

HEALTH SERVICES RESEARCH

2011 Young Investigator Award Winner: Increased Fat Mass Is Associated With High Levels of Low Back Pain Intensity and Disability

Donna M. Urquhart, PhD,* Patricia Berry, B BiomedSci (Hons),*
Anita E. Wluka, MBBS, FRACP, PhD,* Boyd J. Strauss, MBBS, FRACP, PhD,†
Yuan Yuan Wang, MD, PhD,* Joseph Proietto, MBBS, FRACP, PhD,‡
Graeme Jones, MD, MBBS, FRACP,§ John B. Dixon, MBBS, FRACP, PhD,¶ and
Flavia M. Cicuttini, MBBS, FRACP, PhD,*

Study Design. A cross-sectional study.

Objective. To determine whether body composition is associated with low back pain intensity and/or disability.

Summary of Background Data. The relationship between obesity and low back pain and disability is unclear. No study has examined the role of body composition in low back pain and disability.

Methods. A total of 135 participants (25–62 years), with a range of body mass indices (18–55 kg/m²), were recruited for a study examining the relationship between obesity and musculoskeletal disease. Participants completed the Chronic Back Pain Grade Questionnaire, which examines individuals' levels of low back pain intensity and disability. Body composition was assessed using dual radiograph absorptiometry.

Results. Body mass index was associated with higher levels of back pain intensity (Odds ratio [OR] = 1.35; 95% confidence interval

[CI] = 1.09, 1.67) and disability (OR = 1.66; 95% CI = 1.31, 2.09). Higher levels of pain intensity were positively associated with total body (OR = 1.19; 95% CI = 1.04, 1.38) and lower limb fat mass (OR = 1.51; 95% CI = 1.04, 2.20), independent of lean tissue mass. There were also positive associations between higher levels of low back disability and total body (OR = 1.41; 95% CI = 1.20, 1.67) and upper (OR = 1.67; 95% CI = 1.27, 2.19) and lower (OR = 2.29; 95% CI = 1.51, 3.49) limbs fat mass. Similar relationships were observed with trunk, android, and gynoid fat mass. After adjusting for confounders, no measures of lean tissue mass were associated with higher pain intensity or disability ($P > 0.10$).

Conclusion. Greater fat, but not lean tissue mass, was associated with high levels of low back pain intensity and disability. Longitudinal investigation is needed to determine whether fat mass is predictive of low back pain and disability, as this may have important implications for further prevention strategies. Understanding the mechanism for these relationships may provide novel approaches to managing low back pain.

Key words: body composition, disability, fat mass, low back pain.
Spine 2011;36:1320–1325

From the *Department of Epidemiology and Preventive Medicine, School of Public Health and Preventive Medicine, Monash University, Alfred Hospital, Melbourne, Victoria 3004, Australia; †Body Composition Laboratory, Monash Medical Centre, Melbourne, Victoria 3168, Australia; ‡Department of Medicine, University of Melbourne Austin Health, Victoria 3050, Australia; §Menzies Research Institute, Private Bag 23, Hobart, Tasmania, Australia; and; ¶Baker IDI Heart and Diabetes Institute, Commercial Road, Melbourne, Victoria 3004, Australia.

D. M. U. and P. B. are co-first authors.

Acknowledgment date: April 14, 2010. First revision date: June 18, 2010. Acceptance date: August 2, 2010.

The manuscript submitted does not contain information about medical device(s)/drug(s).

Federal funds were received to support this work. No benefits in any form have been or will be received from a commercial party related directly or indirectly to the subject of this manuscript.

Dr. Urquhart was supported by a NHMRC Public Health Capacity Building Grant 546248 and Monash Senior Fellowship. Patricia Berry is the recipient of an Australian Postgraduate Association Scholarship. Dr. Wluka is the recipient of an NHMRC Clinical Career Development Award (level 1, 545876) and Dr. Wang was supported by a NHMRC Public Health (Australia) Fellowship 465142.

