Eur Spine J (2016) 25:1188–1195
DOI 10.1007/s00586-015-4055-2

ORIGINAL ARTICLE

Are obesity and body fat distribution associated with low back pain in women? A population-based study of 1128 Spanish twins

Amabile B. Dario[1] · Manuela L. Ferreira[2] · Kathryn Refshauge[1] · Juan F. Sánchez-Romera[3,4] · Alejandro Luque-Suarez[5] · John L. Hopper[6] · Juan R. Ordoñana[3,4] · Paulo H. Ferreira[1]

Received: 31 August 2014 / Revised: 18 March 2015 / Accepted: 4 June 2015 / Published online: 18 June 2015

Abstract
Purpose To investigate the relationship between different measures of obesity and chronic low back pain (LBP) using a within-pair twin case–control design that adjusts for genetics and early shared environment.
Methods A cross-sectional association between lifetime prevalence of chronic LBP and different measures of obesity (body mass index-BMI; percent body fat; waist circumference; waist–hip ratio) was investigated in 1128 female twins in three stages: (i) total sample analysis; (ii) within-pair case–control analysis for monozygotic (MZ) and dizygotic (DZ) twins together; (iii) within-pair case–control analysis separated by DZ and MZ. Odds ratios (OR) and 95 % confidence intervals (CI) were calculated.
Results BMI (OR 1.12; 95 % CI 1.02–1.26) and percent body fat (OR 1.15; 95 % CI 1.01–1.32) were weakly associated with lifetime prevalence of chronic LBP in the total sample analysis but were absent when shared environment and genetic factors were adjusted for using the within-pair case–control analysis. Greater waist–hip ratios were associated with smaller prevalence estimates of chronic LBP in the within-pair case–control analysis with both MZ and DZ twins (OR 0.67; 95 % CI 0.47–0.94). However, this association did not remain after the full adjustment for genetic factors in the MZ within-pair case–control analysis.
Conclusions BMI, percent of fat mass and greater depositions of fat and mass around the hips are associated with increases in chronic LBP prevalence in women but these associations are small and appear to be confounded by the effects of genetics and early shared environment. Therefore, our results do not support a causal direct relationship between obesity and chronic LBP.

Keywords Obesity · Low back pain · Genetics · Twins

✉ Amabile B. Dario
adar3900@sydney.edu.au

[1] Discipline of Physiotherapy, Faculty of Health Sciences, The University of Sydney, PO Box 170, 75 East Street, Lidcombe, Sydney, NSW 2141, Australia

[2] The George Institute for Global Health & Institute of Bone and Joint Research/The Kolling, Sydney Medical School, The University of Sydney, Level 13, 321 Kent Street, Sydney, NSW 2000, Australia

[3] Murcia Twin Registry, Department of Human Anatomy and Psychobiology, University of Murcia, Campus de Espinardo, 30100 Murcia, Spain

[4] IMIB-Arrixaca, HUVA Virgen de la Arrixaca, 30120 Murcia, Spain

[5] Discipline of Physiotherapy, University of Málaga, Avenida Cervantes, 2, 29071 Málaga, Spain

[6] Australian Twin Registry, Centre for Epidemiology and Biostatistics, The University of Melbourne, Parkville, Melbourne, VIC 3010, Australia

Introduction

Obesity is a pandemic and growing public health concern [1]. It is recognized as the main public health problem in industrialized countries [2] and is linked to morbidity and high mortality rates [1]. Obesity has also been found to be associated with various musculoskeletal disorders, including low back pain (LBP) [3].

LBP is common with the 1-month prevalence being estimated as 23.2 % (95 % CI 20.3–26.1). It is the highest contributor to the number of years that people live with disability in the world [4]. LBP is more common in women

than in men [5] and little is understood about its etiology [6]. There is a consensus in the field that research efforts need to be allocated to the investigation of causes and risk factors for LBP, as this understanding is crucial for effective prevention [7]. From a public health perspective, it would be important to know if lifestyle factors, such as excessive body weight, are contributors to LBP incidence especially when considering that obesity is a potential modifiable risk factor.

Although it is generally assumed that obesity and LBP are associated, the actual path between these conditions remains controversial [6, 8]. For instance, whereas some studies have shown that obesity increases LBP prevalence [9, 10], others have failed to observe any association between the two [11, 12]. Moreover, in a study that used a twin sample [10], the positive association between body mass index (BMI) and LBP found in the total sample analysis disappeared in monozygotic twins dissimilar in body weight, suggesting that genetics possibly influence this relationship.

