Weight Change over Three Decades and the Risk of Osteoporosis in Men

The Norwegian Epidemiological Osteoporosis Studies (NOREPOS)

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The purpose of this study was to assess the effect of weight in middle-aged men and subsequent weight change on the risk of osteoporosis three decades later. The authors utilized data from 1,476 Norwegian men participating in two health screenings in Oslo (1972–1973 and 2000–2001) and Tromsø (1974–1975 and 2001). Height and weight were measured at baseline and follow-up. Total hip bone mineral density (BMD) was assessed at follow-up by dual energy x-ray absorptiometry. Baseline body mass index (BMI) was positively related to BMD three decades later. Subsequent weight change was also strongly related to BMD, and the proportion of persons with osteoporosis decreased from 15.1% among those who lost ≥10% of their body weight to 0.6% among those who gained ≥10% of their body weight. Excluding participants with medical conditions did not change the association between weight change and BMD. Taking both BMI and weight change into account, the prevalence of osteoporosis in the lowest quarter of baseline BMI was 31% (95% confidence interval: 24, 37) in persons losing ≥5% of their weight and 4% (95% confidence interval: 1, 7) in persons gaining ≥5% of their weight. In this cohort of middle-aged men, low baseline BMI and weight loss during the following three decades were both strongly and negatively related to total hip BMD.

Being slim is an established and important risk factor for osteoporosis and osteoporotic fractures in both women and men (1). Weight loss per se is also a risk factor for bone loss (2–4) and later hip fracture (5–7) in both sexes. After conducting a longitudinal study in men, Ensrud et al. (2) reported that both intentional and unintentional weight loss over 1.8 years was positively related to hip bone loss, and this and other studies have strongly suggested a causal relation between weight loss and skeletal ill health.

For middle-aged men, the current risk of osteoporotic fractures is low, but it is important to recognize major risk factors for fracture later in life, and data on these aspects are

Abbreviations: BMD, bone mineral density; BMI, body mass index; CI, confidence interval; SD, standard deviation.
scarce. Previous studies in men have found a relation between short-term weight loss (a few years) and bone mineral density (BMD) (2–4), but we are not aware of any prior studies that have assessed the long-term effect of weight and weight change on the risk of osteoporosis in men.

Lifestyle factors like smoking and physical activity are related to BMD, and they are also related to weight change. In addition, some medical conditions—for example, diabetes mellitus (8) and chronic obstructive lung disease (9)—might influence both body weight and BMD and may thus confound the relation between weight change and osteoporosis if not controlled for.

Our purpose in the present study was to assess the effect of weight in middle-aged men and subsequent weight change on the risk of osteoporosis three decades later when these men entered the period of life with the highest risk of hip fracture.

MATERIALS AND METHODS

Participants

Men included in this analysis participated in two health screenings 26–29 years apart in the Norwegian cities of Oslo and Tromsø. Men from Oslo who had been born in 1924–1925 participated in the Oslo Study during 1972–1973 at ages 47–49 years (10) and participated in the Oslo Health Study during 2000–2001 at ages 75–77 years (11) (www.fhi.no/HUBRO). Men in other age groups were also invited to participate in the Oslo Study in 1972–1973, but they were not invited to participate in the Oslo Health Study in 2000–2001. Men from Tromsø who had been born in 1925–1954 participated in the first survey of the Tromsø Study (Tromsø I) during 1974–1975 at ages 20–50 years and in the fifth survey of the Tromsø Study (Tromsø V) (http://tromsoundersokelsen.no) in 2001 at ages 47–76 years. In order to restrict the age span in this study, we excluded men from Tromsø who were younger than age 65 years in 2001 (born after 1936).

Thus, this analysis included 880 men from Tromsø and 596 men from Oslo (total n = 1,476) with valid hip BMD measurements (only measured at follow-up) and weight at both screenings. Hereafter, the first screening is called baseline and the second screening is called follow-up. These studies are part of the Norwegian Epidemiological Osteoporosis Studies (NOREPOS) collaboration.

