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Overweight and obesity as risk factors for chronic low back pain: a new follow-up in the HUNT Study

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Abstract

Background Numerous studies have examined associations between overweight and obesity and risk of low back pain (LBP), but the exact magnitude of these associations is not yet clear. The purpose of this work was to assess such sex-specific associations in a community-based setting in Norway, taking into account potential relationships with other risk factors.

Methods A cohort study was conducted combining data from two waves of the Trøndelag Health Study, HUNT3 (2006–2008) and HUNT4 (2017–2019). Separate analyses were performed of risk of chronic LBP in HUNT4 among 14,775 individuals without chronic LBP in HUNT3, and of recurrence or persistence in HUNT4 among 5034 individuals with chronic LBP in HUNT3. Relative risks were estimated in generalised linear models for overweight and obesity compared to normal weight. Body size classification was based on values of BMI computed from measurements of height and weight. Chronic LBP was defined as LBP persisting at least 3 months during last year.

Results After adjustment for age, smoking, physical activity in leisure time and work activity, analysis of risk among women produced relative risks 1.11 (95% CI 1.00–1.23) for overweight, 1.36 (95% CI 1.20–1.54) for obesity class I and 1.68 (95% CI 1.42–2.00) for obesity classes II-III. Relative risks among men were 1.10 (95% CI 0.94–1.28) for overweight, 1.36 (95% CI 1.13–1.63) for obesity class I and 1.02 (95% CI 0.70–1.50) for obesity classes II-III, the last estimate being based on relatively few individuals. Analyses of recurrence or persistence indicated similar relationships but with smaller magnitude of relative risks and no drop in risk among obesity classes II-III in men. The change in BMI from HUNT3 to HUNT4 hardly differed between individuals with and without chronic LBP in HUNT3.

Conclusions Risk of chronic LBP increases with higher values of BMI in both sexes, although it is uncertain whether this applies to very obese men. Very obese women carry a particularly large risk. Probabilities of recurrence or persistence of chronic LBP among those already afflicted also increase with higher values of BMI. Adjustment for other factors does not influence relationships with overweight and obesity to any major extent.

Keywords Epidemiology, BMI, Low back pain, Risk, Recurrence, HUNT

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Background

Experiencing low back pain (LBP) may involve great hardships for the individuals affected [1], and the disorder can lead to huge costs for society [2]. According to the Global Burden of Disease Study 2021, LBP is the greatest cause of disability in a worldwide perspective [3]. The etiology of LBP is difficult to explore, especially since the majority of cases (about 90%) remain without any definite pathoanatomical cause [4]. To reduce the prevalence of LBP, epidemiological studies are essential to determine which risk factors are important.

Overweight and obesity constitute established risk factors for cardiovascular disease [5], hypertension [6], type 2 diabetes [7], hip and knee osteoarthritis [8] and certain cancers [9]. At the end of the twentieth century, no clear consensus had emerged regarding potential associations between overweight or obesity and occurrence of LBP. The extensive literature review published in 2000 by Leboeuf-Yde [10] did not reach any definite conclusion, and the review of Mirtz and Greene from 2005 [11] was unable to establish any clear relationship between obesity and LBP. However, the comprehensive meta-analysis performed by Shiri et al. in 2010 [12] indicated that overweight and obesity are associated with prevalence of LBP, although a definite association between overweight and future risk could not be established.

At present, it seems to be more widely accepted that overweight and obesity may increase the risk of certain categories of LBP. In a systematic review including 25 longitudinal studies, a higher body weight was found to be a major risk factor for chronic LBP [13]. Another systematic review with different inclusion criteria found moderate evidence that overweight is a risk factor for chronic LBP [14].

There is still uncertainty, however, concerning the strength of the associations between overweight and obesity and occurrence of LBP, especially when adjustment is made for other risk factors [12]. It is also uncertain whether there are substantial differences between women and men in the potential associations [15, 16]. Moreover, not very much is known about potential associations with measures of body size in follow-up of recurrence or persistence of LBP among individuals who already suffer from the disorder [17]. Finally, it is not clear whether there are important interactions between overweight or obesity and other established risk factors for LBP [18–20].

The present study represents a continuation of work published in 2010 and 2013 [15, 16] examining prevalence, risk and recurrence of chronic LBP in association with overweight and obesity. That work was based on data from the second and third survey waves, HUNT2 and HUNT3, of the Trøndelag Health Study (HUNT) [21] carried out in the former Nord-Trøndelag county in Norway. The current study combines data from the third and fourth survey waves, HUNT3 and HUNT4 [22], moving 11 years forward in time, to explore associations between overweight and obesity and subsequent occurrence of chronic LBP. We focus in particular on the strength of the associations and on potential sex differences.

Methods

Basic study design

The 11 year cohort study was based on information collected in the third and fourth survey waves of the Trøndelag Health Study (HUNT) [22], with HUNT3 (2006–2008) representing the start of follow-up and HUNT4 (2017–2019) indicating the end of follow-up. This cohort study only deals with residents of Nord-Trøndelag county as it existed when HUNT3 was carried out. All individuals aged at least 20 years were invited to participate, completing a questionnaire on basic health and social variables. Each person provided information about physical activity in leisure time and at work and about smoking habits. The respondents were also invited to participate in a clinical consultation including measurements of body height and body weight.

One question in the HUNT3 survey was formulated in this way: "During the last year, have you suffered from pain and/or stiffness in your muscles and joints that has lasted for at least 3 consecutive months?" If a participant answered yes, the following question was asked: "Where did you have these complaints?" The participants were requested to mark the location in a figure showing different parts of the body. Respondents who answered yes to the first question and then selected the lower back as a relevant area were regarded as suffering from chronic LBP in HUNT3. The restriction to cases lasting at least 3 months corresponds to the general definition of chronic LBP [23].