Address correspondence and reprint requests to Dr. Flavia Cicuttini, Department of Epidemiology and Preventive Medicine, School of Public Health and Preventive Medicine, Monash University, Alfred Hospital, Commercial Rd, Melbourne 3004, Victoria, Australia; E-mail: Flavia.Cicuttini@med.monash.edu.au

DOI: 10.1097/BRS.0b013e3181f9fb66

1320 www.spinejournal.com

Copyright © 2011 Lippincott Williams & Wilkins. Unauthorized reproduction of this article is prohibited.

Low back pain is a major public health problem. Episodes of low back pain are commonly considered “nonspecific,” as specific diagnostic tests cannot clearly identify an individual anatomic structure as the source of pain.¹ There is no clear association between “nonspecific” low back pain and spinal abnormalities identified on imaging, particularly as these same abnormalities are observed in asymptomatic individuals.^{2,3} Epidemiologic studies focused on investigating risk factors for nonspecific low back pain have therefore primarily examined the effects of modifiable factors on low back pain and disability, rather than on specific anatomic structures of the spine.

The global burden of obesity is rapidly increasing, with estimates indicating that more than one billion people are overweight and 300 million of these are obese.⁴ It has been hypothesized that an increase in body weight may result in greater mechanical demands on the spine and subsequent structural degeneration and pain. However, a systematic review of 65 studies found that body weight (measured as body

weight and/or body mass index [BMI]) was only a weak risk factor for low back pain.⁵ Such studies have used weight, BMI, and waist-hip ratio (WHR) as measures of obesity,^{6–8} which do not provide information about specific components of body composition, such as muscle and fat mass,⁹ which are increasingly shown to have different roles in the pathogenesis of musculoskeletal disease.¹⁰ No studies have investigated the influence of body composition on low back pain.

The aim of this study was to examine the relationship between fat and lean tissue mass and low back pain intensity and disability, in a population with a weight range from normal to obese.

MATERIALS AND METHODS

Participants

A total of 135 participants, aged 25 to 62 years, who ranged from normal weight to obese, were recruited through local media and community weight loss clinics to take part in a study of obesity and musculoskeletal health. Exclusion criteria included malignancy or inability to complete the study. The study was approved by the Alfred Human Research and Ethics Committee (HREC), the Monash Standing Research Ethics Committee, the Austin Health HREC, and the University of Melbourne Central HREC.

Anthropometric and Physical Activity Data

Height was measured to the nearest 0.1 cm using a stadiometer. Weight was measured to the nearest 0.1 kg using a single pair of electronic scales and with shoes and bulky clothing removed. BMI ($\text{weight} \div \text{height}^2$; kg/m^2) was calculated. Strenuous physical activity was assessed by asking, “On how many days during the last 14 days did you spend at least 20 minutes doing strenuous exercise? *E.g.*, bicycling, brisk walking, *etc.* that was severe enough to raise your pulse rate or cause you to breathe faster,” with frequency options as follows: no days, 1 to 2 days, 3 to 5 days, 6 to 8 days, or 9 or more days. Participation for “3 or more days” was categorized as performing strenuous activity.

Body Composition

Body composition was measured using dual radiograph absorptiometry (GE Lunar Prodigy, using operating system version 9; GE Healthcare, United Kingdom). The machine has a weight limit of approximately 130 kg. Standard regional analyses were used to measure total body, upper and lower limbs, trunk, android, and gynoid fat mass, as well as total body and upper and lower limbs lean tissue mass. Android and gynoid fat mass relates to the distribution of excess fat predominantly around the abdomen, and around the hips, thighs, and buttocks, respectively. Lean tissue mass was used as a measurement of skeletal muscle mass. The skeletal muscle index was calculated as $(\text{arms lean tissue mass} + \text{legs lean tissue mass}) \div \text{height}^2$. Short-term coefficients of variation, assessed in 15 normal young adults, were 1.2% for total body fat mass and 0.4% for total body lean tissue mass.¹¹