One of the limitations of the studies investigating the relationship between obesity and LBP is how obesity has been assessed. The frequent measure used to classify obesity in previous studies was BMI [2, 8, 13] which does not account for the distribution of fat in the body. Although there is evidence that body fat distribution rather than absolute total fat is associated with increases in the risk of diseases such as diabetes and coronary artery disease [14], most studies [2, 8, 13] have not investigated it. To our knowledge, only two studies have looked at the relationship between body fat distribution (where the excessive adipose tissue is stored) and LBP and found higher prevalence of LBP in women with a predominant central obesity, measured by waist circumference and waist–hip ratio [15, 16]. Although familial factors (genetics and early environment) were not investigated in these studies, the findings indicate that body fat distribution, in addition to total fat, should be considered when analyzing the obesity–LBP relationship.

In this current study, we aim to investigate the relationship between chronic LBP and different measures of obesity that account for body fat distribution in female twins. Previous evidence has indicated that women are more likely to report back pain [5, 17], seek medical care more frequently [18], and suffer from pain for longer periods [17] than men. It is likely that women represent a specific subgroup of patients with LBP and deserves special attention particularly when investigating factors such as body fat distribution. By performing a within-pair twin case–control analysis (where one twin has chronic LBP while the co-twin does not), we are able to control for possible genetic and early shared environmental effects on the obesity–LBP relationship.

Methods

Design

Cross-sectional observational study with a within-pair twin case–control design.

Study sample and data collection

All twins included in this study were registered in the Murcia Twin Registry (MTR), a population-based twin registry of adult multiples born between 1940 and 1966 in the region of Murcia, Southeast Spain. Information regarding the MTR characteristics and recruitment procedures can be found elsewhere [19]. All registry and data collection procedures involved in this study were approved by the Murcia University Ethics Committee, and informed consent was obtained from all twins.

Assessment of chronic LBP

The main outcome of this study was lifetime prevalence of chronic LBP with participants being asked the following question: “Have you ever suffered from chronic low back pain?”, based on the corresponding item from the Spanish National Health Survey. The Survey defines ‘chronic’ as a health problem lasting for at least 6 months to screen and eliminate isolated acute processes. This includes seasonal or recurrent episodes. Participants answering “yes” to this question were categorized as having a history of chronic LBP.

Measures of obesity-related measures

Self-reported measures of weight and height were obtained for 38 % of the sample (430 participants). For the other 62 % of the sample (698 participants), standardized anthropometric measurements were obtained on participants by a blinded research assistant for weight, height, waist circumference and percent body fat. BMI was calculated by dividing the individuals’ body weight in kilograms by the square of their height in meters. Percentage of body fat was measured by bioelectrical impedance using TANITA BC-420 MA (Tanita Corporation of America, USA) equipment. A single new and calibrated device was used during the whole study. Subjects were instructed to fast and not practise physical exercise during the previous 4 h, refrain from drinking alcoholic beverages during the last 24 h, and urinate closely prior to the appointment. Waist circumference was measured at the narrowest torso circumference or, alternatively, at the midpoint between the inferior border of the ribcage and the superior aspect of

the iliac crest using an inelastic measuring tape. Hip circumference was measured at the widest point or, alternatively, over the buttocks". Waist–hip ratio (WHR) was calculated as the ratio between their respective components.

Statistical analysis

The analysis was conducted in three stages: (i) total sample analysis; (ii) within-pair case–control analysis for monozygotic (MZ) and dizygotic (DZ) twins together; (iii) within-pair case–control analysis separated by DZ (iii.a) and MZ (iii.b) (Fig. 1). BMI, percent body fat, waist circumference and waist-hip ratio were classified in four categories according to percentile distributions of the data (i.e., category one $\leq$25th lowest percentile; 25th percentile $<$ category two $\leq$ 50th percentile; 50th $<$ category three $\leq$ 75th percentile; category four $>$ 75th percentile). The specific cut-off points used to define the quartiles for obesity-related measures are defined in Table 1.