In the HUNT4 survey a corresponding procedure was used to elicit information on chronic LBP, although the initial question was formulated in a slightly different manner: "Have you suffered from pain in muscles and joints continuously for at least 3 months during the last year?" Otherwise, the HUNT4 survey collected similar relevant information to HUNT3, and also included a question about duration of education.

Study participation

The target county population in HUNT3 for the present study consisted of 64,596 individuals in the age range 30–69 years. Younger or older individuals were excluded because of low participation rates. Of these, 38,106 individuals attended the HUNT3 survey (Fig. 1). A total of 7779 individuals were excluded from the present study

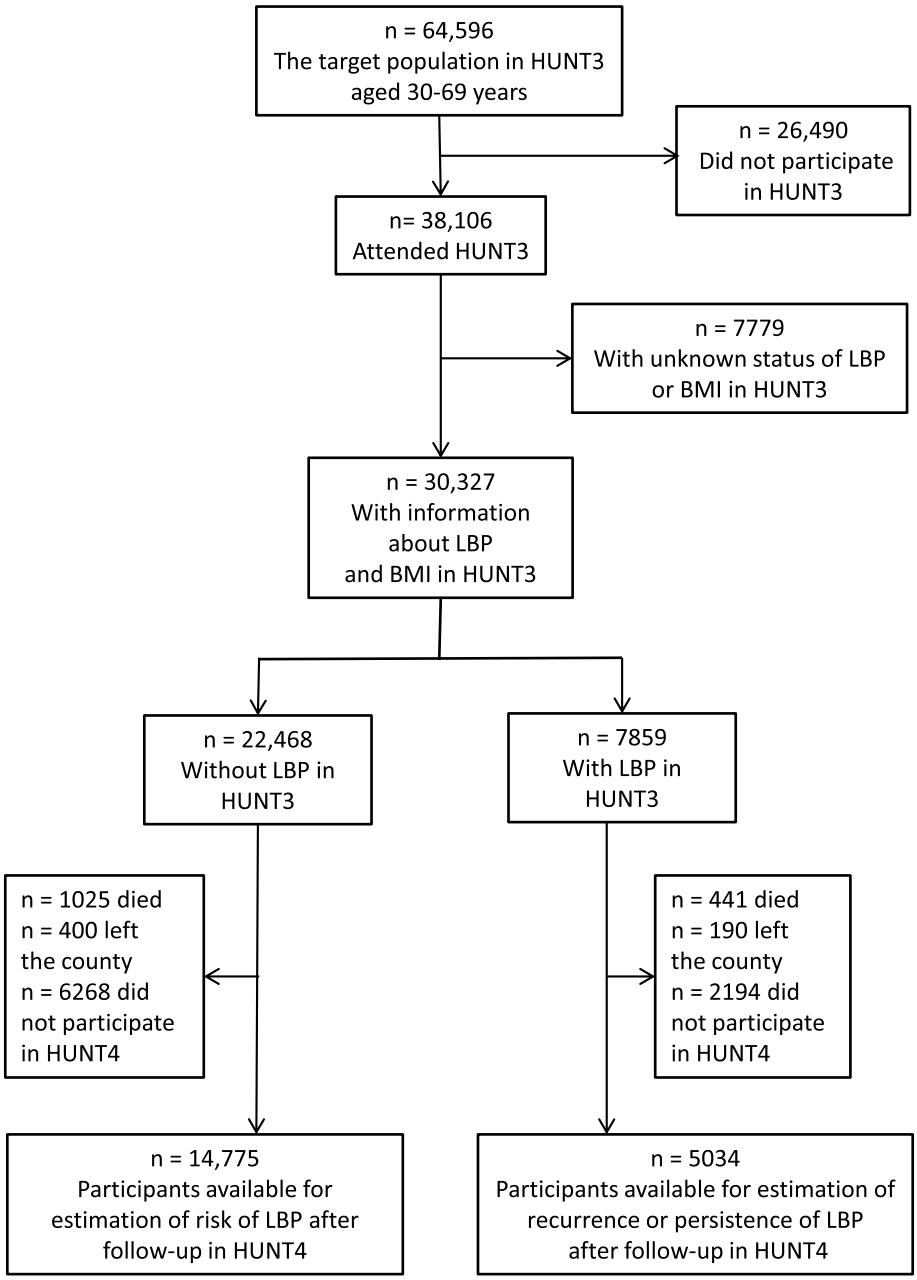

Fig. 1 Flow chart for the cohort analysis of association between overweight and obesity as risk factors for chronic LBP among all individuals regardless of BMI. HUNT, Trøndelag Health Study; LBP, Low back pain; BMI, Body mass index

because of missing information on chronic LBP or BMI. Thus, information on chronic LBP and BMI in HUNT3 was collected from 30,327 individuals, corresponding to a participation rate of 47%.

The cohort consisting of 22,468 individuals who did not report chronic LBP in HUNT3 was included in an 11 year follow-up, dealing with risk of chronic LBP in HUNT4 (Fig. 1). Information on residence status was collected from national registries and linked by the unique identification numbers being used in Norway. During follow-up 1025 individuals in this cohort died and 400 left the county. In addition 6268 individuals in the cohort did not participate in the HUNT4 survey or did not supply information on chronic LBP, despite receiving an invitation. Altogether 14,775 individuals, 8275 women and 6500 men, were available for estimation of risk of chronic LBP after follow-up, representing 70% of the participants remaining in the county and 66% of the original cohort.