Low Back Pain Intensity and Disability Data

The Chronic Pain Grade Questionnaire was administered to obtain information on low back pain intensity and disability over the past 6 months. The Chronic Pain Grade Questionnaire is a reliable and valid instrument for use in population surveys of low back pain.^{12,13} The questionnaire includes seven questions from which a pain intensity score (0–100) and disability points score (0–6) were calculated. To examine the relationship between pain intensity and various participant characteristics, subjects were classified into three groups based on their pain intensity score; no pain ($=0$), low pain intensity (<50), and high pain intensity (≥ 50). Similarly, to investigate risk factors for disability, subjects were categorized into three groups on the basis of their disability points score; no disability ($=0$), low disability (<3), and high disability (≥ 3) as previously described.^{12,13}

Statistical Analysis

The relationship between fat and lean tissue parameters with low back pain intensity and disability were examined using ordinal logistic regression, where the outcome variables, pain and disability, were classified into three ascending levels (no, low, and high). In multivariate analysis, adjustment was made for potential confounders including age, sex, height, and physical activity. Furthermore, to examine the relationship between fat mass, independent of lean tissue mass, and pain intensity and disability, the respective lean tissue mass parameter was included in the multivariate model. Similarly, to examine the relationship of lean tissue mass, independent of fat mass, with pain intensity and disability, the respective fat mass parameter was included in the multivariate model. The statistical analyses were conducted using SPSS Statistics 17.0 (SPSS Institute, Cary, NC). A *P* value of less than 0.05 was considered statistically significant.

RESULTS

The characteristics of the study population ($n = 135$) are presented in Table 1. The mean (SD) age was 47.4 (9.0) years. BMI was approximately normally distributed in this population, with a mean of 32.6 (8.7) kg/m^2 .

The mean total body fat mass was significantly higher in subjects who reported high pain intensity than in those who reported no pain in the previous 6 months (Figure 1A). In contrast, there was no significant difference in mean total body lean tissue mass (Figure 1A). Similarly, mean total body fat mass was significantly higher in subjects who reported high levels of low back disability than in those who did not report disability, while there was no significant difference in mean total body lean tissue mass (Figure 1B).

Relationship Between Body Composition Measures and Low Back Pain Intensity

The univariate analyses showed that BMI and all measures of fat mass including total body, upper and lower limbs, trunk, android, and gynoid fat were positively associated with pain intensity ($P < 0.03$ for all) (Table 2). No significant associations were found between total body, upper and lower

TABLE 1. Participant Characteristics (N = 135)

Characteristics	Values*
Age (yr), mean (SD)	47.4 (9.0)
Sex (female)	113 (83.1)
Body mass index (kg/m ²), mean (SD)	32.6 (8.7)
Participation in strenuous physical activity	42 (30.9)
Ever experienced low back pain	126 (92.6)
Self-reported pain intensity†	
No pain	26 (19.3)
Low pain intensity	80 (59.3)
High pain intensity	29 (21.5)
Self-reported disability†	
No disability	88 (65.2)
Low disability	27 (20.0)
High disability	20 (14.8)
Fat mass (kg), mean (SD)	
Total	37.1 (17.3)
Upper limb	3.6 (1.9)
Lower limb	13.3 (6.7)
Trunk	19.4 (9.2)
Android	3.4 (1.8)
Gynoid	6.7 (2.8)
Lean tissue mass (kg), mean (SD)	
Total	47.4 (9.3)
Upper limb	5.1 (1.3)
Lower limb	15.5 (3.3)

*Values are reported as n (%) unless otherwise stated.
†Refers to the previous 6 mo.

