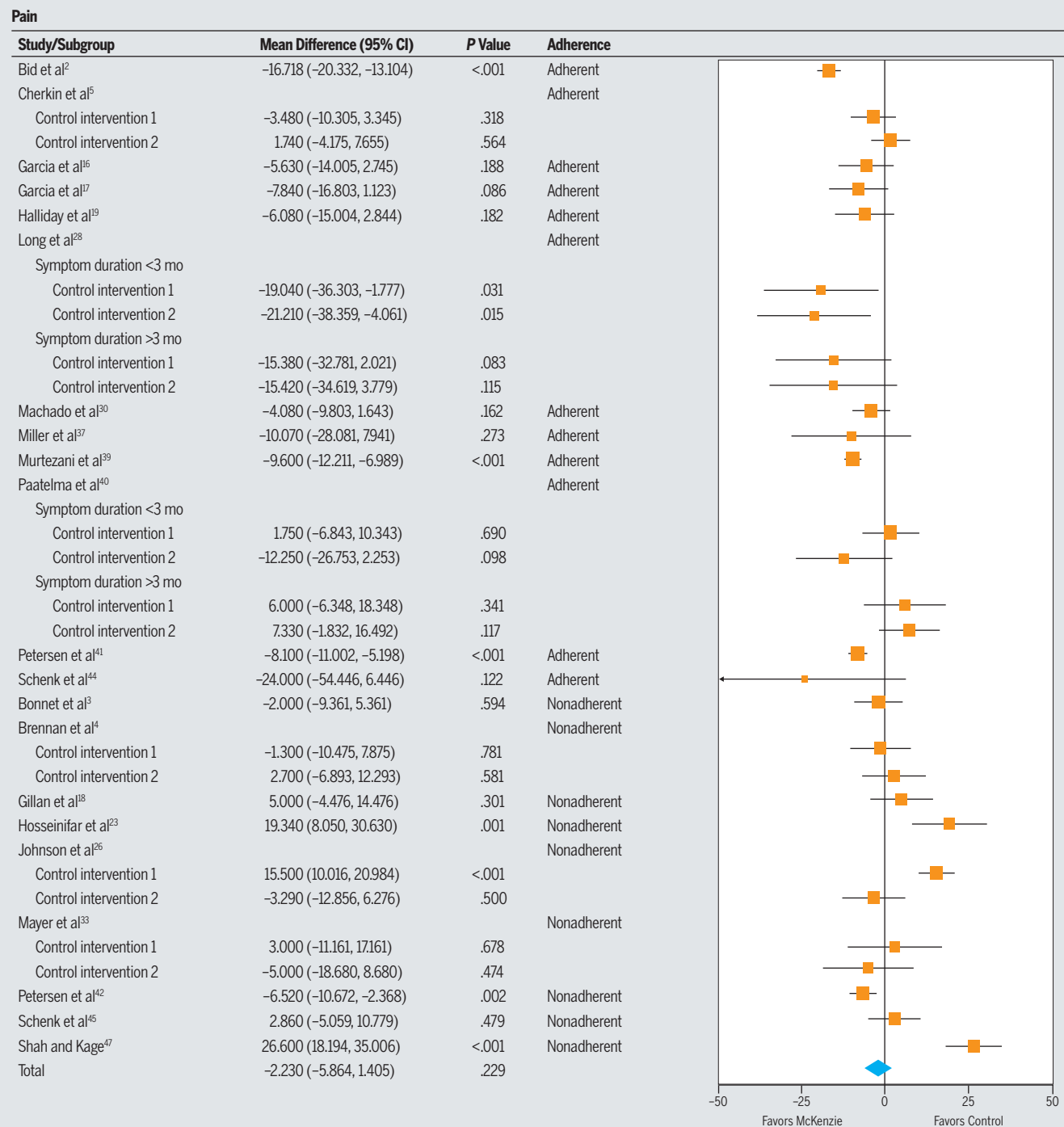


FIGURE 2. Mean differences and 95% CIs by trial for pain.

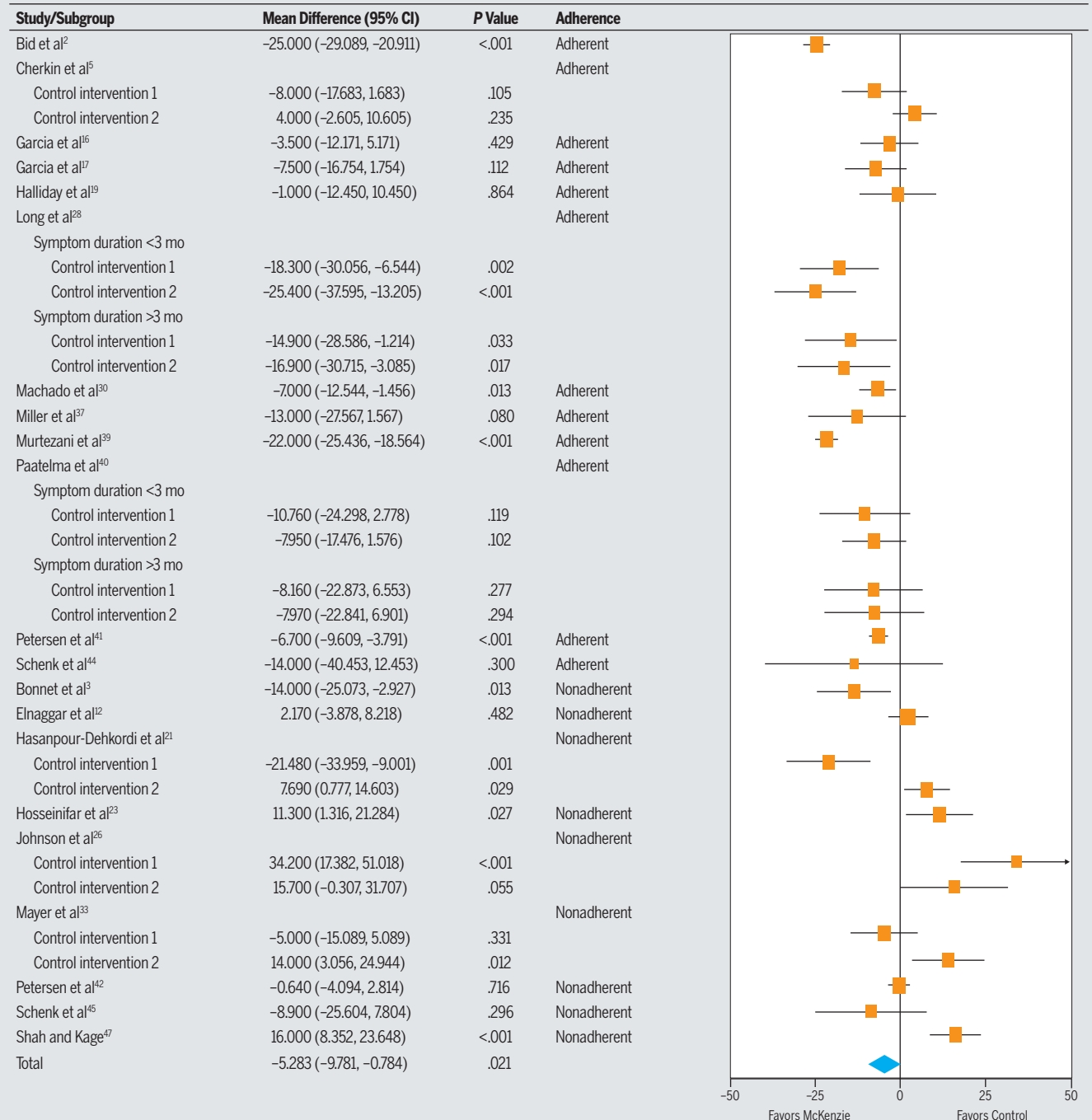
RESEARCH REPORT

have enough power to adjust for factors other than those at the study level. This review included a wide variety of control interventions categorized as either active

or minimal. There were small differences between the adherent and nonadherent trials in terms of median PEDro scale scores, but we did not adjust for this po-

tential confounder. The adherent trials were of slightly higher quality (median, 6.5; IQR, 5-8) compared to the nonadherent trials (median, 5.5; IQR, 4-7), so

Disability



Abbreviation: CI, confidence interval.

FIGURE 3. Mean differences and 95% CIs by trial for disability.

this seems unlikely to be confounding our findings, as lower-quality studies are usually assumed to have inflated effects.⁴⁶ The moderate quality of the included studies also means that the adherent studies may provide biased measures of treatment effects. In addition, while we have used the term *effects* throughout this paper, many of the included trials have characteristics of efficacy studies (eg, carefully selected patients) and may not show the same effects in clinical practice.

Another factor that could have influenced our results was the level of MDT training of the clinicians delivering MDT. Three (27%) of the nonadherent trials reported the delivery of MDT by McKenzie-certified therapists who had received certification from the McKenzie Institute International, compared to 8 (67%) of the adherent trials. However, preliminary evidence suggests that delivery of MDT by certified McKenzie therapists does not produce better outcomes compared to treatment from therapists with no training.⁹ Therefore, this appears unlikely to be an important factor influencing our results.

We observed a small but significant difference in treatment effect size for both pain and disability in adherent MDT trials compared to nonadherent trials (15.2 points on a 100-point pain scale, or 1.5 points on an 11-point numeric pain-rating scale). Some have suggested that this effect size may not be clinically worthwhile, as it has been suggested, for example, that an absolute reduction in pain of at least 2 points on an 11-point scale (20 points on a 100-point scale) is of clinical importance.^{7,14} However, interpreting the clinical importance of the difference in effects is complex and requires consideration of several factors, such as baseline scores, potential harm such as risks and inconvenience, costs, and resource utilization.^{1,14,15,20}

Importantly, our study focused on the difference in effects, and there are potentially no additional risks, inconvenience, or costs required to deliver this therapy according to the core principles. There-

fore, even a small increase in the average effect, as we found, may be worthwhile. However, to determine the clinical importance of the difference in effects, it is important to assess the effect size compared to control in the adherent and nonadherent groups separately.²⁰ Therefore, we conducted unplanned exploratory analyses (not reported in the Results) and found that the effect, compared to control, was 11.1 points less pain in the adherent trials and 3.9 points more pain in the nonadherent trials. Our analysis included trials where the comparison groups varied substantially, from active interventions likely to have similar costs and inconveniences to MDT intervention to minimal or no-intervention controls. When comparing MDT to other similar interventions (eg, manual therapy or an alternative exercise approach), even very small additional effects may be important; however, when comparing MDT to no intervention or placebo, larger effects are required to be considered clinically important.⁷

The strengths of this review include prospective registration of the study protocol on the PROSPERO database, use of PRISMA reporting guidelines,³⁸ and use of a sensitive search strategy. Furthermore, 2 independent reviewers, who did not participate in data extraction, classified trials as adherent or nonadherent.

Despite our prospective guidelines for classifying papers into adherent or nonadherent delivery of MDT, some may dispute the interpretation of the criteria we used, as we were dependent on the information presented in the papers. For example, systematic reviews by Clare et al⁸ and Lam et al²⁷ included RCTs by Gilan et al,¹⁸ Petersen et al,⁴² and Schenk et al,⁴⁵ as the approach was delivered by certified McKenzie therapists. However, we classified these trials as nonadherent because these papers failed to adequately describe the approach. An additional limitation is that several studies did not use any of the 3 core MDT principles, so some might argue that these were not MDT trials. However, we included

these trials because they used terms like *McKenzie exercises*,^{21,23} and some have been included in previous reviews of the McKenzie approach.¹⁸

To eliminate potential confounding and provide a more definitive answer to whether there are differences in effect sizes for the outcomes of pain and disability between adherent and nonadherent MDT, an RCT directly comparing these approaches should be conducted. We also suggest that future clinical practice guidelines for the management of LBP should base their recommendations on evidence from trials and systematic reviews that reported on the delivery of MDT according to the core principles of the approach. Finally, our results suggest that the terms *MDT* or *McKenzie method* should be used only to describe trials that incorporate the core principles of the McKenzie approach.

CONCLUSION

THIS REVIEW PROVIDES PRELIMINARY evidence that the treatment effects of MDT on short-term pain and disability outcomes may be greater when the core principles of MDT—classification of LBP into 1 of the diagnostic syndromes, classification-based treatment using exercise and positions, and appropriate application of force guided by symptom response—are strictly followed. ●

KEY POINTS

FINDINGS: Studies that delivered treatment consistent with the core principles of Mechanical Diagnosis and Therapy (MDT) produced significantly greater reductions in pain and disability compared to studies delivering MDT that did not follow all of the core principles of the approach.

IMPLICATIONS: Based on the available evidence, clinicians who use MDT for the management of low back pain should use an approach consistent with its core principles to optimize outcomes for pain and disability. Researchers investigating the efficacy of MDT for pain and

disability should ensure the intervention reflects the core principles of MDT. Finally, authors of clinical guidelines for the management of low back pain who comment on the efficacy of MDT should be aware that outcomes may differ depending on how closely the core MDT principles were followed.

CAUTION: Classification of trials in this review relied on the descriptions of the MDT intervention provided by the authors of those studies. The results need to be interpreted cautiously, as other differences between studies may have contributed to the differences in outcomes.

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REFERENCES

- Barrett B, Harahan B, Brown D, Zhang Z, Brown R. Sufficiently important difference for common cold: severity reduction. *Ann Fam Med*. 2007;5:216-223. <https://doi.org/10.1370/afm.698>
- Bid DD, Soni NC, Yadav AS, Rathod PV. A study on central sensitization in chronic non-specific low back pain. *Indian J Physiother Occup Ther*. 2017;11:165-175.
- Bonnet F, Monnet S, Otero J. [Short-time effects of a treatment according to the "directional preference" of low back pain patients: a randomized clinical trial]. *Kinesither Rev*. 2011;11:51-59.
- Brennan GP, Fritz JM, Hunter SJ, Thackeray A, Delitto A, Erhard RE. Identifying subgroups of patients with acute/subacute "nonspecific" low back pain: results of a randomized clinical trial. *Spine (Phila Pa 1976)*. 2006;31:623-631. <https://doi.org/10.1097/01.brs.0000202807.72292.a8>
- Cherkin DC, Deyo RA, Battié M, Street J, Barlow W. A comparison of physical therapy, chiropractic manipulation, and provision of an educational booklet for the treatment of patients with low back pain. *N Engl J Med*. 1998;339:1021-1029. <https://doi.org/10.1056/NEJM199810083391502>
- Childs JD, Piva SR, Fritz JM. Responsiveness of the numeric pain rating scale in patients with low back pain. *Spine (Phila Pa 1976)*. 2005;30:1331-1334. <https://doi.org/10.1097/01.brs.0000164099.92112.29>
- Christiansen DH, de Vos Andersen NB, Poulsen PH, Ostelo RW. The smallest worthwhile effect of primary care physiotherapy did not differ across musculoskeletal pain sites. *J Clin Epidemiol*. 2018;101:44-52. <https://doi.org/10.1016/j.jclinepi.2018.05.019>
- Clare HA, Adams R, Maher CG. A systematic review of efficacy of McKenzie therapy for spinal pain. *Aust J Physiother*. 2004;50:209-216. [https://doi.org/10.1016/S0004-9514\(14\)60110-0](https://doi.org/10.1016/S0004-9514(14)60110-0)
- Deutscher D, Werneke MW, Gottlieb D, Fritz JM, Resnik L. Physical therapists' level of McKenzie education, functional outcomes, and utilization in patients with low back pain. *J Orthop Sports Phys Ther*. 2014;44:925-936. <https://doi.org/10.2519/jospt.2014.5272>
- Donelson R. Evidence-based low back pain classification. Improving care at its foundation. *Eura Medicophys*. 2004;40:37-44.
- Donelson R. The McKenzie approach to evaluating and treating low back pain. *Orthop Rev*. 1990;19:681-686.
- Elnaggar IM, Nordin M, Sheikhzadeh A, Parnianpour M, Kahanovitz N. Effects of spinal flexion and extension exercises on low-back pain and spinal mobility in chronic mechanical low-back pain patients. *Spine (Phila Pa 1976)*. 1991;16:967-972.
- Fairbank JC, Couper J, Davies JB, O'Brien JP. The Oswestry low back pain disability questionnaire. *Physiotherapy*. 1980;66:271-273.
- Farrar JT, Portenoy RK, Berlin JA, Kinman JL, Strom BL. Defining the clinically important difference in pain outcome measures. *Pain*. 2000;88:287-294. [https://doi.org/10.1016/S0304-3959\(00\)00339-0](https://doi.org/10.1016/S0304-3959(00)00339-0)
- Ferreira ML, Herbert RD, Ferreira PH, et al. A critical review of methods used to determine the smallest worthwhile effect of interventions for low back pain. *J Clin Epidemiol*. 2012;65:253-261. <https://doi.org/10.1016/j.jclinepi.2011.06.018>
- Garcia AN, Costa LC, da Silva TM, et al. Effectiveness of back school versus McKenzie exercises in patients with chronic nonspecific low back pain: a randomized controlled trial. *Phys Ther*. 2013;93:729-747. <https://doi.org/10.2522/ptj.20120414>
- Garcia AN, Costa LC, Hancock MJ, et al. McKenzie Method of Mechanical Diagnosis and Therapy was slightly more effective than placebo for pain, but not for disability, in patients with chronic non-specific low back pain: a randomised placebo controlled trial with short and longer term follow-up. *Br J Sports Med*. 2018;52:594-600. <https://doi.org/10.1136/bjsports-2016-097327>
- Gillan MG, Ross JC, McLean IP, Porter RW. The natural history of trunk list, its associated disability and the influence of McKenzie management. *Eur Spine J*. 1998;7:480-483. <https://doi.org/10.1007/s005860050111>
- Halliday MH, Pappas E, Hancock MJ, et al. A randomized controlled trial comparing the McKenzie method to motor control exercises in people with chronic low back pain and a directional preference. *J Orthop Sports Phys Ther*. 2016;46:514-522. <https://doi.org/10.2519/jospt.2016.6379>
- Hancock MJ, Kjaer P, Korsholm L, Kent P. Interpretation of subgroup effects in published trials. *Phys Ther*. 2013;93:852-859. <https://doi.org/10.2522/ptj.20120296>
- Hasanpour-Dehkordi A, Dehghani A, Solati K. A comparison of the effects of Pilates and McKenzie training on pain and general health in men with chronic low back pain: a randomized trial. *Indian J Palliat Care*. 2017;23:36-40. <https://doi.org/10.4103/0973-1075.197945>
- Higgins JPT, Green S. *Cochrane Handbook for Systematic Reviews of Interventions*. Oxford, UK: The Cochrane Collaboration; 2011.
- Hosseinfar M, Akbari M, Behtash H, Amiri M, Sarrafzadeh J. The effects of stabilization and McKenzie exercises on transverse abdominis and multifidus muscle thickness, pain, and disability: a randomized controlled trial in nonspecific chronic low back pain. *J Phys Ther Sci*. 2013;25:1541-1545. <https://doi.org/10.1589/jpts.25.1541>
- Hozo I, Schell MJ, Djulbegovic B. Decision-making when data and inferences are not conclusive: risk-benefit and acceptable regret approach. *Semin Hematol*. 2008;45:150-159. <https://doi.org/10.1053/j.seminhematol.2008.04.006>
- Huskisson EC. Measurement of pain. *Lancet*. 1974;304:1127-1131. [https://doi.org/10.1016/S0140-6736\(74\)90884-8](https://doi.org/10.1016/S0140-6736(74)90884-8)
- Johnson OE, Adegoke BO, Ogunlade SO. Comparison of four physiotherapy regimens in the treatment of long-term mechanical low back pain. *J Jpn Phys Ther Assoc*. 2010;13:9-16. <https://doi.org/10.1298/jjpta.13.9>
- Lam OT, Strenger DM, Chan-Fee M, Pham PT, Preuss RA, Robbins SM. Effectiveness of the McKenzie Method of Mechanical Diagnosis and Therapy for treating low back pain: literature review with meta-analysis. *J Orthop Sports Phys Ther*. 2018;48:476-490. <https://doi.org/10.2519/jospt.2018.7562>
- Long A, Donelson R, Fung T. Does it matter which exercise? A randomized control trial of exercise for low back pain. *Spine (Phila Pa 1976)*. 2004;29:2593-2602. <https://doi.org/10.1097/01.brs.0000146464.23007.2a>
- Machado LA, de Souza M, Ferreira PH, Ferreira ML. The McKenzie method for low back pain: a systematic review of the literature with a meta-analysis approach. *Spine (Phila Pa 1976)*. 2006;31:E254-E262. <https://doi.org/10.1097/01.brs.0000214884.18502.93>
- Machado LA, Maher CG, Herbert RD, Clare H, McAuley JH. The effectiveness of the McKenzie method in addition to first-line care for acute low back pain: a randomized controlled trial. *BMC Med*. 2010;8:10. <https://doi.org/10.1186/1741-7015-8-10>
- Maher CG, Sherrington C, Herbert RD, Moseley AM, Elkins M. Reliability of the PEDro scale for rating quality of randomized controlled trials. *Phys Ther*. 2003;83:713-721. <https://doi.org/10.1093/ptj/83.8.713>

32. May S, Donelson R. Evidence-informed management of chronic low back pain with the McKenzie method. *Spine J*. 2008;8:134-141. <https://doi.org/10.1016/j.spinee.2007.10.017>
33. Mayer JM, Ralph L, Look M, et al. Treating acute low back pain with continuous low-level heat wrap therapy and/or exercise: a randomized controlled trial. *Spine J*. 2005;5:395-403. <https://doi.org/10.1016/j.spinee.2005.03.009>
34. McKenzie Institute International. Reference list. Available at: <http://www.McKenzieinstitute.org/clinicians/research-and-resources/reference-list>. Accessed June 30, 2018.
35. McKenzie R, May S. *The Lumbar Spine: Mechanical Diagnosis and Therapy*. 2nd ed. Waikanae, New Zealand: Spinal Publications; 2003.
36. McKenzie RA. *The Lumbar Spine: Mechanical Diagnosis and Therapy*. Christchurch, New Zealand: Spinal Publications; 1981.
37. Miller ER, Schnek RJ, Karnes JL, Rousselle J. A comparison of the McKenzie approach to a specific spine stabilization program for chronic low back pain. *J Man Manip Ther*. 2005;13:103-112. <https://doi.org/10.1179/106698105790824996>
38. Moher D, Liberati A, Tetzlaff J, Altman DG, The PRISMA Group. Preferred reporting items for systematic reviews and meta-analyses: the PRISMA statement. *J Clin Epidemiol*. 2009;62:1006-1012. <https://doi.org/10.1016/j.jclinepi.2009.06.005>
39. Murtezani A, Govori V, Meka VS, Ibraimi Z, Rrecaj S, Gashi S. A comparison of McKenzie therapy with electrophysical agents for the treatment of work related low back pain: a randomized controlled trial. *J Back Musculoskelet Rehabil*. 2015;28:247-253. <https://doi.org/10.3233/BMR-140511>
40. Paatelma M, Kilpikoski S, Simonen R, Heinonen A, Alen M, Videman T. Orthopaedic manual therapy, McKenzie method or advice only for low back pain in working adults: a randomized controlled trial with one year follow-up. *J Rehabil Med*. 2008;40:858-863. <https://doi.org/10.2340/16501977-0262>
41. Petersen T, Kryger P, Ekdahl C, Olsen S, Jacobsen S. The effect of McKenzie therapy as compared with that of intensive strengthening training for the treatment of patients with subacute or chronic low back pain: a randomized controlled trial. *Spine (Phila Pa 1976)*. 2002;27:1702-1709.
42. Petersen T, Larsen K, Nordsteen J, Olsen S, Fournier G, Jacobsen S. The McKenzie method compared with manipulation when used adjunctive to information and advice in low back pain patients presenting with centralization or peripheralization: a randomized controlled trial. *Spine (Phila Pa 1976)*. 2011;36:1999-2010. <https://doi.org/10.1097/BRS.0b013e318201ee8e>
43. Roland M, Morris R. A study of the natural history of back pain. Part I: development of a reliable and sensitive measure of disability in low-back pain. *Spine (Phila Pa 1976)*. 1983;8:141-144.
44. Schenk R, Dionne C, Simon C, Johnson R. Effectiveness of mechanical diagnosis and therapy in patients with back pain who meet a clinical prediction rule for spinal manipulation. *J Man Manip Ther*. 2012;20:43-49. <https://doi.org/10.1179/2042618611Y0000000017>
45. Schenk RJ, Jozefczyk C, Kopf A. A randomized trial comparing interventions in patients with lumbar posterior derangement. *J Man Manip Ther*. 2003;11:95-102. <https://doi.org/10.1179/106698103790826455>
46. Schulz KF, Chalmers I, Hayes RJ, Altman DG. Empirical evidence of bias. Dimensions of methodological quality associated with estimates of treatment effects in controlled trials. *JAMA*. 1995;273:408-412. <https://doi.org/10.1001/jama.1995.03520290060030>
47. Shah SG, Kage V. Effect of seven sessions of posterior-to-anterior spinal mobilisation versus prone press-ups in non-specific low back pain – randomized clinical trial. *J Clin Diagn Res*. 2016;10:YC10-YC13. <https://doi.org/10.7860/JCDR/2016/15898.7485>
48. Simonsen RJ. Principle-centered spine care: McKenzie principles. *Occup Med*. 1998;13:167-183.
49. Stankovic R, Johnell O. Conservative treatment of acute low-back pain. A prospective randomized trial: McKenzie method of treatment versus patient education in "mini back school". *Spine (Phila Pa 1976)*. 1990;15:120-123.
50. Stratford P, Gill C, Westaway M, Binkley J. Assessing disability and change on individual patients: a report of a patient specific measure. *Physiother Can*. 1995;47:258-263. <https://doi.org/10.3138/ptc.47.4.258>



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APPENDIX

SEARCH STRATEGIES

Embase

1. "mechanical diagnosis and therapy".mp. (22)
2. (mechanical diagnosis and therapy).mp. [mp = title, abstract, 'original'title, name of substance word, subject heading word, keyword heading word, protocol supplementary concept word, rare disease supplementary concept word, unique identifier] (24)
3. 1 or 2 (24)
4. (Mechanical diagnosis adj3 therapy).mp. [mp = title, abstract, 'original'title, name of substance word, subject heading word, keyword heading word, protocol supplementary concept word, rare disease supplementary concept word, unique identifier] (24)
5. Directional preference.mp. (202)
6. centralization phenomenon.mp. (15)
7. extension exercise*.mp. (632)
8. flexion exercise*.mp. (332)
9. end range.mp. (244)
10. McKenzie method.mp. (37)
11. McKenzie approach.mp. (11)
12. McKenzie therapy.mp. (13)
13. McKenzie exercise*.mp. (12)
14. 1 or 2 or 3 or 4 or 5 or 6 or 7 or 8 or 9 or 10 or 11 or 12 or 13 (1472)
15. Back Pain/ or Low Back Pain/ (32767)
16. lumbar pain.mp. (1131)
17. Sciatica/ (4807)
18. Radiculopathy/ (4301)
19. 15 or 16 or 17 or 18 (40211)
20. 14 and 19 (202)
21. limit 20 to (clinical study or clinical trial, all or comparative study or controlled clinical trial or randomized controlled trial) (83)
22. limit 21 to humans (83)

MEDLINE

1. "mechanical diagnosis and therapy".mp.
2. (mechanical diagnosis and therapy).mp. [mp = title, original title, abstract, mesh headings, heading words, keyword]
3. 1 or 2
4. (Mechanical diagnosis adj3 therapy).mp. [mp = title, original title, abstract, mesh headings, heading words, keyword]
5. Directional preference.mp.
6. centralization phenomenon.mp.
7. extension exercise*.mp.
8. flexion exercise*.mp.
9. end range.mp.
10. McKenzie method.mp.
11. McKenzie approach.mp.
12. McKenzie therapy.mp.
13. McKenzie exercise*.mp.
14. 1 or 2 or 3 or 4 or 5 or 6 or 7 or 8 or 9 or 10 or 11 or 12 or 13
15. Back Pain/ or Low Back Pain/
16. lumbar pain.mp.
17. Sciatica/
18. Radiculopathy/
19. 15 or 16 or 17 or 18
20. 14 and 19
21. limit 20 to (clinical study or clinical trial, all or comparative study or controlled clinical trial or randomized controlled trial)
22. limit 21 to humans

APPENDIX

PEDro

((mechanical diagnosis and therapy) OR (Directional preference) OR (centralization phenomenon) OR (extension exercise) OR (flexion exercise*) OR (end range) OR (McKenzie method) OR (McKenzie approach) OR (McKenzie therapy) OR (McKenzie exercise*)) AND ((Back Pain) OR (Low Back Pain) OR (lumbar pain) OR (Sciatica) OR (Radiculopathy)) AND (clinical study) OR (clinical trial) OR (comparative study) OR (controlled clinical trial) OR (randomized controlled trial)).

Cochrane Central Register of Controlled Trials

1. "mechanical diagnosis and therapy".mp.
2. (mechanical diagnosis and therapy).mp. [mp = title, 'original'title, abstract, mesh headings, heading words, keyword]
3. 1 or 2
4. (Mechanical diagnosis adj3 therapy).mp. [mp = title, 'original'title, abstract, mesh headings, heading words, keyword]
5. Directional preference.mp.
6. centralization phenomenon.mp.
7. extension exercise*.mp.
8. flexion exercise*.mp.
9. end range.mp.
10. McKenziemethod.mp.
11. McKenzieapproach.mp.
12. McKenzieitherapy.mp.
13. McKenzie exercise*.mp.
14. 1 or 2 or 3 or 4 or 5 or 6 or 7 or 8 or 9 or 10 or 11 or 12 or 13
15. Back Pain/ or Low Back Pain/
16. lumbar pain.mp.
17. Sciatica/
18. Radiculopathy/
19. 15 or 16 or 17 or 18
20. 14 and 19
21. limit 20 to (clinical study or clinical trial, all or comparative study or controlled clinical trial or randomized controlled trial)
22. limit 21 to humans

CINAHL

1. "mechanical diagnosis and therapy" (48)
2. "Directional preference" (38)
3. "centralization phenomenon" (17)
4. "centralization phenomenon" (229)
5. "flexion exercise*" (99)
6. "end range" (177)
7. "McKenzie method" (39)
8. "McKenzie approach" (21)
9. "McKenzie therapy" (14)
10. "McKenzie exercise*" (15)
11. 1 OR 2 OR 3 OR 4 OR 5 OR 6 OR 7 OR 8 OR 9 OR 10 (645)
12. (MH "Low Back Pain") OR (MH "Back Pain+") OR "Back Pain/ or Low Back Pain/" (16923)
13. "lumbar pain" (179)
14. (MH "Sciatica") OR "Sciatica/" (941)
15. (MH "Radiculopathy") OR "Radiculopathy" (1417)
16. 12 OR 13 OR 14 OR 15 (18719)
17. 11 AND 16 (189)
18. 11 AND 16 (Limiters - Human) (132)
19. 11 AND 16 (Limiters - Human; Publication Type: Clinical Trial, Journal Article, Randomized Controlled Trial) (121)

Web of Science

TOPIC: ("mechanical diagnosis and therapy" OR "Directional preference" OR "centralization phenomenon" OR "extension exercise" OR "flexion exercise*" OR "end range" OR "McKenzie method" OR "McKenzie approach" OR "McKenzie therapy" OR "McKenzie exercise*") AND TOPIC: ("Back Pain"

APPENDIX

OR "Low Back Pain" OR "lumbar pain" OR "Sciatica" OR "Radiculopathy") AND TOPIC: ("clinical study" OR "clinical trial" OR "comparative study" OR "controlled clinical trial" OR "randomized controlled trial").