The cohort consisting of 7859 individuals reporting chronic LBP in HUNT3 was included in a separate 11 year follow-up, considering recurrence or persistence of LBP in HUNT4 (Fig. 1). Of these, 441 individuals died and 190 individuals left the county during the follow-up period. A total of 2194 individuals who had the possibility to participate did not attend the HUNT4 survey or did not provide information on LBP. This left 5034 individuals, 3224 women and 1810 men, available for estimation of recurrence or persistence of chronic LBP after follow-up, representing 70% of the participants remaining in the county and 64% of the original cohort.

The relationship between presence or absence of chronic LBP in HUNT3 and mean change in BMI in the period from HUNT3 to HUNT4 was examined among the 19,704 individuals, 11,449 women and 8255 men, with known status of LBP and BMI in HUNT3 and known status of BMI in HUNT4.

Classification of covariates

In both HUNT3 and HUNT4 body height and weight were measured by trained personnel with the participants wearing light clothes without shoes. Height was given in centimetre with one decimal. Weight was given in kg with one decimal.

Body mass index (BMI) in HUNT3, defined as weight/height^2 and specified in kg/m^2, was divided into five groups in the main categorisation: < 18.5 (corresponding to underweight), 18.5–24.9 (normal weight), 25.0–29.9 (overweight), 30.0–34.9 (obesity class I), ≥ 35.0 (obesity classes II and III) [24]. To be able to detect potential heterogeneity in risk of LBP, two separate levels of obesity were considered. Very few underweight individuals in HUNT3 contributed information about the relationship to chronic LBP in HUNT4, only 65 women and 10 men, with 18 and 2 cases of LBP, respectively. For this reason, the underweight individuals were not included in detailed statistical analyses of associations between BMI and risk or recurrence and persistence of LBP, although the category was retained in corresponding crude tabulations of percentages.

Smoking status was described in HUNT3 considering four categories: never smokers, previous smokers, current daily smokers and current occasional smokers. For physical activity in leisure time, including going to work, one category comprised light activity only or hard physical activity (leading to sweating or being out of breath) < 1 h per week. Other categories represented hard physical activity 1–2 and ≥ 3 h per week.

A combined variable, referred to as work activity, was introduced for work status and physical activity at work in HUNT3 among individuals who were currently employed, with a total of six categories. The first four categories represented work mainly involving sitting (e.g. assembly or desk work), work involving much walking (e.g. for clerks, light industry workers or teachers), work requiring much walking and lifting (e.g. for mail carriers, nurses or construction workers) and work involving heavy physical labour (e.g. for foresters, farmers or workers engaged in heavy construction labour). The fifth category of the combined variable included students and those occupied full time with housework. The sixth category represented those not currently employed.

Anxiety and depression at the time of HUNT3 were assessed by total Hospital Anxiety and Depression Scale (HADS) scores [25], which were categorised into five intervals: 0–4, 5–9, 10–14, 15–19 and ≥ 20.

Education, classified in HUNT4, was grouped in three categories according to duration. The first group represented at most 10 years of compulsory primary school, in some cases with an additional one or two years of lower secondary or vocational school. The second group represented 3–4 years of upper secondary or vocational school. The third group corresponded to a college or university education.

Data analysis

Relationships at start of follow-up between BMI and other potential risk factors for LBP in the data set analysed were explored by tabulation of mean values (for age and HADS scores) and frequency distributions (for the remaining categorical variables) over categories of BMI, separately for women and men. To evaluate the importance of differential participation at end of follow-up, similar tabulations were carried out among individuals who were residents of Nord-Trøndelag at end of follow-up but did not participate in HUNT4, among individuals who moved out of the county during follow-up and among those who died. Underweight individuals were excluded from these tabulations.

Crude absolute risk and recurrence or persistence of LBP were described by percentages of LBP at follow-up computed within categories of baseline age and BMI. Corresponding analyses with adjustment for potential confounders were conducted based on generalised linear models with a log link and with BMI as a categorical predictor variable, regarding the normal weight class as the reference category. Initial analyses were adjusted for age only, with the age effect represented by a cubic polynomial to take into account the known non-linear relationship between age and occurrence of chronic LBP [15]. A standard comprehensive adjustment was then introduced with additional predictor variables. The predictors were selected as potential confounders suspected to be associated with both body mass and risk of LBP: smoking

[26, 27], physical activity in leisure time [19, 28], physical activity at work [20, 29] and employment status [30, 31].

Values of the potential confounders were not available for all participants included in the main analyses with comprehensive adjustment, and these analyses were based on a slightly lower number of participants. In particular, HADS scores were unavailable for 1493 of the 19,734 individuals included in analyses of risk or recurrence and persistence of LBP. The HADS scores describe important psychological factors associated with risk of LBP [32], which may constitute potential confounders. For these reasons, computations with additional adjustment for HADS were carried out in special sensitivity analyses. To achieve an accurate assessment of the effect of this adjustment, corresponding analyses without HADS adjustment were also performed including only the individuals with known HADS scores.

The questionnaires used in HUNT3 did not include information on education, but corresponding information was available at the end of follow-up in HUNT4. Separate sensitivity analyses were carried out with additional adjustment for duration of education as assessed in HUNT4 to obtain a crude impression of the importance of this factor.

Particular alternative analyses were carried out with BMI considered as a continuous variable, using the original uncategorised values. This approach also included likelihood ratio tests for interaction between BMI and all other variables in the risk analyses. All predictor variables were consistently regarded as categorical except age and BMI. All analyses were performed separately for women and men, except those involving tests for sex interaction.

Change in BMI during the 11 year follow-up was compared among participants who did or did not report chronic LBP at baseline in HUNT3 by considering estimated marginal means in analysis of covariance with adjustment for potential confounders.