Potential confounders for the total sample analysis included age, engagement in leisure physical activity, engagement in daily physical activity (work and domestic related) and smoking. The same confounders were investigated for the within-pair twin case–control analyses, except age. Leisure physical activity was dichotomised into low/no physical activity engagement in recreational physical activity (mainly sedentary) or moderate/vigorous physical activity engagement (regular physical activity or training several times a week/month, ex: jogging, swimming, cycling). Daily physical activity was dichotomised into low/no engagement in work-related physical activity (mainly sitting or light physical efforts) or moderate/vigorous physical activity engagement (doing tasks that require a strong physical effort). Smoking was dichotomized as ex/never smoker or current smoker. We included confounders in the multivariate logistic regression models if p values for associations in univariate models were $<$0.2.

Total sample analysis

For the total sample analysis, we investigated the association between obesity-related measures (BMI, percent body fat, waist circumference and waist–hip ratio) and lifetime prevalence of chronic LBP using separate multivariate unconditional regression models for each obesity measure. All participants were included and twins were analyzed as individuals rather than pairs.

Within-pair twin case–control analyses

To control for the effect of genetics and early shared environment on a possible association between obesity-related measures and the lifetime prevalence of chronic LBP, we performed a subsequent within-pair twin case–control analysis on all complete and discordant pairs for LBP status (one twin reported chronic LBP while the other did not) using conditional logistic regression. In addition, separated analyses were conducted for DZ and MZ twin pairs. Theoretically, when the magnitude of the association between two variables (i.e., BMI and LBP) increases sequentially from the total sample analysis (no adjustment for genetics or early shared environment) to a DZ within-pair case–control analysis (adjustment for early shared environment and approximately 50 % of genetics occurs) and then to a MZ within-pair case–control analysis (adjustment for early shared environment and approximately 100 % of genetics), the relationship between the two variables is more direct and possibly more consistent with a direct causal path [20].

We set $p < 0.05$ as our level of significance for the estimates of association in the multivariate models and presented estimates as odds ratios (OR) and 95 % confidence intervals (CI). OR represents the odds of having chronic LBP per quartile step. Data analyses were performed using STATA statistical software (version 12.0).

Results

Sample characteristics

Data on lifetime prevalence of chronic LBP for the total sample of 1128 females was estimated as 41.3 % (95 % CI 38.4–44.2) with the prevalence for MZ and DZ estimated as 43.1 % (95 % CI 38.3–47.9) and 40.3 % (95 % CI 36.7–43.9), respectively. Among all twins, the mean age was 54 years with 64 % of the twins being DZ (Table 2). Results for the obesity-related measures for twins with and without chronic LBP are described in Table 3.

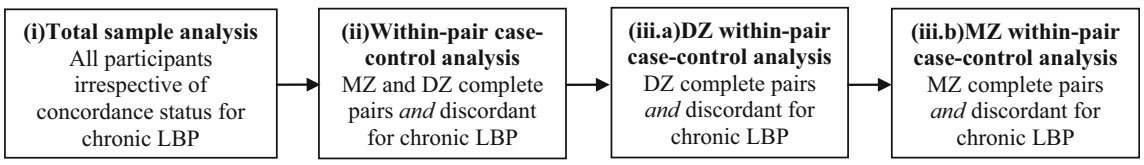

Fig. 1 Statistical analysis schema. *LBP* low back pain, *MZ* monozygotic twins, *DZ* dizygotic twins

Table 1 Cut-off points used for the obesity-related measures

Obesity related measures	1^0 Quartile	2^0 Quartile	3^0 Quartile	4^0 Quartile
Body mass index (kg/m^2)	≤23.71	>23.71 to ≤26.40	>26.40 to ≤30.11	>30.11
Percent body fat (%)	≤29.64	>29.64 to ≤34.93	>34.93 to ≤39.55	>39.55
Waist circumference (cm)	≤76	>76 to ≤84	>84 to ≤93	>93
Waist-hip ratio	≤0.78	>0.78 to ≤0.84	>0.84 to ≤0.89	>0.89

Table 2 Study sample characteristics of anthropometric data and lifestyle factors

Variables	LBP absent		LBP present		Total	
	Mean (SD) or %	*n*	Mean (SD) or %	*n*	Mean (SD) or %	*n*
Age (years)	53.23(7.38)	662	53.59 (7.38)	466	53.38 (7.38)	1128
Height (m)	1.57 (0.07)	662	1.57 (0.07)	466	1.57 (0.07)	1128
Weight (kg)	66.19 (11.24)	662	67.79 (12.39)	466	66.85 (11.75)	1128
Smoking habits[a]	38.75 %	255	42.12 %	195	40.14 %	450
Daily physical activity[b]	15.58 %	103	11.83 %	55	14.03 %	158
Leisure physical activity[b]	53.34 %	351	43.44 %	202	49.24 %	553
DZ twins	59.69 %	428	40.31 %	289	63.56 %	717
MZ twins	56.93 %	234	43.07 %	177	36.44 %	411

