

Relationship Between Obesity and Lumbar Spine Degeneration: A Cross-Sectional Study from the Fifth Korean National Health and Nutrition Examination Survey, 2010–2012

Sang Yoon Lee, MD, PhD,¹ Won Kim, MD, MS,² Shi-Uk Lee, MD, PhD,¹ and Kyoung Hyo Choi, MD, PhD²

Abstract

Background: Although several studies have shown that obesity affects low back pain (LBP), the relationship between degenerative lumbar spine (LSD) and obesity has not been fully investigated. This study evaluated whether obesity is independently associated with LSD in the general population.

Methods: This cross-sectional study used public data from the Fifth Korean National Health and Nutrition Examination Survey (2010–2012). Subjects aged ≥ 50 years who had completed surveys were included (3668 men and 4966 women). Obesity was classified based on the body mass index, and LSD was assessed by lumbar spine radiographs. Independent associations of obesity with LSD or LBP were determined using odds ratios (OR) adjusted by two regression models.

Results: The prevalence of obesity was more frequent in women than in men (38.27% vs. 33.97%, $P < 0.001$). Compared with normal weight women, the risk of LSD was increased in overweight and obese women following adjustments [OR = 1.227, 95% confidence interval (CI): 1.019–1.477; OR = 1.217, 95% CI: 1.024–1.446, respectively]. When obesity was subdivided, the obese II group showed higher odds for LSD in women (OR = 1.797, 95% CI: 1.287–2.510). However, obesity was not correlated with LSD in men. There was no significant association between obesity and LBP in either men or women.

Conclusions: Compared with normal weight women, LSD risk was higher in overweight and obese women, especially those in the obese II subgroup. These findings suggest that maintaining normal body weight may be a preventative factor of LSD.

Keywords: obesity, spondylosis, spine, low back pain, cross-sectional studies

Introduction

OBESITY IS AN IMPORTANT and modifiable risk factor for several disorders of the hip, knee, ankle, and foot.¹ It can also affect the onset and progression of musculoskeletal diseases.² Osteoarthritis (OA) is a disease that is correlated with obesity. The association between OA and obesity is based on intuitive biomechanical theory. As body weight increases so does the cartilage wear.³ In addition to biomechanical effects, the systemic effects of obesity are associated with arthritis because there is an increased incidence of OA of the hand (nonweight bearing joints) in obese patients compared with that of nonobese populations.⁴

Several proinflammatory cytokines from adipose tissue have been linked to OA. Among these cytokines, leptin plays an important role in the pathophysiology of OA.⁵

Although not studied to the same extent as OA, correlations between obesity and low back pain (LBP) have also been described. A large population-based study in Norway including 63,968 subjects reported that a high body mass index (BMI) was associated with an increased prevalence of LBP after adjusting for age.⁶ One recent meta-analysis indicated that the odds ratios (ORs) for LBP among overweight and obese individuals compared with those of normal weight individuals were 1.15 [95% confidence interval (CI): 1.08–1.21] and 1.36 (95% CI: 1.18–1.57),

¹Department of Rehabilitation Medicine, Seoul National University College of Medicine, SMG-SNU Boramae Medical Center, Seoul, Republic of Korea.

²Department of Rehabilitation Medicine, Asan Medical Center, University of Ulsan College of Medicine, Seoul, Republic of Korea.

respectively.⁷ However, unlike OA, the etiologic relationship between obesity and LBP has not been fully studied. In particular, the relationship between degenerative changes (spondylosis) in the lumbar spine and obesity has not been investigated. Therefore, this study aimed to delineate whether obesity is independently associated with lumbar spine degeneration (LSD) in the general population. To identify an independent relationship with obesity, our analysis was adjusted by age group, smoking status, alcohol consumption, level of education, and levels of physical activity (PA), all variables that could affect LSD. We hypothesized that obesity would show a higher correlation with LSD.