LILACS

((mechanical diagnosis and therapy) OR (Directional preference) OR (centralization phenomenon) OR (extension exercise) OR (flexion exercise*) OR (end range) OR (McKenzie method) OR (McKenzie approach) OR (McKenzie therapy) OR (McKenzie exercise*)) AND ((Back Pain) OR (Low Back Pain) OR (lumbar pain) OR (Sciatica) OR (Radiculopathy)) AND (clinical study) OR (clinical trial) OR (comparative study) OR (controlled clinical trial) OR (randomized controlled trial)).

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Promoting the Use of Self-management Strategies for People With Persistent Musculoskeletal Disorders: The Role of Physical Therapists

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Persistent musculoskeletal pain is a worldwide health problem resulting in negative effects on individuals' well-being and substantial costs to society. Recently, there has been discussion about effective treatment approaches to persistent musculoskeletal disorders, including low back pain.^{5,8,15,23,29}

In this Viewpoint, we argue that self-management strategies are essential to the management of persistent musculoskeletal disorders and outline the physical therapist's role in supporting effective self-management.

Persistent Musculoskeletal Disorders

The most common musculoskeletal disorders include osteoarthritis, rheumatoid arthritis, and spine-related neck and back problems.²⁶ Among occupationally active adults, musculoskeletal disorders are the main cause of disability.²⁶ Musculoskeletal disorders are often work re-

lated and a considerable problem in the workplace—they lead to human suffering, lost time due to sickness absence, and reduced work productivity (presenteeism).¹ Musculoskeletal disorders often have a multifactorial origin and are influenced by multifactorial risk factors, including biomechanical, psychosocial, and individual characteristics.¹⁶ Moreover, many of these musculoskeletal disorders are often resistant to current treatments.^{23,26}

A person-centered approach that focuses on self-management and a healthy

lifestyle is important to restore and maintain function, to improve participation in the long term, and to provide a management plan instead of a cure.^{15,23} We believe that self-management support, as an overall approach to persistent musculoskeletal disorders and their multifactorial biopsychosocial origin, may contribute to the long-term management of these conditions.¹⁹ In this way, patients will feel empowered and have the skills and knowledge to actively manage their condition, even after the initial treatment period has ended.

Self-management

Self-management fits the *positive health concept* ("the ability to adapt and to self-manage, in the face of social, physical, and emotional challenges")¹⁸ as an overarching

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approach to prevent long-term disability from persistent musculoskeletal disorders. Barlow et al⁴ defined self-management as “the ability to manage the symptoms, treatment, physical and psychosocial consequences, and lifestyle changes inherent in living with a chronic condition.”

Patients’ expectations need to change, so that people are less likely to expect a diagnosis or a cure for their pain.¹⁵ This kind of thinking reframes the expectation of a passive treatment approach that cures the condition²³ to that of an active approach (eg, self-management and exercise) as central to long-term management.

Effective self-management is based on skills to actively participate in, and take responsibility for, persistent conditions.²² Essential self-management skills include problem solving, decision making, resource utilization, action planning, self-tailoring, self-monitoring, and creating a patient-health professional partnership.^{4,12,22}

Efficacy of Self-management Programs

There is strong evidence to support the use of self-management in many persistent conditions¹⁴; however, for musculoskeletal disorders, the evidence is equivocal.¹¹⁻¹⁴ The variation in outcomes across study findings could be explained by the low levels of reported participation in self-management programs¹⁷ or by the mode of delivery, considering that layperson-led self-management programs may not be as effective as clinician-led programs.^{2,25}

Another reason for variable outcomes could be the way that self-management programs are provided. Traditionally, these programs use lengthy initial periods of contact conducted in a group format, followed by strategies to continue the self-management interventions without additional consultation.⁶ Potential flaws in this model are that the group approach fails to develop a meaningful therapeutic alliance in which the patient and provider work together to develop a patient-preferred approach, including feedback and adjustment of the self-management program.⁶

Self-management Within Physical Therapy Practice

Physical therapists can contribute to reducing or preventing the negative impact of musculoskeletal conditions.²⁷ Physical therapy as an intervention helps to maintain and improve musculoskeletal health and well-being,²⁷ and exercise and physical activity are effective in treating musculoskeletal pain and improving health.²⁷ Therefore, an active lifestyle and exercise should be the cornerstone of a self-management approach. People also need to understand their condition,²⁷ and a meaningful therapeutic alliance, in which the patient and physical therapist work together to develop a patient-preferred approach, is considered important.⁶

The most effective actions to support self-management include providing self-management support for individuals with specific conditions that is integrated into routine health care and interactive online self-management programs.¹⁴ Programs with individual sessions or in a clinical setting might improve adherence.³ With their expertise in musculoskeletal disorders and the duration of patient contact, physical therapists are ideally positioned to support people with persistent musculoskeletal disorders in their self-management.

What Physical Therapists Do Now

Physical therapists regularly spend time prescribing self-management strategies such as exercise, advice, and the use of heat or ice, suggesting that self-management may be an important complement to in-clinic care.²⁸

What “Good” Self-management Support Should Look Like

Self-management support should address biomechanical, psychosocial, and individual characteristics. To facilitate effective self-management, (1) help the patient to identify his or her barriers and goals, (2) assist in identifying optimal strategies to reduce or avoid symptom exacerbation through problem solving, and (3) support

the patient to identify ways to measure the effectiveness of self-management.

Within a self-management approach, physical therapists can apply principles of cognitive behavioral therapy, shared decision making, acceptance and commitment therapy, mindfulness, motivational interviewing, and pain education.

The connotation of prescribing self-management strategies is undesirable—it downplays the importance of the patient being an active participant in decision making, and emphasizes top-down delivery to a passive patient.¹⁴ Self-management strategies using passive approaches (medication, hot packs) increase the likelihood of pain behavior and disability, while active strategies, such as exercise, decrease the likelihood.⁷

Self-management Support Is Not Simply Patient Education

Patient education usually involves clinicians providing disease-specific information, teaching specific disease-related information, and contingency planning. Self-management support focuses on teaching skills that can be generalized and that patients can use to manage their own health conditions independently.²⁴

Adapting the Approach to the Needs of Different Folks

People are different with regard to self-management competency and health locus of control, despite similar symptoms. Some patients may need more support compared to others.³⁰ It is also important to help patients identify and address possible barriers to self-management⁹ with patient-selected strategies to avoid negative thoughts (eg, creating a list of positive statements posted on the refrigerator at home), stay motivated to achieve their goals (engage family and friends to keep the patient on track), and deal with anxiety and depression (discussion with general practitioner).

Providing Self-management Support in Practice

Physical therapists can provide self-management support that can lead to

enhanced self-management skills, self-management behavior, and long-term management of the musculoskeletal disorder (**FIGURE**). Consider using a behavior change framework, such as the attitude-social influence-efficacy model¹⁰ (with attitude, self-efficacy, social influence, knowledge, and skills as determinants of behavior), and include topics based on the patient's needs and goals. Specific self-management strategies, such as goal

setting, modeling, feedback, discussion, self-monitoring, guided practice, and skill training,²⁰ can foster behavior change and equip patients with the skills needed to actively manage their condition.²²

In this individualized self-management approach, physical therapists focus on developing a "management" plan to limit the impact of the condition on the person's well-being, in close collaboration and partnership with the patient. Prob-

lem solving, decision making, resource utilization, forming a patient-provider partnership, action planning, self-tailoring, and self-monitoring are essential components of this approach. Based on patients' characteristics, needs, and goals, physical therapists can focus on patients' attitudes, self-efficacy, social influence, knowledge, and skills with regard to the long-term management of the musculoskeletal disorder. Johnston et al²¹ provided practical tips for physical therapists to incorporate self-management in practice (**TABLE**).

Key Points

- Physical therapists should use a self-management approach to individualized (physical therapy) treatment for patients with persistent musculoskeletal disorders whenever possible.
- Physical therapists should use customized self-management support, targeting biomechanical, psychosocial, and individual characteristics in their treatment of people with persistent musculoskeletal disorders.

TABLE

PRACTICAL TIPS FOR PHYSICAL THERAPISTS TO INCORPORATE SELF-MANAGEMENT INTO PRACTICE*

- | | |
|--|--|
| <ul style="list-style-type: none"> Establish rapport Adopt an active listening communication style Provide information Develop an action plan Goals of treatment are: <ul style="list-style-type: none"> Specific Measurable Action based Realistic Time framed | <ul style="list-style-type: none"> Use a nonjudgmental approach Facilitate problem solving Promote self-efficacy Link patients with resources Encourage patient to maintain personal health records Active follow-up |
|--|--|

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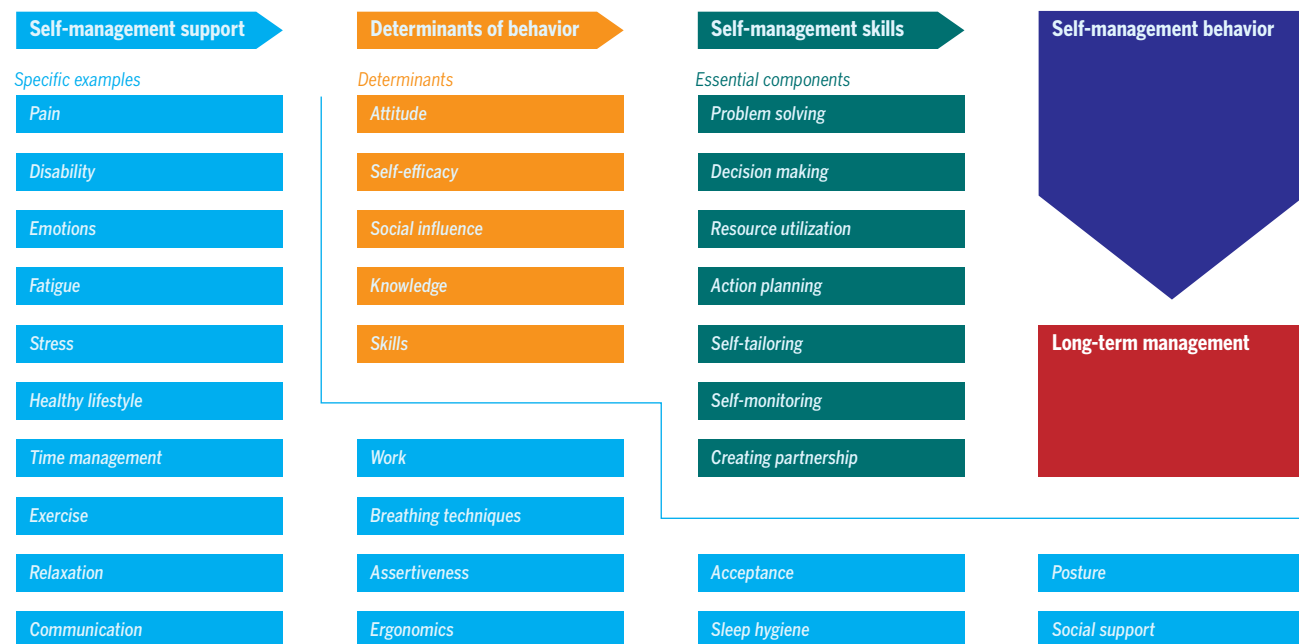


FIGURE. The illustration shows how physical therapist-provided self-management support, with regard to topics based on the patient's characteristics (blue boxes), can influence determinants of behavior (orange boxes) and can lead to enhanced self-management skills (green boxes), practice of self-management behavior (dark blue box), and long-term management (red box) of the musculoskeletal disorder.

- Self-management support should focus on behavior change and an active lifestyle and should target attitude, self-efficacy, social influence, knowledge, and skills with regard to managing the condition.
- Problem solving, decision making, resource utilization, the formation of a patient-provider partnership, action planning, self-tailoring, self-monitoring, and patient-health professional partnership are essential components in this approach. ●

REFERENCES

1. Aas RW, Thingbø C, Holte KA, Lie K, Lode IA. On long term sick leave due to musculoskeletal diseases and disorders. Experiences of work demands. *Work*. 2011;39:233-242. <https://doi.org/10.3233/WOR-2011-1171>
2. Andersen LN, Juul-Kristensen B, Sørensen TL, Herborg LG, Roessler KK, Søgaard K. Efficacy of tailored physical activity or chronic pain self-management programme on return to work for sick-listed citizens: a 3-month randomised controlled trial. *Scand J Public Health*. 2015;43:694-703. <https://doi.org/10.1177/1403494815591687>
3. Bal MI, Sattoe JN, Roelofs PD, Bal R, van Staa A, Miedema HS. Exploring effectiveness and effective components of self-management interventions for young people with chronic physical conditions: a systematic review. *Patient Educ Couns*. 2016;99:1293-1309. <https://doi.org/10.1016/j.pec.2016.02.012>
4. Barlow J, Wright C, Sheasby J, Turner A, Hainsworth J. Self-management approaches for people with chronic conditions: a review. *Patient Educ Couns*. 2002;48:177-187. [https://doi.org/10.1016/S0738-3991\(02\)00032-0](https://doi.org/10.1016/S0738-3991(02)00032-0)
5. Beattie PF, Silfies SP. Improving long-term outcomes for chronic low back pain: time for a new paradigm? *J Orthop Sports Phys Ther*. 2015;45:236-239. <https://doi.org/10.2519/jospt.2015.0105>
6. Beattie PF, Silfies SP, Jordon M. The evolving role of physical therapists in the long-term management of chronic low back pain: longitudinal care using assisted self-management strategies. *Braz J Phys Ther*. 2016;20:580-591. <https://doi.org/10.1590/bjpt-rbf.2014.0180>
7. Blyth FM, March LM, Nicholas MK, Cousins MJ. Self-management of chronic pain: a population-based study. *Pain*. 2005;113:285-292. <https://doi.org/10.1016/j.pain.2004.12.004>
8. Buchbinder R, van Tulder M, Öberg B, et al. Low back pain: a call for action. *Lancet*. 2018;391:2384-2388. [https://doi.org/10.1016/S0140-6736\(18\)30488-4](https://doi.org/10.1016/S0140-6736(18)30488-4)
9. Devan H, Hale L, Hempel D, Saip B, Perry MA. What works and does not work in a self-management intervention for people with chronic pain? Qualitative systematic review and meta-synthesis. *Phys Ther*. 2018;98:381-397. <https://doi.org/10.1093/ptj/pzy029>
10. De Vries H, Mudde AN. Predicting stage transitions for smoking cessation applying the attitude-social influence-efficacy model. *Psychol Health*. 1998;13:369-385. <https://doi.org/10.1080/08870449808406757>
11. Du S, Hu L, Dong J, et al. Self-management program for chronic low back pain: a systematic review and meta-analysis. *Patient Educ Couns*. 2017;100:37-49. <https://doi.org/10.1016/j.pec.2016.07.029>
12. Du S, Yuan C, Xiao X, Chu J, Qiu Y, Qian H. Self-management programs for chronic musculoskeletal pain conditions: a systematic review and meta-analysis. *Patient Educ Couns*. 2011;85:e299-e310. <https://doi.org/10.1016/j.pec.2011.02.021>
13. Elbers S, Wittink H, Pool JJM, Smeets R. The effectiveness of generic self-management interventions for patients with chronic musculoskeletal pain on physical function, self-efficacy, pain intensity and physical activity: a systematic review and meta-analysis. *Eur J Pain*. 2018;22:1577-1596. <https://doi.org/10.1002/ejp.1253>
14. Evidence Centre for National Voices. Supporting Self-management. London, UK: National Voices; 2014.
15. Foster NE, Anema JR, Cherkin D, et al. Prevention and treatment of low back pain: evidence, challenges, and promising directions. *Lancet*. 2018;391:2368-2383. [https://doi.org/10.1016/S0140-6736\(18\)30489-6](https://doi.org/10.1016/S0140-6736(18)30489-6)
16. Hernandez AM, Peterson AL. Work-related musculoskeletal disorders and pain. In: Gatchel RJ, Schultz IZ, eds. *Handbook of Occupational Health and Wellness*. New York, NY: Springer; 2012:63-86.
17. Hoon EA, Gill TK, Pham C, Gray J, Beilby J. A population analysis of self-management and health-related quality of life for chronic musculoskeletal conditions. *Health Expect*. 2017;20:24-34. <https://doi.org/10.1111/hex.12422>
18. Huber M, Knottnerus JA, Green L, et al. How should we define health? *BMJ*. 2011;343:d4163. <https://doi.org/10.1136/bmj.d4163>
19. Hutting N. *Effectiveness of a self-management program for employees with complaints of the arm, neck and/or shoulder* [thesis]. Nijmegen, the Netherlands: HAN University of Applied Sciences; 2015.
20. Hutting N, Johnston V, Richardson J, Walsh N. The role of self-management in the treatment of musculoskeletal disorders [symposium]. World Confederation for Physical Therapy Congress; July 2-4, 2017; Cape Town, South Africa.
21. Johnston V, Jull G, Sheppard DM, Ellis N. Applying principles of self-management to facilitate workers to return to or remain at work with a chronic musculoskeletal condition. *Man Ther*. 2013;18:274-280. <https://doi.org/10.1016/j.math.2013.04.001>
22. Jonkman NH, Schuurmans MJ, Jaarsma T, Shortridge-Baggett LM, Hoes AW, Trappenburg JC. Self-management interventions: proposal and validation of a new operational definition. *J Clin Epidemiol*. 2016;80:34-42. <https://doi.org/10.1016/j.jclinepi.2016.08.001>
23. Lewis J, O'Sullivan P. Is it time to reframe how we care for people with non-traumatic musculoskeletal pain? *Br J Sports Med*. 2018;52:1543-1544. <https://doi.org/10.1136/bjsports-2018-099198>
24. McGowan PT. Self-management education and support in chronic disease management. *Prim Care*. 2012;39:307-325. <https://doi.org/10.1016/j.pop.2012.03.005>
25. Mehlsen M, Hegaard L, Ørnbøl E, Jensen JS, Fink P, Frostholm L. The effect of a lay-led, group-based self-management program for patients with chronic pain: a randomized controlled trial of the Danish version of the Chronic Pain Self-Management Programme. *Pain*. 2017;158:1437-1445. <https://doi.org/10.1097/j.pain.0000000000000931>
26. Murray CJ, Atkinson C, Ali MK, et al. The state of US health, 1990-2010: burden of diseases, injuries, and risk factors. *JAMA*. 2013;310:591-608. <https://doi.org/10.1001/jama.2013.13805>
27. National Institute for Health Research Dissemination Centre. Moving Forward - Physiotherapy for Musculoskeletal Health and Wellbeing. London, UK: National Institute for Health Research; 2018.
28. Peek K, Carey M, Mackenzie L, Sanson-Fisher R. An observational study of Australian private practice physiotherapy consultations to explore the prescription of self-management strategies. *Musculoskeletal Care*. 2017;15:356-363. <https://doi.org/10.1002/msc.1181>
29. Taylor AJ, Kerry R. When chronic pain is not "chronic pain": lessons from 3 decades of pain. *J Orthop Sports Phys Ther*. 2017;47:515-517. <https://doi.org/10.2519/jospt.2017.0606>
30. Wahl AK, Opseth G, Nolte S, Osborne RH, Bjørke G, Mengshoel AM. Is regular use of physiotherapy treatment associated with health locus of control and self-management competency? A study of patients with musculoskeletal disorders undergoing physiotherapy in primary health care. *Musculoskelet Sci Pract*. 2018;36:43-47. <https://doi.org/10.1016/j.msksp.2018.04.008>



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Potential Nervous System Sensitization in Patients With Persistent Lower Extremity Tendinopathies: 3 Case Reports

Tendinopathy is a common condition seen by clinicians in musculoskeletal practice. In the lower extremity, Achilles and patellar tendinopathy have a high incidence and prevalence in the general population and can be functionally disabling.^{1,7,15} Historically, eccentric exercise has been viewed as a standard intervention for chronic tendon dysfunction, with a proposed mechanism of disrupting abnormal collagen cross-linkage to normalize tendon structure.⁴² Current evidence suggests that a variety of exercise-based tendon loading and activity modification prescriptions may be effective, and success is not necessarily linked to a specific protocol.^{10,11,54} Despite the usefulness of exercise-based man-

agement in many cases, a large portion of individuals with tendinopathy report ongoing pain and limited function at 6 to 12 months after intervention, with some being forced to retire from their sport.^{5,9,24} As such, it seems appropriate to continue to investigate optimal inter-

ventions for patients with chronic tendon dysfunction.

Understanding and identifying the pain mechanisms associated with a patient's presentation can be useful in delivering appropriate interventions.⁸ Although tendinopathy is frequently described as local nociceptive pain, the peripheral and central nervous systems demonstrate plasticity in pathological states,²⁷ and sustained peripheral nociceptive activity may lead to the development of peripheral sensitization and/or central sensitization (CS).⁴⁸ If nervous system sensitization has occurred and persists, symptomatic and functional recovery may be enhanced by specifically addressing altered pain mechanisms.⁶¹

Recent investigations have suggested that tendinopathy may be associated with sensitization of the nervous system. A systematic review of primarily upper extremity tendinopathy found that mechanical pressure algometry was reduced locally at the site of tendinopathy as well as remote sites,⁴⁴ the latter being indicative of CS. While animal models support altered central mechanisms in the presence of tendinopathy,² evidence correlating nervous system dysfunction in lower extremity tendinopathy is limited and conflicting. In persons with Achilles tendinopathy, conditioned pain modulation was reduced in individuals with, compared to

● **BACKGROUND:** Tendinopathy is a condition often associated with pain and functional and sport performance limitations. While targeted exercise prescriptions are often effective, many patients with tendinopathy develop persistent symptoms. Emerging evidence suggests a possible link between nervous system sensitization and tendinopathy. If so, identifying and treating specific pain mechanisms may improve outcomes.

● **CASE DESCRIPTION:** Three patients were seen in physical therapy for complaints of ongoing chronic tendon pain and self-reported disability, despite being treated previously and receiving evidence-informed care. Upon examination, each patient demonstrated signs consistent with possible dysfunction of central pain mechanisms. Joint mobilization, pain neuroscience education, and aerobic exercise were primary interventions in each case to decrease pain and improve function.

● **OUTCOMES:** The 3 patients were treated for 5 sessions over the course of 8 weeks. Clinically significant improvement was noted in measures of pain, self-reported function, and pressure pain thresholds. At discharge, all patients were able to run without symptoms, and improvement was maintained at 1-year follow-up.

● **DISCUSSION:** Tendinopathy, while often described as local pain and dysfunction, may be associated with dysfunction of the nervous system. Identifying and treating pain mechanisms in addition to relevant impairments may be an appropriate intervention approach for individuals with tendinopathy.

● **LEVEL OF EVIDENCE:** Therapy, level 4. *J Orthop Sports Phys Ther* 2019;49(4):272-279. Epub 13 Feb 2019. doi:10.2519/jospt.2019.8600

● **KEY WORDS:** central sensitization, manual therapy, pain mechanisms, pain science, tendon

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individuals without, the condition,⁵⁹ and disrupted tactile acuity was reported.¹⁴ In a group of subjects with patellar tendinopathy, mechanical pain and vibration disappearance thresholds were altered.⁶⁰ Alternatively, a blinded case-control study reported that subjects with patellar and Achilles tendinopathy did not exhibit features of CS.⁴⁶ The conflicting results related to nervous system sensitization in lower extremity tendinopathy suggest that more research is warranted.

While a primary pain mechanism may be identified clinically, multiple mechanisms may be at fault in a patient's presentation.⁸ For example, with tendinopathy, even if CS exists, there is likely a peripheral nociceptive component contributing to consistent and predictable aggravating and alleviating activities, and treatment should identify and target both mechanisms. To this end, interventions commonly used in physical therapy practice, namely joint mobilization, pain neuroscience education (PNE), and aerobic exercise, have been associated with pain reduction and dampening of aberrant peripheral and central pain mechanisms.^{4,8,34,38} Joint mobilization has been effective in a variety of musculoskeletal conditions for modulating both peripheral sensitization and CS.^{13,21,64} While PNE and aerobic exercise do not target a specific tissue peripherally, a complex series of interactions may modify top-down sensitization through processes such as deactivation of the periaqueductal gray area or enhancement of the endogenous opioid system.^{26,30} Although each intervention has been shown to be effective in modulating pain independently, the combination of these interventions may have additive effects in managing symptoms of individuals with localized nociceptive pain reports and concurrent nervous system sensitization.^{6,32,47} To date, there have been no reports utilizing a combination of these interventions in the physical therapy management of chronic lower extremity tendinopathy.

The purpose of this article was to describe the physical therapy management

of 3 individuals with lower extremity tendinopathy, using joint mobilization, PNE, and aerobic exercise to modulate possible nervous system sensitization.

CASE DESCRIPTION

THIS CASE SERIES WAS PERFORMED retrospectively on 3 patients seen in physical therapy for complaints of persistent lower extremity tendinopathy. Each patient had ongoing pain, self-reported functional disability for at least 1 year, was treated multiple times without complete resolution, received corticosteroid injections to the involved tendon without benefit, and had magnetic resonance imaging findings suggesting chronic tendinopathic changes. None of the patients had any reported or observed mental health conditions. Patients provided verbal consent to have their data published. The George Washington University Institutional Review Board was contacted and deemed this work exempt from review.

History

Patient 1, a 38-year-old man, presented with a 4-year history of right-sided insidious Achilles pain. Symptoms were localized to the midportion of the tendon and described as a sharp ache. Symptoms were aggravated with running more than 10 minutes, regardless of surface, and relieved transiently with ice massage. Over-the-counter ibuprofen did not help his pain. He did not report substantial functional disability with daily activities, although he was unable to run because of pain. Prior to evaluation, the patient was seen by 3 different physical therapists on 4 different occasions, and despite short-term improvement, no lasting effects were reported. Previous therapies on average lasted 4 to 5 months and included eccentric, concentric, and isometric ankle exercises; low-level laser therapy; calf stretching; and balance exercises, which have been useful in the management of Achilles tendinopathy.³⁶

Patient 2, a 42-year-old woman, presented to physical therapy with a 22-month history of gradual onset of left Achilles pain after training for her first half-marathon. Her symptoms were aggravated with running any distance and walking in high-heeled shoes. During her first bout of therapy, she performed a progression of tendon loading through isometric, concentric, eccentric, and plyometric exercises over the course of 4 months, after which she reported 50% improvement. A second bout of therapy with a second physical therapist 6 months after symptom onset included soft tissue mobilization and trigger point dry needling of the calf, low-level laser therapy to the Achilles tendon, and calf-stretching exercises. Symptomatically, she reported transient improvement but was unable to run or wear heels at discharge, 6 months after her second evaluation.

Patient 3, a 27-year-old man, presented to physical therapy with an 18-month history of basketball-induced right patellar tendon pain. His pain was aggravated with going down stairs and running and alleviated with cessation of those activities. Secondary to pain, he stopped running, his primary form of general exercise and stress reduction. Prior treatment initially consisted of 6 months of chiropractic care, which included lower extremity adjustments, eccentric exercise, electrical stimulation, heat, and ultrasound. He also completed a bout of physical therapy where he performed decline squats, transverse friction mobilization, and gluteal strengthening. Despite treatment, symptoms persisted, and he reported significant frustration related to his inability to exercise.

Examination

Each patient was evaluated and treated by the primary author. Static postural assessment in standing was performed prior to completing observational walking gait analysis. Lumbar screening of active range of motion with overpressure was negative for symptom provocation. Bilateral and unilateral squats, single-

[CASE REPORT]

limb heel raises, and unilateral hop testing were evaluated for symptoms and functional movement patterns. Patients 1 and 2 had their typical pain and weakness during single-limb heel raises and hop tests on the affected side. Patient 3 had his comparable pain during a unilateral squat and single-limb hop testing. Neurodynamic mobility assessment was performed using the straight leg raise and femoral nerve tension tests, with relevant peripheral nerve biasing as appropriate, for foot/ankle and knee symptoms, respectively. Neurodynamic testing did not reproduce symptoms.

Active range of motion with overpressure was performed in all planes for the involved joints. Muscle-length testing of relevant 2-joint muscles was performed prior to joint accessory motion testing. Each patient demonstrated mobility deficits of both the joint and soft tissue at and around the site of dysfunction. Specifically, patients 1 and 2 had restrictions of the subtalar and talocrural joints, while patient 3 had stiffness in the patellofemoral and tibiofemoral joints. Palpation revealed hyperalgesia at the involved tendon in each patient, and while tenderness was present in the associated muscle belly, no active trigger points were noted.

Pressure pain threshold (PPT) measurements were performed as described by Rolke et al.⁵¹ Pressure pain threshold measurements were completed on each patient in an effort to identify altered nociceptive processing. The site of maximal patient-reported tenderness of the involved tendon was chosen, as was the same location on the contralateral tendon for comparison. Additionally, the contralateral thenar eminence was assessed. Reduced PPT readings were noted in all cases at the involved tendon, as well as the contralateral hand. While reduced PPT values at the involved tendon could be indicative of peripheral sensitization, if remote-site values (in this case, the opposite hand) are reduced relative to reference values of healthy individuals,⁵⁰ then one could argue that central pain mechanisms may also be at fault.¹²

The assessment of PPT has been found to be a reliable tool. In a sample of persons with or without acute neck pain, using novice raters, PPT intratester reliability was excellent (intraclass correlation coefficient [ICC] = 0.94-0.97), interrater reliability was acceptable to excellent (ICC = 0.79-0.97), and test-retest reliability was acceptable (ICC = 0.76-0.79).⁶³ Minimal detectable change and minimal clinically important difference have not been established for PPT in lower extremity tendinopathy, but have ranged from 42.7 to 137.0 kPa in the same PPT psychometric evaluation study.⁶³

Numerous outcome measures were used to objectify patient reports. Patients reported their best, worst, and average pain on an 11-item, 0-to-10 numeric pain-rating scale, with 0 indicating no pain and 10 indicating the worst pain imaginable. The Victorian Institute of Sport Assessment has created self-reported disability scales for various tendinopathies. The Achilles and patellar tendon versions were used in these cases. The Victorian Institute of Sport Assessment scales have demonstrated good responsiveness and validity.^{49,62}

Finally, the Central Sensitization Inventory (CSI) was completed by each patient. The CSI is a 2-part questionnaire intended to identify the presence of CS.³⁷ Part A includes 25 questions, each scored 0 to 4, with higher scores indicating a higher likelihood that central pain mechanisms are at fault. Five severity levels have been developed to assist with clinical interpretation of the CSI: 0 to 29, subclinical; 30 to 39, mild; 40 to 49, moderate; 50 to 59, severe; 60 to 100, extreme.⁴⁰ The CSI was found to have good test-retest reliability (0.82), a sensitivity of 0.75, and a specificity of 0.81.³⁹ While some authors have reported the responsiveness of the CSI to be excellent, specific minimal clinically important difference values have not been reported to date.⁵³

Treatment

Following the examination, it was concluded that each patient's diagnosis was

consistent with chronic lower extremity tendinopathy, with a possible component of CS. This conclusion was based on (1) lack of success with previous conservative interventions, (2) the presence of reduced PPT readings locally and at a remote site, and (3) scores on the CSI suggestive of mild CS. Each patient's previous treating clinicians were also contacted after the evaluation to discuss prior treatments. Through conversation, it appeared that previous treatment was evidence based, with appropriate tendon loading progressions integrated and compliance maintained after discharge.^{35,36} Therefore, rather than continuing to primarily address peripheral nociceptive mechanisms, as performed previously, the primary author believed that the application of additional interventions to enhance pain modulation could be beneficial. To this end, treatment consisted of impairment-based joint mobilization, subsequent self-stretching, PNE, and aerobic exercise prescription. Interventions provided for each patient are displayed in TABLE 1.