limb lean tissue mass, and higher pain intensity ($P = 0.21$ – 0.90). In the multivariate analyses adjusting for age, sex, height, and physical activity; total body, upper limb, trunk, android, and gynoid fat mass remained positively associated with higher pain intensity ($P < 0.009$ for all), whereas lower limb fat mass showed a trend toward significance ($P = 0.06$). In addition, upper limb lean tissue mass, unlike total body and lower limb lean tissue measures, reached significance ($P = 0.001$). However, when both fat and lean tissue mass were included in the multivariate model, total body and lower limb fat mass were the only parameters that remained associated with higher pain intensity ($P < 0.03$ for both), with upper limb fat and lean tissue mass not reaching significance ($P = 0.11$ – 0.47). The relationship between total body fat mass and low back pain intensity was similar in men (OR = 1.55; 95% CI = 0.80, 2.99; $P = 0.19$) and women (OR = 1.17; 95% CI = 1.01, 1.36; $P = 0.04$), as was the association between lower limb fat mass and low back pain intensity (Men: OR = 1.41; 95% CI = 0.95, 2.12; $P = 0.09$ and women: OR = 1.48; 95% CI = 1.01, 2.23; $P = 0.05$). However, these results did not reach significance in men. The skeletal muscle index was not significantly associated with low back pain intensity in either univariate (OR = 0.83; 95% CI = 0.62, 1.11; $P = 0.20$) or multivariate (OR = 0.74; 95% CI = 0.52, 1.04; $P = 0.08$) analysis, after adjusting for age, sex, and physical activity.

Relationship Between Body Composition Measures and Low Back Disability

The univariate analyses showed that BMI, total body, upper and lower limbs, trunk, android, and gynoid fat mass were positively associated with low back disability ($P < 0.0001$ for all) (Table 3). No significant association was found between measures of lean tissue mass and levels of disability ($P = 0.21$ – 0.90). In the multivariate analyses adjusting for age, sex, height, and physical activity, the positive associations between BMI and all fat mass parameters and disability remained significant ($P < 0.0001$ for all). Similarly, total body, upper, and lower limbs lean tissue mass, also showed a significant association in multivariate analysis ($P < 0.03$ for all). However,

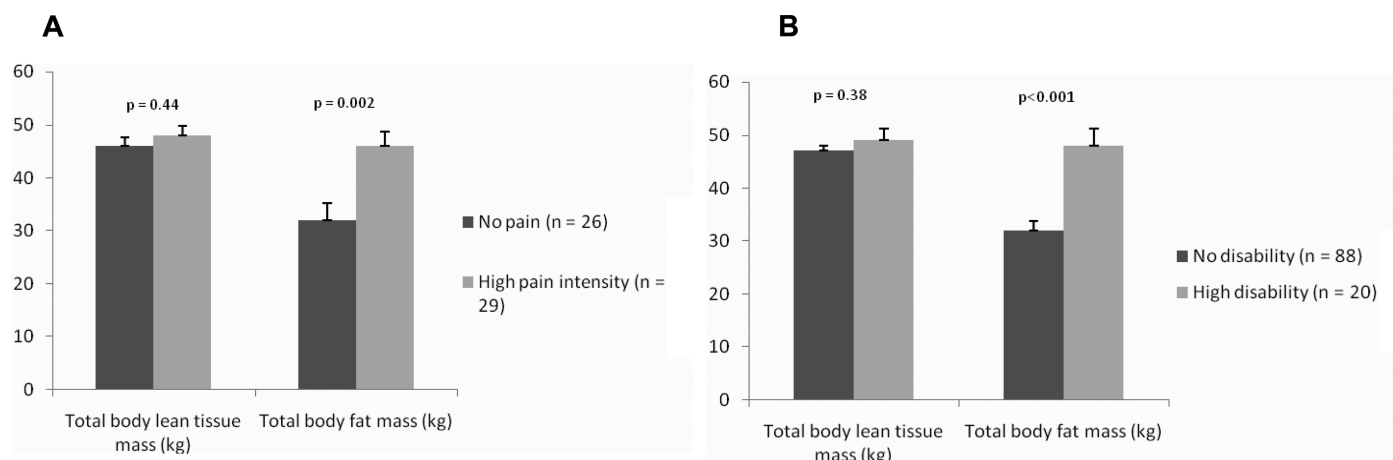


Figure 1. Mean (SE) total body fat and lean tissue mass in subjects with and without high pain intensity (panel A) and disability (panel B)

TABLE 2. Associations Between Self-Reported Levels of Low Back Pain Intensity and Body Mass Index and Body Composition (N = 135)