Materials and Methods

Data source and study population

The data in this study were obtained from the Fifth Korean National Health and Nutrition Examination Survey (KNHANES) conducted from 2010 to 2012 by the Korea Centers for Disease Control and Prevention. The KNHANES is a population-based cross-sectional survey designed to assess health-related behavior, health conditions, and the nutritional state of Koreans (<http://knhanes.cdc.go.kr/>).⁸ The Fifth KNHANES was a nationwide, cross-sectional survey of 25,533 people in 11,520 households. The study used a stratified, multistage, probability-sampling method to select study subjects. Sampling weights were used to represent the entire Korean population. From this pool of data, we included subjects aged ≥ 50 years who had completed surveys on body weight and height; radiographs of the lumbar spine; and a

questionnaire on LBP, smoking, alcohol consumption, educational status, and level of PA. Subjects considered underweight according to BMI $< 18.5 \text{ kg/m}^2$ were excluded. Finally, 3668 men and 4966 women were included in this study (Fig. 1). All participants provided written informed consent. This study was approved by the institutional review board (IRB) of our hospital (IRB No. 2018-0185).

LSD assessment: primary outcome

LSD was assessed by radiographs of the lumbar spine. Anterior–posterior and lateral plain radiographs of the lumbar spine were taken using an SD 3000 Synchro Stand (Accele Ray SYFM Co., Seoul, Korea). The radiographs were centered at the umbilicus in the anterior–posterior view and the iliac crest in the lateral view. Two radiologists evaluated the presence of LSD. A modified version of the Kellgren–Lawrence grading system was used to evaluate LSD as follows: grade 0, normal; grade 1, definite osteophytes; and grade 2, joint or intervertebral space narrowing, bone sclerosis, and large osteophytes.^{9,10} LSD was defined if at least one level of the lumbar spine was scored as grade 2.

LBP assessment: secondary outcome

The presence of LBP was evaluated using the following direct question: “Have you experienced low back pain for more than one month during the last three months?” If the subjects answered “yes,” LBP was considered to be present.

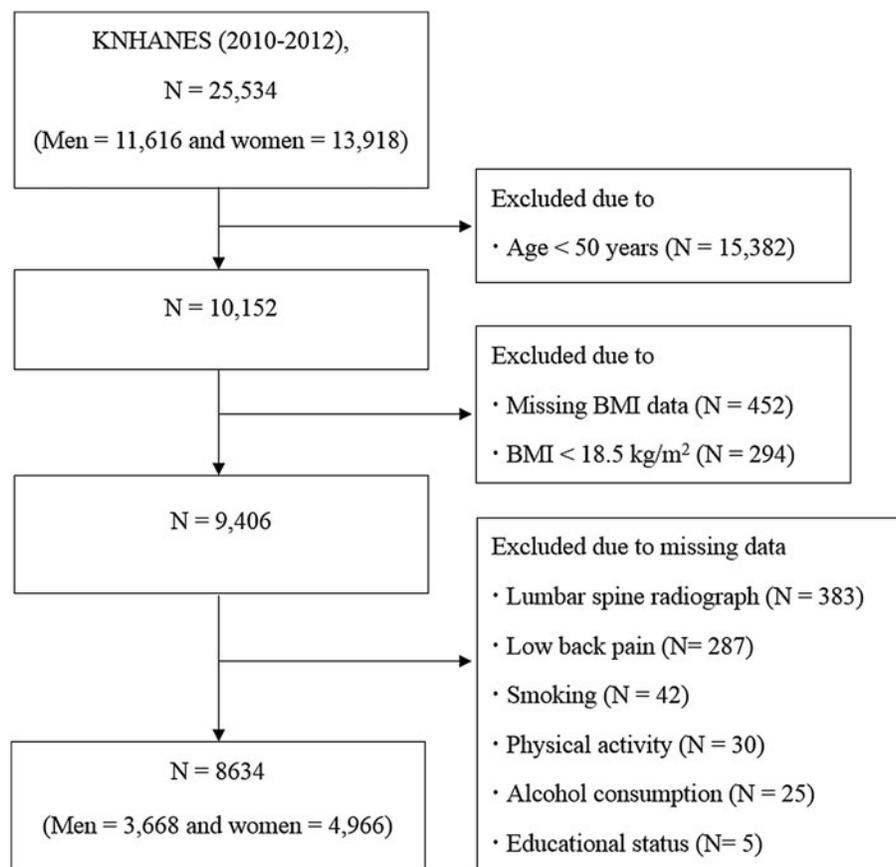


FIG. 1. Flow diagram of study participants. BMI, body mass index; KNHANES, Korean National Health and Nutrition Examination Survey.