LBP low back pain, *DZ* dizygotic, *MZ* monozygotic, *BMI* body mass index, *SD* standard deviation, *n* number of participants

[a] Percentage who smoked

[b] Percentage engaged in physical activity

Table 3 Study sample characteristics of obesity-related measures for the total sample and cases and controls within a twin pair

Variables	LBP absent		LBP present		Total	
	Mean (SD) or %	*n*	Mean (SD) or %	*n*	Mean (SD) or %	*n*
Body mass index						
Total sample	26.81 (4.59)	662	27.68 (5.23)	466	27.17 (4.88)	1128
MZ and DZ pairs	27.42 (4.88)	155	27.68 (5.65)	155	27.55 (5.28)	310
DZ pairs	27.75 (4.83)	77	28.48 (5.42)	77	28.11 (5.13)	154
MZ pairs	27.09 (4.95)	78	26.90 (5.80)	78	26.99 (5.38)	156
Body fat (%)						
Total sample	34.14 (7.03)	374	34.93 (7.60)	313	34.50 (7.30)	687
MZ and DZ pairs	34.85 (7.40)	128	35.71 (7.18)	128	35.28 (7.29)	256
DZ pairs	35.14 (7.14)	65	36.77 (7.06)	65	35.95 (7.12)	130
MZ pairs	34.55(7.71)	63	34.62 (7.20)	63	34.58 (7.43)	126
Waist circumference (cm)						
Total Sample	84.64 (11.94)	378	85.40 (12.50)	316	84.99 (12.20)	694
MZ and DZ pairs	85.79 (11.25)	132	86.42 (12.59)	132	86.10 (12.05)	264
DZ pairs	86.92 (10.85)	67	88.98 (12.46)	67	87.95 (11.69)	134
MZ pairs	84.63 (12.15)	65	83.77 (12.27)	65	84.20 (12.17)	130
Waist-hip ratio						
Total Sample	0.85 (0.08)	371	0.85 (0.08)	311	0.85 (0.08)	682
MZ and DZ pairs	0.85 (0.07)	132	0.85 (0.08)	132	0.85 (0.08)	264
DZ pairs	0.86 (0.08)	67	0.87 (0.08)	67	0.86 (0.08)	134
MZ pairs	0.85 (0.07)	65	0.84 (0.07)	65	0.84 (0.07)	130

LBP low back pain, *SD* standard deviation, *n* number of participants, *MZ* monozygotic, *DZ* dizygotic

BMI

BMI (OR 1.13; 95 % CI 1.02–1.26) was weakly associated with lifetime prevalence of chronic LBP in the total sample analysis but no association was found between BMI and chronic LBP in any of the within-pair twin case–control analyses (Table 4).

Percent body fat

Percent body fat (OR 1.15; 95 % CI 1.01–1.32; $p = 0.05$) was weakly associated with lifetime prevalence of chronic LBP but no association was identified in any of the within-pair case–control analyses.

Waist circumference

No association was found between waist circumference and lifetime prevalence of chronic LBP for the total sample or the within-pair case–control analyses.

Table 4 Total sample analysis and within-pair twin case–control analysis for chronic low back pain

Multivariate models	OR (95 % CI)	*p* value	*n*
Body mass index (kg/m^2)			
Total sample[a]	1.13 (1.02–1.26)	0.026*	1123
MZ and DZ pairs[a]	1.12 (0.83–1.51)	0.449	310
DZ pairs[a]	1.04 (0.69–1.58)	0.842	154
MZ pairs	1.19 (0.78–1.83)	0.444	156
Percent body fat (%)			
Total sample[b]	1.15 (1.01–1.32)	0.047*	682
MZ and DZ pairs	1.27 (0.93–1.75)	0.138	256
DZ pairs[a]	1.41 (0.88–2.26)	0.149	130
MZ pairs	1.23 (0.78–1.94)	0.369	126
Waist circumference (cm)			
Total sample[b]	1.06 (0.93–1.22)	0.378	689
MZ and DZ pairs	0.84 (0.62–1.15)	0.277	264
DZ pairs[a]	0.83 (0.54–1.26)	0.374	134
MZ pairs	0.89 (0.56–1.42)	0.638	130
Waist-hip ratio			
Total sample[b]	1.02 (0.89–1.17)	0.779	677
MZ and DZ pairs	0.67 (0.47–0.94)	0.022*	264
DZ pairs[a]	0.59 (0.35–0.98)	0.040*	134
MZ pairs	0.77 (0.48–1.25)	0.289	130