Session 1: Initial Evaluation After the examination, each patient was educated on the clinical diagnosis and the neurophysiology of pain. None of the patients had ever received PNE. Patients were informed that tissue injury and pain perception were 2 separate concepts, and that various factors can contribute to the persistence of pain through top-down sensitization (ie, stress, anxiety, depression) or bottom-up sensitization (ie, overuse).⁵⁷ Patients were instructed to reflect on what they had learned and bring any questions to subsequent visits.

Session 2: First Follow-up, 1 Week After Evaluation This visit included approximately 45 minutes of PNE. During this time, the patient's current pain beliefs and health condition knowledge were explored. The physical therapist attempted to normalize abnormality, imparting to the patient that physical or biological impairment does not always correlate to symptoms; rather, numerous studies suggest the presence of abnormality in

asymptomatic individuals. The physical therapist also discussed neuroanatomy, including nociceptive pathways, neurons, synapses, action potentials, ascending and descending spinal symptom modulation mechanisms, peripheral sensitization and CS, and neuroplasticity, using non-threatening, patient-friendly metaphors, as needed.³³ A previously published case report detailed examples of language used during the PNE process, should readers prefer additional guidance.⁶⁵

After checking for understanding and answering questions, impairment-based manual therapy was directed to the involved joints to improve mobility, reduce strain on the involved tendon, and reduce pain. A pragmatic approach was applied, using techniques previously described by Maitland.²⁰ If a joint felt hypomobile to the physical therapist, grade III to IV mobilizations were performed until the tissue mobility improved. To increase

technique specificity, pluses or minuses can be used to describe the range of tissue resistance the mobilization was performed within.²⁰ Each technique was followed by a self-stretch²³ to enhance mobility gains, to be performed as part of the patient's home exercise program. Each self-stretch was held for 1 minute and repeated 5 times daily.

Session 3: Second Follow-up, 2 Weeks After Evaluation Pressure pain threshold readings of the involved tendon and the contralateral hand were taken at the beginning of the session. Improvements were noted in each patient. Manual therapy was performed again, as mobility restrictions and pain remained. In attempts to enhance pain modulation through multiple mechanisms, each patient was prescribed an aerobic exercise program.³⁸ The mode of exercise was self-selected, as long as the patient enjoyed it and was able to exercise at a moderate to

high intensity for 30 to 45 minutes most days of the week, without aggravation of symptoms. Patients were instructed to keep an exercise log, which was checked for compliance. Due to therapist and patient scheduling challenges, each patient was instructed to continue with aerobic exercise and self-stretching until the next follow-up visit 2 weeks later.

Session 4: Third Follow-up, 4 Weeks After Evaluation Pressure pain threshold readings were again assessed at the onset of the session, with continued improvement noted. Self-reported functional scales were administered again, with improvement noted in each case; however, pain and disability remained. Upon assessment, no clinically detectable joint restrictions were present, thus manual therapy was discontinued. Good compliance with aerobic exercise was noted, with each patient reporting performance at least 4 days each week at the recommended intensity

TABLE 1

INTERVENTIONS

Treatment Protocol	Patient 1	Patient 2	Patient 3
Session 1: initial evaluation			
Education	Pain science, pain mechanisms, contributing factors to tendinopathy	Pain science, pain mechanisms, contributing factors to tendinopathy	Pain science, pain mechanisms, contributing factors to tendinopathy
Session 2: first follow-up, 1 wk after evaluation			
Education	45 min of PNE	45 min of PNE	45 min of PNE
Manual therapy	Posterior glide to right talocrural joint, grade III-; lateral glide to right subtalar joint, grade III	Posterior glide to left talocrural joint, grade III+; lateral glide to left subtalar joint, grade III+	Inferior glide to right PFJ, grade III+; medial glide to right PFJ, grade III; posterior glide to right tibiofemoral joint, grade III
HEP modifications	Self-stretching of the gastrocnemius (1-min duration, 5 times per day)	Self-stretching of the gastrocnemius (1-min duration, 5 times per day)	Self-stretching of the quadriceps (1-min duration, 5 times per day)
Session 3: second follow-up, 2 wk after evaluation			
Manual therapy	Posterior glide to right talocrural joint, grade IV+; lateral glide to right subtalar joint, grade IV++	Posterior glide to left talocrural joint, grade IV; lateral glide to left subtalar joint, grade IV	Inferior glide to right PFJ, grade IV++; posterior glide to right tibiofemoral joint, grade IV++
HEP modifications	Aerobic exercise (swimming for 30-45 min, moderate/high intensity, most days of the week)	Aerobic exercise (swimming for 30-45 min, moderate/high intensity, most days of the week)	Aerobic exercise (elliptical for 30-45 min, moderate/high intensity, most days of the week)
Session 4: third follow-up, 4 wk after evaluation			
Treatment	Jog/walk program on treadmill (5 min of running at 8.0 kph, 1 min of walking at 4.8 kph, 4 bouts)	Jog/walk program on treadmill (6 min of running at 8.0 kph, 1 min of walking at 4.8 kph, 3 bouts)	Jog/walk program on treadmill (10 min of running at 8.9 kph, 1 min of walking at 4.8 kph, 2 bouts)
HEP modifications	Running program (2-3 times per week)	Running program (2-3 times per week)	Running program (2-3 times per week)
Session 5: fourth follow-up, 8 wk after evaluation	NA (reassessment performed)	NA (reassessment performed)	NA (reassessment performed)

Abbreviations: HEP, home exercise program; NA, not applicable; PFJ, patellofemoral joint; PNE, pain neuroscience education.

[CASE REPORT]

and duration. Provocative testing from the initial examination was reassessed. Patient 3 no longer had pain with squats, while patients 1 and 2 no longer had pain with heel raises or hop tests.

With running being the primary activity restriction for each patient, running gait was evaluated on a treadmill. Each patient was able to run at least 5 to 10 minutes prior to pain, and when testing was complete, pain reduced back to baseline immediately, indicating low symptom irritability. Each patient was then instructed to initiate a return-to-running program, whereby he or she would run until symptom onset, walk until symptoms were back to baseline, and run again until symptom onset. Total duration was to be limited by the patient's general fatigue. Running was to be performed 2 to 3 times each week, on days when not performing additional aerobic exercise. Running duration and frequency were increased as tolerated. Patients were told to provide weekly status updates to the treating physical therapist via e-mail, but unless symptoms were not improving, follow-up was scheduled for 1 month later.

Session 5: Fourth Follow-up, 8 Weeks After Evaluation Pressure pain threshold readings were taken at the beginning of the session, with equivalent values noted bilaterally. Each patient reported the ability to run at least 30 minutes without aggravation of primary symptoms and was satisfied with progress. Self-reported functional scales were reassessed, with clinically significant improvements recorded. Given improvements, each patient was discharged from formal therapy, with instruction to continue progressing his or her exercise capacity as tolerated and to contact the physical therapist should questions arise.

OUTCOMES

EACH PATIENT WAS TREATED FOR 5 sessions over the course of 2 months. Primary outcomes are presented in **TABLE 2**. One hundred percent compliance with the home exercise program was reported by each patient. Clinically significant improvements were noted in pain and self-reported function. Pressure pain threshold readings at the involved tendon improved by an average of 169%,

suggesting less pain sensitivity at previously hyperalgesic locations. Contralateral tendon and contralateral hand PPT readings also improved by an average of 126% and 46%, respectively, suggesting that the effects of pain modulation were widespread, not just local to the patient's primary site of pain. Improvement was maintained at 1-year e-mail follow-up, when each patient reported being symptom free and having no self-reported functional limitation.

DISCUSSION

THIS REPORT DETAILS 3 INDIVIDUALS with persistent lower extremity tendinopathy who responded positively to a combination of joint mobilization, aerobic exercise, and PNE. Although each patient reported localized peripheral pain, each failed numerous bouts of evidence-based treatment targeted at the local nociceptive structures. Each patient's persistent pain contributed to functional limitations, mechanical hyperalgesia locally and remotely, as well as findings reported on the CSI suggesting a component of impaired central pain

TABLE 2

OUTCOMES

Outcome Measure	Patient 1			Patient 2			Patient 3		
	Examination	Discharge	1-y Follow-up	Examination	Discharge	1-y Follow-up	Examination	Discharge	1-y Follow-up
Pain (NPRS)	0/10 at rest, 8/10 with activity	0/10 at rest, 0/10 with activity*	0/10 at rest, 0/10 with activity*	1/10 at rest, 7/10 with activity	0/10 at rest, 0/10 with activity*	0/10 at rest, 0/10 with activity*	0/10 at rest, 7/10 with activity	0/10 at rest, 0/10 with activity*	0/10 at rest, 0/10 with activity*
Self-reported function	VISA-A, 46%	VISA-A, 93%*	VISA-A, 100%*	VISA-A, 53%	VISA-A, 91%*	VISA-A, 100%*	VISA-P, 65%	VISA-P, 91%*	VISA-P, 100%*
CSI part A	30	19	12	35	21	9	38	26	15
PPT, kPa			NT			NT			NT
Involved tendon	208	597		168	501		351	780	
Contralateral tendon	256	609		199	492		420	806	
Contralateral hand	367	523		249	402		403	541	
Functional movement	Pain with single-limb heel raise, pain with hop testing	No pain reported with heel raise or hop testing	NT	Pain with single-limb heel raise, pain with hop testing	No pain reported with heel raise or hop testing	NT	Pain with unilateral squat, pain with hop testing	No pain reported with squat or hop testing	NT

Abbreviations: CSI, Central Sensitization Inventory; NPRS, numeric pain-rating scale; NT, not tested; PPT, pressure pain threshold; VISA-A, Victorian Institute of Sport Assessment-Achilles; VISA-P, Victorian Institute of Sport Assessment-patella.

*Clinically significant improvement, according to the outcome measure's minimal clinically important or detectable change.

mechanisms and altered nociceptive processing. Addressing central and peripheral nociceptive mechanisms through joint mobilization, pain education, and gradual tendon reloading programs was associated with symptomatic and functional improvement in each case.

Consistent with the typical presentation of tendinopathy, peripheral nociceptive pain is frequently classified by localized pain, clear proportionate mechanical aggravating and alleviating factors, and the absence of dysesthesia or vascular complaints.⁵⁶ Alternatively, CS is often characterized by widespread, disproportionate, nonanatomical pain distributions that are severe and functionally disabling.⁴¹ Interestingly, centrally mediated symptoms have been noted in individuals with localized symptom reports as well, such as subacromial impingement, patellofemoral pain, or knee osteoarthritis.^{18,43,52} In regard to tendinopathy, various reports have identified altered central pain mechanisms. For example, sensory and motor deficits were found to exist on the contralateral limb in the presence of unilateral tendon pain and disability.¹⁹ In a cross-sectional study, psychological factors were more strongly associated with condition severity than were strength deficits in persons with gluteal tendinopathy, suggesting a central or top-down contribution to symptoms.⁴⁵

Among its numerous effects, manual therapy has been proposed to improve joint biomechanics and motion.^{3,29} One could argue that improved mobility could theoretically reduce excessive strain on local structures, such as tendons, potentially modifying local peripheral nociceptive pain. Additionally, joint mobilization has been shown to enhance descending pain modulation in a variety of painful musculoskeletal conditions, indicating that effects of joint mobilization are, at least in part, centrally mediated.^{13,55,58} Given the local peripheral nociceptive presentation of tendinopathy and evidence suggestive of possible nervous system sensitization, joint mobilization seems reasonable in

targeting multiple pain mechanisms in individuals with tendinopathy.

The underlying physiological mechanisms behind aerobic exercises' modulatory effects on chronic pain continue to be explored. However, popular theories include enhanced conditioned pain modulation¹⁶ and neurotransmitter activation within the endocannabinoid and endogenous opioid systems.^{25,26} In a recent functional magnetic resonance imaging study evaluating persons with fibromyalgia, exercise was associated with greater activity in the left dorsolateral prefrontal cortex as compared to rest.¹⁷ By enhancing descending pain inhibition through exercise, patients with persistent pain may be better able to participate in daily and recreational activity. While aerobic exercise has been widely regarded as a method for reducing chronic pain, it is unclear as to which type, frequency, intensity, and duration are most appropriate to elicit optimal results. Common components for managing pain with aerobic exercise seem to be a longer-duration threshold (greater than 10 minutes) and exercises completed at higher intensities.^{22,28} The dosage prescribed for each patient was offered based on these components.

Pain neuroscience education is an educational intervention describing the neurobiology and neurophysiology of pain and nociceptive processing by the nervous system. One of the primary aims of PNE is to identify and describe how various mechanisms and brain processing can affect and modify a person's pain experience, rather than utilizing the traditional biomedical model, in which continued local tissue injury or dysfunction is the primary driver of pain.³⁰ A previous review found evidence supporting the use of PNE to improve pain, self-reported disability, catastrophization, and physical performance.³⁰ It also has been suggested that manual therapy with PNE may help to sharpen body schema maps,^{31,47} which tend to be altered in persons with CS.²⁷ Thus, an impairment-based approach that includes PNE as described in this case series may be useful for tendinopathy.

There are several limitations associated with this report. The small number of patients, lack of control, and a multimodal treatment approach limit the generalizability of the outcomes. Additionally, more information on pain mechanisms could be elucidated with vibratory detection thresholds or assessment of hypoesthesia. Furthermore, order bias or acclimation to testing was possible. Finally, a case series can only describe the process and outcomes and not infer causality.

Tendinopathy is frequently managed successfully with appropriate exercise prescription. However, if additional factors such as stress, depression, or CS are contributing to symptoms, then addressing the localized tissue or nociceptive input alone may not be effective. As such, identifying and addressing the primary pain mechanism(s) at fault may be useful for persons with chronic pain and functional limitation. In the case of chronic lower extremity tendinopathy, a pain-mechanisms approach to management has not been described to date. While evidence in this area of research is still emerging and currently conflicting, the outcomes for these patients suggest that higher-quality studies using a similar approach would be appropriate. ●

REFERENCES

1. Albers IS, Zwerver J, Diercks RL, Dekker JH, Van den Akker-Scheek I. Incidence and prevalence of lower extremity tendinopathy in a Dutch general practice population: a cross sectional study. *BMC Musculoskelet Disord*. 2016;17:16. <https://doi.org/10.1186/s12891-016-0885-2>
2. Andersson G, Forsgren S, Scott A, et al. Tenocyte hypercellularity and vascular proliferation in a rabbit model of tendinopathy: contralateral effects suggest the involvement of central neuronal mechanisms. *Br J Sports Med*. 2011;45:399-406. <https://doi.org/10.1136/bjsm.2009.068122>
3. Bialosky JE, Bishop MD, Price DD, Robinson ME, George SZ. The mechanisms of manual therapy in the treatment of musculoskeletal pain: a comprehensive model. *Man Ther*. 2009;14:531-538. <https://doi.org/10.1016/j.math.2008.09.001>
4. Bishop MD, Torres-Cueco R, Gay CW, Lluch-Girbés E, Beneciuk JM, Bialosky JE. What effect can manual therapy have on a patient's

[CASE REPORT]

pain experience? *Pain Manag.* 2015;5:455-464. <https://doi.org/10.2217/pmt.15.39>

5. Bot SD, van der Waal JM, Terwee CB, van der Windt DA, Bouter LM, Dekker J. Course and prognosis of elbow complaints: a cohort study in general practice. *Ann Rheum Dis.* 2005;64:1331-1336. <https://doi.org/10.1136/ard.2004.030320>
6. Brage K, Ris I, Falla D, Sogaard K, Juul-Kristensen B. Pain education combined with neck- and aerobic training is more effective at relieving chronic neck pain than pain education alone – a preliminary randomized controlled trial. *Man Ther.* 2015;20:686-693. <https://doi.org/10.1016/j.math.2015.06.003>
7. Cassel M, Baur H, Hirschmüller A, Carlsohn A, Fröhlich K, Mayer F. Prevalence of Achilles and patellar tendinopathy and their association to intratendinous changes in adolescent athletes. *Scand J Med Sci Sports.* 2015;25:e310-e318. <https://doi.org/10.1111/sms.12318>
8. Chimenti RL, Frey-Law LA, Sluka KA. A mechanism-based approach to physical therapist management of pain. *Phys Ther.* 2018;98:302-314. <https://doi.org/10.1093/ptj/pzy030>
9. Cook JL, Khan KM, Harcourt PR, Grant M, Young DA, Bonar SF. A cross sectional study of 100 athletes with jumper's knee managed conservatively and surgically. The Victorian Institute of Sport Tendon Study Group. *Br J Sports Med.* 1997;31:332-336. <https://doi.org/10.1136/bjism.31.4.332>
10. Cook JL, Purdam CR. Is tendon pathology a continuum? A pathology model to explain the clinical presentation of load-induced tendinopathy. *Br J Sports Med.* 2009;43:409-416. <https://doi.org/10.1136/bjism.2008.051193>
11. Couppé C, Svensson RB, Silbernagel KG, Langberg H, Magnusson SP. Eccentric or concentric exercises for the treatment of tendinopathies? *J Orthop Sports Phys Ther.* 2015;45:853-863. <https://doi.org/10.2519/jospt.2015.5910>
12. Courtney CA, Kavchak AE, Lowry CD, O'Hearn MA. Interpreting joint pain: quantitative sensory testing in musculoskeletal management. *J Orthop Sports Phys Ther.* 2010;40:818-825. <https://doi.org/10.2519/jospt.2010.3314>
13. Courtney CA, Steffen AD, Fernández-de-la-Peñas C, Kim J, Chmell SJ. Joint mobilization enhances mechanisms of conditioned pain modulation in individuals with osteoarthritis of the knee. *J Orthop Sports Phys Ther.* 2016;46:168-176. <https://doi.org/10.2519/jospt.2016.6259>
14. Debenham J, Butler P, Mallows A, Wand BM. Disrupted tactile acuity in people with Achilles tendinopathy: a preliminary case-control investigation. *J Orthop Sports Phys Ther.* 2016;46:1061-1064. <https://doi.org/10.2519/jospt.2016.6514>
15. de Jonge S, van den Berg C, de Vos RJ, et al. Incidence of midportion Achilles tendinopathy in the general population. *Br J Sports Med.* 2011;45:1026-1028. <https://doi.org/10.1136/bjsports-2011-090342>
16. Ellingson LD, Koltyn KF, Kim JS, Cook DB. Does exercise induce hypoalgesia through conditioned

pain modulation? *Psychophysiology.* 2014;51:267-276. <https://doi.org/10.1111/psyp.12168>

17. Ellingson LD, Stegner AJ, Schwabacher JJ, Koltyn KF, Cook DB. Exercise strengthens central nervous system modulation of pain in fibromyalgia. *Brain Sci.* 2016;6:8. <https://doi.org/10.3390/brainsci6010008>
18. Fingleton C, Smart K, Moloney N, Fullen BM, Doody C. Pain sensitization in people with knee osteoarthritis: a systematic review and meta-analysis. *Osteoarthritis Cartilage.* 2015;23:1043-1056. <https://doi.org/10.1016/j.joca.2015.02.163>
19. Heales LJ, Lim EC, Hodges PW, Vicenzino B. Sensory and motor deficits exist on the non-injured side of patients with unilateral tendon pain and disability—implications for central nervous system involvement: a systematic review with meta-analysis. *Br J Sports Med.* 2014;48:1400-1406. <https://doi.org/10.1136/bjsports-2013-092535>
20. Hengeveld E, Banks K, Maitland GD, Wells P. *Maitland's Peripheral Manipulation.* 4th ed. Oxford, UK: Butterworth-Heinemann; 2005.
21. Herd CR, Meserve BB. A systematic review of the effectiveness of manipulative therapy in treating lateral epicondylalgia. *J Man Manip Ther.* 2008;16:225-237. <https://doi.org/10.1179/106698108790818288>
22. Hoffman MD, Shepanski MA, Ruble SB, Valic Z, Buckwalter JB, Clifford PS. Intensity and duration threshold for aerobic exercise-induced analgesia to pressure pain. *Arch Phys Med Rehabil.* 2004;85:1183-1187. <https://doi.org/10.1016/j.apmr.2003.09.010>
23. Jenkins J, Beazell J. Flexibility for runners. *Clin Sports Med.* 2010;29:365-377. <https://doi.org/10.1016/j.csm.2010.03.004>
24. Kettunen JA, Kvist M, Alanen E, Kujala UM. Long-term prognosis for jumper's knee in male athletes. A prospective follow-up study. *Am J Sports Med.* 2002;30:689-692. <https://doi.org/10.1177/03635465020300051001>
25. Koltyn KF. Analgesia following exercise: a review. *Sports Med.* 2000;29:85-98. <https://doi.org/10.2165/00007256-200029020-00002>
26. Koltyn KF, Brellenthin AG, Cook DB, Sehgal N, Hillard C. Mechanisms of exercise-induced hypoalgesia. *J Pain.* 2014;15:1294-1304. <https://doi.org/10.1016/j.jpain.2014.09.006>
27. Latremoliere A, Woolf CJ. Central sensitization: a generator of pain hypersensitivity by central neural plasticity. *J Pain.* 2009;10:895-926. <https://doi.org/10.1016/j.jpain.2009.06.012>
28. Lee HS. The effects of aerobic exercise and strengthening exercise on pain pressure thresholds. *J Phys Ther Sci.* 2014;26:1107-1111. <https://doi.org/10.1589/jpts.26.1107>
29. Loudon JK, Reiman MP, Sylvain J. The efficacy of manual joint mobilisation/manipulation in treatment of lateral ankle sprains: a systematic review. *Br J Sports Med.* 2014;48:365-370. <https://doi.org/10.1136/bjsports-2013-092763>
30. Louw A, Diener I, Butler DS, Puenteadura EJ. The effect of neuroscience education on

pain, disability, anxiety, and stress in chronic musculoskeletal pain. *Arch Phys Med Rehabil.* 2011;92:2041-2056. <https://doi.org/10.1016/j.apmr.2011.07.198>

31. Louw A, Farrell K, Wettach L, Uhl J, Majkowski K, Welding M. Immediate effects of sensory discrimination for chronic low back pain: a case series. *N Z J Physiother.* 2015;43:58-63.
32. Louw A, Nijs J, Puenteadura EJ. A clinical perspective on a pain neuroscience education approach to manual therapy. *J Man Manip Ther.* 2017;25:160-168. <https://doi.org/10.1080/10669817.2017.1323699>
33. Louw A, Zimney K, O'Hotto C, Hilton S. The clinical application of teaching people about pain. *Physiother Theory Pract.* 2016;32:385-395. <https://doi.org/10.1080/09593985.2016.1194652>
34. Louw A, Zimney K, Puenteadura EJ, Diener I. The efficacy of pain neuroscience education on musculoskeletal pain: a systematic review of the literature. *Physiother Theory Pract.* 2016;32:332-355. <https://doi.org/10.1080/09593985.2016.1194646>
35. Malliaras P, Cook J, Purdam C, Rio E. Patellar tendinopathy: clinical diagnosis, load management, and advice for challenging case presentations. *J Orthop Sports Phys Ther.* 2015;45:887-898. <https://doi.org/10.2519/jospt.2015.5987>
36. Martin RL, Chimenti R, Cuddeford T, et al. Achilles pain, stiffness, and muscle power deficits: midportion Achilles tendinopathy revision 2018. *J Orthop Sports Phys Ther.* 2018;48:A1-A38. <https://doi.org/10.2519/jospt.2018.0302>
37. Mayer TG, Neblett R, Cohen H, et al. The development and psychometric validation of the Central Sensitization Inventory. *Pain Pract.* 2012;12:276-285. <https://doi.org/10.1111/j.1533-2500.2011.00493.x>
38. Naugle KM, Fillingim RB, Riley JL, 3rd. A meta-analytic review of the hypoalgesic effects of exercise. *J Pain.* 2012;13:1139-1150. <https://doi.org/10.1016/j.jpain.2012.09.006>
39. Neblett R, Cohen H, Choi Y, et al. The Central Sensitization Inventory (CSI): establishing clinically significant values for identifying central sensitivity syndromes in an outpatient chronic pain sample. *J Pain.* 2013;14:438-445. <https://doi.org/10.1016/j.jpain.2012.11.012>
40. Neblett R, Hartzell MM, Mayer TG, Cohen H, Gatchel RJ. Establishing clinically relevant severity levels for the central sensitization inventory. *Pain Pract.* 2017;17:166-175. <https://doi.org/10.1111/papr.12440>
41. Nijs J, Torres-Cueco R, van Wilgen CP, et al. Applying modern pain neuroscience in clinical practice: criteria for the classification of central sensitization pain. *Pain Physician.* 2014;17:447-457.
42. Öhberg L, Lorentzon R, Alfredson H. Eccentric training in patients with chronic Achilles tendinosis: normalised tendon structure and decreased thickness at follow up. *Br J Sports Med.* 2004;38:8-11. <https://doi.org/10.1136/bjism.2001.000284>
43. Pazzinatto MF, de Oliveira Silva D, Barton C, Rathleff MS, Briani RV, de Azevedo FM. Female

adults with patellofemoral pain are characterized by widespread hyperalgesia, which is not affected immediately by patellofemoral joint loading. *Pain Med.* 2016;17:1953-1961. <https://doi.org/10.1093/pm/pnw068>

44. Plinsinga ML, Brink MS, Vicenzino B, van Wilgen CP. Evidence of nervous system sensitization in commonly presenting and persistent painful tendinopathies: a systematic review. *J Orthop Sports Phys Ther.* 2015;45:864-875. <https://doi.org/10.2519/jospt.2015.5895>
45. Plinsinga ML, Coombes BK, Mellor R, et al. Psychological factors not strength deficits are associated with severity of gluteal tendinopathy: a cross-sectional study. *Eur J Pain.* 2018;22:1124-1133. <https://doi.org/10.1002/ejp.1199>
46. Plinsinga ML, van Wilgen CP, Brink MS, et al. Patellar and Achilles tendinopathies are predominantly peripheral pain states: a blinded case control study of somatosensory and psychological profiles. *Br J Sports Med.* 2018;52:284-291. <https://doi.org/10.1136/bjsports-2016-097163>
47. Puenteadura EJ, Flynn T. Combining manual therapy with pain neuroscience education in the treatment of chronic low back pain: a narrative review of the literature. *Physiother Theory Pract.* 2016;32:408-414. <https://doi.org/10.1080/09593985.2016.1194663>
48. Rio E, Moseley L, Purdam C, et al. The pain of tendinopathy: physiological or pathophysiological? *Sports Med.* 2014;44:9-23. <https://doi.org/10.1007/s40279-013-0096-z>
49. Robinson JM, Cook JL, Purdam C, et al. The VISA-A questionnaire: a valid and reliable index of the clinical severity of Achilles tendinopathy. *Br J Sports Med.* 2001;35:335-341. <https://doi.org/10.1136/bjsm.35.5.335>
50. Rolke R, Baron R, Maier C, et al. Quantitative sensory testing in the German Research Network on Neuropathic Pain (DFNS): standardized protocol and reference values. *Pain.* 2006;123:231-243. <https://doi.org/10.1016/j.pain.2006.01.041>

51. Rolke R, Magerl W, Campbell KA, et al. Quantitative sensory testing: a comprehensive protocol for clinical trials. *Eur J Pain.* 2006;10:77-88. <https://doi.org/10.1016/j.ejpain.2005.02.003>
52. Sanchis MN, Lluch E, Nijs J, Struyf F, Kangasperko M. The role of central sensitization in shoulder pain: a systematic literature review. *Semin Arthritis Rheum.* 2015;44:710-716. <https://doi.org/10.1016/j.semarthrit.2014.11.002>
53. Scerbo T, Colasurdo J, Dunn S, Unger J, Nijs J, Cook C. Measurement properties of the Central Sensitization Inventory: a systematic review. *Pain Pract.* 2018;18:544-554. <https://doi.org/10.1111/papr.12636>
54. Silbernagel KG, Thomeé R, Eriksson BI, Karlsson J. Continued sports activity, using a pain-monitoring model, during rehabilitation in patients with Achilles tendinopathy: a randomized controlled study. *Am J Sports Med.* 2007;35:897-906. <https://doi.org/10.1177/0363546506298279>
55. Sluka KA, Skyba DA, Radhakrishnan R, Leeper BJ, Wright A. Joint mobilization reduces hyperalgesia associated with chronic muscle and joint inflammation in rats. *J Pain.* 2006;7:602-607. <https://doi.org/10.1016/j.jpain.2006.02.009>
56. Smart KM, Blake C, Staines A, Doody C. The discriminative validity of "nociceptive," "peripheral neuropathic," and "central sensitization" as mechanisms-based classifications of musculoskeletal pain. *Clin J Pain.* 2011;27:655-663. <https://doi.org/10.1097/AJP.0b013e318215f16a>
57. Steeds CE. The anatomy and physiology of pain. *Surgery.* 2009;27:507-511. <https://doi.org/10.1016/j.mpsur.2009.10.013>
58. Sterling M, Pedler A, Chan C, Puglisi M, Vuvan V, Vicenzino B. Cervical lateral glide increases nociceptive flexion reflex threshold but not pressure or thermal pain thresholds in chronic whiplash associated disorders: a pilot randomised controlled trial. *Man Ther.* 2010;15:149-153. <https://doi.org/10.1016/j.math.2009.09.004>
59. Tompra N, van Dieën JH, Coppieters MW.