Obesity classification: exposure variables

Obesity was defined and classified using the criteria for the Asia-Pacific region as follows: normal weight (BMI 18.5–22.9 kg/m²), overweight (BMI 23–24.9 kg/m²), and obese (BMI ≥25 kg/m²).¹¹ Obesity was subdivided into obese I (BMI 25–29.9 kg/m²) and obese II (BMI ≥30 kg/m²) groups.

Confounders and effect modifiers

Age group, smoking status, alcohol consumption, education, and PA were included in the analysis as potential confounders. Age and PA are well-known risk factors of LBP.^{12,13} Education level, which influences occupation and lifestyle, has also been reported as an affecting factor of LBP.^{12,14} Smoking and alcohol consumption are important risk factors of general health, and several studies have also suggested an association with LBP.^{14–16} The subjects were categorized into four age groups: 50–59, 60–69, 70–79, or ≥80 years. Smoking status was divided into current smokers or nonsmokers. Alcohol consumption was divided into “drinking biweekly or more” or “once a week or less.” Education levels were categorized as elementary school graduate or below, middle school graduate, high school graduate, or college graduate or above. PA was assessed using the Korean version of the International Physical Activity Questionnaire-Short Form (IPAQ-SF), which assessed three activities: vigorous activity, moderate-intensity activity, and walking.^{17,18} PA was defined as follows: vigorous PA, vigorous activity of at least 20 min for ≥3 days per week; or moderate PA, moderate-intensity activity of at least 30 min for ≥5 days per week.¹⁷

Statistical analysis

The subjects' characteristics for men and women were compared by Student's *t*-test for continuous variables and chi-squared tests for categorical variables. Univariate and multivariate logistic regression analyses were performed to evaluate the effects of obesity on LSD and LBP. All logistic regression analyses were performed separately for each sex. Only those who completed all surveys and examinations were included in the regression analysis. Obesity was included in the analyses as three (normal weight, overweight, and obese) or four categories (normal weight, overweight, and obese I and II). Model 1 was adjusted by age group. Model 2 was adjusted by age group, smoking, alcohol consumption, education, and levels of PA. Sampling weights were used for each participant's data to represent the entire Korean population. Statistical analyses were performed using SAS, version 9.4 (SAS Institute, Cary, NC).

Results

Clinical characteristics of the study subjects

The prevalence of obesity was higher in women than in men (38.27% vs. 33.97%; *P* < 0.001), especially in the obese II group (5.00% vs. 1.22%). LSD was also more frequent in women than in men (34.27% vs. 26.18%; *P* < 0.001). LBP occurred 2.4 times more frequently among women than among men (31.93% vs. 13.52%; *P* < 0.001) (Table 1). LSD was more frequent in obese II (50.4%) than in normal weight (33.9%) women. However, LSD was less frequent in

obese II (18.2%) than in normal weight (33.6%) men (Supplementary Table S1; Supplementary Data are available online at www.liebertpub.com/met).

Association between obesity and the risk of LSD

Univariate analysis of the risk of LSD using three obesity categories in men revealed lower OR in the overweight (OR = 0.755, 95% CI: 0.609–0.935) and obese (OR = 0.799, 95% CI: 0.648–0.986) groups compared with the normal weight group. However, these associations were not significant in multivariate analyses. In women, the risk of LSD was increased in the overweight and obese groups in multivariate analyses. After adjusting for age (Model 1), the ORs were 1.248 (95% CI: 1.039–1.499) in the overweight and 1.253 (95% CI: 1.057–1.485) in the obese groups. After adjusting for all other potential confounding factors (Model 2), the ORs were 1.227 (95% CI: 1.019–1.477) in the overweight and 1.217 (95% CI: 1.024–1.446) in the obese groups (Table 2).