All statistical analyses were carried out using IBM SPSS version 28 (IBM Corp., Armonk, New York).

Results

Associations between BMI and other risk factors for LBP

In the data set analysed with regard to risk or recurrence and persistence of LBP, the largest percentage of daily smokers was observed in both women and men with BMI in the normal weight range (Additional file 1: Supplementary Table 1). Physical activity in leisure time showed a clear inverse association with BMI. The percentage of individuals not currently employed was considerably greater among individuals with a large BMI, in particular among women. Duration of education evaluated at end of follow-up was inversely related to baseline BMI in both sexes.

Associations between risk factors and follow-up status

The participants who died during follow-up were on average 10 years older at baseline than participants in other follow-up categories (Additional file 1: Supplementary Table 2). More daily smokers were seen among those who died during follow-up and also to some extent among non-participants, compared to participants at end of follow-up. Percentages of hard physical activity were quite similar among participants and non-participants, but were lower among those who died during follow-up. No major differences in work activity were seen comparing participants and non-participants, although the percentage of individuals not currently employed was slightly greater among non-participants, especially in women (Additional file 1: Supplementary Table 2).

Associations between BMI and risk of chronic LBP

Among the 8275 women who did not report chronic LBP at baseline, 1587 women (19.2%) reported chronic LBP at end of follow-up (Table 1). Among the 6500 men without chronic LBP at baseline, 967 men (14.9%) reported chronic LBP after follow-up. Compared to men, women showed higher percentages of chronic LBP at end of follow-up in all 10 year age intervals at baseline and all intervals of BMI (Table 1).

Crude absolute risks of chronic LBP increased with increasing BMI among women (Table 1), although the difference between the overweight and normal weight categories was smaller than the difference between obese groups. A similar positive relationship was only partly supported over the range of BMI values in men (Table 1). Analyses based on generalised linear models with age adjustment only, among the 8228 women and 6493 men with BMI $\geq$ 18.5 kg/m^2, revealed uniformly increasing risk of chronic LBP depending on BMI (Table 2). The sole exception was provided by the rather small category of obese men with BMI $\geq$ 35.0 kg/m^2, with an estimated relative risk of about the same magnitude as in overweight men.

In analyses with standard comprehensive adjustment among the 7899 women and 6253 men with information on smoking, physical and work activity, the relationships between BMI and risk of LBP were only slightly weakened (Table 2). The estimated linear relationship was stronger in women than in men but the sex difference was not significant ($P=0.54$). According to the categorical estimates, both women and men with BMI in the interval 30.0–34.9 kg/m^2 had a 36% higher risk of LBP than individuals in the normal BMI range. Obese women with BMI $\geq$ 35.0 kg/m^2 showed a 68% increase in risk.

Additional adjustment for HADS, performed in sensitivity analyses in the data with known HADS scores, produced only small changes in risk estimates (Additional

Table 1 Crude absolute risk of chronic LBP among individuals without chronic LBP at baseline, by baseline age and BMI[a]

	Women		Men	
	Total	**With chronic LBP at end of follow-up (%)**	**Total**	**With chronic LBP at end of follow-up (%)**
Complete data set	8275	1587 (19.2)	6500	967 (14.9)
Age at baseline (year)				
30–39	1594	287 (18.0)	827	122 (14.8)
40–49	2280	491 (21.5)	1635	246 (15.0)
50–59	2509	440 (17.5)	2198	324 (14.7)
60–69	1892	369 (19.5)	1840	275 (14.9)
BMI (kg/m^2)				
< 18.5	47	6 (12.8)	7	0 (0.0)
18.5–24.9	3451	585 (17.0)	1520	201 (13.2)
25.0–29.9	3185	597 (18.7)	3674	537 (14.6)
30.0–34.9	1200	283 (23.6)	1112	202 (18.2)
≥ 35.0	392	116 (29.6)	187	27 (14.4)

[a] Among all individuals regardless of BMI

Table 2 Associations between baseline BMI and risk of chronic LBP, among individuals without chronic LBP at baseline[a]

	Women		Men	
	RR (95% CI) with adjustment for age only	**RR (95% CI) with standard comprehensive adjustment[b]**	**RR (95% CI) with adjustment for age only**	**RR (95% CI) with standard comprehensive adjustment[b]**
Number included	8228	7899	6493	6253
BMI (kg/m^2)				
18.5–24.9	1.00 (reference)	1.00 (reference)	1.00 (reference)	1.00 (reference)
25.0–29.9	1.11 (1.00–1.23)	1.11 (1.00–1.23)	1.10 (0.95–1.28)	1.10 (0.94–1.28)
30.0–34.9	1.39 (1.23–1.58)	1.36 (1.20–1.54)	1.37 (1.15–1.64)	1.36 (1.13–1.63)
≥ 35.0	1.75 (1.48–2.07)	1.68 (1.42–2.00)	1.09 (0.75–1.58)	1.02 (0.70–1.50)
Per 5 kg/m^2 [c]	1.17 (1.12–1.22)	1.16 (1.11–1.21)	1.14 (1.05–1.23)	1.12 (1.04–1.22)
Per standard deviation[c,d]	1.16 (1.11–1.20)	1.15 (1.10–1.19)	1.09 (1.03–1.16)	1.09 (1.03–1.15)
P for linear trend[c]	< 0.001	< 0.001	0.002	0.006

[a] Among individuals with BMI ≥ 18.5 kg/m^2

[b] Adjustment for age, smoking, physical activity in leisure time and work activity

[c] BMI considered as a continuous variable

[d] Standard deviation of BMI: 4.60 for women and 3.53 for men

file 1: Supplementary Table 3), in particular among men. Adjustment for duration of education at end of follow-up hardly affected risk estimates with regard to BMI (Additional file 1: Supplementary Table 4).