Central pain processing is altered in people with Achilles tendinopathy. *Br J Sports Med.* 2016;50:1004-1007. <https://doi.org/10.1136/bjsports-2015-095476>

60. van Wilgen CP, Konopka KH, Keizer D, Zwerver J, Dekker R. Do patients with chronic patellar tendinopathy have an altered somatosensory profile? – A Quantitative Sensory Testing (QST) study. *Scand J Med Sci Sports.* 2013;23:149-155. <https://doi.org/10.1111/j.1600-0838.2011.01375.x>
61. Vardeh D, Mannion RJ, Woolf CJ. Toward a mechanism-based approach to pain diagnosis. *J Pain.* 2016;17:T50-T69. <https://doi.org/10.1016/j.jpain.2016.03.001>
62. Visentini PJ, Khan KM, Cook JL, et al. The VISA score: an index of severity of symptoms in patients with jumper's knee (patellar tendinosis). *J Sci Med Sport.* 1998;1:22-28. [https://doi.org/10.1016/S1440-2440\(98\)80005-4](https://doi.org/10.1016/S1440-2440(98)80005-4)
63. Walton DM, MacDermid JC, Nielson W, Teasell RW, Chiasson M, Brown L. Reliability, standard error, and minimum detectable change of clinical pressure pain threshold testing in people with and without acute neck pain. *J Orthop Sports Phys Ther.* 2011;41:644-650. <https://doi.org/10.2519/jospt.2011.3666>
64. Xu Q, Chen B, Wang Y, et al. The effectiveness of manual therapy for relieving pain, stiffness, and dysfunction in knee osteoarthritis: a systematic review and meta-analysis. *Pain Physician.* 2017;20:229-243.
65. Zimney K, Louw A, Puenteadura EJ. Use of Therapeutic Neuroscience Education to address psychosocial factors associated with acute low back pain: a case report. *Physiother Theory Pract.* 2014;30:202-209. <https://doi.org/10.3109/09593985.2013.856508>



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STEVEN J. KAMPER, PhD¹

Reliability and Validity: Linking Evidence to Practice

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The previous Evidence in Practice article introduced the idea of the “construct,” or what you are interested in measuring, for example, pain, disability, or strength. As there are often numerous measures available for any given construct, how do you choose which to use? Of the number of considerations that go into this, none are more important than reliability and validity.

It is no overstatement to say that if a measure is not both sufficiently reliable and valid, then it is not fit for purpose.

Reliability

Formally, reliability is the extent to which a measurement is free from error. In practice, a reliable measure is one that gives you the same answer when you measure the same construct several times. Consider the example of measuring height (the construct) with a tape measure (the measure). You might measure a person's height in millimeters 3 times with the tape measure; the extent to which the number of millimeters is the same on each occasion is the reliability of the measure.

The implications of unreliable measures are serious. If an unreliable diagnostic test (measure) was applied to a patient several times, then the same patient might be diagnosed as both having and not having the condition on different occasions or by different people. If an unreliable measure of symptom severity was collected from a patient before and after an intervention, then it would be impossible to tell whether that symptom improved, stayed the same, or got worse. Essentially, data collected from unreli-

able measures do not provide useful information; a measure that is not reliable cannot be valid.

There are several different types of reliability, each uniquely relevant to situations in which the measures might be used. Intrarater reliability refers to the situation where the same rater takes the measure on one patient on several occasions, and reliability is the extent to which the scores from the successive measurements are the same. Interrater reliability is relevant when multiple raters use the same measure on a single person, and reliability is the extent to which scores from the different raters are the same.

Validity

Validity is the extent to which the score on a measure truly reflects the construct it is supposed to measure. This is relatively straightforward when it comes to things like height or strength, but waters quickly become murky when we consider unobservable or “latent” constructs such as pain, quality of life, or disability. For these sorts of constructs, we collect indirect measures, such as self-reported experiences and behaviors, or recall of beliefs and emotions, and assume that these reflect the construct. For example,

we might ask a patient to answer the 24 questions of the Roland-Morris Disability Questionnaire (the measure) and score his or her level of back pain-related disability (construct) by adding up the number of “yes” responses. The patient's score out of 24 is valid to the extent that the questions really reflect the construct of disability and to the extent that having difficulty with more of the items reflects greater disability.

There are several different types of validity relevant to clinical measures, the most commonly assessed being construct validity. When researchers assess the construct validity of a measure, they are ideally able to compare their measure to a “gold standard.” For example, arthroscopic visualization of the anterior cruciate ligament is considered a gold standard of anterior cruciate ligament rupture, so a study might compare results from Lachman's test to the findings from arthroscopy to assess the validity of Lachman's test.

Unfortunately, there are no gold standards for many constructs in which we are interested (eg, latent constructs such as disability and pain). In these cases, construct validity is tested against a “reference standard,” which is a sort of imperfect gold standard. When there is no gold standard, the best way to test validity is via hypothesis testing. This involves setting out a series of hypotheses before collecting the data. These hypotheses are theorized relationships between a score on the measure and other characteris-

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tics, for example, that the score will be strongly correlated with scores on another measure of the same construct and less strongly correlated with scores on a different but related construct. The extent to which the data are consistent with the predetermined hypotheses will be evidence supporting validity of the measure.

Statistics

Testing reliability and validity generally involves assessing agreement between 2 scores, either scores on the same measure collected twice (reliability) or scores on different measures (validity). The statistics used to describe agreement depend on whether the measures are dichotomous (eg, kappa, sensitivity/specificity) or continuous (eg, intraclass correlation

coefficients, correlations, limits of agreement, R^2).

Conclusion

There are a couple of important general points to note about reliability and validity. First, both are on a spectrum, so measures are not “unreliable” or “reliable,” but more or less reliable and more or less valid. Of course, this makes choosing measures more difficult; we need to make a subjective judgment as to whether a measure is “reliable enough” and “valid enough” in a particular situation. General guidelines exist to help interpret reliability and validity statistics, but these guidelines do not and should not replace clinical judgment. The second point is that no measure sits on the very end of the spectrum; there is

no perfectly reliable and perfectly valid measure. Even in the case of measuring height with a tape measure, successive measures are likely to differ by a few millimeters here and there. Finally, there are practical concerns when it comes to choosing a measure, including how long it takes to administer, whether the patient can comprehend text or instructions, and how data will be stored and used.

Measurement is an entire field of research by itself. Although the general concepts are quite straightforward, you do not have to scratch too far below the surface before things become complicated. When reading research, you should look for information that reassures you that the measures used are sufficiently reliable and valid. The take-home message: be very cautious about using, or trying to interpret, information from a measure if you have no information about its reliability and validity. ●

“Essentially, data collected from unreliable measures do not provide useful information.”

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Maintaining Lumbar Spine Stability: A Study of the Specific and Combined Effects of Abdominal Activation and Lumbosacral Orthosis on Lumbar Intrinsic Stiffness

Inadequate lumbar stability is hypothesized as a potential mechanism explaining low back pain (LBP) and disability.³⁰ Lumbar stability is achieved through numerous mechanisms, including the intrinsic mechanics of the lumbar spine,⁵ feedforward motor control or preplanned muscle activity,³⁷ and feedback motor control or reflex responses.^{7,29} Sudden perturbations of the trunk may lead to poorly

controlled lumbar motion and injury, especially if trunk muscle reflexes are delayed,^{7,29} as observed in some patients with LBP.^{32,34} Considering the inherent delay of reflex responses and, consequently, their limited trainability,^{31,44} interventions to enhance lumbar stability must concentrate on enabling its mechanisms to operate before anticipated trunk efforts or perturbations. Two interventions that can achieve this goal are the active cocontraction of the abdominal and back muscles and the passive use of a lumbosacral orthosis (LSO).

The cocontraction of the abdominal muscles, which enhances lumbar stability through feedforward motor control, can be trained via a lumbar stabilization exercise program. There are 2 strategies to activate the abdominal muscles: (1) isolated activation of the transversus abdominis, which is achieved through abdominal hollowing, also known as the abdominal drawing-in maneuver (ADIM)³⁵; and (2) the global cocontraction of the entire abdominal wall, also known as “bracing.”²² These isolated and global cocontraction approaches must be learned, especially

● **BACKGROUND:** Two potential interventions for enhancing lumbar stability are to actively increase abdominal muscle activity, either through the abdominal drawing-in maneuver (ADIM) or bracing, and passively increase lumbar stiffness using a lumbosacral orthosis (LSO).

● **OBJECTIVE:** To compare the increase in lumbar stiffness after 2 active interventions (ADIM versus bracing) and 1 passive intervention (LSO), and to evaluate the combined effect of active (abdominal bracing) and passive interventions.

● **METHODS:** In this experimental and comparative study, lumbar stiffness, a surrogate measure of lumbar stability, was estimated in 25 healthy individuals during 7 trunk perturbation conditions: (1) control, (2) ADIM, (3) bracing at 5% of right external oblique maximal voluntary activation (5% bracing), (4) bracing at 10% of right external oblique maximal voluntary activation (10% bracing), (5) LSO, (6) LSO plus 5% bracing, and (7) LSO plus 10% bracing. Electromyographic biofeedback of the external oblique was provided on a

monitor, while ultrasound was used for the ADIM to ensure a sustained contraction of the transversus abdominis.

● **RESULTS:** The ADIM, 5% bracing, and 10% bracing active interventions generated comparable lumbar stiffness. However, considering that bracing can range from 10% to 20%, it may be superior to hollowing, as further estimated with a mixed-effect statistical model. Combining bracing and an LSO resulted in an additive effect on lumbar stiffness.

● **CONCLUSION:** Bracing and ADIM produced comparable lumbar stiffness, as they were performed at the same overall abdominal activation levels (5% and 10% maximal voluntary activation). The independent effects of bracing and LSO raises the possibility of combining these interventions in some circumstances. *J Orthop Sports Phys Ther* 2019;49(4):262-271. Epub 18 Jan 2019. doi:10.2519/jospt.2019.8565

● **KEY WORDS:** abdominal drawing-in maneuver, bracing, hollowing, lumbosacral orthosis, surface electromyography

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the ADIM, which is known to require practice.²⁷ The ADIM involves the selective neuromuscular control of the transversus abdominis and the internal oblique, with minimal to no contraction of other superficial abdominal and paraspinal muscles.¹³ The sole study that has compared these 2 approaches (ADIM versus bracing) to counteract sudden trunk perturbations found that bracing was effective in reducing the kinematic response to perturbation, whereas the ADIM was not.⁴² A limitation of abdominal cocontraction is that it relies on maintained increases in muscle activation that cannot be sustained for extended periods and therefore is not helpful in counteracting unexpected perturbations. An LSO, in contrast to muscle cocontraction,⁹ effectively counters unpredictable perturbations by passively increasing lumbar intrinsic stiffness,^{4,18,25} without increasing lumbar compression forces.¹ Of note is that lumbar stiffness represents a proxy measure of lumbar stability.²⁶

Cholewicki et al³ looked at the individual and combined effects of generating intra-abdominal pressure (IAP) and wearing an LSO on lumbar stiffness, using a paradigm of sudden trunk unloading. It has been shown that cocontraction of the trunk muscles and IAP are concurrent,² and that raising IAP or cocontracting the trunk muscles and wearing an LSO, independently or in combination, can increase lumbar spine stiffness. This suggests the possibility of combining abdominal cocontraction and an LSO in certain functional situations. In the study by Cholewicki et al,³ individuals were instructed to use the Valsalva maneuver to raise IAP.³ Because these findings were limited in their generalizability to tasks with a closed glottis (short-duration tasks), it was of interest to extend this study to tasks of longer duration that require continuous breathing, such as tasks of daily living or work. This would likely correspond to a lower IAP (or trunk muscle cocontraction) and lumbar stiffness,² changing the relative efficacy of these active and passive interventions.

The aim of the present study was to compare the lumbar intrinsic stiffness of 2 active interventions (ADIM versus bracing) and 1 passive intervention, and to assess the combined effect of active (bracing only) and passive interventions during a task requiring continuous breathing. It was hypothesized that both active and passive interventions would increase lumbar intrinsic stiffness during trunk perturbations, but that the ADIM would produce lower intrinsic stiffness than bracing.⁴² It was further hypothesized that the combination of active and passive interventions would produce more lumbar intrinsic stiffness compared to either intervention used independently. These hypotheses were tested by assessing lumbar stiffness during different combinations of the ADIM, bracing, and LSO.

METHODS

Participants

TWENTY-FIVE HEALTHY PARTICIPANTS (11 men, 14 women), between 18 and 65 years of age, participated in the study (TABLE 1). Participants were excluded if they met any of the following criteria: LBP in the past month; surgery of the pelvis or spinal column; a specific lumbar congenital malformation (spondylolysis, intervertebral fusion, sacralization) or scoliosis that might affect lumbar stability; any systemic or degenerative disease or history of neurological disease (balance disorders, history of stroke); body mass index greater than 30 kg/m²; positive response to the Physical Activity

Monitor providing readiness Questionnaire⁴⁰ to identify people having a heart or cardiovascular condition; abnormal blood pressure (systolic pressure greater than 140 mmHg and diastolic pressure greater than 90 mmHg) or taking medications to control high blood pressure (to ensure safety when wearing an LSO); treatment with anticonvulsive, antidepressive, and anxiolytic medication or other medication that might influence neuronal excitability; pregnancy; claustrophobia; or inability to correctly perform the ADIM, as assessed in the first session, external obliquely 3 potential volunteers screened for the study did not meet these criteria.

All participants provided written, informed consent prior to all experimental procedures. The rights of the participants were protected. All procedures were approved by the ethics committee of the Center for Interdisciplinary Research in Rehabilitation of Greater Montreal (registration number CRIR-1071-0415).

Protocol

Trunk Perturbation Protocol A detailed description of the trunk perturbation apparatus has previously been published¹⁸ and is only summarized briefly here. Participants stood upright in the apparatus, with the immobilized pelvis and thorax in a rigid harness (FIGURE 1). Trunk force and motion signals were collected during small (4 mm), quick (less than 40 milliseconds), front and back perturbations of the trunk/harness, delivered randomly and repeatedly (275 perturbations) within a 75-second period. During these

TABLE 1

PARTICIPANT CHARACTERISTICS
AT THE INITIAL MEASUREMENT SESSION*

Variable	Men (n = 11)	Women (n = 14)
Age, y	23.7 ± 3.1	22.9 ± 1.4
Height, cm	175.6 ± 11.4	167.8 ± 7.6
Weight, kg	77.8 ± 9.8	60.3 ± 8.7
BMI, kg/m ²	24.1 ± 2.3	21.5 ± 2.4

Abbreviation: BMI, body mass index.

*Values are mean ± SD.

perturbations, participants were asked to maintain torques of 15 Nm for men and 10 Nm for women, which corresponds to approximately 5% of back strength, while aided with real-time visual feedback of back extension torque.

The trunk perturbation protocol was performed under 7 experimental conditions: (1) minimization of abdominal muscle contraction (control), (2) transversus

abdominis muscle contraction (ADIM), (3) bracing at 5% of maximal external oblique activation (BR5), (4) bracing at 10% of maximal external oblique activation (BR10), (5) wearing the LSO (LSO), (6) wearing the LSO and bracing at 5% (LSO+BR5), and (7) wearing the LSO and bracing at 10% (LSO+BR10). Bracing at 5% and 10% of maximal external oblique activation corresponds to the range of abdominal-wall activation levels that are achieved when performing the ADIM,⁴² allowing a fair comparison between bracing and ADIM interventions, even though bracing can reasonably be expected to range between 10% and 25% of maximal voluntary activation (MVA).^{8,42} This also allows participants to perform the 75-second trunk perturbation trials without muscle fatigue and interference with breathing, which might be expected at higher levels of effort.²³

A flexible and extensible LSO (LumboLux; Breg, Inc, Carlsbad, CA) without additional dorsal or ventral panels was used for the experimental conditions of LSO, LSO+BR5, and LSO+BR10. The LSO was 15 cm high in the front (abdominal side) and 25 cm in the back (dorsal side). The tension of the LSO was adjusted with the participant in quiet standing, using a force-sensing resistor sensor (model FSR 400; Interlink Electronics, Inc, Westlake Village, CA), attached on the skin between the lateral aspect of the left iliac crest and the 12th rib. Participants adjusted the LSO tension to reach a pressure of 60 mmHg or 8.0 kPa.

Study Design and Procedures

The protocol required highly skilled abdominal contractions that had to be sustained during the 75-second trunk perturbations, while simultaneously producing a sustained trunk extension torque. Consequently, the task proved quite challenging. To ensure that participants performed the task appropriately, 2 similar experimental sessions separated by 2 to 7 days were conducted. The aim of the first session was to familiarize the participants with the ADIM and with all

the steps of the study protocol, including instrumentation (electromyography [EMG]), producing a maximal voluntary contraction (MVC) in an isometric dynamometer, and undergoing trunk perturbations to assess lumbar intrinsic stiffness.

The participant had to perform the ADIM first in the supine position, then during upright standing, with real-time ultrasound feedback given by the experimenter until the maneuver was successfully repeated. Second, after EMG electrodes were applied, the participant was asked to generate an MVC of the trunk muscles for EMG normalization purposes. An MVC in each of 6 directions (flexion, extension, left lateral bending, right lateral bending, left axial rotation, and right axial rotation) was collected, while participants stood in a custom-made isometric dynamometer and received real-time visual feedback of their performance and verbal encouragement from the assessor.¹⁷ A monitor located in front of the participant provided visual feedback of the increase in L5-S1 lumbar torque progressively generated by the participant for each direction of interest.

Before the participants were positioned in the perturbation apparatus, a maximal bracing contraction of the abdominals was performed in the upright posture to collect the maximal EMG of the left external oblique. The instructions were, "Please contract your abdominal muscles progressively to your maximum in about 3 seconds, hold 1 second, and relax. It is important not to bend the trunk and not to contract other muscles during this effort." This value served as the 100% reference for performing the abdominal bracing contractions at 2 effort levels (5% and 10%). Once in the apparatus, the participants were told to perform three 30-second perturbation trials without any contraction of the abdominal muscles. This was done to ensure the participants really understood the task. Then, for familiarization purposes, participants had to perform one 75-second trial of the ADIM, BR5, and BR10 experimental conditions.

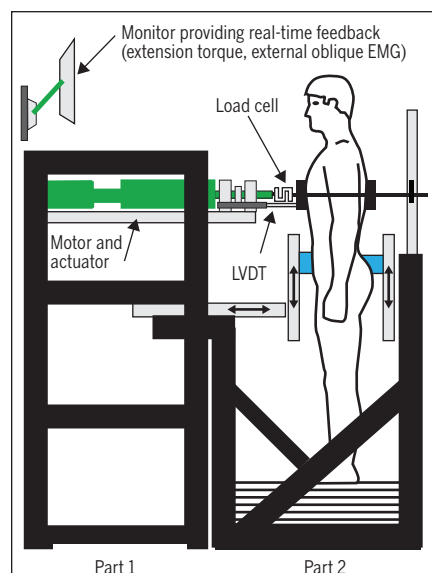


FIGURE 1. The trunk perturbation apparatus consisted of 2 independent steel structures: the first supporting the motor/actuator system and the second allowing stabilization of the participant's pelvis (sacrum and both anterosuperior iliac spines). A linear actuator (operating range, 100 mm; model T13-B4020MS040; Thomson Industries, Inc, Radford, VA) was attached to a servomotor (model AKM54K-ANC2DB00; Kollmorgen, Radford, VA), the latter being controlled with a SERVOSTAR 600 controller (model S610-30-AS; Kollmorgen). A load cell (capacity, 5000 N; model SM; Interface Inc, Scottsdale, AZ) and a rigid harness were fixed at the end of the actuator shaft to allow compression of the trunk at the T8 vertebral level. The harness was supported behind the participant by a frictionless system allowing its horizontal displacement, the latter being measured with an LVDT (operating range, 50 mm; model LD610-50; OMEGA Engineering, Inc, Norwalk, CT) attached on the side of the actuator and the front surface of the harness. A monitor was attached on the wall to provide a real-time video display of the applied trunk extension torque (preloading). All signals (load cell, LVDT) were collected at a 1000-Hz sampling rate. Abbreviations: EMG, electromyography; LVDT, linear variable differential transformer.

Session 2 followed a similar protocol, except that the ADIM training was minimal (recall), and all 7 experimental conditions were performed in a randomized sequence. Before recording in the conditions that required a specific abdominal contraction, participants were familiarized with the task by performing a 30-second trial followed by a 1-minute rest. Next, two 75-second trials, each followed by a 2-minute rest, were performed for each experimental condition. An additional 5-minute rest was provided after 10 trials to ensure that fatigue did not impact performance throughout the protocol.

Biological Feedback Ultrasonographic feedback was used in the ADIM condition to ensure proper activation of the transversus abdominis muscle. The process was similar to that of our previous work,¹⁶ with the exception that the transducer was positioned at the level of the umbilicus, then moved laterally to the right side to allow visualization of the medial edge of the transversus abdominis (fascial tip) and its lateral slide during the ADIM. This lateral slide of the transversus abdominis (instead of its thickness increase) was the sole criterion of ADIM success in the trunk perturbation apparatus, as it was the only perceptible movement during trunk perturbations due to vibrations of the abdomen and ultrasound apparatus.

However, during training out of the apparatus (in supine and standing positions), more feedback was provided. Participants were verbally cued to slowly draw their lower abdominal wall toward their spine. By visual inspection and palpation, the experimenter ensured that the participant had no posterior rotation of the pelvis, minimal thickness increase of the superficial external oblique (as seen on ultrasound images), and did not hold his or her breath during the maneuver. The internal oblique was not considered because it generally accompanies transversus abdominis activation.¹³ Though it was not feasible to provide the ultrasound feedback to the participant during

the perturbations, this feedback was used by the experimenter to confirm that the ADIM was sustained (transversus abdominis slide) throughout the 75-second perturbations, which was further quantified as detailed below.

To ensure constant muscle activation for the bracing trials, visual feedback of the filtered external oblique EMG was provided. The EMG signal was high-pass filtered (30 Hz; second-order Butterworth infinite impulse response filter) to remove low-frequency movement and electrocardiographic artifacts,³³ and then rectified and low-pass filtered (1 Hz; second-order Butterworth infinite impulse response filter) to generate an EMG envelope free of high-frequency modulations generated by the trunk perturbation apparatus. When the participant did not sustain the ADIM or bracing level appropriately (qualitative appraisal by the experimenter), the trial was repeated.

EMG Measures Electromyographic signals were recorded from 1 pair of lumbar spine muscles (longissimus at L3, 3 cm from midline), 1 pair of abdominal muscles (rectus abdominis, 3 cm above the navel and 3 cm from the midline), and the left external oblique (at the navel and nipple intersection) with a Bagnoli-16 system (DS-B04; Delsys Inc, Natick, MA), at a sampling rate of 1000 Hz and bandwidth of 20 to 450 Hz, using 5 bipolar surface electrodes (model DE-2.1; Delsys Inc). A custom-made cushion surrounded each electrode,¹⁴ preventing it from being compressed by the LSO. The internal obliques were not monitored, as they are known to coactivate with the transversus abdominis¹⁹ and the pelvis restraints would have interfered with electrodes in the trunk perturbation apparatus.

Analysis

Estimation of Lumbar Stiffness Total lumbar stiffness was separated into intrinsic and reflexive components based on the delay between the perturbation and the resultant force.¹⁸ Intrinsic stiffness (h) is defined by the contributions

of this restoring force (F) in the first 60 milliseconds following a perturbation (x), as defined in the following equation:

$$F(t) = \int_{-0.06}^{0.06} h(\tau)x(t-\tau)d\tau$$

This intrinsic stiffness arises from the mechanical resistance of the trunk to being displaced and is due to the viscoelastic properties of the muscles and tissues of the trunk. The reflex component arises from the muscle contractions produced in response to the trunk displacements. Intrinsic trunk stiffness has the advantage of producing restoring forces immediately following trunk displacements, while reflexive or voluntary restoring forces are delayed. Our previous work showed that the reflex force produced in response to these trunk displacements is very small compared to the force due to the intrinsic component,¹⁸ and these reflex forces often disagree with EMG measures,²⁰ so our analysis focused on the study of the intrinsic component only.

The elastic component (K) of intrinsic stiffness was computed by integrating the intrinsic stiffness, using the following equation:

$$K = \int_{-0.06}^{0.06} h(\tau)d\tau$$

This is the main measure reported here, as it has been shown to be a component that changes from task to task, as well as the most widely studied component of dynamic stiffness.^{28,45} This elastic component details the steady-state force produced to counteract a displacement of a specific amplitude, like spring stiffness, and is essential for counteracting the destabilizing force of gravity, which acts like a negative (destabilizing) spring. Furthermore, previous studies have shown that the viscous component of intrinsic stiffness changes proportionally with the elastic component, so quantifying the elastic component is sufficient for understanding the intrinsic stiffness changes with muscle activation and the LSO.¹⁵

[RESEARCH REPORT]

This methodology to quantify lumbar stiffness contrasts with the methods in the study by Vera-Garcia et al,⁴² which measured the amplitude of lumbar movement following sudden perturba-

tions. Although both protocols produce sudden (unanticipated) trunk perturbations, the approach used in the current study had several advantages: (1) it required less time to make measurements

(275 perturbations within a 75-second period), (2) it provided dynamic quantification of lumbar stiffness, (3) it separated the intrinsic and reflexive mechanical contributions responsible for lumbar stiffness, and (4) it provided quantification of the lumbar stiffness independent of the perturbation.

Ultrasound and EMG Data Analyses An **ONLINE VIDEO** of ultrasound images, as well as an **APPENDIX** containing detailed analyses and results, is provided at www.jospt.org. Briefly, the ADIM was successfully sustained during the whole 75-second perturbation period.

The EMG signals collected during the MVCs were processed (detrended; band-pass filtered between 30 and 450 Hz with an eighth-order dual-pass Butterworth filter; root-mean-square amplitude quantification using 90% overlapped, 250-millisecond time windows) to obtain the MVA values for EMG normalization purposes. The EMG signals collected during the trunk perturbations were processed as detailed elsewhere.²⁰ Briefly, the filtered signals were separated into background and reflex components by aligning the EMG signal to each rising and falling edge of the perturbation signal, essentially aligning the EMG signal to each forward and backward perturbation separately. The EMG signals were then ensemble averaged (across 135 forward or backward perturbations), and the samples corresponding to the EMG background (−250 to 0 milliseconds) were averaged and, finally, normalized to the MVA values. These normalized EMG values for the longissimus, rectus abdominis, and external oblique were further averaged across homologous muscles, except for the external oblique, for which the EMG values were collected on the left side only.

Statistical Analyses Statistical tests were run to test our 2 hypotheses, first of the effect of the active interventions (without the 3 LSO conditions), and second of the combined effects of bracing and the LSO. To assess our first hypothesis,

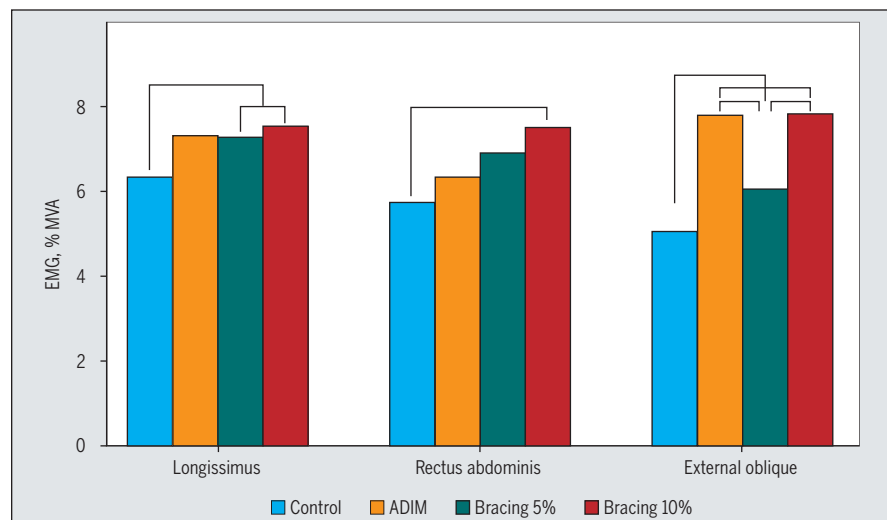


FIGURE 2. Mean muscle activation (25 participants) of the longissimus, rectus abdominis, and left external oblique across 4 experimental conditions. Standard deviations (range across muscles and conditions, 3.2%–6.4% MVA) were not displayed for clarity. Statistically significant differences between experimental conditions are identified by lines on top of the bars. Abbreviations: ADIM, abdominal drawing-in maneuver; EMG, electromyography; MVA, maximal voluntary activation.

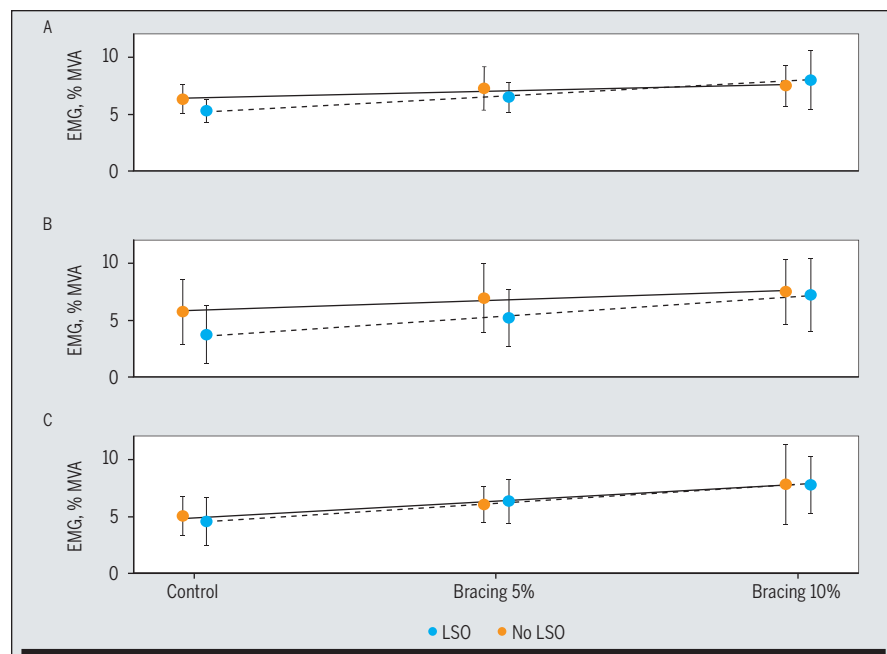


FIGURE 3. Background muscle activation (EMG) of the (A) longissimus, (B) rectus abdominis, and (C) external oblique as a function of bracing level (0%, 5%, and 10% MVA) and wearing an LSO. The lines represent the linear regression parameters (intercept, slope) resulting from the mixed-effect models. Abbreviations: EMG, electromyography; LSO, lumbosacral orthosis; MVA, maximal voluntary activation.

a 1-way analysis of variance (ANOVA) for repeated measures on the factor of experimental condition (control, ADIM, BR5, BR10) was computed. Significant interactions or main effects were further analyzed using a post hoc Tukey-Kramer test, as it represents a good balance between the control of type 1 and type 2 errors. To test our second hypothesis, a mixed-effects model was used, with LSO as a fixed categorical variable, bracing as a continuous fixed factor, and participant as a random factor. All analyses were done with a significance level set at $P<.05$.