Analyses based on four categories in men revealed a lower OR in the overweight (OR = 0.755, 95% CI: 0.609–0.935) and obese II (OR = 0.395, 95% CI: 0.157–0.996) groups only in univariate analysis; the associations were insignificant in multivariate analyses. In women, the risk of LSD was higher in the obese II group compared with the normal weight group in all analysis models (univariate OR = 1.826, 95% CI: 1.321–2.522; Model 1 OR = 1.853, 95% CI: 1.333–2.577; Model 2 OR = 1.797, 95% CI: 1.287–2.510). In addition, the risks of LSD were increased in the overweight group in Model 1 (OR = 1.248, 95% CI:

TABLE 1. SUBJECT CHARACTERISTICS

	Men (n = 3668)	Women (n = 4966)	P
Age (years)	61.1 ± 0.2	62.8 ± 0.2	<0.0001
Weight (kg)	67.1 ± 0.2	57.7 ± 0.2	<0.0001
Height (cm)	167.1 ± 0.1	153.6 ± 0.1	<0.0001
BMI (kg/m ²)	24.0 ± 0.1	24.4 ± 0.1	<0.0001
Obesity classification			
Normal weight	1394 (36.0)	1751 (35.2)	<0.0001
Overweight	1064 (30.0)	1312 (26.5)	
Obese I	1166 (32.8)	1665 (33.3)	
Obese II	44 (1.2)	238 (5.0)	
Lumbar spine degeneration	1121 (26.2)	1824 (34.3)	<0.0001
Low back pain	527 (13.5)	1645 (31.9)	<0.0001
Current smoker	1138 (35.6)	176 (4.6)	<0.0001
Alcohol drinking (biweekly or more)	1450 (42.7)	265 (6/1)	<0.0001
Education			
≤Elementary school	1156 (30.0)	2952 (59.2)	<0.0001
Middle school	707 (20.9)	785 (17.2)	
High school	1093 (31.3)	920 (18.1)	
≥College	712 (17.8)	309 (5.6)	
PA			
Vigorous PA	557 (16.0)	467 (9.4)	<0.0001
Moderate PA	335 (9.2)	444 (8.6)	0.4425
Walking	1552 (41.1)	1713 (34.5)	<0.0001

Values are expressed as the mean ± standard error or as numbers (%). *P* values were calculated using Student's *t*-test or chi-squared test. BMI, body mass index; PA, physical activity.

TABLE 2. ODDS RATIOS FOR LUMBAR SPINE DEGENERATION ACCORDING TO NORMAL WEIGHT, OVERWEIGHT, AND OBESE GROUPS

	Univariate analysis			Model 1 ^a			Model 2 ^b		
	OR	95% CI	P	OR	95% CI	P	OR	95% CI	P
Men									
Normal	Ref.			Ref.			Ref.		
Overweight	0.755	0.609–0.935	0.010	0.915	0.724–1.155	0.453	0.916	0.725–1.159	0.465
Obese	0.799	0.648–0.986	0.036	0.988	0.790–1.237	0.919	0.998	0.794–1.255	0.989
Women									
Normal	Ref.			Ref.			Ref.		
Overweight	1.180	0.996–1.398	0.055	1.248	1.039–1.499	0.018	1.227	1.019–1.477	0.031
Obese	1.228	1.042–1.447	0.015	1.253	1.057–1.485	0.009	1.217	1.024–1.446	0.026

P values <0.05 are in *boldface*.

^aModel 1 was adjusted for age group.

^bModel 2 was adjusted for smoking, alcohol consumption, education, and levels of PA. CI, confidence interval; OR, odds ratio.

1.038–1.499) and Model 2 (OR=1.226, 95% CI: 1.019–1.477) (Table 3).

Association between obesity and the risk of LBP

There were no significant associations between obesity and LBP in either the three- or four-category analyses for both men and women. Although higher ORs for LBP were observed in women in the obese II category in univariate and Model 1 and 2 analyses, none were statistically significant (Tables 4 and 5).

Discussion

The most important finding of this study was the increased risk of LSD in overweight and obese women compared with that in normal weight women. Analysis based on four obesity categories revealed significant correlations to LSD in the obese II group of women. There were obvious sex differences, and obesity was not correlated with LSD in men. There were no significant associations between obesity and LBP in both men and women.