OR odds ratio, *CI* confidence interval, *MZ* monozygotic, *DZ* dizygotic, *n* number of participants in each analytical step

* Statistically significant $p < 0.05$

[a] Adjusted for leisure physical activity

[b] Adjusted for smoking and leisure physical activity

Waist-hip ratio

No association was found between waist–hip ratio and lifetime prevalence of chronic LBP for the total sample analysis. Waist–hip ratio was associated with chronic LBP in the within-pair case–control analysis with MZ and DZ twins included (OR 0.67; 95 % CI 0.47–0.94). When the analyses were performed separately for zygosity, a stronger association was found for DZ twins (OR 0.59; 95 % CI 0.35–0.98) and the association disappeared when the analysis was conducted for MZ twins (OR 0.77; 95 % CI 0.48–1.25).

Discussion

To our knowledge, this is the first study investigating the obesity–LBP relationship in females that considered not only traditional measures of obesity such as BMI but also measures of body fat distribution and explored the effects of genetics and early shared environment. We found that lifetime prevalence of chronic LBP was weakly associated with measures of obesity. However, the association was no longer present after the full adjustment for genetics and early shared environmental factors in MZ twins dissimilar for LBP status. These results suggest that a causal direct link between obesity and chronic LBP is unlikely.

BMI and percent body fat

Our results demonstrated that lifetime prevalence of chronic LBP was associated with BMI and percent body fat when the total sample of twins (with no adjustment for genetics or early shared environment among twins) was used. The association was small (OR 1.1 and OR 1.2 for BMI and percent body fat, respectively), but in agreement with previous cross-sectional studies [21–23].

Obesity and LBP are complex traits resulting from multiple interactions between genetic and environmental factors. For example, 35–60 % of the body fat [24], and 67 % of LBP [25] variances can be accounted for by the transmission of genetic and familial environmental factors. Consequently, the true extent of the effect of obesity on LBP is difficult to estimate, and for a clear and more direct identification of obesity–LBP relationship other factors, including familial factors, should be considered.

We found that after the adjustment for familial factors, using the within-pair twin case–control design, the significant association between chronic LBP and both BMI and percent body fat did not persist. This pattern of attenuated associations after controlling for familial factors is in agreement with the previous twin studies that investigated the obesity and LBP relationship using a within-pair twin

case–control design [10, 21]. It suggests that genetics and common shared environment facts play an important role when measures such as BMI and percent body fat are investigated as part of the obesity–LBP relationship.

Waist circumference and waist–hip ratio

Our results did not reveal any association between waist circumference and lifetime prevalence of chronic LBP for the total sample analysis or for any of the within-pair case–control analyses. In addition, no association was found between waist–hip ratio and lifetime prevalence of chronic LBP for the total sample analysis. However, the within-pair twin case–control analysis, with both MZ and DZ twins included, showed a significant inverse relationship between waist–hip ratio and chronic LBP (OR 0.7; 95 % CI 0.5–0.9), meaning that women with greater waist–hip ratios had lower estimates of prevalence of chronic LBP.

When we sequentially separated the analysis for DZ and then MZ twins pairs, even though the pattern of association remained the same (inverse relationship between waist–hip ratio and chronic LBP), the association was significant only in DZ twins (OR 0.6; 95 % CI 0.4–1.0), disappearing after the full adjustment for genetic factors in the MZ twins (OR 0.8; 95 % CI 0.5–1.3). The pattern of a significant relationship being observed in the within-pair twin case–control analysis and in the within-pair twin DZ only case–control analysis but not in the total sample analysis indicates that when genetics and early shared environment among twins are considered, a relationship between waist–hip ratio and chronic LBP is stronger and possibly more direct. This association is attenuated and was not statistically significant when only MZ twins are analyzed (OR 0.8; early shared environment and genetic component are fully controlled for) as opposed to DZ twins only (OR 0.6; early shared environment fully controlled for but genetic component only partially controlled for). Although there is not a clear explanation for this effect, this finding could point to the possibility that when a full adjustment for genetics and early environment is implemented, the significant relationship initially observed in DZ twins disappears. Thus, genetics could be responsible for the possible relationship between waist–hip ratio and chronic LBP.