Participant Characteristics	Pain Intensity					
	Univariate Analysis*		Multivariate Analysis†		Multivariate Analysis‡	
	Coefficient (95% CI)	P	Coefficient (95% CI)	P	Coefficient (95% CI)	P
Body mass index (kg/m ²)	1.38 (1.12, 1.68)	0.002	1.35 (1.09, 1.67)	0.005	NA	NA
Fat mass (kg)						
Total	1.16 (1.05, 1.28)	0.003	1.16 (1.04, 1.29)	0.009	1.19 (1.04, 1.38)	0.02
Upper limb	1.32 (1.10, 1.58)	0.003	1.31 (1.07, 1.60)	0.008	1.18 (0.93, 1.50)	0.17
Lower limb	1.32 (1.03, 1.71)	0.03	1.31 (0.99, 1.74)	0.06	1.51 (1.04, 2.20)	0.03
Trunk	1.37 (1.13, 1.66)	0.01	1.35 (1.11, 1.65)	0.003	NA	NA
Android	1.37 (1.13, 1.67)	0.001	1.35 (1.10, 1.64)	0.004	NA	NA
Gynoid	1.21 (1.07, 1.37)	0.003	1.21 (1.05, 1.39)	0.008	NA	NA
Lean tissue mass (kg)						
Total	1.07 (0.89, 1.28)	0.47	1.28 (0.98, 1.67)	0.07	0.89 (0.64, 1.23)	0.47
Upper limb	1.17 (0.91, 1.51)	0.21	2.37 (1.45, 3.89)	0.001	1.57 (0.91, 2.71)	0.11
Lower limb	1.04 (0.63, 1.71)	0.90	1.41 (0.66, 2.97)	0.38	0.55 (0.22, 1.36)	0.20
Per 5-kg/m ² increase in body mass index, 5-kg increase for total body fat and lean tissue mass, lower limb fat and lean tissue mass, and trunk fat mass. Per 1-kg increase for upper limb fat and lean tissue mass, android and gynoid fat mass.						
*Odds ratio per unit increase in respective body composition measurement.						
†Odds ratio per unit increase in respective body composition measurement adjusted for age, sex, height, and physical activity.						
‡Odds ratio per unit increase in respective body composition measurement adjusted for age, sex, and physical activity, and fat or lean tissue mass.						
CI indicates confidence interval; NA, not applicable.						

when both fat mass and lean tissue mass were included in the multivariate analyses with adjustments for age, sex, and physical activity, all fat mass parameters remained positively associated with disability ($P < 0.0001$ for all), whereas all lean tissue mass parameters did not reach significance ($P = 0.12$ – 0.70). The skeletal muscle index was not significantly associated with low back pain disability in either univariate (OR = 0.86; 95% CI = 0.63, 1.17; $P = 0.34$) or multivariate analysis (OR = 0.79, 95% CI = 0.53, 1.18; $P = 0.25$), after adjusting for age, sex, and physical activity. Similar results were obtained when men and women were analyzed separately (data not shown).

DISCUSSION

This study found that individuals with a greater fat mass of the whole body and upper and lower limbs were more likely to have higher levels of low back pain intensity and disability, independent of lean tissue mass. Similar associations were found with trunk, android, and gynoid fat mass. In contrast, we found no relationship between lean tissue mass and pain intensity and disability of the lower back, independent of fat mass and after adjusting for confounders. Although increased spinal loading associated with excess fat may explain the relationship between fat mass and back pain and disability,

it is also possible that metabolic processes associated with excess adipose tissue may play an important role.