RESULTS

ONLY A FEW TRIALS NEEDED TO BE repeated due to participant error (3 participants had to repeat 1 trial each), showing the effectiveness of the familiarization session.

Background Muscle Activation

Background EMG values varied between different active intervention conditions. The different active interventions (ADIM, BR5, BR10) and the control condition resulted in a significant difference in background EMG for each muscle (FIGURE 2), as assessed by an ANOVA ($P<.01$). For the longissimus, post hoc analyses showed increased EMG activity for the BR5 and BR10 conditions relative to the control condition. For the rectus abdominis, the BR10 condition was higher than the control condition. For the external oblique, several differences were detected: the control condition was less than the ADIM, BR5, and BR10 conditions; the ADIM condition was greater than the BR5 condition; and the BR5 condition was less than the BR10 condition.

Background muscle activation increased with increased bracing and was also affected by using an LSO (TABLE 2,

FIGURE 3). For all muscles, as the bracing level increased, background muscle activity increased. Additionally, wearing the LSO decreased background EMG in all muscles. The significant bracing-by-LSO interaction observed for EMG activity of the longissimus and rectus abdominis showed that wearing an LSO decreased activity at low bracing levels (0% and 5%), but this difference disappeared at higher levels (FIGURE 3). Regarding EMG activity of the external oblique, no effect was observed, as expected; the activity of the external oblique was controlled experimentally to reach either 5% or 10% MVA, using visual feedback under both the LSO and non-LSO conditions.

Lumbar Intrinsic Stiffness

Both of the active interventions increased lumbar stiffness. An ANOVA testing the effect of the different active interventions (ADIM, BR5, BR10) and control

TABLE 2

STATISTICAL RESULTS CORRESPONDING TO THE MIXED-EFFECT MODEL FOR BACKGROUND MUSCLE ACTIVATION OR LUMBAR INTRINSIC STIFFNESS AS THE DEPENDENT VARIABLE

Measure/Model	Estimate*	SE	t	df	P Value	R ²
EMG: longissimus						0.87
Intercept	6.5 (4.7, 8.3)	0.9	7.08	296	<.001	
Bracing	0.1 (0.0, 0.2)	0.0	3.32	296	.001	
LSO	-1.2 (-1.8, -0.5)	0.3	-3.69	296	<.001	
Interaction	0.1 (0.0, 0.2)	0.0	2.95	296	.003	
EMG: rectus abdominis						0.73
Intercept	5.9 (4.0, 7.8)	1.0	6.02	296	<.001	
Bracing	0.2 (0.1, 0.3)	0.1	3.05	296	.003	
LSO	-2.2 (-3.2, -1.1)	0.5	-4.09	296	<.001	
Interaction	0.2 (0.0, 0.3)	0.1	2.09	296	.038	
EMG: external oblique (left side)						0.83
Intercept	5.0 (2.9, 7.0)	1.0	4.73	296	<.001	
Bracing	0.3 (0.2, 0.4)	0.0	6.00	296	<.001	
LSO	-0.3 (-1.1, 0.6)	0.4	-0.58	296	.559	
Interaction	0.0 (-0.1, 0.2)	0.1	0.54	296	.593	
Lumbar intrinsic stiffness						0.73
Intercept	2286 (1755, 2818)	269	8.51	146	<.001	
Bracing	100 (55, 144)	22	4.45	146	<.001	
LSO	725 (322, 1129)	204	3.55	146	.001	
Interaction	2 (-60, 65)	32	0.07	146	.945	

Abbreviations: EMG, electromyography; LSO, lumbosacral orthosis; SE, standard error.

*Values in parentheses are 95% confidence interval.

intervention was significant ($P < .001$), while post hoc comparisons showed a significantly higher stiffness for the ADIM (mean $\pm$ SD, 2910 ± 1337 N/m) and BR10 (mean $\pm$ SD, 3361 ± 1489 N/m) conditions than for the control condition (mean $\pm$ SD, 2365 ± 970 N/m). Stiffness in the BR10 condition was also significantly higher than that in the BR5 condition (mean $\pm$ SD, 2627 ± 1211 N/m). Consequently, the ADIM condition was not significantly different from the BR5 and BR10 conditions.

Lumbar intrinsic stiffness depended on both the level of bracing as well as whether the participant wore an LSO (TABLE 2, FIGURE 4), increasing by 100 N/m for each percent increase in EMG activity of the external oblique (bracing) and by 725 N/m when wearing an LSO. Furthermore, bracing and the LSO were found to act independently, as the interaction term was not significant. Lumbar stiffness reached 4011 N/m for the LSO+BR10 condition and was as high as 5011 N/m when extrapolated to LSO and bracing at an external oblique activation level of 20%.

DISCUSSION

THE AIM OF THIS STUDY WAS TO ASSESS the relative effectiveness of active and passive lumbar spine stabilization interventions on lumbar intrinsic stiffness. The main findings were that (1) the ADIM and bracing active interventions were equally efficient when bracing was performed at the same overall abdominal activation level as the ADIM, and (2) combining bracing and wearing an LSO had an additive effect.

Control of Experimental Manipulations

The steps taken to control the abdominal activation (familiarization session, monitoring with ultrasound imaging, and EMG biofeedback) were partly successful in ensuring the ability to perform this challenging task (trunk perturbations). Overall, the EMG measures made in this study are consistent with those made in previous studies, both for the control^{27,42} and active conditions.^{27,42} There was a gradual and statistically significant increase of external oblique activation from the control to the BR5 and then

the BR10 conditions; approximately the same pattern was seen for the longissimus and rectus abdominis, as indicated by some significant differences between conditions.

However, regarding the ADIM condition, although the ultrasound measures showed that the transversus abdominis lateral slide was sustained throughout the trunk perturbation trials, the background muscle activation of the superficial trunk muscles was mostly consistent with the definition of the ADIM, mainly because the external oblique increased to the same level observed in the BR10 condition. It appears that meeting the criteria for a successful ADIM during dynamic tasks is much more difficult than during static tasks, which concurs with previous findings from repeated sub-maximal trunk flexion/extension cycles³⁸ and challenges the premise that isolated transversus abdominis contractions are achievable during dynamic tasks. Unfortunately, we are not aware of other studies that have measured abdominal-wall activation while attempting to produce the ADIM during dynamic tasks. We conclude that the continuous trunk perturbation protocol might have induced involuntary external oblique activation throughout the perturbation interval.

The use of an LSO did have an effect on the activation of the trunk muscles. We did not expect and did not find any changes in the EMG activity of the external oblique, as participants were instructed to match their external oblique muscle activation target levels. Looking instead at the longissimus and rectus abdominis, we found that wearing an LSO decreased trunk muscle activity, which is consistent with the passive support that is theoretically provided by an LSO.¹ Relative to the control condition, this corresponds to a decrease of 1.1% (longissimus) and 1.9% (rectus abdominis) MVA, or an average decrease of 1.5% MVA. These results concur with the decrease in back muscle activation (0.7%-2.2% MVA) measured with the use of an LSO during an unstable sitting task.⁶

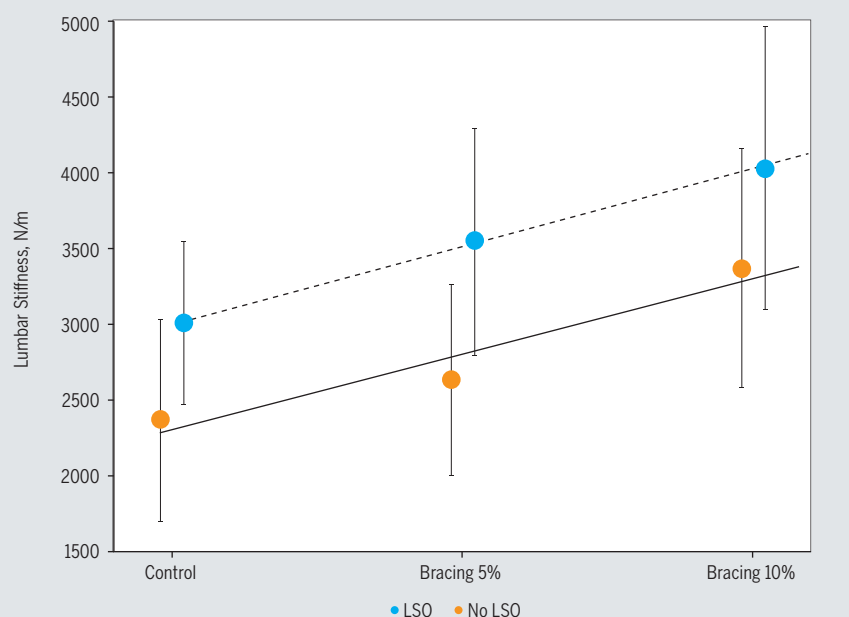


FIGURE 4. Lumbar intrinsic stiffness as a function of bracing level (0%, 5%, and 10% of maximal voluntary activation) and wearing an LSO. The lines represent the linear regression parameters (intercept, slope) resulting from the mixed-effect model. Abbreviation: LSO, lumbosacral orthosis.

Lumbar Intrinsic Stiffness

Effect of Different Active Modalities (ADIM Versus Bracing) All active interventions significantly increased lumbar stiffness, including the ADIM. This is consistent with measures of posteroanterior spinal stiffness, as measured with a protocol of inducing an indented load applied to the L4 spinous process with the use of a servomotor.³⁹ The increase in stiffness during these active interventions means that these interventions would result in a decrease in amplitude of lumbar movement following a sudden force perturbation. Our results for the ADIM differ from the findings of Vera-Garcia et al⁴² that the ADIM was not effective for reducing the amplitude of lumbar movement following sudden perturbations. At first glance, this might be attributed to the unsuccessful control of external oblique activation during the ADIM, as well as to the overall trunk muscle activation (longissimus, rectus abdominis, external oblique) at levels similar to those found in the bracing conditions. However, as discussed earlier, if it is not possible to meet the criteria of a successful ADIM during dynamic tasks, then the trunk muscle activation observed here may be the best that can be achieved, considering that the criterion of a sustained transversus abdominis lateral slide was met. We intentionally selected bracing activation levels (5% and 10% MVA) corresponding to the range of abdominal-wall activation levels achieved when performing the ADIM⁴² to allow for a fair comparison between the bracing and ADIM active modalities. Consequently, it might not be so surprising that no differences were observed. If this is so, then the question of which abdominal maneuver (ADIM versus bracing) provides more lumbar stability during dynamic activities becomes obsolete. Further studies are needed to address this question during dynamic tasks. How the ADIM is operationally defined should also be considered, as various definitions exist.

Another important issue is that Vera-Garcia et al⁴² provided the biofeedback of internal oblique activity as a surro-

gate measure of transversus abdominis activity to reach 10%, 15%, and 20% MVA. Although surface EMG can be used to measure the combined activity of the transversus abdominis and internal oblique,^{21,24} the use of ultrasound biofeedback to train the ADIM may lead to different results, as we simply asked participants to perform the ADIM to generate a maximal lateral slide of the transversus abdominis fascial tip. This might have ensured that the transversus abdominis was pulling on the medial layer of the thoracolumbar fascia. Interestingly, a balanced tension between the deep abdominal (transversus abdominis and internal oblique) and lumbar spinal muscles would allow for applying tension on the medial and posterior layers of the thoracolumbar fascia, respectively, which, in turn, would increase the stability of the lumbar spine.⁴³ Our methodology requires back muscles to produce a 10- to 15-Nm back extension torque, thus allowing this lumbar stability mechanism to operate, which may further explain why the ADIM significantly increased lumbar stiffness in the present study.

While both active interventions increased lumbar stiffness to similar levels, bracing has the potential to increase lumbar stiffness to a much greater extent than the ADIM. Considering that bracing can range between 10% and 20%, as might be reasonably expected,^{8,42} bracing would result in stiffness values that would be significantly higher than those produced by the ADIM (TABLE 2, FIGURE 4). Effectively, assuming a linear relationship between trunk muscle cocontraction and lumbar stiffness between 10% and 20% MVA, as supported by previous findings,^{3,41,42} bracing at 20% MVA would increase lumbar stiffness to 4286 N/m instead of 3286 N/m at 10% MVA, which represents a substantial 30% increase. This would concur with previous findings looking at the amplitude of lumbar movement following sudden perturbations,⁴² as well as with measures of posteroanterior stiffness at L4 using an indented load.³⁹

Combined Effect of Active (Bracing) and Passive (LSO) Modalities Bracing can increase lumbar stability more than an LSO. Bracing increased lumbar stiffness by 100 N/m for each percent increase of external oblique activation, reaching 2000 N/m for BR10. This is higher than the increase estimated in lumbar stiffness generated through wearing an LSO computed in the present study (725 N/m) and in our previous study (1036 N/m).¹⁸ However, biomechanical modeling simulations have shown that this is at the expense of increasing lumbar compression,⁹ contrary to the use of an LSO.¹

The use of an LSO and bracing acted independently in increasing lumbar stiffness, as the interaction term between bracing and the use of an LSO was not significant. This concurs with previous findings,³ which are extended here during a prolonged task requiring breathing. It is, therefore, conceivable to combine these 2 treatment approaches in certain situations, keeping in mind that the training of abdominal muscle activation should first be looked at and considering a careful prescription of an LSO. This might be beneficial for patients who have initiated the training of abdominal bracing but have not yet mastered the maneuver, and are considered to need some external support to perform home or work physical activities safely. An LSO would be used temporarily, until the clinician judges that the patient correctly performs the bracing maneuver. Such a treatment strategy may promote a faster resumption of physical activities, as recommended in all clinical practice guidelines. The LSO could then be used solely when acute flare-ups occur, allowing the patient to maintain or resume physical/work activities until the pain subsides to a manageable level. Lumbosacral orthoses have been shown to induce an immediate and clinically significant pain decrease.³⁶ In addition, although it reduces trunk muscle activity to a small extent, such a decrease of the tonic muscle activity of several trunk muscles, when applied over a long period of time, has the potential

to reduce pain symptoms as a result of the lower forces imposed on the lumbar spine.¹⁰⁻¹² Consequently, wearing an LSO can be considered a pain self-management strategy for the patient.

Study Limitations

The findings of the present study are limited to healthy participants. With regard to the ADIM, additional studies are required to test whether it can be sustained during dynamic tasks. Another limitation is that the experimental protocol used to answer the questions required the control of many confounding variables, which may affect the generalizability of the findings to normal activities. Finally, only a flexible and extensible LSO was considered, and bracing was limited to 10% MVA, which is at the lower end of the possible range of abdominal-wall activation. Consequently, predictions of stiffness values at bracing levels of greater than 10% MVA are speculative and should be interpreted with caution.

CONCLUSION

THE ADIM AND BRACING ACTIVE INTERVENTIONS were equally efficient at increasing lumbar stiffness when bracing was performed at the same overall abdominal activation level as the ADIM. However, extrapolation through statistical modeling suggests that bracing at higher activation levels may lead to greater lumbar stiffness than that provided by the ADIM. It was also shown that abdominal bracing and wearing an LSO have an additive effect. This raises the possibility of combining these 2 treatment approaches in certain clinical situations. However, the individual and combined clinical effects of these active and passive interventions remain to be tested longitudinally in patients with LBP. ●

KEY POINTS

FINDINGS: The abdominal drawing-in maneuver provides lumbar stiffness similar to bracing when the latter is performed

at the same overall abdominal activation levels (5%-10%). Combining bracing and a lumbosacral orthosis has an additive effect on lumbar stiffness.

IMPLICATIONS: The independent effect of bracing and a lumbosacral orthosis raises the possibility of combining these interventions in some circumstances.

CAUTION: The individual and combined clinical effects of bracing and a lumbosacral orthosis remain to be tested longitudinally in patients with low back pain.

REFERENCES

- Cholewicki J. The effects of lumbosacral orthoses on spine stability: what changes in EMG can be expected? *J Orthop Res*. 2004;22:1150-1155. <https://doi.org/10.1016/j.jorthres.2004.01.009>
- Cholewicki J, Ivancic PC, Radebold A. Can increased intra-abdominal pressure in humans be decoupled from trunk muscle co-contraction during steady state isometric exertions? *Eur J Appl Physiol*. 2002;87:127-133. <https://doi.org/10.1007/s00421-002-0598-0>
- Cholewicki J, Juluru K, Radebold A, Panjabi MM, McGill SM. Lumbar spine stability can be augmented with an abdominal belt and/or increased intra-abdominal pressure. *Eur Spine J*. 1999;8:388-395. <https://doi.org/10.1007/s005860050192>
- Cholewicki J, Lee AS, Reeves NP, Morrisette DC. Comparison of trunk stiffness provided by different design characteristics of lumbosacral orthoses. *Clin Biomech (Bristol, Avon)*. 2010;25:110-114. <https://doi.org/10.1016/j.clinbiomech.2009.10.010>
- Cholewicki J, McGill SM. Mechanical stability of the *in vivo* lumbar spine: implications for injury and chronic low back pain. *Clin Biomech (Bristol, Avon)*. 1996;11:1-15. [https://doi.org/10.1016/0268-0033\(95\)00035-6](https://doi.org/10.1016/0268-0033(95)00035-6)
- Cholewicki J, Reeves NP, Everding VQ, Morrisette DC. Lumbosacral orthoses reduce trunk muscle activity in a postural control task. *J Biomech*. 2007;40:1731-1736. <https://doi.org/10.1016/j.jbiomech.2006.08.005>
- Cholewicki J, Silfies SP, Shah RA, et al. Delayed trunk muscle reflex responses increase the risk of low back injuries. *Spine (Phila Pa 1976)*. 2005;30:2614-2620.
- Coenen P, Campbell A, Kemp-Smith K, O'Sullivan P, Straker L. Abdominal bracing during lifting alters trunk muscle activity and body kinematics. *Appl Ergon*. 2017;63:91-98. <https://doi.org/10.1016/j.apergo.2017.04.009>
- Grenier SG, McGill SM. Quantification of lumbar stability by using 2 different abdominal activation strategies. *Arch Phys Med Rehabil*. 2007;88:54-62. <https://doi.org/10.1016/j.apmr.2006.10.014>
- Healey EL, Burden AM, McEwan IM, Fowler NE. Diurnal variation in stature: do those with chronic low-back pain differ from asymptomatic controls? *Clin Biomech (Bristol, Avon)*. 2011;26:331-336. <https://doi.org/10.1016/j.clinbiomech.2010.11.017>
- Healey EL, Burden AM, McEwan IM, Fowler NE. The impact of increasing paraspinal muscle activity on stature recovery in asymptomatic people. *Arch Phys Med Rehabil*. 2008;89:749-753. <https://doi.org/10.1016/j.apmr.2007.09.044>
- Healey EL, Fowler NE, Burden AM, McEwan IM. Raised paraspinal muscle activity reduces rate of stature recovery after loaded exercise in individuals with chronic low back pain. *Arch Phys Med Rehabil*. 2005;86:710-715. <https://doi.org/10.1016/j.apmr.2004.10.026>
- Henry SM, Westervelt KC. The use of real-time ultrasound feedback in teaching abdominal hollowing exercises to healthy subjects. *J Orthop Sports Phys Ther*. 2005;35:338-345. <https://doi.org/10.2519/jospt.2005.35.6.338>
- Jorgensen MJ, Marras WS. The effect of lumbar back support tension on trunk muscle activity. *Clin Biomech (Bristol, Avon)*. 2000;15:292-294. [https://doi.org/10.1016/S0268-0033\(99\)00067-4](https://doi.org/10.1016/S0268-0033(99)00067-4)
- Kearney RE, Hunter IW. System identification of human joint dynamics. *Crit Rev Biomed Eng*. 1990;18:55-87.
- Larivière C, Gagnon D, De Oliveira E, Jr., Henry SM, Mecheri H, Dumas JP. Reliability of ultrasound measures of the transversus abdominis: effect of task and transducer position. *PM R*. 2013;5:104-113. <https://doi.org/10.1016/j.pmrj.2012.11.002>
- Larivière C, Gagnon D, Genest K. Offering proper feedback to control for out-of-plane lumbar moments influences the activity of trunk muscles during unidirectional isometric trunk exertions. *J Biomech*. 2009;42:1498-1505. <https://doi.org/10.1016/j.jbiomech.2009.03.045>
- Larivière C, Ludvig D, Kearney R, Mecheri H, Caron JM, Preuss R. Identification of intrinsic and reflexive contributions to low-back stiffness: medium-term reliability and construct validity. *J Biomech*. 2015;48:254-261. <https://doi.org/10.1016/j.jbiomech.2014.11.036>
- Lee NG, You JS, Kim TH, Choi BS. Unipedal postural stability in nonathletes with core instability after intensive abdominal drawing-in maneuver. *J Athl Train*. 2015;50:147-155. <https://doi.org/10.4085/1062-6050-49.3.91>
- Ludvig D, Larivière C. Trunk muscle reflexes are elicited by small continuous perturbations in healthy subjects and patients with low-back pain. *J Electromyogr Kinesiol*. 2016;30:111-118. <https://doi.org/10.1016/j.jelekin.2016.06.007>
- Marshall P, Murphy B. The validity and reliability of surface EMG to assess the neuromuscular response of the abdominal muscles to rapid limb movement. *J Electromyogr Kinesiol*. 2003;13:477-489. [https://doi.org/10.1016/S1050-6411\(03\)00027-0](https://doi.org/10.1016/S1050-6411(03)00027-0)
- McGill S. *Low Back Disorders: Evidence-Based*

Prevention and Rehabilitation. Champaign, IL: Human Kinetics; 2002.

23. McGill S. *Ultimate Back Fitness and Performance*. Waterloo, Canada: Wabuno Publishers; 2004.
24. McGill S, Juker D, Kropf P. Appropriately placed surface EMG electrodes reflect deep muscle activity (psoas, quadratus lumborum, abdominal wall) in the lumbar spine. *J Biomech*. 1996;29:1503-1507. [https://doi.org/10.1016/0021-9290\(96\)84547-7](https://doi.org/10.1016/0021-9290(96)84547-7)
25. McGill S, Seguin J, Bennett G. Passive stiffness of the lumbar torso in flexion, extension, lateral bending, and axial rotation. Effect of belt wearing and breath holding. *Spine (Phila Pa 1976)*. 1994;19:696-704.
26. McGill SM, Grenier S, Kavcic N, Cholewicki J. Coordination of muscle activity to assure stability of the lumbar spine. *J Electromyogr Kinesiol*. 2003;13:353-359. [https://doi.org/10.1016/S1050-6411\(03\)00043-9](https://doi.org/10.1016/S1050-6411(03)00043-9)
27. McPhee M, Tucker KJ, Wan A, MacDonald DA. Perceived task complexity of trunk stability exercises. *Musculoskelet Sci Pract*. 2017;27:57-63. <https://doi.org/10.1016/j.msksp.2016.11.009>
28. Mirbagheri MM, Barbeau H, Kearney RE. Intrinsic and reflex contributions to human ankle stiffness: variation with activation level and position. *Exp Brain Res*. 2000;135:423-436. <https://doi.org/10.1007/s002210000534>
29. Moorhouse KM, Granata KP. Role of reflex dynamics in spinal stability: intrinsic muscle stiffness alone is insufficient for stability. *J Biomech*. 2007;40:1058-1065. <https://doi.org/10.1016/j.jbiomech.2006.04.018>
30. Panjabi MM. Clinical spinal instability and low back pain. *J Electromyogr Kinesiol*. 2003;13:371-379. [https://doi.org/10.1016/S1050-6411\(03\)00044-0](https://doi.org/10.1016/S1050-6411(03)00044-0)
31. Pedersen MT, Essendrop M, Skotte JH, Jørgensen K, Schibye B, Fallentin N. Back muscle response to sudden trunk loading can be modified by training among healthcare workers. *Spine (Phila Pa 1976)*. 2007;32:1454-1460. <https://doi.org/10.1097/BRS.0b013e318060a5a7>
32. Radebold A, Cholewicki J, Panjabi MM, Patel TC. Muscle response pattern to sudden trunk loading in healthy individuals and in patients with chronic low back pain. *Spine (Phila Pa 1976)*. 2000;25:947-954.
33. Redfern M, Hughes R, Chaffin D. High-pass filtering to remove electrocardiographic interference from torso EMG recordings. *Clin Biomech (Bristol, Avon)*. 1993;8:44-48. [https://doi.org/10.1016/S0268-0033\(05\)80009-9](https://doi.org/10.1016/S0268-0033(05)80009-9)
34. Reeves NP, Cholewicki J, Milner TE. Muscle reflex classification of low-back pain. *J Electromyogr Kinesiol*. 2005;15:53-60. <https://doi.org/10.1016/j.jelekin.2004.07.001>
35. Richardson C, Hodges P, Hides J. *Therapeutic Exercise for Lumbopelvic Stabilization: A Motor Control Approach for the Treatment and Prevention of Low Back Pain*. 2nd ed. Edinburgh, UK: Churchill Livingstone; 2004.
36. Shahvarpour A, Preuss R, Sullivan MJL, Negrini A, Larivière C. The effect of wearing a lumbar belt on biomechanical and psychological outcomes related to maximal flexion-extension motion and manual material handling. *Appl Ergon*. 2018;69:17-24. <https://doi.org/10.1016/j.apergo.2018.01.001>
37. Silfies SP, Mehta R, Smith SS, Karduna AR. Differences in feedforward trunk muscle activity in subgroups of patients with mechanical low back pain. *Arch Phys Med Rehabil*. 2009;90:1159-1169. <https://doi.org/10.1016/j.apmr.2008.10.033>
38. Southwell DJ, Hills NF, McLean L, Graham RB. The acute effects of targeted abdominal muscle activation training on spine stability and neuromuscular control. *J Neuroeng Rehabil*. 2016;13:19. <https://doi.org/10.1186/s12984-016-0126-9>
39. Stanton T, Kawchuk G. The effect of abdominal stabilization contractions on posteroanterior spinal stiffness. *Spine (Phila Pa 1976)*. 2008;33:694-701. <https://doi.org/10.1097/BRS.0b013e318166e034>
40. Thomas S, Reading J, Shephard RJ. Revision of the Physical Activity Readiness Questionnaire (PAR-Q). *Can J Sport Sci*. 1992;17:338-345.
41. Vera-Garcia FJ, Brown SH, Gray JR, McGill SM. Effects of different levels of torso coactivation on trunk muscular and kinematic responses to posteriorly applied sudden loads. *Clin Biomech (Bristol, Avon)*. 2006;21:443-455. <https://doi.org/10.1016/j.clinbiomech.2005.12.006>
42. Vera-Garcia FJ, Elvira JL, Brown SH, McGill SM. Effects of abdominal stabilization maneuvers on the control of spine motion and stability against sudden trunk perturbations. *J Electromyogr Kinesiol*. 2007;17:556-567. <https://doi.org/10.1016/j.jelekin.2006.07.004>
43. Vleeming A, Schuenke MD, Danneels L, Willard FH. The functional coupling of the deep abdominal and paraspinal muscles: the effects of simulated paraspinal muscle contraction on force transfer to the middle and posterior layer of the thoracolumbar fascia. *J Anat*. 2014;225:447-462. <https://doi.org/10.1111/joa.12227>
44. Wilder DG, Aleksiev AR, Magnusson ML, Pope MH, Spratt KF, Goel VK. Muscular response to sudden load. A tool to evaluate fatigue and rehabilitation. *Spine (Phila Pa 1976)*. 1996;21:2628-2639.
45. Zhang LQ, Nuber G, Butler J, Bowen M, Rymer WZ. *In vivo* human knee joint dynamic properties as functions of muscle contraction and joint position. *J Biomech*. 1997;31:71-76. [https://doi.org/10.1016/S0021-9290\(97\)00106-1](https://doi.org/10.1016/S0021-9290(97)00106-1)



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APPENDIX

ULTRASOUND MEASURES OF THE ABDOMINAL DRAWING-IN MANEUVER DURING TRUNK PERTURBATIONS

All quantitative ultrasound examinations were conducted using a Philips HD11 1.0.6 ultrasound machine (Koninklijke Philips NV, Amsterdam, the Netherlands). A 12- to 5-MHz, 50-mm, linear-array transducer (model L12-5; Koninklijke Philips NV) was positioned to image the 3 layers of the abdominal-wall muscles in the transverse plane: transversus abdominis (TrA), internal oblique, and external oblique. The transducer was positioned at the navel level and then laterally, in order to see the medial end of the TrA (fascial tip), as further explained and seen in **FIGURE 1**.

Collecting videos during the abdominal drawing-in maneuver and quantifying the TrA lateral slide (off line) were challenging, as images were oscillating due to trunk perturbations (see **ONLINE VIDEO**). Also, the transducer was likely not perfectly maintained at the same position throughout the 75-second trials, which forced the use of different measurement strategies to get a fair appreciation of activation of the TrA.

The lateral slide of the TrA tip, representing the shortening of the TrA muscle, was computed using a custom-made MATLAB program (The MathWorks, Inc, Natick, MA), by calculating the distance between the tip identified at rest and during contraction, using x and y coordinates (**FIGURE 1**). More specifically, as illustrated in **FIGURE 2**, measures were taken as follows: (1) at rest before (R_{beg}) and after (R_{end}) performing the abdominal drawing-in maneuver and trunk perturbations; (2) during the contraction of the TrA just before (C_{beg}) and just after (C_{end}) trunk perturbations; and (3) during the abdominal drawing-in maneuver and trunk perturbations at each 15-second interval (C_{T0} , C_{T15} , C_{T30} , C_{T45} , C_{T60} , C_{T75}).