The direction of the relationship between waist–hip ratio and chronic LBP were somewhat unexpected. Our findings suggest that chronic LBP prevalence is smaller in women with a higher waist–hip ratio. Thus, those individuals with greater hip circumferences (accumulation of fat and corresponding weight around the hip bones) were more likely to have chronic LBP. Our results are in agreement with a population-based cross-sectional study in middle-aged women, which showed that even after the adjustment of many possible confounder factors such as work-related and physical activity, high waist-to-hip ratio was still inversely associated with the risk of severe LBP [26]. However, this study did not control for genetics factors and early shared environment. These findings are in contrast to other earlier studies that used samples of women from the general population where greater levels of central obesity were associated with LBP [15, 16]. From a biomechanical perspective, it is plausible that greater fat mass around the waist area loads the spine through gravity. However, it is also mechanically plausible that greater hip to waist mass could potentially unbalance the forces around the spine, leading to lumbar–pelvic instability and LBP [27]. It is important to note that the assessment of waist–hip ratio not only incorporates the distribution of fat but also is a reflection of bony anatomical features such as the shape of the pelvis. However, it is possible that the results found in the intermediate analytical steps are a result of genetics confounding the association between waist–hip ratio and chronic LBP. It is important to note that this finding was only present in the intermediate analytical steps (MZ/DZ analysis and DZ analysis) where DZ pairs are included, and consequently 50 % of genetics are not controlled for.

Limitations

We acknowledge several limitations in our study design. Firstly this was a cross-sectional analysis, which limits possible insights on a causation path between the variables of interest. Secondly, the measure of chronic LBP used in this study was somewhat simple and did not include additional assessments of the severity, frequency, as well as disability levels associated with LBP. This assessment of LBP might have influenced the results as patients' understanding of what constitutes chronic LBP and degrees or patterns of chronicity may vary. Thirdly, the accuracy of BMI data could have been affected by the combination of self-reported with a direct measure of weight and height used to calculate BMI given that self-reported assessment methods seem to underestimate weight and overestimate height values [28]. In our sample, the difference between the subgroups for BMI data, measured (27.53 kg/m^2) and self-reported (26.58 kg/m^2), was 0.96 kg/m^2. We have tested the association between LBP and both subgroups for BMI for all analytical steps, and the difference in OR found were very small ($\neq$OR < 0.2) and clinically not significant. Therefore, we believe that the combination of self-reported with a direct measure of weight and height have little or no effect in our results.

Also, in spite of being a practical and widely used assessment method, uncertainty has been raised regarding the validity and reliability of bioelectrical impedance measurements for estimation of body fat [29]. However, we should take into account the homogeneous character of our

sample and the measurement conditions, and the fact that the objective of this study is not to determine the exact value of body fat percentage but analyzing its possible association with LBP. Consequently, we believe that the method of body fat measurement does not have a relevant effect on our results and conclusions. Lastly, we recognize that the smaller sample size in the case–control analyses reduced the power required to identify a relationship between obesity-related measures and chronic LBP, if in fact, the relationship exists. Therefore, we cannot exclude that a much larger twin sample would show a significant result. Although smaller samples and larger confidence intervals observed in the case–control analyses could add uncertainty to the results of our study, the imprecision of the data is unlikely to be the explanation for this finding. Firstly, the average values between LBP and non LBP groups are very small for all variables in all analysis steps. Furthermore, the magnitude of the ORs for the significant predictors in the total sample (BMI and percent body fat) is still similar to the all within-pair twin case–control analyses, which points to the fact that the association between obesity-related measures and chronic LBP in fact was weak and non-reliable when twins were considered as individuals. Secondly, according to a systematic review recently published, the reduced association between LBP and obesity seems to be consistent across studies when genetics and the environment factors are considered [30]. Pooled results of two MZ case–control studies with greater sample sizes than ours, 442 [10] and 413 [21] pairs, has shown no association between obesity and LBP (OR 1.4; 95 % CI 0.8–2.3) [30]. Notwithstanding, this study represents a step forward in the investigation of the relationship between obesity and chronic LBP relationship because we used a comprehensive assessment of obesity that accounts for body fat distribution and employed a within-pair case–control design to allow for more direct and precise estimates of obesity–LBP relationship.