Our finding that total body, limb, trunk, android, and gynoid fat mass is associated with higher levels of low back pain intensity and disability is novel. No previous studies have examined the relationship between fat mass and low back pain. Rather, they have used general measures of obesity, such as weight, BMI, and WHR, which do not provide specific information on mass of fat and lean tissue. Studies that have used measures of WHR to determine whether there is an association between central adiposity and chronic low back pain have reported conflicting findings. Although a case-control study of community-based women reported a high WHR ratio to be associated with a lower risk of severe low back pain,⁸ a cross-sectional, community-based study reported women with a high BMI and large WHR to have a greater likelihood of low back pain⁶ and a subgroup analysis of a case-control study reported an increased WHR in women with chronic low back pain and a negative straight leg raise test.⁷ Moreover, interpretation of the latter study is difficult, as it was based on a subgroup analysis, which failed to find an association in women with a positive straight leg raise test, or in men, regardless of their straight leg raise test result. It is well recognized that weight and BMI do not differentiate lean

TABLE 3. Associations Between Self-Reported Levels of Low Back Disability and Body Mass Index and Body Composition (N = 135)

	Disability					
	Univariate Analysis*		Multivariate Analysis†		Multivariate Analysis‡	
Participant Characteristics	Coefficient (95% CI)	P	Coefficient (95% CI)	P	Coefficient (95% CI)	P
Body mass index (kg/m ²)	1.64 (1.32, 2.03)	0.0001	1.66 (1.31, 2.09)	0.0001	NA	NA
Fat mass (kg)						
Total	1.29 (1.16, 1.45)	0.0001	1.32 (1.16, 1.49)	<0.001	1.41 (1.20, 1.67)	0.0001
Upper limb	1.64 (1.33, 2.02)	0.0001	1.73 (1.38, 2.18)	<0.001	1.67 (1.27, 2.19)	0.0001
Lower limb	1.78 (1.35, 2.34)	0.0001	1.93 (1.40, 2.65)	<0.001	2.29 (1.51, 3.49)	0.0001
Trunk	1.60 (1.29, 1.97)	<0.001	1.61 (1.28, 2.00)	<0.001	NA	NA
Android	1.57 (1.27, 1.94)	<0.001	1.57 (1.27, 1.95)	<0.001	NA	NA
Gynoid	1.38 (1.20, 1.58)	<0.001	1.42 (1.21, 1.66)	<0.001	NA	NA
Lean tissue mass (kg)						
Total	1.07 (0.89, 1.28)	0.50	1.44 (1.09, 1.90)	0.01	0.77 (0.54, 1.09)	0.14
Upper limb	1.08 (0.84, 1.39)	0.55	2.69 (1.57, 4.60)	<0.001	1.13 (0.61, 2.09)	0.70
Lower limb	1.14 (0.68, 1.92)	0.62	2.38 (1.07, 5.26)	0.03	0.46 (0.17, 1.23)	0.12

Per 5-kg/m² increase in body mass index, 5-kg increase for total body fat and lean tissue mass, lower limb fat and lean tissue mass, and trunk fat mass. Per 1-kg increase for upper limb fat and lean tissue mass, android and gynoid fat mass.

*Odds ratio per unit increase in respective body composition measurement.

†Odds ratio per unit increase in respective body composition measurement adjusted for age, sex, height, and physical activity.

‡Odds ratio per unit increase in respective body composition measurement adjusted for age, sex, and physical activity, and fat or lean tissue mass. CI indicates confidence interval; NA, not applicable.

tissue and fat mass.⁹ In addition, a recent systematic review of 65 epidemiologic studies has shown only a weak association between weight and BMI and low back pain.⁵

Although imaging studies have previously reported a reduction in cross-sectional area of specific trunk muscles in patients with low back pain,^{14,15} measures of lean tissue of the total body or limbs have not been performed. While Toda *et al*⁷ reported reduced trunk and lower extremity muscle mass in female patients with chronic low back pain and a negative straight leg raise test compared with controls, these findings were only evident in a specific subgroup of 71 women, who were defined according to the straight leg raise test, which has been reported to have limited diagnostic accuracy.¹⁶ Furthermore, no adjustment for confounders, particularly the independent effect of muscle and fat mass, was performed even though both are strongly associated with BMI and individually may simply reflect obesity. These findings suggest that although there is evidence for atrophy of specific muscles surrounding the spine, there is a paucity of data indicating significant changes in muscle mass of the total body or upper and lower limbs associated with low back pain.