Obesity and LSD were significantly associated among women in this study and not among men. Shiri et al. also suggested that the association between overweight or obe-

sity and LBP is stronger in women than in men.¹⁹ With regard to joint degeneration, women are also more likely than men to suffer hip and knee joint pain.²⁰ These sex differences can be explained by several mechanisms. Although obesity has usually been defined by the BMI, derived from simple mass (weight) and height measurements, body composition differs between men and women, with a higher fat mass in women compared with men.²¹ Thus, if the weight of a man and a woman is the same, the effect of the fat mass will be higher in women. Fat mass may play a role in the systemic effects of obesity rather than the biomechanical effects. A population-based study also reported that the risk of LBP increased with higher fat mass but not with fat-free mass.²² Differences in hormonal influences in men and women are another mechanism that may explain the sex difference in LSD. Sex hormones play a significant role in the prevalence of osteoporosis in both men and women, and osteoporosis is an important risk factor for LSD.²³ This difference in sex hormones may have caused the LSD differences observed in men and women.

In this study, we classified obesity into three categories, and also analyzed “obese” status after further dividing obesity into two categories (obese I and II) according to the BMI standards. In the three-category analyses, both

TABLE 3. ODDS RATIOS FOR LUMBAR SPINE DEGENERATION ACCORDING TO NORMAL WEIGHT, OVERWEIGHT, OBESE I, AND OBESE II CATEGORIES

	Univariate analysis			Model 1 ^a			Model 2 ^b		
	OR	95% CI	P	OR	95% CI	P	OR	95% CI	P
Men									
Normal	Ref.			Ref.			Ref.		
Overweight	0.755	0.609–0.935	0.010	0.915	0.724–1.155	0.453	0.917	0.725–1.159	0.466
Obese I	0.817	0.661–1.009	0.061	1.009	0.805–1.265	0.937	1.018	0.809–1.283	0.877
Obese II	0.395	0.157–0.996	0.049	0.501	0.192–1.310	0.159	0.524	0.202–1.360	0.183
Women									
Normal	Ref.			Ref.			Ref.		
Overweight	1.180	0.996–1.398	0.055	1.248	1.038–1.499	0.018	1.226	1.019–1.477	0.031
Obese I	1.153	0.972–1.367	0.101	1.178	0.987–1.406	0.069	1.143	0.956–1.368	0.142
Obese II	1.826	1.321–2.522	<0.001	1.853	1.333–2.577	<0.001	1.797	1.287–2.510	<0.001

P values <0.05 are in *boldface*.

^aModel 1 was adjusted for age group.

^bModel 2 was adjusted for smoking, alcohol consumption, education, and levels of PA.

TABLE 4. ODDS RATIOS FOR LOW BACK PAIN ACCORDING TO NORMAL WEIGHT, OVERWEIGHT, AND OBESE CATEGORIES

	<i>Univariate analysis</i>			<i>Model 1^a</i>			<i>Model 2^b</i>		
	<i>OR</i>	<i>95% CI</i>	<i>P</i>	<i>OR</i>	<i>95% CI</i>	<i>P</i>	<i>OR</i>	<i>95% CI</i>	<i>P</i>
Men									
Normal	Ref.			Ref.			Ref.		
Overweight	1.129	0.846–1.506	0.410	1.228	0.918–1.642	0.166	1.283	0.957–1.718	0.095
Obese	0.805	0.619–1.046	0.104	0.878	0.670–1.151	0.346	0.936	0.717–1.223	0.628
Women									
Normal	Ref.			Ref.			Ref.		
Overweight	0.880	0.724–1.070	0.199	0.882	0.722–1.077	0.218	0.857	0.697–1.054	0.144
Obese	1.046	0.891–1.227	0.582	1.036	0.877–1.224	0.680	0.982	0.829–1.164	0.838

^aModel 1 was adjusted for age group.

^bModel 2 was adjusted for smoking, alcohol consumption, education, and levels of PA.

overweight and obesity were significantly correlated with LSD in women (OR=1.227 and 1.217, respectively). However, in the four-category analysis, “obese I” was not associated with LSD (OR=1.143, 95% CI: 0.956–1.368, *P*=0.142), although “obese II” was significantly correlated with LSD in women (OR=1.797, 95% CI: 1.287–2.510, *P*<0.001). Therefore, when considering the effects of obesity on LSD, it is necessary to confirm the obese II (BMI ≥30 kg/m²) status by dividing the obesity into two sub-categories. The definition of obesity for Asian people was based on the Asia-Pacific region criteria of BMI=25 kg/m², as mentioned above. However, as Asian people’s eating habits have become more westernized, the cutoff point for obesity needs to be increased to BMI=30 kg/m²,²⁴ as recommended by the World Health Organization (WHO).²⁵ In our study, there was no marked difference in the ORs for LSD in the overweight and obese I categories in women. The overweight and obese I groups may not have a linear relationship because several environmental factors, which were not adjusted in our regression model, might have affected the correlations. The effects of obesity on LSD may have prevailed these environmental factors only in the obese II group.