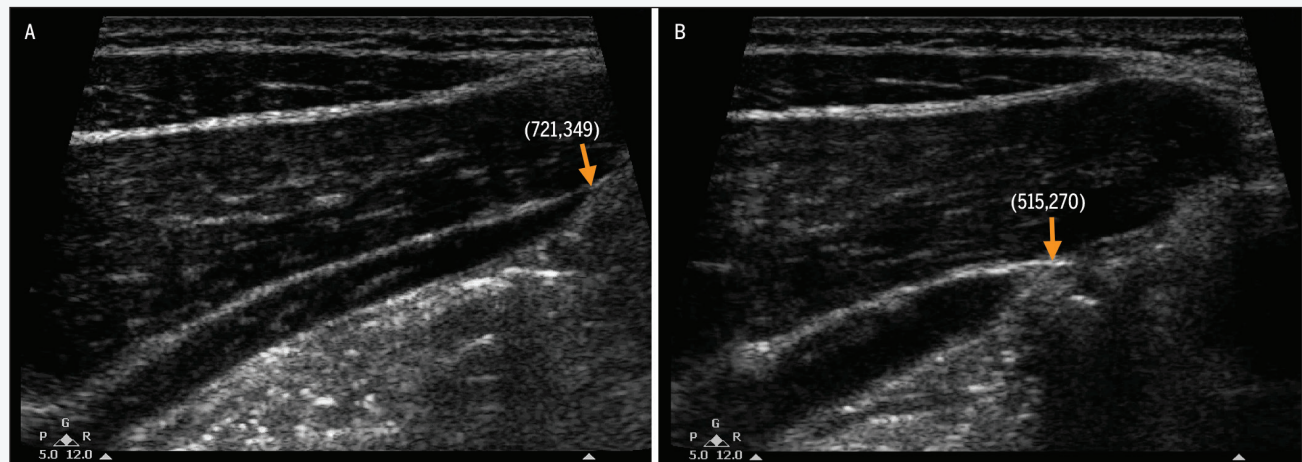


FIGURE 1. Image and measures of the transversus abdominis lateral slide, from rest (A) to contraction (B), during the abdominal drawing-in maneuver. The transducer was positioned so that the fascial tip of the transversus abdominis (identified with an arrow in the images) was at some distance from the border of the image (approximately 20% of the image width, as illustrated in [A]). This allowed us to capture the movement of the fascial tip (lateral slide) between (A) rest and (B) contraction. This movement was quantified with the use of the Cartesian coordinates corresponding to the pixel identified using the custom-made MATLAB (The MathWorks, Inc, Natick, MA) program. The image calibration allowed us to transform the pixel position to millimeters.

A TrA lateral slide index (I) was computed for each time interval (T_0 - T_{75}) of the 75-second trunk perturbation trial, with the use of 2 rest references (R_{beg} and R_{end}) and normalized with 2 values (C_{beg} and C_{end}) in order to obtain percentages. To account for the possibility that the ultrasound probe position changed between the beginning and end of the trial, the index was computed with “beginning” and “end” references as follows (T_0 example):

$$I_{T_0}(R_{\text{beg}}, C_{\text{beg}}) = [(C_{T_0} - R_{\text{beg}}) / C_{\text{beg}}] \times 100$$

$$I_{T_0}(R_{\text{end}}, C_{\text{end}}) = [(C_{T_0} - R_{\text{end}}) / C_{\text{end}}] \times 100$$

The results showed that the TrA slide index was on average very close to 100%, or a little over (**TABLE**) the rest reference values (R_{beg} or R_{end}) or values used for normalization (C_{beg} or C_{end}). The SD values were relatively large (range, 8%-21%), which is explained by interindividual variability as well as measurement errors due to image oscillations during trunk perturbations.

Four 1-way analyses of variance for repeated measures were conducted to test whether the abdominal drawing-in maneuver was sustained through the 75-second trunk perturbation task (T_0 , T_{15} , T_{30} , T_{45} , T_{60} , T_{75}). None reached statistical significance, P values being the same (.211) for the 2 approaches to compute the TrA lateral slide index.

APPENDIX

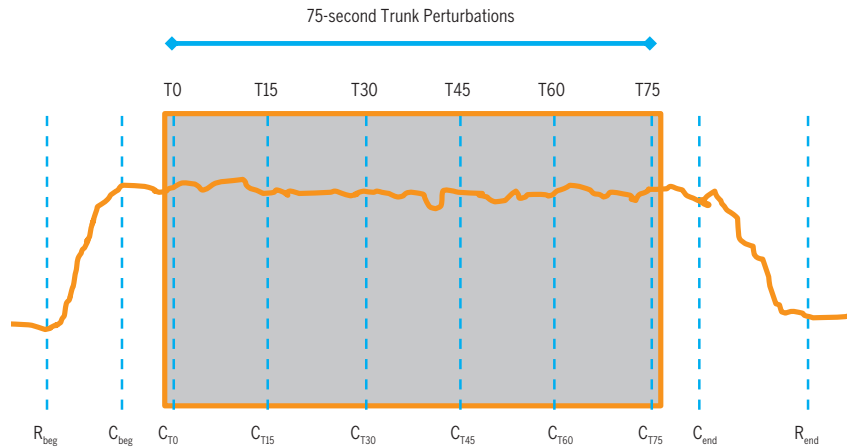


FIGURE 2. Illustration of the transversus abdominis lateral slide (not a true signal) and of the time frames where the x and y coordinates (**FIGURE 1**) of the fascial tip of the transversus abdominis were digitized. The gray area represents the trunk perturbations' time interval. Abbreviations: C_{beg}, beginning of contraction; C_{end}, end of contraction; R_{beg}, beginning of rest; R_{end}, end of rest; T, time interval.

TABLE

Transversus Abdominis Lateral Slide Index, Computed at Each Time Interval With the Use of 2 Rest References and Normalized With 2 Values*

Variable	T0	T15	T30	T45	T60	T75
I (R _{beg} , C _{beg})	99 ± 8	105 ± 14	102 ± 16	106 ± 21	107 ± 21	107 ± 20
I (R _{end} , C _{end})	99 ± 20	104 ± 16	100 ± 12	105 ± 19	104 ± 12	103 ± 9

Abbreviations: C_{beg}, beginning of contraction; C_{end}, end of contraction; I, lateral slide index; R_{beg}, beginning of rest; R_{end}, end of rest; T, time interval.

*Values are mean ± SD (n = 25 participants).

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The Epidemiology of Low Back Pain and Injury in Dance: A Systematic Review

Dance is a physical pursuit that boasts high global popularity. In Australia, it has the highest participation rate for all cultural, sporting, and leisure activities among girls, and the second highest participation rate for children of both sexes.⁵ In the United States, dance is estimated to account for 39% of the total moderate to vigorous physical activity achieved by adolescent girls and 23% of the total achieved by adolescent boys.⁵⁵

The physically demanding nature of dance has been well documented. Students from preprofessional ballet schools in the United Kingdom complete more training hours than commonly reported by other adolescent athlete populations.²¹

Australian professionals, including both company and independent dancers, typically complete in excess of 30 dance-hours per week in class, rehearsal, and performance, which they often manage alongside multiple other roles within the dance industry.⁸¹ Moreover, it has been established that dancers are vulnerable to a high degree of musculoskeletal pain



and injury,^{35,39,42} a significant proportion of which includes pain and injury in the lower back.⁵⁸

Observational studies have documented high prevalence rates of low back pain (LBP) in

contemporary and ballet dancers, which has been associated with activity modification, care seeking, and medication use.^{76,77} Furthermore, LBP and low back injury (LBI) have been identified as common and often severe causes of time-loss injury in both preprofessional and professional dancers.^{4,21} This problem has been attributed to the unique and highly physical movement demands of dance.^{56,59}

Indeed, spinal pathologies such as spondylolysis, a defect caused by alternating full flexion and extension movements,¹ are more common in ballet dancers than in the general population.⁶⁵ Further, the incidence of spine stress fractures in professional ballet dancers appears to increase with dance-hours completed.⁴⁰

Due to the heterogeneous injury definitions and reporting methods used in dance injury surveillance studies,^{42,46,75} and the complexities of assessing pain and chronic injury outcomes,⁶ determining the extent to which LBP and LBI are a problem in dance is not straightforward. Therefore, to advance the understanding of LBP and LBI in dance, the primary aim

• **BACKGROUND:** Dance is a physical pursuit that involves loading the spine through repetitive dynamic movements and lifting tasks. As such, low back pain (LBP) and low back injury (LBI) have been identified as common health problems in contemporary and classical ballet dancers. However, clarity regarding the experience of LBP and LBI in dance is lacking.

• **OBJECTIVES:** To systematically review and synthesize the epidemiology of LBP and LBI in dance populations.

• **METHODS:** A comprehensive search of 6 electronic databases, back catalogs of dance science-specific journals, and reference lists of relevant articles and a forward citation search were performed.

• **RESULTS:** Fifty full-text articles were included in the final systematic review. There was considerable

methodological heterogeneity among the included studies. The median (range) point, yearly, and lifetime prevalence of LBP was 27% (17%-39%), 73% (41%-82%), and 50% (17%-88%), respectively. The lower back contributed to 11% (4%-22%) of time loss and 11% (5%-23%) of medical-attention injuries.

• **CONCLUSION:** Dancers are vulnerable to LBP and LBI. The use of definitions that are sensitive to the complexity of LBP and LBI would facilitate improved understanding of the problem within dance, inform health care strategies, and allow for monitoring LBP-specific intervention outcomes. *J Orthop Sports Phys Ther* 2019;49(4):239-252. Epub 18 Jan 2019. doi:10.2519/jospt.2019.8609

• **KEY WORDS:** ballet, contemporary dance, incidence, prevalence, risk factors

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of this review was to systematically assess the available evidence on the prevalence and incidence of LBP and LBI in preprofessional and professional dance populations. A secondary aim was to identify any risk factors in these populations for LBP and LBI.

METHODS

THIS SYSTEMATIC REVIEW IS STRUCTURED in accordance with the Preferred Reporting Items for Systematic Reviews and Meta-Analyses (PRISMA) statement.⁵² The review was registered via PROSPERO (CRD42017073428) prior to commencement.

Search Strategy

Relevant publications were identified through systematic searches of the following 6 electronic databases up until June 25, 2018: MEDLINE, SPORTDiscus, Web of Science, Embase, CINAHL, and the ProQuest Performing Arts Periodicals Database. The search strategy included a combination of controlled vocabulary (eg, Medical Subject Headings of the National Library of Medicine) and free-text terms (TABLE 1). In addition, the Online Dance Medicine and Science Bibliography, back catalogs of dance-specific journals, including the *Journal of Dance Medicine and Science* and *Medical Problems of Performing Artists*, and reference lists from comprehensive reviews and identified studies were hand searched for possible references not otherwise found. Forward citation searching via Google Scholar was

also performed. The search was limited to those articles published in English, but no date limits for publication were set.

Inclusion/Exclusion Criteria

Cohort or cross-sectional studies were included if they examined the prevalence and incidence of LBP/LBI in dancers, or risk factors for LBP/LBI in dancers, and met the following inclusion criteria. Studies of dancers of both sexes and all ages, including children participating in a structured dance program as well as adults dancing either at a tertiary or professional level, were eligible, but to control for current exposure, studies with only retired dancers were not. To ensure consistency in the type of physical exposure, dance styles including ballet, contemporary, modern, and dance theatre or similar were eligible, whereas other forms of artistic dance (eg, Irish dancing or salsa) or social forms of dance (eg, weddings) were excluded.

All possible definitions of pain and injury (eg, any complaint, medical consultation, disabling/time loss) and duration (eg, acute, chronic) were considered. However, the studies had to clearly report outcomes for the low back or lumbar spine region; studies reporting pain and injury to the back, spine, or lumbopelvic region were excluded. A risk factor was defined as any pre-existing factor that may increase the potential for LBP or LBI in dancers, and was identified through a prospective research design. Studies investigating factors associated with LBP/LBI cross-sectionally that were unable to describe whether the risk factor preceded

the episode of pain were excluded from this component of the review. Studies that reported risk factors for injury but did not delineate the site of the injury were also excluded.

Data Extraction and Risk-of-Bias Assessment

Two reviewers (C.S. and E.B.) independently checked the titles and/or abstracts of all studies returned by the search results. Studies that were clearly not relevant were excluded. The full text of all subsequent studies was assessed to determine whether the selection criteria were met. Any disagreement between review authors was resolved through discussion. Data extraction and risk-of-bias assessment were performed by 2 reviewers (C.S. and D.W.) using a standardized, pre-piloted form. Extracted information included study details (authors, year, country, design, duration), participant information (dance style, level, sex, age, sample size), definition of pain/injury used, collection methods, LBP/LBI estimates (prevalence, incidence, etc), exposure variables (ie, risk factors), reported significance of associations between risk factors and LBP/LBI, and risk factors not significantly associated with LBP/LBI.

The classification of LBP/LBI estimates was dependent on the case definition and data-collection methods used by individual studies. Outcomes from studies that used a definition of pain were classified as LBP, whereas outcomes extracted from studies that used a definition of injury were classified as LBI. The risk-of-bias assessment was performed using a tool to assess risk of bias in prevalence studies (APPENDIX, available at www.jospt.org).³⁷ This tool contains 10 items that address external validity (selection and nonresponse bias) and internal validity (measurement and analysis bias).³⁷ Thus, each study received a score out of 10, with a lower score indicating a lower risk of bias. Risk-of-bias assessment was performed in relation to the assessment and reporting of LBP and LBI outcomes.

TABLE 1

MEDLINE SEARCH STRATEGY AND RESULTS

Data Source	Search Strategy	Hits, n
MEDLINE Complete (EBSCO)	1. Dancing (MeSH) OR Danc* OR Ballet	13850
	2. Back pain (MeSH) OR Low back pain (MeSH) OR Spinal Injuries (MeSH) OR Athletic injuries (MeSH) OR Wounds and Injuries (MeSH) OR Back ache OR Lumbar pain OR Spin* pain OR Lumbago OR Sports Injur*	142841
	3. 1 AND 2	377
	4. Limit to English Language	330

RESULTS

THE LITERATURE SEARCH RETURNED a total of 4121 articles. Following duplication removal and a review of titles and abstracts, 144 full texts were screened, with 98 subsequently excluded. Fifty studies were included in the final review (FIGURE).

Description of the Studies

Of the studies included in this review, 22 were cross-sectional in design, 19 were retrospective, and 9 were prospective. Thirty studies presented data collected from a single cohort or medical center, and 20 included multiple cohorts. Ballet was the predominant style for 31 studies, contemporary or modern for 6, musical theatre for 2, and either a combination of styles or nonexclusive style was featured in 11 studies. Twenty-two studies featured professionals exclusively, 17 featured nonprofessionals, and 11 had a mix of professional and nonprofessional dancers. Descriptive data extracted from the included studies are represented in TABLES 2 and 3.

Risk of Bias

The median risk-of-bias score was 4.5/10. Five studies were judged to have a low risk of bias (deemed as 3/10 or less), which equated to 10% of the studies included in the final review. Studies with a

low risk of bias commonly incorporated a tool with established reliability and validity to measure pain or injury (item 7), provided an adequate anatomical description of the low back (item 6), and obtained a sample that was judged to reflect a national dance population (item 1).

Prevalence of LBP

Fourteen studies^{20,26,33,47,54,57,58,61,62,65,76-78,87} that reported LBP prevalence met the inclusion criteria for this review. Thirteen of these were cross-sectional and 1 was prospective. Multiple tools and LBP definitions were used. Seven studies reported point prevalence,^{76,77} “pain now,”⁸⁷ “recent pain,”⁷⁸ or pain experienced in the last 7 days.^{33,57,58} These studies reported a median (range) prevalence of 27% (17%-39%). Only 1 study⁷⁷ reported monthly prevalence of activity-limiting LBP (LBP that resulted in missed or modified dance practice), which was 22%. Six studies reported LBP experienced for an academic (9 months)⁷⁷ or full (12 months)^{57,58,61,65,76} calendar year. These studies had a median (range) prevalence of 73% (41%-82%) for any LBP, and 33% (25%-52%) for LBP that was associated with activity limitation or disability. One study⁷⁷ identified a 24% prevalence of chronic LBP, which was defined as 3 consecutive monthly episodes of pain, recorded over a 9-month period. The lifetime history of LBP, re-

ported by 6 studies,^{26,47,62,65,76,87} ranged between 17% and 88% and had a median value of 50%. Using only estimates from studies with a low risk of bias had minimal impact on the median (range) values observed for point (27% [17%-39%]) and yearly (78% [70%-82%]) LBP prevalence.

Prevalence of LBI

Five studies reported the prevalence of LBI in dancers.^{8,16,22,58,78} These used a range of designs, definitions, and time periods. The point prevalence of LBI that limited participation was 8% in a single study of predominantly professional contemporary dancers.⁷⁸ During a 7-month season, 25% of professional female dancers and 0% of male dancers experienced an LBI, although this was based on a sample of only 13 dancers (8 female).⁸ Nineteen percent of West End performers reported experiencing an LBI during their current production, albeit with varied time periods of each production.²² History of LBI in professional contemporary and ballet dancers was reported by 2 studies, and history of major LBI (causing more than 1 month away from dance) was reported by 1 study. These values were 23%,¹⁶ 32%,⁷⁸ and 20%,⁵⁸ respectively.

Incidence of LBI

Only 2 studies reported incidence of LBI using a dance-exposure or dance-hour denominator. Incidences of 0.78 per 1000 dance-exposures and 0.53 per 1000 dance-hours were observed in ballet students.⁹ Reported incidence in professional ballet dancers was 0.63 and 0.55 per 1000 dance-hours in females and males, respectively.⁴

LBP and LBI as a Percentage of All Injuries Experienced by Dancers

Thirty-three studies reported the percentage of all injuries sustained by dancers that were to the lower back. Of these, 11 studies (12 estimates) used a time- or activity-loss definition,^{4,7,9,11,12,19,59,66,82,83,85} 16 studies (12 estimates) used a med-

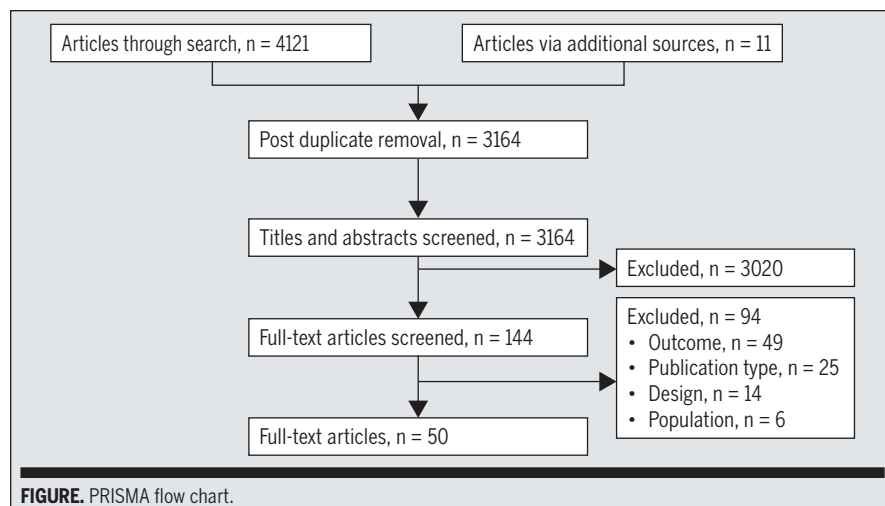


FIGURE. PRISMA flow chart.

[RESEARCH REPORT]

ical-attention or medical-cost definition,^{25,27-30,45,53,56,63,67-71,74,88} and 6 used a definition that was not dependent on time loss or medical attention.^{19,43,44,72,84,86} The median percentage (range) was 11% (4%-22%) for studies that used a time-loss definition, 11% (5%-23%) for studies that used a medical-attention definition, and 12% (6%-21%) for studies that employed separate injury definition criteria. No studies that examined LBI scored a

low risk of bias for this outcome. However, including only studies with a risk-of-bias score equal to or below the median (4.5 or less) had minimal impact on the median (range) of observed values for time-loss (10% [4%-20%]), medical-attention (12% [5%-18%]), or other (10% [9%-11%]) LBI incidence definitions.

The percentage of all injuries accounted for by the lower back was higher in studies that used professional cohorts

exclusively rather than preprofessional cohorts. In preprofessional dancers, the median percentage (range) was 10% (4%-22%) in studies that used a time-loss definition^{7,9,12,21} and 8% (5%-12%) in studies that applied a medical-attention injury definition.^{28,45,63,74,88} In professionals, the median (range) was higher, at 13% (6%-20%) for time-loss definitions^{4,11,59,66,82,83} and 14% (12%-23%) for medical-attention definitions.^{29,53,67-71}

TABLE 2

PREVALENCE OF LBP IN DANCE

Study	Study Type	Level of Evidence	Risk of Bias	Country	Cohort Description	n (% Female)	Age, y*	Definition of LBP	Collection Methods	LBP Prevalence Estimates, %
Drężewska and Śliwiński ²⁰	Cross-sectional	IV	6.5	Poland	1 ballet school	71 (63)	16.5 (15-18)	Pain measured via visual analog scale	Self-report	Period unclear: 62
Gamboa et al ²⁶	Prospective (LBP acquired cross-sectionally)	IV	4	United States	1 ballet school	359 (80) [†]	14.7 ± 1.9	"Subjective history with specific focus on LBP"	Part of medical history pre-screening	Lifetime: 33
Grego Muniz de Araújo et al ³³	Cross-sectional	IV	2	Brazil	1 dance festival. Ballet and other. Professional and other	163 (77)	28.7 ± 9.8	Nordic musculoskeletal questionnaire	Self-report	Last 7 d: all, 39; female, 39; male, 38
Liederbach et al ⁴⁷	Cross-sectional	IV	6	United States	Schools and companies. Classically trained	947 (65)	18-35	"A history of chronic or recurrent LBP"	Self-report	History of chronic/recurrent LBP: all dancers, 17; dancers with scoliosis, 23
Nunes et al ⁵⁴	Cross-sectional	IV	6	Canada	2 dance studios. Young dance students	31 (100)	8-20	Pain identified on a body chart	Self-report	Last month: 8.3
Ramel and Moritz ⁵⁷	Cross-sectional	IV	1	Sweden	3 ballet companies	128 (59)	Female, 27 (18-43); male, 28 (17-47)	Nordic musculoskeletal questionnaire	Self-report	Past 12 mo: 70 Time loss in last 12 mo: 25 Past 7 d: 27
Ramel et al ⁵⁸	Cross-sectional	IV	2	Sweden	3 ballet companies	51 (67)	32 (28-37)	Nordic musculoskeletal questionnaire; major injury defined as one that stopped dance for more than 1 mo	Self-report	Past 12 mo: 82 Time loss in last 12 mo: 33 Past 7 d: 37 Major low back injury history: 19.6
Roussel et al ⁶²	Prospective (LBP acquired cross-sectionally)	IV	4	Belgium	1 preprofessional dance program	32 (81)	20 ± 2	Unspecified	Visual analog scale	History: 63
Roussel et al ⁶¹	Cross-sectional	IV	4	Belgium	1 preprofessional dance program	40 (95)	20.3 ± 2.4 (17-26)	LBP for at least 2 consecutive days	Visual analog scale	Past 12 mo: 41
Seitsalo et al ⁶⁵	Cross-sectional	IV	4.5	Finland	1 ballet company	60 (58) [‡]	28 (21-43)	Any LBP, lumbago, sciatic pain, non-specific LBP	Self-report	History: any LBP, 88; lumbago, 12; sciatic pain, 10 Past 12 mo: 76

Table continues on page 243.

Risk Factors for LBP and LBI

Only 2 studies assessed risk for LBP and adjusted for confounding variables.^{76,77} Other studies performed only univariable analysis, or delineated LBP and injury outcomes based on a potential risk factor but did not perform statistical analysis on these variables. Studies that did perform univariable analysis lacked consistency in reporting the significance of associations between risk factors and LBP. They also did not present or inter-

pret the magnitudes of any identified associations.

Seventeen studies examined sex as a risk factor for LBP and LBI, or delineated outcomes based on sex (TABLE 4).^{4,7,12,16,30,33,57,65,67-70,76,77,82,84,85} No sex-related differences were reported in 11 studies.^{12,33,57,65,67-70,76,77,84} One study observed a higher percentage of self-reported and a lower percentage of physical therapist-reported LBIs in male dancers compared to female dancers.⁷ Four injury studies observed that male dancers expe-

rienced a greater percentage of injuries to the low back than female dancers.^{16,30,82,85} One study observed a higher incidence of LBI in female dancers, although significance was not reported.⁴

Five studies tested for or delineated differences in age (TABLE 4).^{20,30,76,77,87} The prevalence of LBP or proportion of LBI increased as the age of dancers increased in 3 studies.^{20,30,87} Conversely, age was not associated with LBP prevalence in 2 studies that adjusted for confounding

TABLE 2

PREVALENCE OF LBP IN DANCE (CONTINUED)

Study	Study Type	Level of Evidence	Risk of Bias	Country	Cohort Description	n (% Female)	Age, y*	Definition of LBP	Collection Methods	LBP Prevalence Estimates, %
Swain et al ⁷⁶	Cross-sectional	IV	4	Australia	1 ballet school, 2 contemporary universities, 1 ballet company	110 (83)	Female, 179 ± 2.6; male, 17 ± 3.6	Pain in the posterior aspect of the body, from the lower margin of the 12th ribs to the lower gluteal folds	Self-report	Point: all, 12; female, 25.3; male, 15.8 Past 12 mo: all, 64 History: all, 74; female, 78.9; male, 72.5
Swain et al ⁷⁷	Prospective (duration, 9 mo)	II	3	Australia	1 ballet school, 2 contemporary universities, 1 ballet company	119 (84)	Female, 179 ± 2.7; male, 17 ± 3.7	Pain in the posterior aspect of the body, from the lower margin of the 12th ribs to the lower gluteal folds Any episode, activity-limiting LBP (causes modification or missed class), chronic LBP (3 consecutive monthly episodes)	Self-report	9 mo Any LBP: all, 78; female, 78; male, 79 Activity-limiting LBP: all, 52; female, 54; male, 42 Chronic LBP: all, 24; female, 23; male, 32 Point prevalence: 8-25 Monthly prevalence: any LBP, 19-58; activity-limiting LBP, 11-34
Thomas and Tarr ⁷⁸	Cross-sectional	IV	5.5	United Kingdom	Student, professional, retired, and other. Predominantly contemporary dancers	204 (86)	16 to >45	Pain (subjective), injury: participation impact	Questionnaires and semi-structured narrative interviews	"Recent" LBP, 29.9; current low back injury, 8.3; past low back injury, 32.4
Wójcik and Siatkowski ⁸⁷	Cross-sectional	IV	2.5	Poland	Ballet students at the primary, junior high, and high school levels	237 (sex distribution not reported)	Primary, 11.2 ± 0.8; junior, 14.0 ± 0.8; high, 17.0 ± 0.8	Pain: a numeric rating scale	Self-report	Pain "now" All, 23.2; primary, 12.5; junior, 16.1; high, 46.9 Pain "before" All, 36.7; primary, 18.8; junior, 33.3; high, 64.1

Abbreviation: LBP, low back pain.

*Values are mean (range), range, mean ± SD, or mean ± SD (range).

†Prevalence data available for n = 198.

‡Prevalence data available for n = 50.

TABLE 3

PREVALENCE AND INCIDENCE OF LBI IN DANCE

Study	Study Type (Duration)	Level of Evidence	Risk of Bias	Country	Cohort Description	n (% Female)	Age, y*	Definition of LBI	Collection Methods	LBI Estimates
Allen et al ⁴	Prospective (1 y)	II	3.5	United Kingdom	1 professional ballet company	52 (49) [†]	Female, 25 ± 6; male, 23 ± 5	Time loss	Physical therapist	Incidence, n (%) All, 47 (13.2); female, 26 (15.1); male, 21 (11.5) Incidence (per 1000 dance-h) Female, 0.63; male, 0.55
Baker et al ⁷	Cross-sectional/retrospective (9.5 mo)	IIIb	5.5	United Kingdom	1 contemporary dance school (first years)	57 (83)	Female, 20 ± 2.5; male, 21 ± 3	Time loss	Self-report and physical therapist records	Incidence, n (%) All, 6 (8.6); female, 3 (5.4); male, 3 (21.4) Physical therapist recorded All, 9 (14.3); female, 8 (15.4); male, 1 (9.1)
Berlet et al ⁸	Prospective (7 mo)	II	5.5	United States	1 ballet company	13 (62)	Female, 26.89 ± 2.98; male, 28.83 ± 3.31	Time loss/medical-attention grading	Self-report	Prevalence, % All, 15; female, 25; male, 0
Bowerman et al ⁹	Prospective (6 mo)	II	5.5	Australia	1 ballet school	46 (65) 4 dropouts (1 female, 3 male)	16 ± 1.58	Time loss	Physical therapist	Incidence, n (%): 13 (22.0) Incidence (per 1000 dance-exposures): 0.78 Incidence (per 1000 dance-h): 0.53
Byhring and Bø ¹¹	Prospective (19 wk)	II	4.5	Norway	1 ballet company	41 (66)	Female, 26 ± 5.7; male, 27 ± 4.6	Combined time loss/medical attention	Physical therapist	Incidence, %: -7.5-8.5
Caine et al ¹²	Cross-sectional/retrospective (8.5 mo)	IIIb	3.5	Canada	1 ballet school	71 (62)	Female, 16.41; male, 17.37; all, 11-23	Time loss	Self-report	Incidence, n (%) All, 4 (3.5); female, 2 (2.8); male, 2 (4.7)
Costa et al ¹⁶	Cross-sectional	IIIb	5	Brazil	2 professional ballet companies and controls	53 (59), 57 controls	Female, 34.2 ± 6.3; male, 34.1 ± 7.3	Injuries, regardless of time loss/medical attention	Self-report	Prevalence, % Dancers: all, 22.6; female, 6.5; male, 45.5 Controls, 5.3
DiPasquale et al ¹⁹	Prospective (4 mo)	II	6.5	United States	1 modern dance university program (not audition based)	46 (89)	19.61 ± 1.31	Injuries, regardless of time loss/medical attention	Self-report	Incidence, n (%): 5 (10.9)
Ekegren et al ²¹	Prospective (1 academic year)	II	3.5	United Kingdom	3 ballet schools	266 (58)	17.2 ± 1.21 (15-23)	Time loss	Physical therapists	Incidence, n (%): 36 (9.5)

Table continues on page 245.