Although we found that the mean amount of fat mass was significantly higher in those who reported high levels of pain intensity (Figure 1A) or disability (Figure 1B) than in those

who did not, we found no significant difference in the mean amount of lean tissue mass. This suggests that the effect of obesity is mediated by fat mass, with lean mass remaining relatively stable, despite differences in degrees of obesity. The mechanism by which fat mass effects back pain and disability may be via excess fat placing increased load on the spine. Alternatively, the effect of fat mass may occur through systemic processes. This is supported by our findings that all measures of fat mass throughout the body, including those of the trunk and upper and lower limbs, were positively associated with pain intensity and disability.

Indeed, there is growing evidence to suggest that metabolic activity may be important in the pathogenesis of musculoskeletal disorders. For example, it is well recognized that obesity is a risk factor for hand osteoarthritis, which is a non-weight-bearing joint,^{17,18} and that fat mass is a risk factor for knee cartilage defects,¹⁹ which are strong predictors of knee pain and progression of osteoarthritis.^{20,21} Adipose tissue is metabolically active, releasing a multitude of proinflammatory cytokines and key mediators of metabolism termed the “adipokines.”²² Given high concentrations of macrophage, secreted cytokines have also been shown to be involved in the development of symptomatic disc disease through nerve ingrowth and neovascularization,²³ and inflammatory cytokines

have been demonstrated in lumbar facet joints in degenerative spinal conditions.²⁴ It is possible that inflammatory factors released from adipose tissue may heighten inflammatory changes surrounding the spine, subsequently increasing the associated pain and disability. Moreover, such inflammatory processes, including those involving the neural system, can play a large role in the destruction of tissue surrounding the spine and the development of chronic pain conditions.²⁵

Although a limitation of our study was that we had a smaller proportion of men, our sample had several strengths, in that it included participants with a range of weight extending from normal to obese. We also adjusted for confounders, such as age, sex, height, and physical activity, as well as fat and lean tissue mass, which are known to have a colinear relationship. Similar results were found when analyses were performed in men and women separately. Although our assessment of pain and disability was questionnaire-based, these measures were performed using validated tools. While this study was cross-sectional and had a relatively modest sample size, we were able to identify statistically significant and consistent relationships using independent measures of fat mass. Future longitudinal studies, in larger populations with a wide spectrum of back pain and disability, will be needed to determine the predictive nature of our findings.

We found that increased fat mass (total body, upper and lower limb, trunk, and android and gynoid) was associated with higher levels of low back pain intensity and disability. Longitudinal investigation is needed to determine whether fat mass is predictive of low back pain and disability, as this may have important implications for future prevention strategies. Moreover, understanding the mechanism of action by which increased adipose tissue is associated with back pain and disability may provide insights into therapeutic strategies for the prevention of low back pain.

➤ Key Points

- ❑ Greater fat, but not lean tissue mass, was associated with high levels of low back pain intensity and disability.
- ❑ Although the associations between fat mass and back pain and disability are likely to be multifactorial, spinal loading and metabolic processes may play an important role.
- ❑ Longitudinal investigation is needed to determine whether fat mass is predictive of low back pain and disability, because this may have important implications for further prevention strategies.