Positive relationships between BMI and risk of chronic LBP were seen in all 10 year age intervals, except among men aged 40–49 years (Table 3). However, no significant age interaction could be demonstrated in either sex ($P=0.73$ for women and $P=0.15$ for men). Among the other potential confounders, only duration of education in women showed a significant interaction with BMI ($P=0.012$) (Additional file 1: Supplementary Table 5). Women with a particularly short or long duration of education showed clear positive relationships between BMI and risk of chronic LBP, in contrast to the group of women with a medium duration of education (Additional file 1: Supplementary Table 5).

Associations between BMI and recurrence or persistence of chronic LBP

A total of 3224 women and 1810 men reported chronic LBP at baseline. Among these, 1872 women (58.1%) and

Table 3 Associations between baseline BMI and risk of chronic LBP, among individuals without chronic LBP at baseline, by age[a]

	Women		Men	
	Number included	RR (95% CI) per 5 kg/m^2 with standard comprehensive adjustment[b,c]	Number included	RR (95% CI) per 5 kg/m^2 with standard comprehensive adjustment[b,c]
Age (year)				
30–39	1531	1.18 (1.07–1.30)	799	1.13 (0.91–1.39)
40–49	2211	1.11 (1.03–1.20)	1579	0.91 (0.76–1.08)
50–59	2385	1.19 (1.09–1.31)	2119	1.18 (1.03–1.36)
60–69	1772	1.16 (1.04–1.28)	1756	1.29 (1.11–1.50)
P for interaction between BMI and age[c]		0.73		0.15

[a] Among individuals with BMI ≥ 18.5 kg/m^2

[b] Adjustment for age, smoking, physical activity in leisure time and work activity

[c] BMI considered as a continuous variable

899 men (49.7%) reported chronic LBP at end of follow-up (Table 4). A higher percentage of chronic LBP at end of follow-up was seen in women than in men in all 10 year age intervals. A similar sex difference was also observed in all intervals of BMI except in the very small group of 18 women and 3 men with BMI < 18.5 kg/m^2 (Table 4).

Crude percentages of recurrence or persistence of LBP increased with increasing BMI above the 18.5 kg/m^2 threshold, both among women and men (Table 4). Percentages displayed larger increases among categories of obese individuals than among those with normal weight or overweight. Age-adjusted analyses based on generalised linear models revealed consistent positive relationships between BMI and the probability of recurrence or persistence of LBP (Table 5). Additional adjustment for smoking and physical and work activity did not affect the relationships to any appreciable extent. The relationships were quite similar in women and men ($P = 0.75$ for equality).

Additional adjustment for HADS scores (Additional file 1: Supplementary Table 6) or duration of education at end of follow-up (Additional file 1: Supplementary Table 7) did not produce essential changes in estimated relative probabilities of recurrence or persistence. However, the relationship with BMI among men was no longer statistically significant after adjustment for education (Additional file 1: Supplementary Table 7).

Table 4 Crude recurrence or persistence of chronic LBP among individuals with chronic LBP at baseline, by baseline age and BMI[a]

	Women		Men	
	Total	With chronic LBP at end of follow-up (%)	Total	With chronic LBP at end of follow-up (%)
Complete data set	3224	1872 (58.1)	1810	899 (49.7)
Age at baseline (year)				
30–39	412	223 (54.1)	184	87 (47.3)
40–49	793	439 (55.4)	391	194 (49.6)
50–59	1086	648 (59.7)	687	342 (49.8)
60–69	933	562 (60.2)	548	276 (50.4)
BMI (kg/m^2)				
< 18.5	18	12 (66.7)	3	2 (66.7)
18.5–24.9	1044	572 (54.8)	363	173 (47.7)
25.0–29.9	1323	742 (56.1)	982	477 (48.6)
30.0–34.9	582	368 (63.2)	371	192 (51.8)
≥ 35.0	257	178 (69.3)	91	55 (60.4)

[a] Among all individuals regardless of BMI

Table 5 Associations between baseline BMI and recurrence or persistence of chronic LBP, among individuals with chronic LBP at baseline[a]

	Women		Men	
	RR (95%CI) with adjustment for age only	RR (95% CI) with standard comprehensive adjustment[b]	RR (95% CI) with adjustment for age only	RR (95% CI) with standard comprehensive adjustment[b]
Number included	3206	3036	1807	1735
BMI (kg/m^2)				
18.5–24.9	1.00 (reference)	1.00 (reference)	1.00 (reference)	1.00 (reference)
25.0–29.9	1.02 (0.95–1.09)	1.03 (0.95–1.10)	1.02 (0.90–1.16)	1.02 (0.89–1.16)
30.0–34.9	1.14 (1.05–1.24)	1.11 (1.02–1.21)	1.09 (0.94–1.26)	1.07 (0.92–1.25)
≥ 35.0	1.26 (1.14–1.39)	1.22 (1.11–1.34)	1.27 (1.04–1.54)	1.22 (1.00–1.50)
Per 5 kg/m^2 [c]	1.08 (1.05–1.11)	1.07 (1.04–1.10)	1.08 (1.02–1.15)	1.07 (1.00–1.14)
Per standard deviation[c,d]	1.08 (1.05–1.10)	1.07 (1.04–1.10)	1.06 (1.02–1.11)	1.05 (1.00–1.10)
P for linear trend[c]	< 0.001	< 0.001	0.011	0.044

[a] Among individuals with BMI ≥ 18.5 kg/m^2

[b] Adjustment for age, smoking, physical activity in leisure time and work activity