In summary, BMI, percent of fat mass and greater depositions of fat and mass around the hips are associated with increases in chronic LBP prevalence in women. However, these associations are small and disappear with the full adjustment for genetics and early shared environment effects. Therefore, our results do not support a causal direct relationship between obesity and chronic LBP. We advocate that the results observed in this study should be tested in the future in a longitudinal twin research design.

Acknowledgments The authors would like to acknowledge the participation of the twins and the assistance of staffs and researchers from the Murcia Twin Registry for the implementation of this study. We also acknowledge the financial support to the Murcia Twin Registry by the Fundación Séneca, Regional Agency for Science and Technology, Murcia, Spain (08633/PHCS/08 and 15302/PHCS/10) and Ministry of Science and Innovation, Spain (PSI11560–2009). Funding for this project has been also received from Fundación MAPFRE (2012). MLF is a Sydney Medical Foundation Fellow. ABD is supported by the National Council for Scientific and Technological Development (CNPq) program "Science without Borders"—Brazil.

Conflict of interest None.

References

1. Haslam DWJW (2005) Obesity. Lancet 336(1):1197–1209
2. Vismara LMF, Zaina F, Galli M, Negrini S, Capodaglio P (2010) Effect of obesity and low back pain on spinal mobility: a cross sectional study in women. J Neuroeng Rehabil 7(3):1–8. doi:10.1186/1743-0003-7-3
3. Guh DPZW, Bansback N, Amarsi Z, Birmingham CL, Anis AH (2009) The incidence of co-morbidities related to obesity and overweight: a systematic review and meta-analysis. BMC Public Health 9(88):1–20. doi:10.1186/1471-2458-9-88
4. Murray CJL, Vos T, Lozano R, Naghavi M, Flaxman AD (2012) Disability-adjusted life years (DALYs) for 291 diseases and injuries in 21 regions, 1990–2010: a systematic analysis for the Global Burden of Disease Study 2010. Lancet 380(9859):2197–2223
5. McBeth JJK (2007) Epidemiology of chronic musculoskeletal pain. Best Pract Res Clin Rheumatol 21(3):403–425. doi:10.1016/j.berh.2007.03.003
6. Leboeuf-Yde C (2000) Weight and low back pain a systematic literature review of 56 journal articles reporting on 65 epidemiologic studies. Spine 25(2):226–237
7. Junqueira DR, Ferreira ML, Refshauge K, Maher CG, Hopper JL, Hancock M, Carvalho MG, Ferreira PH (2014) Heritability and lifestyle factors in chronic low back pain: results of the Australian Twin Low Back Pain Study (The AUTBACK study). Eur J Pain. doi:10.1002/ejp.506
8. Mirtz TAGL (2005) Is obesity a risk factor for low back pain? An example of using the evidence to answer a clinical question. Chiropr Osteopat 13(2):1–6. doi:10.1186/1746-1340-13-2
9. Shiri RKJ, Leino-Arjas P, Solovieva S, Viikari-Juntura E (2009) The association between obesity and low back pain: a meta-analysis. Am J Epidemiol. doi:10.1093/aje/kwp356
10. Leboeuf-Yde CKK, Bruun NH (1999) Low back pain and life style. Part II—obesity. Spine 24(8):779–784
11. Tsuritani I, Honda R, Noborisaka Y, Ishida M, Ishizaki M, Yamada Y (2002) Impact of obesity on musculoskeletal pain and difficulty of daily movements in Japanese middle-aged women. Maturitas 42(1):23–30
12. Lee CY, Kratter R, Duvoisin N, Taskin A, Schilling J (2005) Cross-sectional view of factors associated with back pain. Int Arch Occup Environ Health 78(4):319–324. doi:10.1007/s00420-004-0589-z
13. Deere KCCJ, Holliday K, McBeth J, Crawley EM, Sayers A, Palmer SDR, Clark EM, Tobias JH (2012) Obesity is a risk factor for musculoskeletal pain in adolescents: findings from a population-based cohort. Pain 123(2012):1932–1938. doi:10.1016/j.pain.2012.06.006
14. Perry AC, Applegate EB, Allison MD, Jackson ML, Miller PC (1998) Clinical predictability of the waist-to-hip ratio in assessment of cardiovascular disease risk factors in overweight, premenopausal women. Am J Clin Nutr 68(5):1022–1027
15. Toda YSNTTMTOR (2000) Lean body mass and body fat distribution in participants with chronic low back pain. Arch Intern Med 160(21):3265–3269. doi:10.1001/archinte.160.21.3265
16. Han TS, Schouten JS, Lean ME, Seidell JC (1997) The prevalence of low back pain and associations with body fatness, fat