References

1. Airaksinen O, Brox JJ, Cedraschi C, et al. Chapter 4. European guidelines for the management of chronic nonspecific low back pain. *Eur Spine J* 2006;15(suppl 2):S192–300.
2. Jensen M, Brant-Zawadzki MN, Obuchowski N, et al. Magnetic resonance imaging of the lumbar spine in people without back pain. *N Engl J Med* 1994;331(2):69–73.
3. van Tulder M, Assendelft WJ, Koes BW, et al. Spinal radiographic findings and nonspecific low back pain. A systematic review of observational studies. *Spine* 1997;22(4):427–34.
4. World Health Organization. *Global Strategy on Diet, Physical Activity, and Health*. Geneva, Switzerland: World Health Organization; 2011. <http://www.who.int/dietphysicalactivity/publications/facts/obesity/en./2010>.
5. Leboeuf-Yde C. Body weight and low back pain. A systematic literature review of 56 journal articles reporting on 65 epidemiologic studies. *Spine* 2000;25(2):226–37.
6. Han T, Schouten JSAG, Lean MEJ, et al. The prevalence of low back pain and associations with body fatness, fat distribution, and height. *Int J Obes Relat Metab Disord* 1997;21:600–7.
7. Toda Y, Segal N, Toda T, et al. Lean body mass and body fat distribution in participants with chronic low back pain. *Arch Intern Med* 2000;160(21):3265–9.
8. Yip Y, Ho S, Chan S. Tall stature, overweight, and the prevalence of low back pain in Chinese middle-aged women. *Int J Obes Relat Metab Disord* 2001;25(6):887–92.
9. Roubenoff R. Applications of bioelectrical impedance analysis for body composition to epidemiologic studies. *Am J Clin Nutr* 1996;64(3):459S–62S.
10. Sandell L. Obesity and osteoarthritis: is leptin the link? *Arthritis Rheumatism* 2009;60(10):2858–60.
11. Bennell K. A prospective study investigating the skeletal response to intense athletic activity. Melbourne, Australia: The University of Melbourne; 1995.
12. Von Korf M, Ormel J, Keefe FJ, et al. Grading the severity of chronic pain. *Pain* 1992;50:133–49.
13. Smith B, Penny KI, Purves AM, et al. The Chronic Pain Grade questionnaire: validation and reliability in postal research. *Pain Suppl* 1997;71:141–7.
14. Danneels L, Vanderstraeten GG, Cambier DC, et al. CT imaging of trunk muscles in chronic low back pain patients and healthy control subjects. *Euro Spine J* 2000;9(4):266–72.
15. Wallwork T, Stanton WR, Freke M, et al. The effect of chronic low back pain on size and contraction of the lumbar multifidus muscle. *Manual Therapy* 2009;14(5):496–500.
16. Devillé W, van der Windt DA, Dzaferagić A, et al. The test of Lasègue: systematic review of the accuracy in diagnosing herniated discs. *Spine* 2000;25(9):1140–7.
17. Cicuttini F, Baker J, Spector T. The association of obesity with osteoarthritis of the hand and knee in women: a twin study. *J Rheumatol* 1996;23(7):1221–6.
18. Oliveria S, Felson DT, Cirillo PA, et al. Body weight, body mass index, and incident symptomatic osteoarthritis of the hand, hip, and knee. *Epidemiology* 1999;10(2):161–6.
19. Wang Y, Wluka AE, English DR, et al. Body composition and knee cartilage properties in healthy, community-based adults. *Ann Rheum Dis* 2007;66(9):1244–8.
20. Cicuttini F, Ding C, Wluka A, et al. Association of cartilage defects with loss of knee cartilage in healthy, middle-age adults: a prospective study. *Arthritis Rheum* 2005;52(7):2033–9.
21. Ding C, Cicuttini F, Scott F, et al. Association of prevalent and incident knee cartilage defects with loss of tibial and patellar cartilage: a longitudinal study. *Arthritis Rheum* 2005;52(12):3918–27.
22. Pottie P, Presle N, Terlain B, et al. Obesity and osteoarthritis: more complex than predicted! *Ann Rheumatic Dis* 2006;65:1403–5.
23. Freemont A, Peacock TE, Goupille P, et al. Nerve ingrowth into diseased intervertebral disc in chronic back pain. *Lancet* 1997;350:178–81.
24. Igarashi A, Kikuchi S, Konno S, et al. Inflammatory cytokines released from the facet joint tissue in degenerative lumbar spinal disorders. *Spine* 2004;29(19):2091–5.
25. Willard F. Neuroendocrine-immune network, nociceptive stress, and the general adaptive response. In: Everett T, Dennis M, Ricketts E, eds. *Physiotherapy in Mental Health: A Practical Approach*. Oxford, United Kingdom: Butterworth Heinemann Ltd; 2005:102–6.