[c] BMI considered as a continuous variable

[d] Standard deviation of BMI: 4.60 for women and 3.53 for men

Associations between baseline LBP and change in BMI

The mean change in BMI during follow-up, evaluated with standard comprehensive adjustment for age, smoking and physical and work activity, did not differ significantly between individuals of the same sex with and without chronic LBP at baseline (Table 6). Estimated mean changes were marginally greater among those reporting chronic LBP at baseline, but absolute differences in mean change were less than 0.1 kg/m^2. Analyses with adjustment for age only or with no adjustment

Table 6 Mean change in BMI during follow-up by status of chronic LBP at baseline[a]

	Women		Men	
	Number included	Mean change in BMI (kg/m^2) (95% CI)[b]	Number included	Mean change in BMI (kg/m^2) (95% CI)[b]
No adjustment				
Status at baseline				
No chronic LBP	8241	0.41 (0.35–0.46)	6456	0.16 (0.12–0.21)
With chronic LBP	3208	0.28 (0.19–0.38)	1799	0.24 (0.14–0.33)
P		0.029		0.17
Adjustment for age				
Status at baseline				
No chronic LBP	8241	0.41 (0.36–0.47)	6456	0.26 (0.21–0.31)
With chronic LBP	3208	0.40 (0.30–0.49)	1799	0.38 (0.28–0.47)
P		0.73		0.026
Standard comprehensive adjustment[c]				
Status at baseline				
No chronic LBP	7915	0.46 (0.35–0.57)	6219	0.37 (0.28–0.45)
With chronic LBP	3036	0.52 (0.39–0.65)	1727	0.45 (0.34–0.57)
P		0.27		0.11

[a] Among all individuals regardless of BMI

[b] Estimated marginal means computed with adjustment by analysis of covariance

[c] Adjustment for age, baseline BMI, smoking, physical activity in leisure time and work activity

produced slightly different results, but in no case was any major difference found in mean change of BMI depending on LBP status at baseline (Table 6).

Discussion

Summary of findings

The current follow-up study showed definite positive associations between baseline BMI and risk of chronic LBP in both women and men. Women displayed a conspicuous monotone relationship with estimated risk in the entire range of BMI, but among men the estimated risk was reduced in the relatively small group of very obese individuals compared to other obese. The probability of recurrence or persistence of LBP followed similar monotone relationships with BMI in both sexes, although the associations were apparently weaker than those observed for risk of LBP. Adjustment for potential confounders did not affect risk estimates to any large extent. No remaining difference in mean change of BMI during follow-up could be demonstrated between individuals with and without baseline LBP after adjustments.

Strengths and limitations

Our study has several strengths. It incorporates a population-based follow-up of a large number of individuals over a relatively long period. Body height and weight were measured at baseline and at end of follow-up. Information on chronic LBP was also collected at the same time. This made it possible to analyse associations with risk of LBP among participants unaffected by LBP at baseline, as well as recurrence of LBP among those already suffering from LBP. It was also possible to compare weight gain during follow-up in those with and without baseline LBP. Moreover, information was available on most relevant potential confounders.

Certain limitations must still be taken into account in the interpretation of our results. The baseline participation rate in the entire county considering information on LBP and BMI in HUNT3 was relatively low (47%), although participation rates in HUNT4 in the cohorts followed were considerably larger (70%). Participation in HUNT4 depended to some extent on potential confounders but was essentially independent of BMI and presence or absence of LBP in HUNT3. The basic information about LBP relied on self-reports supplied in a questionnaire. No information on LBP was available in the interval between baseline and end of follow-up, and information about pain intensity was not collected. The questions used to elicit information about chronic LBP in HUNT3 and HUNT4 were not identical, and it is difficult to assess the effect this had on results in the present study.

It was unfortunately not possible to include the small underweight category in the statistical models considered for risk of LBP, and because of few individuals in obesity class III, it was necessary to combine obesity classes II and III. Thus risk of LBP for extreme values of BMI may not be accurately reflected by our estimates.

Despite the adjustments carried out in the statistical analysis, residual confounding may have affected risk estimates. In the data file, information about education was only available as a variable recorded in HUNT4, not at baseline. For this reason, adjustment for duration of education was restricted to separate sensitivity analyses. However, at start of follow-up in HUNT3, the participants in the present study were in the age range 30–69 years, when the great majority would already have attained their final level of education. No serious error should thus be introduced by adjusting for information on education in HUNT4 rather than the more appropriate values in HUNT3. The classification based on duration of education served as a substitute for a more detailed classification of social class [33].

Previous studies

The results reported from earlier study waves in the HUNT survey provide an opportunity to assess changes over time in associations between BMI and occurrence of chronic LBP. For the 11 year follow-up from HUNT2 to HUNT3 [16], a comparison must take into account that associations were expressed in terms of odds ratios, not relative risks, and that all obese individuals were assigned to a single risk category. Adjusted estimates converted to approximate relative risks [34] were RR = 1.15 for women in the overweight category, compared to RR = 1.11 in the present study (Table 2). For obese women with BMI ≥ 30.0 kg/m^2, the previous study led to RR = 1.17, compared to RR = 1.36 and RR = 1.68 in the two separate obese categories in the current study. Thus the estimated risk of LBP associated with obesity in women may appear to be greater now, with a further gradient between the two levels of obesity.

For overweight men, the risk estimates were similar in the previous [16] and current studies based on HUNT data (RR = 1.11 and RR = 1.10). The estimate RR = 1.29 for all obese men in the previous study should be compared to the current values RR = 1.36 and RR = 1.02 (Table 2). Considering the small sample size of the very obese category in men, this does not necessarily reflect any major difference between studies. An even earlier follow-up between the study waves HUNT1 and HUNT2 [35] produced estimates of the relative risk of LBP among overweight and obese of similar magnitude to those found for the period from HUNT2 to HUNT3.