There was no significant association between BMI-based obesity and LBP in our study (both men and women). One recent cross-sectional study of the 2015 National Health Interview Survey with 32,060 respondents reported that the

adjusted ORs of LBP in overweight (OR=1.21, 95% CI: 1.11–1.32) and obese (OR=1.55, 95% CI: 1.44–1.67) subjects were higher than those in normal weight subjects.²⁶ One cohort study with 975 participants reported that obesity was associated with the presence of intervertebral disc degeneration measured by magnetic resonance imaging.²⁷ However, a population-based twin study suggested that the association between BMI and chronic LBP was absent after adjusting for shared environment and genetic factors²⁸; the authors concluded that a direct causal relationship between obesity and chronic LBP could not be supported. Recently, Dario et al. also reported that none of the obesity-related measures, including BMI, increased the risk of chronic LBP with or without adjustment for familial factors.²⁹ The reasons for the contrasting results among these studies included differences in the definition of back pain, population groups, and adjustment variables.

Because our results suggest that obesity II was correlated with LSD, LBP was also expected to have a significant relationship with obesity, but this was not the case. This is because LBP is affected not only by LSD but also by other several factors. In fact, radiological spinal degeneration is not consistent with back pain symptoms.³⁰ In addition, obesity decreases the amount of PA, which can cause pain³¹; thus, a sedentary lifestyle may reduce the incidence of LBP. LBP may also be affected by a variety of other diseases besides LSD. For example, in the case of a compression

TABLE 5. ODDS RATIOS FOR LOW BACK PAIN ACCORDING TO NORMAL WEIGHT, OVERWEIGHT, OBESE I, AND OBESE II CATEGORIES

	<i>Univariate analysis</i>			<i>Model 1^a</i>			<i>Model 2^b</i>		
	<i>OR</i>	<i>95% CI</i>	<i>P</i>	<i>OR</i>	<i>95% CI</i>	<i>P</i>	<i>OR</i>	<i>95% CI</i>	<i>P</i>
Men									
Normal	Ref.			Ref.			Ref.		
Overweight	1.129	0.846–1.506	0.410	1.228	0.918–1.642	0.166	1.283	0.957–1.718	0.095
Obese I	0.818	0.628–1.066	0.138	0.892	0.678–1.173	0.414	0.950	0.725–1.245	0.710
Obese II	0.456	0.148–1.410	0.172	0.505	0.167–1.530	0.227	0.562	0.176–1.794	0.330
Women									
Normal	Ref.			Ref.			Ref.		
Overweight	0.880	0.724–1.070	0.199	0.882	0.722–1.077	0.218	0.857	0.697–1.054	0.144
Obese I	1.013	0.854–1.201	0.881	1.009	0.843–1.206	0.925	0.959	0.799–1.149	0.647
Obese II	1.283	0.953–1.727	0.100	1.226	0.903–1.666	0.192	1.148	0.842–1.565	0.382

^aModel 1 was adjusted for age group.

^bModel 2 was adjusted for smoking, alcohol consumption, education, and levels of PA.

fracture that can cause LBP, obesity increases bone density,³² which may reduce the incidence of compression fractures. Because this was a cross-sectional study, there were limitations on controlling for these factors. Therefore, whether obesity directly affects LBP requires prospective studies that have controlled for the different variables that can cause LBP.