TABLE 3

PREVALENCE AND INCIDENCE OF LBI IN DANCE (CONTINUED)

Study	Study Type (Duration)	Level of Evidence	Risk of Bias	Country	Cohort Description	n (% Female)	Age, y*	Definition of LBI	Collection Methods	LBI Estimates
Evans et al ²²	Cross-sectional	IIIb	3.5	United Kingdom	Multiple West End productions	58 (64)	Female, 25.8 ± 5.4; male, 25.0 ± 5.4	Injuries, regardless of time loss/medical attention	Self-report	Prevalence, %: 18.5
Fulton et al ²⁵	Retrospective (3 y)	IIIb	4.5	United States	Summer dance intensive. Modern and other styles. Recreational to professional	321 who sought care	(12-50)	Medical attention	Clinic records	Incidence, n (%): 41 (10) for years 2-3 (year 1 NR)
Garrick ²⁷	Retrospective (5 y)	IIIb	5	United States	1 sports medicine clinic. Primarily treats professional and preprofessional ballet dancers	1055 injuries treated	No age restrictions	Medical attention	Physician/sports medicine clinic records	Incidence, n (%): -63 (6); 95 (9) involved the spine. Two thirds were the lumbar spine
Garrick and Requa ²⁹	Retrospective (3 y)	IIIb	5	United States	1 professional ballet company	~70 contracted dancers and 12 apprentices ~200 students covered for injuries sustained during performances or rehearsals with the company	NR	Medical expenses	Insurance documents and medical records	Incidence, n (%): 71 (23)
Garrick and Requa ³⁰	Retrospective (17 y)	IIIb	3.5	United States	1 sports medicine clinic with medical responsibilities for 2 professional companies and 1 preprofessional ballet school	3960 injuries treated (75)	11 to >26	Medical attention	Physician/sports medicine clinic records	Incidence, n (%) All, 293 (7.4); female, 200 (6.5); male, 93 (10.6)
Garrick ²⁸	Retrospective (2 y)	IIIb	4	United States	1 ballet school	59	13-18	Medical attention	Medical records	Incidence, n (%): 9 (4.6)
Klemp and Learmonth ⁴³	Retrospective (10 y)	IIIb	4	South Africa	1 ballet company	47 (64)	27.8 (19-47)	Injuries, regardless of time loss/medical attention	Workers' compensation records	Incidence, n (%) Ligament injuries, 6 (8.5); tendon injuries, 0 (0); muscle injuries, not possible to discern
Krasnow et al ⁴⁴	Cross-sectional	IV	5.5	Canada	Females from modern, ballet, and gymnastics	65 (100); 35 dancers, 30 gymnasts	15.5 ± 0.5 (12-18)	Injuries, regardless of time loss/medical attention	Self-report	Incidence, % Ballet, 12; modern, 21; gymnast, 18

Table continues on page 246.

TABLE 3

PREVALENCE AND INCIDENCE OF LBI IN DANCE (CONTINUED)

Study	Study Type (Duration)	Level of Evidence	Risk of Bias	Country	Cohort Description	n (% Female)	Age, y*	Definition of LBI	Collection Methods	LBI Estimates
Leander-son et al ⁴⁵	Retrospective (7 y)	IIIb	4	Sweden	1 ballet school	476 (62)	10-21	Medical attention	Medical records	Incidence, n (%) Female, 23 (5.3); male, 22 (5.1) of LBP diagnosis of all injuries
Nilsson et al ⁵³	Retrospective/ prospective (5 y)	II-IIIb	4.5	Sweden	1 ballet company	98 (51)	28.3 (17-47)	Medical attention	Physician records	Incidence, n (%): all, 60 (15)
Quirk ⁵⁶	Retrospective (15 y)	IIIb	6	Australia	1 professional ballet company and a ballet school	664 (71)	NR	Medical attention	Physician records	Incidence, n (%): 180 (8.5)
Ramkumar et al ⁵⁹	Retrospective (10 y)	IIIb	4.5	United States	1 professional ballet company	153 (53)	27.5	Combined time loss/ medical diagnosis	Workers' compensation and physician diagnosis	Incidence, n (%): 117 (20)
Rovere et al ⁶³	Retrospective (9 mo)	IIIb	4	United States	Ballet and modern dancers from 1 tertiary dance program	218 (74)	NR	Medical attention	Physician records	Incidence, n (%): 43 (12.2)
Shah et al ⁶⁶	Cross-sectional	IV	3.5	United States	National survey of professional modern dancers	184 (73)	30.1 ± 7.3 (18-55)	Time loss	Self-report	Incidence, n (%): 40 (17)
Sobrino et al, ⁶⁷ Sobrino and Guillelén ⁶⁸	Retrospective (5 y)	IIIb	4.5	Spain	4 professional ballet and contemporary ballet companies	145 (52)	All, 25.8 ± 5.7; female, 26.3 ± 5.9; male, 25.2 ± 5.4	Medical attention	Insurance records	Incidence, n (%): 49 (13.4)
Solomon and Micheli ⁷²	Cross-sectional	IV	5	United States	Multiple modern dance companies	164 (77)	26.15 ± 6.43 (16-48)	"Debilitating" injuries, regardless of time loss/ medical attention	Self-report	Incidence, n (%) All, 45 (15.3); Cunningham, 9 (14.3); Graham, 10 (16.3); Horton, 8 (21.6); Humphrey-Weidman, 2 (6.1); Limon, 16 (15.4) [†]
Solomon et al, ⁶⁹ Solomon et al, ⁷⁰ Solomon et al ⁷¹	Retrospective/ prospective (5 y)	II-IIIb	3.5	United States	1 ballet company	Year 1, 70 (57); year 2, 60 (NR); year 3, 60; year 4, 60; year 5, 59	All, 17-35	Reported injury that may or may not have required medical attention	Company records	Incidence, n (%) Year 1, 12 (8); year 2, 12 (8.4); year 3, 13 (13); 5-y average, 14 (12)
Straccioli et al, ⁷⁴ Yin et al ⁸⁸	Retrospective (9 y)	IIIb	4.5	United States	1 sports medicine clinic. Pediatric dancers	181 (95) 171 (100)	14.8 ± 2	Medical attention	Random sampling of medical charts of a sports medicine clinic	Incidence, % 11.5 11.7

Table continues on page 247.

TABLE 3

PREVALENCE AND INCIDENCE OF LBI IN DANCE (CONTINUED)

Study	Study Type (Duration)	Level of Evidence	Risk of Bias	Country	Cohort Description	n (% Female)	Age, y*	Definition of LBI	Collection Methods	LBI Estimates
Wanke et al ⁸⁵	Retrospective (17 y)	IIIb	3.5	Germany	6 professional ballet companies and 1 state ballet school	Occupational accidents, 291 (63)	All, 30.1; female, 29.5; male, 30.8	Time-loss injuries attributed to dance floors	Work accident reports	Incidence, % Female, 4.2; male, 14.1
Wanke et al ⁸⁴	Cross-sectional	IV	5	Germany	1 musical theatre school	37 (46)	All, 21.3 ± 2.2; female, 21.1 ± 2.2; male, 21.5 ± 2.2	Injuries, regardless of time loss/medical attention	Self-report	Incidence, n (%) All, 12 (13.2); acute, 3 (6.1); chronic, 9 (21.4)
Wanke et al ⁸²	Retrospective (17 y)	IIIb	3.5	Germany	3 ballet companies	Occupational accidents, 745 (48)	All, 28.7 ± 5.3; female, 28.9 ± 5.2; male, 28.5 ± 5.4	Time loss	Work accident reports	Incidence, % All, 8.5; female, 9.8; male, 17.3
Wanke et al ⁸³	Retrospective (2 y, 17 y apart)	IIIb	3.5	Germany	3 ballet companies	Occupational accidents, 241 (46)	1994/95: all, 28.0; female, 28.3; male, 27.7 2011/12: all, 29.5; female, 29.5; male, 29.5	Time loss	Work accident reports	Incidence, % 1994/95, 5.8; 2011/12, 20.3
Washing-ton ⁸⁶	Cross-sectional	IV	7	United States/ international	International survey of dancers as well as medical and support staff	NR	NR	Injuries, regardless of time loss/medical attention	Self-report	Incidence, n (%) Individual reports, 52 (12); group reports, 81 (6)

Abbreviations: LBI, low back injury; LBP, low back pain; NR, not reported.
*Values are mean (range), range, mean ± SD, or mean ± SD (range).
†Injury analysis for 50 dancers only.
‡Some injuries were counted more than once if a dancer studied multiple dance styles.

variables, including factors related to sex and maturation, anthropometry, cohort type, and LBP history.^{76,77} Years dancing was not associated with LBP prevalence in 3 studies.^{20,76,77}

Additional exploration of risk factors included a history of LBP and anthropometric data (eg, height, body mass). History was a significant predictor for activity-limiting LBP in 1 study (adjusted odds ratio = 3.98; 95% confidence interval: 1.44, 11.00).⁷⁷ Higher prevalence of LBP history was observed in dancers with scoliosis, although statistical anal-

ysis was not performed.⁴⁷ A body mass index lower than 18.5 was associated with higher risk of LBP in 1 study,²⁰ but no association between LBP and height, body mass, or body mass index was observed in 2 studies using multivariable analysis.^{76,77}

DISCUSSION

Findings

THE PURPOSE OF THIS SYSTEMATIC review was to synthesize the epidemiology of LBP and LBI in pre-

professional and professional dance populations. The median point (27%), yearly (73%), and lifetime (50%) prevalence of LBP observed in dancers were similar to or above rates that have been previously reported in the global population (18%, 48%, and 49%, respectively)³⁶ and in a meta-analysis of sub-elite to elite participants in Olympic sports (24%, 55%, and 61%, respectively).⁷⁹ These findings must be interpreted with substantial caution, as significant methodological heterogeneity was present among the included studies. Specifically,

[RESEARCH REPORT]

there were inconsistencies in targeted populations, sex balance, study designs, time periods, anatomical definitions, and statistical reporting. A higher median yearly percentage than lifetime percentage highlights the difficulties in obtaining

accurate estimates of LBP and emphasizing the importance of synthesizing results from a range of studies.

Comparison between studies that report LBP and those that report LBI is difficult. Studies of LBP used prevalence

statistics more frequently compared to studies of LBI, which most often presented the frequency of LBI as a percentage of all injuries experienced by dancers. Nonetheless, studies included in the review indicated that approximately 73% of

TABLE 4

ASSOCIATION OF SEX AND AGE WITH LOW BACK PAIN AND LOW BACK INJURY IN DANCE

Risk Factor/Study	Observation	Reported Significance	Confounders Controlled for
Sex			
Allen et al ⁴	Male less than female (11.5% versus 15.1%; 0.55/1000 dance-hours versus 0.63/1000 dance-hours)	Not reported	None
Baker et al ⁷	Self-report: male greater than female (21.4% versus 5.4%) Physical therapist records: male less than female (9.1% versus 15.4%)	Not reported	None
Caine et al ¹²	Male equal to female (4.7% versus 2.8%)	Not reported	None
Costa et al ¹⁶	Male greater than female (45.5% versus 6.5%)	$P < .01$	None
Garrick and Requa ³⁰	Male greater than female (10.6% versus 6.5%)	Not reported	None
Grego Muniz de Araújo et al ³³	Male equal to female (39% versus 38%)	Not reported	None
Ramel and Moritz ⁵⁷	Male equal to female. Delineated values not presented	"No significant difference in pain locations in men versus women." P value not reported	None
Seitsalo et al ⁶⁵	Spondylolysis prevalence: male equal to female (40% versus 26%)	$P = .08$	None
Sobrinho and Guillén, ⁶⁸ Sobrinho et al ⁶⁷	Male equal to female (24% versus 25%)	Not reported	None
Solomon et al, ⁶⁹ Solomon et al ⁷⁰	Male equal to female (9% versus 10%)	Not reported	None
Swain et al ⁷⁶	Point prevalence: male equal to female (16% versus 25%) Lifetime prevalence: male equal to female (73% versus 79%)	Point: AOR = 1.20; 95% CI: 0.19, 7.69; $P = .85$ Lifetime: AOR = 1.18; 95% CI: 0.26, 5.39; $P = .83$	Age, body mass, body mass index, cohort type, years dancing Age, height, body mass
Swain et al ⁷⁷	Male equal to female (42% versus 54%)	$P > .25$	None
Wanke et al ⁸²	Male greater than female (17.3% versus 9.8%)	"Significant gender (sic) specific differences were observed in the spine region ... particularly with the more than twice affected lumbar spine in male dancers." P value not reported	None
Wanke et al ⁸⁶	Injuries to the lumbar spine that were attributed to dance floors were more common in male dancers (14.1%) than in female dancers (4.2%)	$P = .023$	None
Wanke et al ⁸⁴	Male equal to female. Delineated values not reported	"No statistically significant gender (sic) differences in the location of acute injuries were found." P value not reported	None
Age			
Drężewska and Śliwiński ²⁰	Older greater than younger	$P < .05$	None
Garrick and Requa ³⁰	Older greater than younger	Not reported	None
Swain et al ⁷⁶	Age not significant	Point: AOR = 1.06; 95% CI: 0.76, 1.48; $P = .71$ Lifetime: AOR = 1.11; 95% CI: 0.91, 1.40; $P = .31$	Sex, height, body mass, cohort type, years dancing Sex, height, body mass
Swain et al ⁷⁷	Age not significant	AOR = 0.99; 95% CI: 0.76, 1.29; $P = .93$	Age started dancing, cohort type, low back pain history
Wójcik and Siatkowski ⁸⁷	Older greater than younger	Not reported	None

Abbreviations: AOR, adjusted odds ratio; CI, confidence interval.

dancers will experience at least 1 episode of LBP each year; however, the lower back will only be identified as the cause of time loss or medical attention for 11% of cases.

This disparity may be indicative of 2 realities. First, dancers experience many injuries at sites that do not include the lower back,⁴² which in effect may lower the relative contribution of LBI to the total injury count. Second, the impact of an episode of LBP will often fall short of a time-loss or medical-attention threshold,⁷⁷ and many dancers may be able to maintain a high level of performance even in the presence of pain.³⁸ In this respect, traditional definitions of injury are only capable of providing a partial overview of the problem. This finding is consistent with observations from general,²⁴ sporting,⁶ and dance populations.⁴¹ For example, based on a review of 7 population studies, the pooled prevalence of care seeking in persons with LBP was 58%,²⁴ which indicates that medical records are not suitable for determining the overall prevalence of a condition. Based on current evidence, it is unclear whether this behavior is more pronounced in dancers compared to general and sporting populations.

Consistent with previous reviews of pain and injury in dance,^{35,39,42} significant heterogeneity of definitions among the studies included was observed. For example, for time-loss injuries, collection methods included self-report^{7,66} as well as health professional registration,^{4,7,9,21,82,83,85} and the minimum threshold for registration included activity modification or partial absence,^{7,9,11,12,66} complete absence for at least 1 day,^{4,21,59} or time-limiting incident without a threshold defined.^{82,83,85} Furthermore, the interpretation of severity varied between studies that used a time-loss definition. For instance, Bowerman et al⁹ used 3 levels to classify injury severity in preprofessional ballet students: (1) modified class, (2) off class for up to 3 days, (3) off class for more than 3 days. In contrast, in professional ballet dancers, Allen et al⁴ categorized injuries as transient (return within 7 days), mild

(return after 7 to 28 days), moderate (return within 29 to 84 days), and severe (return after 84 days).

A second aim of the review was to identify risk factors for LBP and LBI. Overall, few studies deliberately focused on risk factors, and, collectively, interpreting factors associated with LBP and LBI was limited by an absence of appropriate statistical analysis and magnitude-based statistics. One prospective cohort study indicated that a history of LBP was a significant predictor of future episodes of activity-limiting LBP.⁷⁷ This is in accord with LBP literature, which has consistently described history as a primary contributor toward future LBP.^{2,23} The implication is that LBP is rarely limited to a single episode.

The prevalence of LBP and percentage of all injuries located in the lower back appeared to increase with age and dance level.^{20,30,87} However, multivariable statistical analyses have not yet demonstrated a significant relationship between age, years of training, or dance level and LBP.^{76,77} As a relationship with age and dance level may provide important information about biological or workplace factors that contribute to LBP and LBI in a dance population, further investigation is required.

There was mixed information describing sex as a risk factor. Previously, it has been suggested that male dancers may be more vulnerable to LBP and LBI, due, in part, to the lifting demands required of men in ballet.³ While this still may be the case, both males and females from ballet and contemporary dance are exposed to a variety of physical factors beyond lifting that may increase risk of LBP and LBI. In addition to physical factors, biological and psychosocial factors contribute to the initiation, maintenance, and perception of pain,¹⁴ and these factors are pertinent to both male and female dancers. Overall, the current evidence does not support that dancers are materially different, with respect to risk factors for LBP, from other athletic or broader general populations.

Recommendations

Definitions that are sensitive to the nature of LBP in dance are needed. This is not simple. Pain is a subjective experience that fluctuates within and between individuals.^{48,64} It need not be associated with identifiable tissue damage to be valid,^{10,34,51} and, although the impact can be severe, many dancers who experience pain are able to maintain their ability to perform.³⁸ Given this, the injury definition endorsed by the International Association for Dance Medicine and Science, which considers injury as an anatomic tissue-level impairment, as diagnosed by a health care practitioner, that results in full time loss from activity for 1 or more days beyond the day of onset,⁴⁶ may not be best suited for determining the prevalence of LBP. However, an initial intent of the measurement of the International Association for Dance Medicine and Science definition was to encourage the standardization of measurement of risk factors and injury reporting,⁴⁶ which the current review endorses. To achieve this in LBP epidemiology, Dionne et al¹⁸ proposed a minimal definition (“In the past 4 weeks, have you had pain in your low back?”) that should be combined with a minimum severity criterion. Where possible, a description or diagram of the lower back area should accompany this definition.¹⁸ In sports medicine, the Oslo Sports Trauma Research Center questionnaire has been identified as a sensitive and valid tool capable of documenting patterns of injury in athletic populations,¹⁵ and has also been proposed as a suitable tool for dance epidemiology.⁴¹

Because a key function of surveillance is to assess the effectiveness of an intervention,⁸⁰ outcomes specific to the site of pain and injury are needed. For the lower back, prevalence, which refers to the proportion of the population with the condition at a given time,⁶⁰ should be used.⁶ Due, in part, to high childhood and adolescent prevalence of LBP,¹³ as well as the recurrent nature of LBP and LBI,^{49,73} the incidence of first-time episodes of LBP

is difficult to determine. Furthermore, the percentage of all injuries located in the lower back area may be influenced by the total number of other injuries in a cohort, as well as by multiple injuries in a single dancer at the same site, suggesting that this outcome may have less value for assessing outcomes of site-specific interventions.

Multisite studies, potentially with control groups, are needed. Single-site studies may be more sensitive to site-specific effects (eg, repertoire, touring, injury reporting cultures, etc). In addition, as dance cohorts are composed of highly specialized populations, they are limited in numbers of potential participants. Multisite studies are more likely to recruit enough participants to facilitate multivariable analysis, allowing more valid conclusions. Furthermore, as LBP symptoms are prevalent in non-dance populations,^{13,36} the inclusion of control groups in future studies will allow researchers to determine the proportion of LBP symptoms observed that can be attributed to dance participation.

Limitations

To limit the focus and clarity of the present review, studies that reported results that were not specific to the lower back or lumbar spine and studies that used general language to describe the site of injury were not included. As such, some studies investigating back pain or injury in dancers were not eligible for inclusion.⁵⁰ Furthermore, inclusion criteria were limited to peer review. Although this is a strength of the study, several national reports were subsequently excluded (eg, Safe Dance reports I-IV).^{17,31,32,81} It is also possible that relevant studies were not included due to the search terminology employed.

No minimum sample size was set as an inclusion criterion in this study. This was due to the aim of the study, which was to synthesize all available evidence for LBP and LBI in dance, and the fact that many medium-sized dance companies consist of few permanently em-

ployed dancers. However, the utility of determining prevalence from studies with small samples should be considered when interpreting findings from specific studies. Finally, due to the range of definitions used, a meta-analysis of reported data was not possible.

CONCLUSION

LOW BACK PAIN AND INJURY ARE COMMON in dance and reflect levels reported in other athletic populations. Available evidence is unable to determine whether the experience of LBP in dance is distinct from that of nondancers, or which LBP risk factors, if any, are of increased importance in a dance population. Multisite prospective cohort studies that employ definitions suitable to capture LBP and LBI, with outcomes clearly reported, would enable improved comparison with non-dance populations. Such studies would also facilitate improved identification of risk factors to better identify dancers who may need injury prevention or pain management strategies, inform dance-appropriate clinical management, and allow for monitoring of low back-specific interventions within dance. ●

KEY POINTS

FINDINGS: Dancers are at least as vulnerable to low back pain and injury as other athletic populations.

IMPLICATIONS: Strategies are needed to improve prevention and management of low back pain and injury in dance.

CAUTION: Traditional definitions of injury underrepresent the problem, as they only partially capture the impact of low back pain.

REFERENCES

1. Adams MA, Dolan P. Spine biomechanics. *J Biomech*. 2005;38:1972-1983. <https://doi.org/10.1016/j.jbiomech.2005.03.028>
2. Adams MA, Mannion AF, Dolan P. Personal risk factors for first-time low back pain. *Spine (Phila Pa 1976)*. 1999;24:2497-2505.

3. Alderson J, Hopper L, Elliott B, Ackland T. Risk factors for lower back injury in male dancers performing ballet lifts. *J Dance Med Sci*. 2009;13:83-89.
4. Allen N, Nevill A, Brooks J, Koutedakis Y, Wyon M. Ballet injuries: injury incidence and severity over 1 year. *J Orthop Sports Phys Ther*. 2012;42:781-790. <https://doi.org/10.2519/jospt.2012.3893>
5. Australian Bureau of Statistics. Children's Participation in Cultural and Leisure Activities, Australia. Canberra, Australia: Australian Bureau of Statistics; 2012.
6. Bahr R. No injuries, but plenty of pain? On the methodology for recording overuse symptoms in sports. *Br J Sports Med*. 2009;43:966-972. <https://doi.org/10.1136/bjsm.2009.066936>
7. Baker J, Scott D, Watkins K, Keegan-Turcotte S, Wyon M. Self-reported and reported injury patterns in contemporary dance students. *Med Probl Perform Art*. 2010;25:10-15.
8. Berlet GC, Kiebzak GM, Dandar A, et al. Prospective analysis of body composition and SF36 profiles in professional dancers over a 7-month season: is there a correlation to injury? *J Dance Med Sci*. 2002;6:54-61.
9. Bowerman E, Whatman C, Harris N, Bradshaw E, Karin J. Are maturation, growth and lower extremity alignment associated with overuse injury in elite adolescent ballet dancers? *Phys Ther Sport*. 2014;15:234-241. <https://doi.org/10.1016/j.ptsp.2013.12.014>
10. Brinjikji W, Luetmer PH, Comstock B, et al. Systematic literature review of imaging features of spinal degeneration in asymptomatic populations. *AJNR Am J Neuroradiol*. 2015;36:811-816. <https://doi.org/10.3174/ajnr.A4173>
11. Byhring S, Bø K. Musculoskeletal injuries in the Norwegian National Ballet: a prospective cohort study. *Scand J Med Sci Sports*. 2002;12:365-370. <https://doi.org/10.1034/j.1600-0838.2002.01262.x>
12. Caine D, Bergeron G, Goodwin BJ, et al. A survey of injuries affecting pre-professional ballet dancers. *J Dance Med Sci*. 2016;20:115-126. <https://doi.org/10.12678/1089-313X.20.3.115>
13. Calvo-Muñoz I, Gómez-Conesa A, Sánchez-Meca J. Prevalence of low back pain in children and adolescents: a meta-analysis. *BMC Pediatr*. 2013;13:14. <https://doi.org/10.1186/1471-2431-13-14>
14. Chimenti RL, Frey-Law LA, Sluka KA. A mechanism-based approach to physical therapist management of pain. *Phys Ther*. 2018;98:302-314. <https://doi.org/10.1093/ptj/pty030>
15. Clarsen B, Rønnes O, Myklebust G, Flørenes TW, Bahr R. The Oslo Sports Trauma Research Center questionnaire on health problems: a new approach to prospective monitoring of illness and injury in elite athletes. *Br J Sports Med*. 2014;48:754-760. <https://doi.org/10.1136/bjsports-2012-092087>
16. Costa MS, Ferreira AS, Orsini M, Silva EB, Felicio LR. Characteristics and prevalence of

musculoskeletal injury in professional and non-professional ballet dancers. *Braz J Phys Ther.* 2016;20:166-175. <https://doi.org/10.1590/bjpt-rbf.2014.0142>

17. Crookshanks D. Safe Dance III: A Report on the Occurrence of Injury in the Australian Professional Dance Population. Braddon, Australia: Australian Dance Council; 1999.
18. Dionne CE, Dunn KM, Croft PR, et al. A consensus approach toward the standardization of back pain definitions for use in prevalence studies. *Spine (Phila Pa 1976)*. 2008;33:95-103. <https://doi.org/10.1097/BRS.0b013e31815e7f94>
19. DiPasquale S, Becker N, Green S, Sauers K. Self-reported injury and management in a liberal arts college dance department. *Med Probl Perform Art.* 2015;30:224-230. <https://doi.org/10.21091/mppa.2015.4041>
20. Dreżewska M, Śliwiński Z. Lumbosacral pain in ballet school students. Pilot study. *Ortop Traumatol Rehabil.* 2013;15:149-158.
21. Ekegren CL, Qvested R, Brodrick A. Injuries in pre-professional ballet dancers: incidence, characteristics and consequences. *J Sci Med Sport.* 2014;17:271-275. <https://doi.org/10.1016/j.jsams.2013.07.013>
22. Evans RW, Evans RI, Carvajal S. Survey of injuries among West End performers. *Occup Environ Med.* 1998;55:585-593. <https://doi.org/10.1136/oem.55.9.585>
23. Ferguson SA, Marras WS. A literature review of low back disorder surveillance measures and risk factors. *Clin Biomech (Bristol, Avon)*. 1997;12:211-226. [https://doi.org/10.1016/S0268-0033\(96\)00073-3](https://doi.org/10.1016/S0268-0033(96)00073-3)
24. Ferreira ML, Machado G, Latimer J, Maher C, Ferreira PH, Smeets RJ. Factors defining care-seeking in low back pain – a meta-analysis of population based surveys. *Eur J Pain.* 2010;14:747e1-747e7. <https://doi.org/10.1016/j.ejpain.2009.11.005>
25. Fulton J, Burgi C, Canizares RC, Sheets C, Butler RJ. Injuries presenting to a walk-in clinic at a summer dance intensive program: a three-year retrospective data analysis. *J Dance Med Sci.* 2014;18:131-135. <https://doi.org/10.12678/1089-313X.18.3.131>
26. Gamboa JM, Roberts LA, Maring J, Fergus A. Injury patterns in elite preprofessional ballet dancers and the utility of screening programs to identify risk characteristics. *J Orthop Sports Phys Ther.* 2008;38:126-136. <https://doi.org/10.2519/jospt.2008.2390>
27. Garrick JG. Ballet injuries. *Med Probl Perform Art.* 1986;1:123-127.
28. Garrick JG. Early identification of musculoskeletal complaints and injuries among female ballet students. *J Dance Med Sci.* 1999;3:80-83.
29. Garrick JG, Requa RK. An analysis of epidemiology and financial outcome. *Am J Sports Med.* 1993;21:586-590. <https://doi.org/10.1177/036354659302100417>
30. Garrick JG, Requa RK. The relationship between

age and sex and ballet injuries. *Med Probl Perform Art.* 1997;12:79-82.

31. Geeves T. Safe Dance II: National Injury and Lifestyle Survey of Australian Adolescents in Pre-professional Dance Training. Braddon, Australia: Australian Dance Council; 1997.
32. Geeves T. Safe Dance: A Report on Dance Injury Prevention and Management in Australia. Braddon, Australia: Australian Dance Council; 1990.
33. Grego Muniz de Araújo L, Luiz Monteiro H, Marcelo Pastre C. Musculoskeletal disorders (MSDs) in dancers and former dancers participating in the largest dance festival in the world. *Sci Sports.* 2013;28:146-154. <https://doi.org/10.1016/j.scispo.2012.04.011>
34. Hancock MJ, Maher CG, Latimer J, et al. Systematic review of tests to identify the disc, SIJ or facet joint as the source of low back pain. *Eur Spine J.* 2007;16:1539-1550. <https://doi.org/10.1007/s00586-007-0391-1>
35. Hincapié CA, Morton EJ, Cassidy JD. Musculoskeletal injuries and pain in dancers: a systematic review. *Arch Phys Med Rehabil.* 2008;89:1819-1829. <https://doi.org/10.1016/j.apmr.2008.02.020>
36. Hoy D, Bain C, Williams G, et al. A systematic review of the global prevalence of low back pain. *Arthritis Rheum.* 2012;64:2028-2037. <https://doi.org/10.1002/art.34347>
37. Hoy D, Brooks P, Woolf A, et al. Assessing risk of bias in prevalence studies: modification of an existing tool and evidence of interrater agreement. *J Clin Epidemiol.* 2012;65:934-939. <https://doi.org/10.1016/j.jclinepi.2011.11.014>
38. Jacobs CL, Cassidy JD, Côté P, et al. Musculoskeletal injury in professional dancers: prevalence and associated factors: an international cross-sectional study. *Clin J Sport Med.* 2017;27:153-160. <https://doi.org/10.1097/JSM.0000000000000314>
39. Jacobs CL, Hincapié CA, Cassidy JD. Musculoskeletal injuries and pain in dancers: a systematic review update. *J Dance Med Sci.* 2012;16:74-84.
40. Kadel NJ, Teitz CC, Kronmal RA. Stress fractures in ballet dancers. *Am J Sports Med.* 1992;20:445-449. <https://doi.org/10.1177/036354659202000414>
41. Kenny SJ, Palacios-Derflinger L, Whittaker JL, Emery CA. The influence of injury definition on injury burden in preprofessional ballet and contemporary dancers. *J Orthop Sports Phys Ther.* 2018;48:185-193. <https://doi.org/10.2519/jospt.2018.7542>
42. Kenny SJ, Whittaker JL, Emery CA. Risk factors for musculoskeletal injury in preprofessional dancers: a systematic review. *Br J Sports Med.* 2016;50:997-1003. <https://doi.org/10.1136/bjsports-2015-095121>
43. Klemp P, Learmonth ID. Hypermobility and injuries in a professional ballet company. *Br J Sports Med.* 1984;18:143-148. <https://doi.org/10.1136/bjism.18.3.143>
44. Krasnow D, Mainwaring L, Kerr G. Injury, stress,

and perfectionism in young dancers and gymnasts. *J Dance Med Sci.* 1999;3:51-58.