The two largest studies to date [36, 37] on relationships between BMI and occurrence of LBP were based on data collected in adolescents and young adults in military service. One cross-sectional study [36], including more than 800,000 women and men, showed adjusted odds ratios for overweight and obesity of about the same magnitude as those found in HUNT from HUNT2 to HUNT3 [16], with no major sex differences. A cohort study with an 18 month follow-up of occurrence of serious LBP [37], with over 600,000 participants, produced similar odds ratios in pooled analysis including both women and men.

A large Korean study with an 8 year continuous follow-up [38] aimed primarily at developing a prediction model for risk of LBP. The model was derived considering a cohort comprising more than 290,000 individuals. The final adjusted model, for women and men combined, included BMI as a predictor variable, with hazard ratios HR = 1.11 for overweight and HR = 1.13 for obese. A relatively large cross-sectional study of chronic LBP in the United States [39], with more than 30,000 individuals, also led to adjusted odds ratios of the same magnitude, with a more pronounced relationship indicated in women than in men. A cross-sectional study of LBP in Spain [40], with nearly 20,000 participants, gave odds ratio estimates indicating a stronger relation with overweight and obesity. It is difficult, however, to compare cross-sectional estimates with estimates for risk or recurrence in cohort studies.

Other cohort studies of relationships between BMI and risk of LBP have generally included less than 10,000 individuals. Results have not been unambiguous, but overweight and obese individuals have in some cases shown substantial increases in risk compared to individuals with normal weight. Thus in a British 9 year follow-up of a birth cohort of about 9000 individuals [41], obese women were estimated to have an odds ratio of 1.78 compared to normal weight women, although no increased risk could be demonstrated in men. In an extensive occupational cohort study of about 6500 individuals in the United States with a 4 year follow-up [42], odds ratios for low back disorders as large as 2.02 were found for overweight women and men and 2.77 for obese individuals.

Two meta-analyses have been carried out dealing with the association between BMI and risk of LBP. The first one [12] covered articles published until May 2009. Considering cohort studies focusing on LBP occurring in the past 12 months, with adjustment for potential confounders, the overall estimated odds ratio for overweight compared to normal weight was OR = 1.08. The corresponding estimate for obese individuals was OR = 1.42. However, these estimates were based on only 2 and 3 separate studies which were not very large. The second meta-analysis [43] comprised 10 cohort studies published before December 2015, including those considered in the first meta-analysis. The particular study based on data from HUNT2 and HUNT3 [16] contributed by far the largest data set. The odds ratio estimates were OR = 1.15 for overweight individuals and 1.36 for obese, relative to normal weight individuals [43]. The associations were generally quite similar in females and males, with only slightly stronger associations suggested in females.

Few studies have been carried out of the association between BMI and recurrence or persistence of chronic LBP. The study based on data from HUNT2 and HUNT3 [16] showed associations of magnitude similar to those found in the current study, although the current study revealed a greater probability of recurrence or persistence in very obese individuals not previously reported.

The large Korean study [38] established a prediction model for recurrence of LBP during a 5 year period based on a cohort including more than 90,000 women and men. In a model with adjustment for other relevant risk predictors, the estimated hazard ratios were HR = 1.05 for both overweight and obese individuals. A study in Sweden [44] found estimates RR = 1.05 for overweight and RR = 1.12 for obese, considering recurrence of LBP over 2 year periods in a cohort of more than 5000 women and men.

There are very few previous studies assessing the potential influence of back pain on subsequent weight change. The cohort study with follow-up from HUNT2 to HUNT3 [16] indicated that any additional weight gain in individuals with chronic LBP was negligible. In contrast, a British cohort study [41] found an increase in estimated weight gain over a 10 year period of 1.10 kg among young women with chronic back pain compared to those without pain. In a study of Danish schoolchildren [45], individuals with spinal pain showed an increased risk of becoming overweight after a 2 year follow-up.

Interpretation

Several large studies of associations between BMI and LBP have produced rather similar estimates of risk among overweight individuals relative to individuals of normal weight. This also applies to estimates of relative risk for obese, although there is more variation between studies in this case. This general characterisation seems to hold true regardless of the choice of risk factors adjusted for in the statistical analyses, which may differ considerably between studies. Reverse causation, with occurrence of LBP affecting BMI at a later stage seems unlikely in view of the analyses with BMI as a dependent variable.

Taking the overall evidence into account, it seems reasonable to conclude that the higher risks observed in overweight and even more in obese individuals reflect a real underlying association between body composition

and LBP. Several early studies of these relationships were quite small, and risk estimates were subject to large sampling errors. Associations were not necessarily significant in the statistical sense even with large estimates of relative risk. Reviews [10, 11, 46] emphasised the lack of statistical significance in many separate studies and may have given an incomplete impression by referring to results as inconsistent or conflicting. With the larger data sets available today, it seems more likely that most of the variation between study results is due to random factors. Some variation can be expected between populations in different geographical areas because of essential differences in distributions of body measures [47], but otherwise the results should be representative.

According to the present study, being overweight with BMI in the interval 25.0–29.9 kg/m^2 is associated with an increase in risk of LBP of magnitude about 10% for both women and men. Being obese with BMI in the interval 30.0–34.9 kg/m^2 apparently implies an increase in risk of about 35%, although this number may represent a slight overestimate. It is unlikely that the differences in risk estimates between the relevant follow-up periods in the HUNT Study reflect actual changes over time in underlying associations. These numbers also seem consistent with the overall estimates from the earlier meta-analyses [12, 43], with no major differences between women and men.