Our study has several limitations: first, it was difficult to establish a causal relationship between obesity and LSD because of the inherent limitations of cross-sectional studies, although this was a nationwide, representative, large population-based study. Subjects with LSD might be physically less active due to pain and become obese. Second, information was obtained through self-reporting; therefore, self-selection and recall biases were possible.³¹ Third, the assessments of LSD and LBP were not precise in this population-based study. For LSD measurements, early degenerative changes, which can be confirmed by magnetic resonance imaging or computed tomography, could not be included since a simple X-ray was performed and grade 2 or more was defined as degeneration in the Kellgren–Lawrence grading system. Because LBP was also evaluated only as the presence of back pain for ≥ 1 month over the last 3 months, it was impossible to evaluate the intensity of pain, and it may not distinguish among the type of LBP.³³ Therefore, further studies with more precise measurements of LSD and LBP are needed. Fourth, we could not include several important variables such as metabolic diseases, which may affect the risk of LSD and LBP, and the control of confounding factors might be imperfect. In addition, sample size of obese II group was too small than other groups. In men, it was only 1.2% of total male population. Thus, relatively small sample size of only one group can raise a bias in this study.³⁴

Conclusions

The risk of LSD was increased in overweight and obese women, especially among those in the obese II category, compared with normal weight women. In men, obesity was not correlated with LSD. There were no statistically significant associations between obesity and LBP in either men or women. These findings imply that maintaining normal body weight may be a factor for the prevention of LSD.

Acknowledgment

This study was supported by a grant (2017-661) from the Asan Institute for Life Sciences, Asan Medical Center, Seoul, Korea.

Author Disclosure statement

No conflicting financial interests exist.

References

- Hootman JM, Macera CA, Ainsworth BE, et al. Association among physical activity level, cardiorespiratory fitness, and risk of musculoskeletal injury. *Am J Epidemiol* 2001;154:251–258.
- Wearing SC, Hennig EM, Byrne NM, et al. Musculoskeletal disorders associated with obesity: A biomechanical perspective. *Obes Rev* 2006;7:239–250.
- Koonce RC, Bravman JT. Obesity and osteoarthritis: More than just wear and tear. *J Am Acad Orthop Surg* 2013;21:161–169.
- Grotle M, Hagen KB, Natvig B, et al. Obesity and osteoarthritis in knee, hip and/or hand: An epidemiological study in the general population with 10 years follow-up. *BMC Musculoskelet Disord* 2008;9:132.
- Dumond H, Presle N, Terlain B, et al. Evidence for a key role of leptin in osteoarthritis. *Arthritis Rheum* 2003;48:3118–3129.
- Heuch I, Hagen K, Heuch I, et al. The impact of body mass index on the prevalence of low back pain: The HUNT study. *Spine (Phila Pa 1976)* 2010;35:764–768.
- Zhang TT, Liu Z, Liu YL, et al. Obesity as a risk factor for low back pain: A Meta-Analysis. *Clin Spine Surg* 2018;31:22–27.
- Park HA. The Korea national health and nutrition examination survey as a primary data source. *Korean J Fam Med* 2013;34:79.
- Kellgren JH, Lawrence JS. Radiological assessment of osteo-arthrosis. *Ann Rheum Dis* 1957;16:494–502.
- Yang S, Kim W, Choi KH, et al. Influence of occupation on lumbar spine degeneration in men: The Korean National Health and Nutrition Examination Survey 2010–2013. *Int Arch Occup Environ Health* 2016;89:1321–1328.
- Lim JU, Lee JH, Kim JS, et al. Comparison of World Health Organization and Asia-Pacific body mass index classifications in COPD patients. *Int J Chron Obstruct Pulmon Dis* 2017;12:2465–2475.
- Hoy D, Brooks P, Blyth F, et al. The Epidemiology of low back pain. *Best Pract Res Clin Rheumatol* 2010;24:769–781.
- Kim W, Jin YS, Lee CS, et al. Relationship between the type and amount of physical activity and low back pain in Koreans aged 50 years and older. *PM R* 2014;6:893–899.
- Hurwitz EL, Randhawa K, Torres P, et al. The Global Spine Care Initiative: A systematic review of individual and community-based burden of spinal disorders in rural populations in low- and middle-income communities. *Eur Spine J* 2017. doi: 10.1007/s00586-017-5393-z.
- Ondrejovicova A, Petrovics G, Svitkova K, et al. Is non-pharmacological treatment an effective option for chronic low back pain? *Neuro Endocrinol Lett* 2017;38:169–172.
- Iizuka Y, Iizuka H, Mieda T, et al. Prevalence of chronic nonspecific low back pain and its associated factors among middle-aged and elderly people: An analysis based on data from a musculoskeletal examination in Japan. *Asian Spine J* 2017;11:989–997.
- Kim W, Chung SG, Kim K, et al. The relationship between body fat and bone mineral density in Korean men and women. *J Bone Miner Metab* 2014;32:709–717.
- Craig CL, Marshall AL, Sjostrom M, et al. International physical activity questionnaire: 12-country reliability and validity. *Med Sci Sports Exerc* 2003;35:1381–1395.
- Shiri R, Karppinen J, Leino-Arjas P, et al. The association between obesity and low back pain: A meta-analysis. *Am J Epidemiol* 2010;171:135–154.
- O'Connor MI. Sex differences in osteoarthritis of the hip and knee. *J Am Acad Orthop Surg* 2007;15 Suppl 1:S22–S25.
- Pasco JA, Nicholson GC, Brennan SL, et al. Prevalence of obesity and the relationship between the body mass index and body fat: Cross-sectional, population-based data. *PLoS One* 2012;7:e29580.
- Chou L, Brady SR, Urquhart DM, et al. The association between obesity and low back pain and disability is affected