distribution and height. Int J Obes Relat Metab Disord 21(7):600–607
17. Leboeuf-Yde C, Nielsen J, Kyvik K, Fejer R, Hartvigsen J (2009) Pain in the lumbar, thoracic or cervical regions: do age and gender matter? A population-based study of 34,902 Danish twins 20–71 years of age. BMC Musculoskelet Disord 10(1):1–12. doi:10.1186/1471-2474-10-39
18. Weimer MB, Macey TA, Nicolaidis C, Dobscha SK, Duckart JP, Morasco BJ (2013) Sex differences in the medical care of va patients with chronic non-cancer pain. Pain Med. doi:10.1111/pme.12177
19. Ordonana JR, Rebollo-Mesa I, Carrillo E, Colodro-Conde L, Garcia-Palomo FJ, Gonzalez-Javier F, Sanchez-Romera JF, Aznar Oviedo JM, de Pancorbo MM, Perez-Riquelme F (2013) The Murcia Twin Registry: a population-based registry of adult multiples in Spain. Twin Res Hum Genet 16(1):302–306. doi:10.1017/thg.2012.66
20. Brian S. Everitt DH (2005) Encyclopedia of statistics in behavioral science, vol 1, 1st edn. Wiley
21. Hestbaek L, Leboeuf-Yde C, Kyvik KO (2006) Are lifestyle-factors in adolescence predictors for adult low back pain? A cross-sectional and prospective study of young twins. BMC Musculoskelet Disord. doi:10.1186/1471-2474-7-27
22. Leboeuf-Yde C, Kyvik KO, Bruun NH (1999) Low back pain and lifestyle. Part II—obesity. Information from a population-based sample of 29,424 twin subjects. Spine 24(8):779–783 **(Discussion 783–774)**
23. Livshits G, Popham M, Malkin I, Sambrook PN, MacGregor AJ, Spector T, Williams FMK (2011) Lumbar disc degeneration and genetic factors are the main risk factors for low back pain in women: the UK Twin Spine Study. Ann Rheum Dis 70(10):1740–1745. doi:10.1136/ard.2010.137836
24. Chaput JP, Perusse L, Despres JP, Tremblay A, Bouchard C (2014) Findings from the Quebec Family Study on the etiology of obesity: genetics and environmental highlights. Curr Obes Rep 3:54–66. doi:10.1007/s13679-013-0086-3
25. Ferreira PHBP, Maher CG, Hopper JL, Ferreira ML (2013) Nature or nurture in low back pain? Results of a systematic review of studies based on twin samples. Eur J Pain 1(1):1–15. doi:10.1002/j.1532-2149.2012.00277
26. Yip YB, Ho SC, Chan SG (2001) Tall stature, overweight and the prevalence of low back pain in Chinese middle-aged women. Int J Obes 25(6):887–892. doi:10.1038/sj.ijo.0801557
27. O'Sullivan PB (2000) Lumbar segmental 'instability': clinical presentation and specific stabilizing exercise management. Man Ther 5(1):2–12. doi:10.1054/math.1999.0213
28. Gorber SC, Tremblay M, Moher D, Gorber B (2007) A comparison of direct vs. self-report measures for assessing height, weight and body mass index: a systematic review. Obes Rev 8(4):307–326. doi:10.1111/j.1467-789X.2007.00347.x
29. Talma H, Chinapaw MJ, Bakker B, HiraSing RA, Terwee CB, Altenburg TM (2013) Bioelectrical impedance analysis to estimate body composition in children and adolescents: a systematic review and evidence appraisal of validity, responsiveness, reliability and measurement error. Obes Rev Off J Int Assoc Study Obes 14(11):895–905. doi:10.1111/obr.12061
30. Dario AB, Ferreira ML, Refshauge K, Lima T, Ordonana JR, Ferreira PH (2015) The relationship between obesity, low back pain and lumbar disc degeneration when genetics and the environment are considered: a systematic review of twin studies. Spine J. doi:10.1016/j.spinee.2015.02.001