The group of obese individuals may, however, be heterogeneous with regard to risk of LBP. For women with BMI $\geq$ 35.0 kg/m^2 this study suggests a substantial increase in risk compared to normal weight women of magnitude about 68%. As this relatively small category was not dealt with separately in most previous studies, it is difficult to find comparable estimates from other populations. For men with BMI $\geq$ 35.0 kg/m^2, a much lower risk estimate was found in the present study. It is not possible to state with any certainty whether this sex difference is real and indeed whether the estimate found for men is too low. A very large BMI in men may reflect a different body composition from that found in women, which could affect the risk of LBP [48]. Future epidemiological studies of risk of LBP should pay more attention to the very obese categories in women and men.

Statistically significant interaction between duration of education and BMI was observed among women in the present study, but not among men. The general positive association between BMI and risk of LBP was clearly present among women with an education of short or long duration, but could not be established among those with an education of medium education. There appears to be no natural explanation of this kind of relationship, which may represent a spurious finding. Potential interaction between BMI and physical activity has been discussed extensively in connection with risk of LBP [18], but no such interaction was indicated in the present study or in the previous HUNT follow-ups [16, 19, 35].

Assessment of overweight and obesity is conventionally based on BMI, but other body measures may possibly provide a better basis. In a comparison of measures, considering the follow-up from HUNT2 to HUNT3 [48], body weight and waist and hip circumference were also associated with risk of LBP after adjustment for other factors, but waist-hip-ratio was not. This may indicate that central obesity is unlikely to play a major role in the development of LBP, although the amount of total fat mass can be important. However, with mutual adjustment for the remaining body measures, only simple body weight still showed an association with risk. This suggests that mechanical or structural elements play an essential role [12].

Meta-analyses have concluded that body fat percentage [49], waist circumference, waist-hip-ratio and total fat mass [50] are all related to occurrence of LBP. Adipose tissue secretes cytokines and hormones, possibly related to development of musculoskeletal pain [49]. Hormonal factors have been shown to be associated with risk of LBP among women in the data set considered in the present study [51]. The effects of BMI on back pain have been confirmed by Mendelian randomisation [52, 53], but it is not at present clear which underlying factors are responsible for the relationship between measures of body size and risk of LBP.

It is difficult to develop realistic models for short term prognostic effects on recurrence of LBP [54], and there are few studies of such effects [55]. It is not always evident whether recovery from LBP has occurred between two particular points in time when LBP is present. For this reason no distinction was made in the current study between recurrence or persistence. The results in the few large long-term studies of recurrence [16, 38, 44] are generally consistent with the results of the present study, with positive associations with BMI of somewhat smaller magnitude than the association for risk among unaffected, evaluated by relative risks. However, as the absolute probability of recurrence is generally much larger than the absolute incidence among unaffected, recurrence of LBP may still be quite important from a population point of view.

Conclusions

In general, the risk of chronic LBP among individuals who are not currently affected increases with higher levels of BMI, both in women and men. However, it is uncertain whether this statement applies to obese men with

BMI ≥ 35.0 kg/m^2. Obese women with BMI ≥ 35.0 kg/m^2 may carry a particularly large risk. Adjustment for other risk factors does not influence the relationship with BMI to any major extent. The probability of recurrence or persistence of chronic LBP among those already affected also increases with higher levels of BMI.

Abbreviations

BMI	Body mass index
CI	Confidence interval
HADS	Hospital Anxiety and Depression Scale
HR	Hazard ratio
HUNT	Trøndelag Health Study
LBP	Low back pain
OR	Odds ratio
RR	Relative risk

Supplementary Information

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Supplementary Material 1. Supplementary Table 1. Description of data: Descriptive statistics of potential risk factors for LBP, by baseline BMI. **Supplementary Table 2.** Descriptive statistics of potential risk factors for LBP at baseline in HUNT3, by follow-up status. **Supplementary Table 3.** Associations between baseline BMI and risk of chronic LBP, among individuals without chronic LBP at baseline, with and without adjustment for HADS. **Supplementary Table 4.** Associations between baseline BMI and risk of chronic LBP, among individuals without chronic LBP at baseline, with and without adjustment for education at end of follow-up in HUNT4. **Supplementary Table 5.** Associations between baseline BMI and risk of chronic LBP, within categories of education in HUNT4. **Supplementary Table 6.** Associations between baseline BMI and recurrence or persistence of chronic LBP, among individuals with chronic LBP at baseline, with and without adjustment for HADS. **Supplementary Table 7.** Associations between baseline BMI and recurrence or persistence of chronic LBP, among individuals with chronic LBP at baseline, with and without adjustment for education at end of follow-up in HUNT4.

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Authors' contributions
InH, IvH, KH and J-AZ contributed to the study design. InH and IvH performed the analysis of the data. InH wrote the paper. All authors discussed the results, commented on the manuscript and approved the paper.

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Availability of data and materials
The data set analysed belongs to a third party, the HUNT Study (the Trøndelag Health Study). The authors of the current manuscript have been given permission to analyse the data after obtaining the necessary Norwegian permits. Because of the confidentiality requirements, a data set of this kind with information from a complete county at the individual level cannot be made public. However, research groups wishing to analyse data from the HUNT Study may apply to the HUNT organisation (http://www.ntnu.edu/hunt) to get access, after having obtained the permits needed according to Norwegian law.

Declarations

Ethics approval and consent to participate
The work was approved by the Regional Committee for Medical and Health Research Ethics South East Norway (number 2019/43095). The HUNT was also approved by the Norwegian Data Inspectorate. Each participant in the HUNT surveys signed a written informed consent regarding the collection and use of data for research purposes.

Consent for publication
Not applicable.

Competing interests
The authors declare no competing interests.

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