- by mood disorders: A population-based, cross-sectional study of men. *Medicine (Baltimore)* 2016;95:e3367.
23. Yoshimura N, Muraki S, Oka H, et al. Prevalence of knee osteoarthritis, lumbar spondylosis, and osteoporosis in Japanese men and women: the research on osteoarthritis/osteoporosis against disability study. *J Bone Miner Metab* 2009;27:620–628.
 24. Jih J, Mukherjea A, Vittinghoff E, et al. Using appropriate body mass index cut points for overweight and obesity among Asian Americans. *Prev Med* 2014;65:1–6.
 25. Physical status: The use and interpretation of anthropometry. Report of a WHO Expert Committee. *World Health Organ Tech Rep Ser* 1995;854:1–452.
 26. Peng T, Perez A, Pettee Gabriel K. The Association Among Overweight, Obesity, and Low Back Pain in U.S. Adults: A Cross-Sectional Study of the 2015 National Health Interview Survey. *J Manipulative Physiol Ther* 2018;41:294–303.
 27. Teraguchi M, Yoshimura N, Hashizume H, et al. Prevalence and distribution of intervertebral disc degeneration over the entire spine in a population-based cohort: The Wakayama Spine Study. *Osteoarthritis Cartilage* 2014;22:104–110.
 28. Dario AB, Ferreira ML, Refshauge K, et al. Are obesity and body fat distribution associated with low back pain in women? A population-based study of 1128 Spanish twins. *Eur Spine J* 2016;25:1188–1195.
 29. Dario AB, Loureiro Ferreira M, Refshauge K, et al. Obesity does not increase the risk of chronic low back pain when genetics are considered. A prospective study of Spanish adult twins. *Spine J* 2017;17:282–290.
 30. Borenstein DG, O'Mara JW, Jr., Boden SD, et al. The value of magnetic resonance imaging of the lumbar spine to predict low-back pain in asymptomatic subjects: A seven-year follow-up study. *J Bone Joint Surg Am* 2001;83-A:1306–1311.
 31. Kim H, Min TJ, Kang SH, et al. Association between walking and low back pain in the Korean population: A cross-sectional study. *Ann Rehabil Med* 2017;41:786–792.
 32. Cao JJ. Effects of obesity on bone metabolism. *J Orthop Surg Res* 2011;6:30.
 33. Koes BW, van Tulder M, Lin CW, et al. An updated overview of clinical guidelines for the management of non-specific low back pain in primary care. *Eur Spine J* 2010;19:2075–2094.
 34. Faber J, Fonseca LM. How sample size influences research outcomes. *Dental Press J Orthod* 2014;19:27–29.

Address correspondence to:

Won Kim, MD, MS
 Department of Rehabilitation Medicine
 Asan Medical Center
 University of Ulsan College of Medicine
 88, Olympic-Ro 43-Gil
 Songpa-gu
 Seoul 05505
 Republic of Korea

E-mail: duocl79@gmail.com