45. Leanderson C, Leanderson J, Wykman A, Strender LE, Johansson SE, Sundquist K. Musculoskeletal injuries in young ballet dancers. *Knee Surg Sports Traumatol Arthrosc.* 2011;19:1531-1535. <https://doi.org/10.1007/s00167-011-1445-9>
46. Liederbach M, Hagins M, Gamboa JM, Welsh TM. Assessing and reporting dancer capacities, risk factors, and injuries: recommendations from the IADMS Standard Measures Consensus Initiative. *J Dance Med Sci.* 2012;16:139-153.
47. Liederbach M, Spivak J, Rose DJ. Scoliosis in dancers: a method of assessment in quick-screen settings. *J Dance Med Sci.* 1997;1:107-112.
48. Maher C, Underwood M, Buchbinder R. Non-specific low back pain. *Lancet.* 2017;389:736-747. [https://doi.org/10.1016/S0140-6736\(16\)30970-9](https://doi.org/10.1016/S0140-6736(16)30970-9)
49. Maul I, Läubli T, Klipstein A, Krueger H. Course of low back pain among nurses: a longitudinal study across eight years. *Occup Environ Med.* 2003;60:497-503. <https://doi.org/10.1136/oem.60.7.497>
50. McMeeken J, Tully E, Stillman B, Nattrass C, Bygott IL, Story I. The experience of back pain in young Australians. *Man Ther.* 2001;6:213-220. <https://doi.org/10.1054/math.2001.0410>
51. Merskey H, Bogduk N. *Classification of Chronic Pain: Descriptions of Chronic Pain Syndromes and Definitions of Pain Terms*. 2nd ed. Seattle, WA: IASP Press; 1994.
52. Moher D, Liberati A, Tetzlaff J, Altman DG, The PRISMA Group. Preferred reporting items for systematic reviews and meta-analyses: the PRISMA statement. *PLoS Med.* 2009;6:e1000097. <https://doi.org/10.1371/journal.pmed.1000097>
53. Nilsson C, Leanderson J, Wykman A, Strender LE. The injury panorama in a Swedish professional ballet company. *Knee Surg Sports Traumatol Arthrosc.* 2001;9:242-246. <https://doi.org/10.1007/s001670100195>
54. Nunes NM, Haddad JJ, Bartlett DJ, Obright KD. Musculoskeletal injuries among young, recreational, female dancers before and after dancing in pointe shoes. *Pediatr Phys Ther.* 2002;14:100-106.
55. O'Neill JR, Pate RR, Liese AD. Descriptive epidemiology of dance participation in adolescents. *Res Q Exerc Sport.* 2011;82:373-380. <https://doi.org/10.1080/02701367.2011.10599769>
56. Quirk R. Ballet injuries: the Australian experience. *Clin Sports Med.* 1983;2:507-514.
57. Ramel E, Moritz U. Self-reported musculoskeletal pain and discomfort in professional ballet dancers in Sweden. *Scand J Rehabil Med.* 1994;26:11-16.
58. Ramel EM, Moritz U, Jarnlo GB. Recurrent musculoskeletal pain in professional ballet dancers in Sweden: a six-year follow-up. *J Dance Med Sci.* 1999;3:93-100.
59. Ramkumar PN, Farber J, Arnouk J, Varner KE, McCulloch PC. Injuries in a professional ballet dance company: a 10-year retrospective study.

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- J Dance Med Sci.* 2016;20:30-37. <https://doi.org/10.12678/1089-313X.20.1.30>
60. Rothman KJ, Greenland S, Lash TL. *Modern Epidemiology*. 3rd ed. Philadelphia, PA: Wolters Kluwer/Lippincott Williams & Wilkins; 2008.
 61. Roussel N, De Koning M, Schutt A, et al. Motor control and low back pain in dancers. *Int J Sports Med.* 2013;34:138-143. <https://doi.org/10.1055/s-0032-1321722>
 62. Roussel NA, Nijs J, Mottram S, Van Moorsel A, Truijen S, Stassijns G. Altered lumbopelvic movement control but not generalized joint hypermobility is associated with increased injury in dancers. A prospective study. *Man Ther.* 2009;14:630-635. <https://doi.org/10.1016/j.math.2008.12.004>
 63. Rovere GD, Webb LX, Gristina AG, Vogel JM. Musculoskeletal injuries in theatrical dance students. *Am J Sports Med.* 1983;11:195-198. <https://doi.org/10.1177/036354658301100402>
 64. Schneider S, Jungheuerl DU, Keefe FJ, Schwartz JE, Stone AA, Broderick JE. Individual differences in the day-to-day variability of pain, fatigue, and well-being in patients with rheumatic disease: associations with psychological variables. *Pain.* 2012;153:813-822. <https://doi.org/10.1016/j.pain.2012.01.001>
 65. Seitsalo S, Anttila H, Karihaho T, et al. Spondylolysis in ballet dancers. *J Dance Med Sci.* 1997;1:51-54.
 66. Shah S, Weiss DS, Burchette RJ. Injuries in professional modern dancers: incidence, risk factors, and management. *J Dance Med Sci.* 2012;16:17-25.
 67. Sobrino FJ, de la Cuadra C, Guillén P. Overuse injuries in professional ballet: injury-based differences among ballet disciplines. *Orthop J Sports Med.* 2015;3:2325967115590114. <https://doi.org/10.1177/2325967115590114>
 68. Sobrino FJ, Guillén P. Overuse injuries in professional ballet: influence of age and years of professional practice. *Orthop J Sports Med.* 2017;5:232596711712704. <https://doi.org/10.1177/232596711712704>
 69. Solomon R, Micheli LJ, Solomon J, Kelly T. The "cost" of injuries in a professional ballet company: anatomy of a season. *Med Probl Perform Art.* 1995;10:3-10.
 70. Solomon R, Micheli LJ, Solomon J, Kelly T. The "cost" of injuries in a professional ballet company: a three year perspective. *Med Probl Perform Art.* 1996;11:67-74.
 71. Solomon R, Solomon J, Micheli LJ, McGray E, Jr. The "cost" of injuries in a professional ballet company: a five-year study. *Med Probl Perform Art.* 1999;14:164-170.
 72. Solomon RL, Micheli LJ. Technique as a consideration in modern dance injuries. *Phys Sportsmed.* 1986;14:83-90. <https://doi.org/10.1080/00913847.1986.11709150>
 73. Stanton TR, Henschke N, Maher CG, Refshauge KM, Latimer J, McAuley JH. After an episode of acute low back pain, recurrence is unpredictable and not as common as previously thought. *Spine (Phila Pa 1976).* 2008;33:2923-2928. <https://doi.org/10.1097/BRS.0b013e31818a3167>
 74. Straccioliini A, Yin AX, Sugimoto D. Etiology and body area of injuries in young female dancers presenting to sports medicine clinic: a comparison by age group. *Phys Sportsmed.* 2015;43:342-347. <https://doi.org/10.1080/00913847.2015.1076326>
 75. Swain CT, Ekegren CL. Upholding standards of reporting in the synthesis of dance epidemiology literature: letter to the editor re: prevalence and profile of musculoskeletal injuries in ballet dancers: a systematic review and meta-analysis [letter]. *Phys Ther Sport.* 2016;22:129-130. <https://doi.org/10.1016/j.ptsp.2016.08.001>
 76. Swain CT, Bradshaw EJ, Whyte DG, Ekegren CL. Life history and point prevalence of low back pain in pre-professional and professional dancers. *Phys Ther Sport.* 2017;25:34-38. <https://doi.org/10.1016/j.ptsp.2017.01.005>
 77. Swain CT, Bradshaw EJ, Whyte DG, Ekegren CL. The prevalence and impact of low back pain in pre-professional and professional dancers: a prospective study. *Phys Ther Sport.* 2018;30:8-13. <https://doi.org/10.1016/j.ptsp.2017.10.006>
 78. Thomas H, Tarr J. Dancers' perceptions of pain and injury: positive and negative effects. *J Dance Med Sci.* 2009;13:51-59.
 79. Trompeter K, Fett D, Platen P. Prevalence of back pain in sports: a systematic review of the literature. *Sports Med.* 2017;47:1183-1207. <https://doi.org/10.1007/s40279-016-0645-3>
 80. van Mechelen W, Hlobil H, Kemper HC. Incidence, severity, aetiology and prevention of sports injuries. A review of concepts. *Sports Med.* 1992;14:82-99. <https://doi.org/10.2165/00007256-199214020-00002>
 81. Vassallo AJ. Safe Dance IV: Investigating Injuries in Australia's Professional Dancers. Braddon, Australia: Australian Dance Council; 2017.
 82. Wanke EM, Arendt M, Mill H, Groneberg DA. Occupational accidents in professional dance with focus on gender differences. *J Occup Med Toxicol.* 2013;8:35. <https://doi.org/10.1186/1745-6673-8-35>
 83. Wanke EM, Koch F, Leslie-Spinks J, Groneberg DA. Traumatic injuries in professional dance—past and present: ballet injuries in Berlin, 1994/95 and 2011/12. *Med Probl Perform Art.* 2014;29:168-173. <https://doi.org/10.21091/mppa.2014.3034>
 84. Wanke EM, Kunath EK, Koch F, et al. Survey of health problems in musical theater students: a pilot study. *Med Probl Perform Art.* 2012;27:205-211.
 85. Wanke EM, Mill H, Wanke A, Davenport J, Koch F, Groneberg DA. Dance floors as injury risk: analysis and evaluation of acute injuries caused by dance floors in professional dance with regard to preventative aspects. *Med Probl Perform Art.* 2012;27:137-142.
 86. Washington EL. Musculoskeletal injuries in theatrical dancers: site, frequency, and severity. *Am J Sports Med.* 1978;6:75-98. <https://doi.org/10.1177/036354657800600207>
 87. Wójcik M, Siatkowski I. Assessment of spine pain presence in children and young persons studying in ballet schools. *J Phys Ther Sci.* 2015;27:1103-1106. <https://doi.org/10.1589/jpts.27.1103>
 88. Yin AX, Sugimoto D, Martin DJ, Straccioliini A. Pediatric dance injuries: a cross-sectional epidemiological study. *PM R.* 2016;8:348-355.



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APPENDIX

RISK-OF-BIAS ASSESSMENT

Item	Level/Example
1. Was the study's target population a close representation of the national population?	Low risk: the study's target population was a close representation of the national population The study sampled multiple cohorts in multiple locations High risk: the study's target population was clearly not representative of the national population The study sampled a single cohort only or multiple cohorts limited to a single city
2. Was the sampling frame a true or close representation of the target population?	Low risk: the sampling frame was a true or close representation of the target population The target population was professional ballet dancers and the sampling frame was a professional ballet company High risk: the sampling frame was not a true or close representation of the target population The sampling frame was limited to only injured dancers
3. Was some form of random selection used to select the sample, or was a census undertaken?	Low risk: a census was undertaken, or some form of random selection was used to select the sample An entire cohort was invited to participate High risk: a census was not undertaken; random selection was not used Only dancers treated by 1 health professional were sampled
4. Was the likelihood of nonresponse bias minimal?	Low risk: the response rate for the study was 75% or greater or there were no significant differences in relevant demographic characteristics between responders and nonresponders High risk: the response rate was less than 75%, and there were significant demographic differences between responders and nonresponders, or differences between responders and nonresponders were not reported
5. Were data collected directly from the subjects (as opposed to a proxy)?	Low risk: all data were collected directly from the subjects High risk: data were collected from a proxy Physical therapist records were used to determine prevalence or incidence
6. Was an acceptable case definition used in the study?	Low risk: an acceptable definition of pain or injury was used, and the low back region was clearly defined Low back pain was defined as pain experienced between the lower 12th rib and upper gluteal fold and was accompanied by a diagram High risk: an acceptable definition of pain or injury was not used, and/or no description of the low back was provided No threshold for injury reporting was provided and no description of the low back region was provided
7. Was the study instrument that measured the parameter of interest shown to have reliability and validity?	Low risk: the instrument used had been shown to have reliability and validity High risk: reliability or validity had not been established
8. Was the same mode of data collection used for all subjects?	Low risk: the same mode of data collection was used for all subjects High risk: the same mode of data collection was not used for all subjects
9. Was the length of the shortest prevalence period for the parameter of interest appropriate?	Low risk: the shortest prevalence period for the parameter of interest was appropriate The study reports point prevalence, 1-month prevalence, or injury was registered upon occurrence High risk: the shortest prevalence period for the parameter of interest was not appropriate The study reports prevalence greater than 1-month recall
10. Were the numerators and denominators accurate and appropriate?	Low risk: there were no errors in the reporting of the numerator and denominator for the parameters of interest High risk: there were clear errors in the numerator and the denominator reported

Adapted from Hoy et al.³⁷

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Is Cadence Related to Leg Length and Load Rate?

Cadence, or the number of steps per minute, has received much attention in the recent running literature. Studies have related cadence to numerous aspects of running, including impact mechanics,¹⁷ joint loading,⁸ efficiency,⁷ plantar loading,¹⁵ performance,¹² and injuries.¹⁰ The optimal running cadence is often considered to be 180 steps per minute, a recommendation that is likely based on a report on the 1984 Los Angeles Olympics by Daniels,² who noted that elite male and female runners competing in events between 3 km and the marathon were typically exhibiting a cadence of 180 steps per minute.

Earlier research by Cavanagh and Kram in 1989¹ identified weak associations between anthropometric variables, including leg length, and stride length. While they did not measure actual cadence, their findings suggest that cadence

was lower in runners with longer legs due to their longer stride. Although generating important findings, this investigation was conducted with a small population of 12 uninjured male runners 18 to 40 years of age.

It has been suggested that lower cadence may be associated with increased vertical load rate.¹⁷ Increased vertical load rate, in turn, has been associated with

running injuries, including stress fractures, plantar fasciitis, and patellofemoral pain.^{3,4,11,13} Lieberman et al⁹ reported a weak and insignificant relationship between cadence and load rate when assessing cadences that were increased and decreased from habitual cadence. In another step-rate perturbation study, Wille et al¹⁶ found no relationship between cadence and load rate. Futrell et al⁶ reported similar findings in a study of habitual cadence and load rate in both uninjured and injured runners.

It is plausible that runners' leg length may influence their cadence, with shorter legs exhibiting higher cadences. Therefore, leg length may be a cofactor in the relationship between cadence and load rate. If so, normalizing cadence by leg length may reveal a relationship between cadence and load rate that wasn't noted by Futrell et al⁶ or Wille et al.¹⁶ It is also possible that the relationship between normalized cadence and load rate may be stronger in injured runners compared to their uninjured counterparts.

To further our understanding of injury risk and treatment in relation to cadence, a more contemporary study with a larger sample size that assesses both injured and uninjured runners is warranted. Therefore, the purpose of this study was to examine the relationship between cadence and leg length in injured and uninjured runners who display a rearfoot strike pattern.

We chose to limit the population studied to runners with a rearfoot strike pattern because 95% of runners in conventional

• **BACKGROUND:** Increasing cadence is often recommended to reduce load rate and to lower injury risk. However, habitual cadence was recently shown to be unrelated to load rate. Cadence is likely influenced by leg length. If so, then cadence may be related to load rate when it is normalized to leg length.

• **OBJECTIVES:** To examine the relationship between cadence and leg length in both injured and uninjured runners with a rearfoot strike pattern. We hypothesized that increased leg length would be associated with lower cadence. We also evaluated the relationship between cadence normalized to leg length and the vertical average load rate (VALR), expecting that as cadence normalized to leg length increased, VALR would decrease.

• **METHODS:** In this cross-sectional cohort, laboratory-based study, 40 uninjured and 42 injured recreational runners with a rearfoot strike pattern were measured at self-selected speeds. The relationship of cadence to leg length was

measured between groups by injury status. A secondary analysis evaluated the relationship between cadence normalized to leg length and VALR. The data were analyzed using a multiple linear regression, with injury status as a covariate. Alpha was set to .05.

• **RESULTS:** Accounting for injury status, leg length had a moderate negative association with cadence ($P < .001$, $r = 0.449$, standardized $\beta = -0.443$). There were no associations of VALR with cadence normalized to leg length by injury status or across participants.

• **CONCLUSION:** Lower cadence was observed in recreational runners with longer legs, regardless of injury status. However, cadence was not related to load rate when normalized to leg length. *J Orthop Sports Phys Ther* 2019;49(4):280-283. doi:10.2519/jospt.2019.8420

• **KEY WORDS:** biomechanics, cadence, injury, running

¹Spaulding National Running Center, Spaulding Rehabilitation Hospital, Cambridge, MA. ²Spaulding Rehabilitation Hospital, Harvard Medical School, Boston, MA. The Partners HealthCare Human Research Committee approved this study. The authors certify that they have no affiliations with or financial involvement in any organization or entity with a direct financial interest in the subject matter or materials discussed in the article. Address correspondence to Dr Adam S. Tenforde, 1575 Cambridge Street, Cambridge, MA 02138. E-mail: atenforde@partners.org • Copyright ©2019 *Journal of Orthopaedic & Sports Physical Therapy*®

footwear land this way. We hypothesized that increased leg length would be associated with lower cadence. As a secondary analysis, we investigated the association of cadence normalized to leg length with load rate in runners. As cadence is often related to injury, we examined the relationship between cadence normalized to leg length and load rate in both injured and uninjured runners. We hypothesized that a significant negative relationship between cadence and load rate would be noted when cadence was normalized to leg length, and that this finding would be most significant in the injured runners.

METHODS

A TOTAL OF 82 RUNNERS WITH A rearfoot strike pattern, as confirmed by video, were included in this study (42 injured, 40 uninjured). The 2 populations of runners were derived from samples of convenience. The uninjured runners provided written informed consent as part of an Institutional Review Board–approved study evaluating running mechanics in uninjured recreational runners at the Spaulding National Running Center laboratory. A portion of the runners studied (19 injured and 12 uninjured) were part of a prior investigation.⁶ Uninjured runners were defined as volunteer recreational runners with no injury in the past 6 months and who ran with no pain during the study.

The injured runners were patients who sought physical therapy treatment at the Spaulding National Running Center clinic and were identified through review of medical charts. A running injury was defined as pain of more than 7 days in duration that leads a runner to seek medical treatment.¹⁸ Injuries involved 1 or more anatomical locations and could be unilateral or bilateral. To minimize the influence of pain on running mechanics, only injured runners who reported pain of 3/10 or less on a visual analog scale during the testing were included. The Partners HealthCare Human Research Committee approved this research pro-

ocol. As the injured runners' data were collected as part of the standard of care, a waiver of informed consent was granted for these participants.

Participants were provided a 5-minute warm-up in which they ran at a self-selected speed on an instrumented treadmill (Advanced Mechanical Technology, Inc, Watertown, MA) sampling at 1500 Hz. Inclusion was limited to runners with self-selected speeds of 2.57 ± 0.10 m/s, to minimize the effect of speed on the outcome measures. Injured runners wore their own running shoes, which is standard procedure for running-clinic assessments. The uninjured runners wore laboratory-standard neutral shoes (Pegasus; Nike, Inc, Beaverton, OR), according to the study protocol.

Cadence and vertical average load rate (VALR) were determined from the vertical force data from the instrumented treadmill. Data were averaged over 10 consecutive foot strikes. Forces were normalized to body weight, and VALR was calculated along the rise to the impact peak, as previously described.¹⁴ The left and right limb lengths (measured from the center of the greater trochanter to the medial malleolus) were measured using a flexible measuring tape that was pulled taut by the examiner. The values for left and right limbs were averaged to generate a leg length for each participant.

Statistics

The primary outcome measure in this analysis was the correlation between leg length and cadence for uninjured and injured runners. A secondary analysis of the correlation between cadence normalized to leg length and VALR was also conducted.

Data were examined for linearity using scatter plots and tested for normality using Shapiro-Wilk tests, with alpha set at .05. The Pearson product-moment correlation was used to investigate correlations. A Spearman rank-order correlation was used when data were not normally distributed. Multiple linear regression analyses were used to investigate the effect of in-

jury status. We performed evaluation of all runners with a covariate of injury status (uninjured, 0; injured, 1). An alpha of .05 was used to determine the overall statistical significance of each predictor's coefficient. The coefficient of determination (R^2) and the adjusted R^2 were calculated for each model, along with the 95% confidence intervals for the unstandardized regression coefficients (β). Squared semi-partial correlation coefficients (R^2) were reported for the regressions to investigate the unique contribution (variance) of each predictor. When only 1 predictor is entered (ie, no other covariates), the standardized β is equal to the Pearson product-moment correlation coefficient (r).

RESULTS

THE DEMOGRAPHICS ACROSS THE injured and uninjured groups were very similar in terms of sex, age, and leg length (TABLE 1). Pain in the injured group was less than 1/10, on average. These results suggest that pain did not influence the outcomes. There were no differences in average cadence or cadence normalized to leg length between groups (TABLE 1). However, the uninjured runners exhibited significantly lower VALR compared to the injured runners.

A significant correlation between cadence and leg length was present for all runners combined ($r = 0.449$, $P < .001$) (FIGURE 1). Cadence normalized to leg length was not correlated to VALR ($r = 0.167$, $P = .133$) (FIGURE 2); therefore, no regression models were developed to further assess this association.

When accounting for injury status as a covariate, leg length was negatively associated with cadence (standardized $\beta = -0.443$) (TABLE 2). Leg length, not injury status, accounted for most of the variance in the model.

DISCUSSION

THE PRIMARY FINDINGS FROM THIS study suggest that leg length is moderately correlated with cadence and

RESEARCH REPORT

injury status does not influence this association. Our results suggest that only a portion of variance in cadence was accounted for by leg length. When looking at the scatter plot of the data in **FIGURE 1**, the runners with the longest legs do tend to have the lowest cadence, and vice versa. This suggests that leg length should be considered when evaluating the cadence of a runner, especially in those with the longest or the shortest legs. However, there is a large spread in the data in the center of the graph (ie, leg length between

0.85 and 0.95 m), demonstrating both high and low cadences for very similar leg lengths. Our findings are consistent with a prior investigation that identified low associations of anthropometric characteristics, including leg length, with stride length.¹ It is possible that the relative lengths of the thigh and lower leg or mass distributions of each segment may also play a role, but these were not assessed in our investigation.

In a cross-sectional investigation, Futrell et al⁶ found no association between habitual cadence and VALR in both uninjured and injured runners. However, the moderate association we found between cadence and leg length

suggests that leg length should be considered when interpreting cadence in a runner. Our exploratory investigation evaluating whether cadence normalized to leg length was related to VALR was unrevealing. Combined with the findings of Futrell et al⁶ and a prior report by Wille et al,¹⁶ our results suggest that cadence alone and cadence adjusted for leg length are clearly not associated with VALR in either uninjured or injured runners. The concept of a potential optimal cadence was derived from elite runners and may have more to do with running performance than with running load rate.

In terms of limitations, it should be noted that the current study population

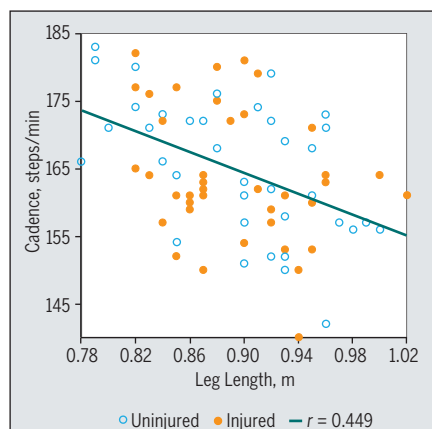


FIGURE 1. Scatter plot of leg length versus cadence. The solid line represents the linear regression line of best fit for the full population, accounting for injury status.

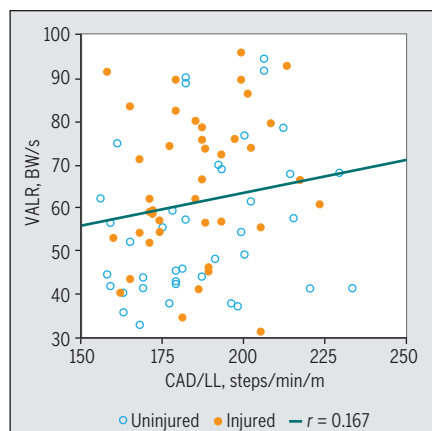


FIGURE 2. Scatter plot of CAD/LL and VALR. The solid line represents the linear regression line of best fit, demonstrating a weakly positive association for the full population ($P = .133$). Abbreviations: CAD/LL, cadence normalized to leg length; VALR, vertical average load rate.

TABLE 1

PARTICIPANT DEMOGRAPHICS AND OUTCOME MEASURES*

Measure	Uninjured (n = 40)	Injured (n = 42)	All Runners (n = 82)
Sex (female), n	23	24	47
Age, y	32 ± 10	34 ± 10	33 ± 10
Pain (VAS)	0.00 ± 0.00	0.40 ± 0.76 [†]	0.20 ± 0.57
Leg length, m	0.90 ± 0.06	0.89 ± 0.05	0.89 ± 0.05
Speed, m/s	2.57 ± 0.10	2.56 ± 0.10	2.57 ± 0.10
Cadence, steps/min	165 ± 10	164 ± 9	165 ± 10
Cadence normalized to leg length, steps/min/m	186 ± 21	184 ± 17	185 ± 19
VALR, BW/s	56.5 ± 17.4	65.7 ± 16.8 [†]	61.2 ± 17.6

Abbreviations: BW, body weight; VALR, vertical average load rate; VAS, visual analog scale.

*Values are mean ± SD unless otherwise indicated.

[†]Statistically significant difference between uninjured and injured runners ($P < .05$).

TABLE 2

MULTIPLE REGRESSION EVALUATING ASSOCIATION OF LEG LENGTH WITH CADENCE

Model	All Runners	
	Leg Length	Injury Status
β^*	-78.4 (-113.9, -43.0)	-1.8 (-5.6, 2.1)
Standardized β	-0.443 [†]	-0.091
$R^{2\ddagger}$	18.60%	0.80%
Model $r^{\S}$	0.449	
Model adjusted $R^{2\ddagger}$	18.2% [†]	
Model P value	<.001	

*Unstandardized beta coefficient. Values in parentheses are 95% confidence interval.

[†]Statistically significant predictor variable ($P < .05$).

[‡]Semi-partial correlation coefficient.

[§]Multiple correlation coefficient.

[†]Statistically significant model ($P < .05$).

was primarily composed of recreational runners with a rearfoot strike pattern. Additionally, they had an average cadence of 165 steps per minute within a limited range of speeds. Therefore, the results cannot be extrapolated to runners with a forefoot strike pattern or to more elite runners, who typically run at higher cadences and speeds.

Another limitation was the difference in footwear used between the groups. Using samples of convenience in the study, those in the injured group used their habitual footwear and those in the uninjured group used standardized lab footwear. However, the correlational results were similar between groups, suggesting that any influence of footwear was minimal.

CONCLUSION

BASED ON OUR FINDINGS, RUNNERS with longer legs have lower cadences than runners with shorter legs. However, leg length only explained a small amount of the variance in cadence. Normalizing cadence to leg length did not result in significant correlations with VALR in runners, regardless of injury status, further supporting the notion that cadence is not related to vertical load rate. ●

KEY POINTS

FINDINGS: Leg length may account for some variation in observed cadence. There is no association between cadence normalized to leg length and vertical average load rate.

IMPLICATIONS: While the relationship between leg length and cadence is moderate, runners with relatively shorter or longer legs may naturally have a higher or lower cadence, respectively. However, even when normalizing cadence to leg length, it is not related to load rate.

CAUTION: The investigation was conducted in runners with a rearfoot strike pattern and within a limited range of running speeds in a laboratory setting. Therefore, extrapolation of these results to runners at different speeds, with different foot-strike patterns, and on outdoor terrain should be done with caution.

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REFERENCES

1. Cavanagh PR, Kram R. Stride length in distance running: velocity, body dimensions, and added mass effects. *Med Sci Sports Exerc.* 1989;21:467-479.
2. Daniels J. *Daniels' Running Formula*. Champaign, IL: Human Kinetics; 1998.
3. Davis IS, Bowser BJ, Mullineaux DR. Greater vertical impact loading in female runners with medically diagnosed injuries: a prospective investigation. *Br J Sports Med.* 2016;50:887-892. <https://doi.org/10.1136/bjsports-2015-094579>
4. Davis IS, Milner CE, Hamill J. Does increased loading during running lead to tibial stress fractures? A prospective study [abstract]. *Med Sci Sports Exerc.* 2004;36:S58.
5. de Almeida MO, Saragiotto BT, Yamato TP, Lopes AD. Is the rearfoot pattern the most frequently foot strike pattern among recreational shod distance runners? *Phys Ther Sport.* 2015;16:29-33. <https://doi.org/10.1016/j.ptsp.2014.02.005>
6. Futrell EE, Jamison ST, Tenforde AS, Davis IS. Relationships between habitual cadence, footstrike, and vertical load rates in runners. *Med Sci Sports Exerc.* 2018;50:1837-1841. <https://doi.org/10.1249/MSS.0000000000001629>
7. Hafer JF, Brown AM, deMille P, Hillstrom HJ, Garber CE. The effect of a cadence retraining protocol on running biomechanics and efficiency: a pilot study. *J Sports Sci.* 2015;33:724-731. <https://doi.org/10.1080/02640414.2014.962573>
8. Heiderscheit BC, Chumanov ES, Michalski MP, Wille CM, Ryan MB. Effects of step rate manipulation on joint mechanics during running. *Med Sci Sports Exerc.* 2011;43:296-302. <https://doi.org/10.1249/MSS.0b013e3181ebdf4>
9. Lieberman DE, Warrener AG, Wang J, Castillo ER. Effects of stride frequency and foot position at landing on braking force, hip torque, impact peak force and the metabolic cost of running in humans. *J Exp Biol.* 2015;218:3406-3414. <https://doi.org/10.1242/jeb.125500>
10. Luedke LE, Heiderscheit BC, Williams DS, Rauh MJ. Influence of step rate on shin injury and anterior knee pain in high school runners. *Med Sci Sports Exerc.* 2016;48:1244-1250. <https://doi.org/10.1249/MSS.0000000000000890>
11. Milner CE, Ferber R, Pollard CD, Hamill J, Davis IS. Biomechanical factors associated with tibial stress fracture in female runners. *Med Sci Sports Exerc.* 2006;38:323-328. <https://doi.org/10.1249/01.mss.0000183477.75808.92>
12. Ogueta-Alday A, Morante JC, Gómez-Molina J, García-López J. Similarities and differences among half-marathon runners according to their performance level. *PLoS One.* 2018;13:e0191688. <https://doi.org/10.1371/journal.pone.0191688>
13. Pohl MB, Hamill J, Davis IS. Biomechanical and anatomic factors associated with a history of plantar fasciitis in female runners. *Clin J Sport Med.* 2009;19:372-376. <https://doi.org/10.1097/JSM.0b013e3181b8c270>
14. Tenforde AS, Ruder MC, Jamison ST, Singh PP, Davis IS. Is symmetry of loading improved for injured runners during novice barefoot running? *Gait Posture.* 2018;62:317-320. <https://doi.org/10.1016/j.gaitpost.2018.03.043>
15. Wellenkötter J, Kernozek TW, Meardon S, Suchomel T. The effects of running cadence manipulation on plantar loading in healthy runners. *Int J Sports Med.* 2014;35:779-784. <https://doi.org/10.1055/s-0033-1363236>
16. Wille CM, Lenhart RL, Wang S, Thelen DG, Heiderscheit BC. Ability of sagittal kinematic variables to estimate ground reaction forces and joint kinetics in running. *J Orthop Sports Phys Ther.* 2014;44:825-830. <https://doi.org/10.2519/jospt.2014.5367>
17. Willy RW, Buchenick L, Rogacki K, Ackerman J, Schmidt A, Willson JD. In-field gait retraining and mobile monitoring to address running biomechanics associated with tibial stress fracture. *Scand J Med Sci Sports.* 2016;26:197-205. <https://doi.org/10.1111/sms.12413>
18. Yamato TP, Saragiotto BT, Lopes AD. A consensus definition of running-related injury in recreational runners: a modified Delphi approach. *J Orthop Sports Phys Ther.* 2015;45:375-380. <https://doi.org/10.2519/jospt.2015.5741>



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