Of 2,254 men born in 1925–1936 who attended the first Tromsø Study survey, 624 were deceased when the follow-up study started. Of the remaining 1,630 men, 880 were included in the present analysis and the remaining 750 either were not invited, did not participate in the follow-up study, or participated but had no BMD measurement. Compared with these 750 men, men with BMD measurements had a slightly lower body mass index (BMI; weight (kg)/height (m)²) at baseline (BMI = 0.28; p = 0.044) but did not differ in terms of age (p = 0.32), smoking prevalence (p = 0.23), or physical activity during leisure time (p = 0.11). In Oslo, 3,576 men born in 1924–1925 participated in the baseline study. Of these men, 1,341 were deceased when the follow-up study started and 596 were included in the present analysis, whereas the remaining 1,640 either were not invited, did not participate in the follow-up study, or participated but had no BMD measurement. Compared with these 1,640 men, men with BMD measurements had a lower BMI at baseline (BMI = 0.44; p = 0.001), smoked less at baseline (current smoking: 38 percent vs. 55 percent; p < 0.001), and were more physically active during leisure time (p < 0.001).

The study protocols for the Oslo Health Study and the Tromsø Study were evaluated by the Regional Committee for Medical Research Ethics and approved by the Norwegian Data Inspectorate. Written informed consent was obtained from the participants.

Measurements

At baseline and follow-up, height (in centimeters) and weight (in kilograms) were measured while participants stood without shoes and in light indoor clothing at a screening station. In 2000–2001, measurements were recorded to one decimal digit. Based on the measurements at baseline and follow-up, respectively, BMI was calculated as weight divided by height squared.

At follow-up, total hip BMD was measured at the left hip (nondominant) by dual energy x-ray absorptiometry. If the left hip measurement was missing, a valid right hip scan was used. Right hip scans were used for 4.7 percent of participants in Tromsø and 2.6 percent of participants in Oslo. A Lunar DPX-L device was used in the Oslo Health Study and a GE Lunar Prodigy device was used in the Tromsø Study; both scanners were made by the same manufacturer (GE Lunar, Madison, Wisconsin). All scans were performed following the same protocol. They were reviewed and reanalyzed if necessary, and technically incorrect scans and scans with metal in the region of interest were excluded.

According to a previous cross-calibration study, BMD data from the Oslo Health Study were recalculated to the scale of the GE Lunar Prodigy device in the Tromsø Study (12; Tone K. Omsland, University of Oslo, personal communication, 2008).

Questionnaire information

At baseline and follow-up, information was collected via self-administered questionnaires. The questionnaires were sent with the letter of invitation and were handed in at the screening stations. Information collected at baseline included information on smoking, physical activity, and chronic diseases. At follow-up, information concerning education, physical activity, smoking habits, alcohol use, general health status, and the occurrence of chronic diseases was collected.

Statistical methods

We calculated weight change as percentage weight change from baseline (weight at follow-up minus weight at baseline divided by weight at baseline). We then constructed weight change categories according to the categories of Langlois et al. (5): loss of ≥10 percent, loss of 5–<10 percent, loss or gain of <5 percent, gain of 5–<10 percent, and gain of
\( \geq 10 \text{ percent. We also created categories combining the weight change categories with quarters of baseline BMI (cutpoints: 22.6, 24.2, and 26.0). Because there were few participants in the two lowest weight change categories, these were combined in the latter.} \)

We also performed analyses entering weight change as a continuous variable. Since the interval between the first and second screenings was longer in Oslo (median, 28 years) than in Tromsø (median, 27 years), we entered percent weight change per year (calculated for each individual) in a supplementary analysis. However, this did not change the results (data not shown). In an additional stratified analysis, we found that the relations between weight change and BMD were similar in Tromsø and Oslo (data not shown). Finally, an alternative analysis using absolute weight change in kilograms instead of percent weight change gave similar results (data not shown).

We used analysis of variance to calculate adjusted mean BMDs in categories of weight change. Linear regression was used to assess the relation between weight change as a continuous variable and BMD.

In the basic models, we adjusted for age and baseline BMI. We then controlled for factors on which data were collected at follow-up that might be related to BMD and weight change. Smoking was entered as never smoking, ex-smoking, and current smoking in three categories (<15 cigarettes/day, \( \geq 15 \) cigarettes/day, or pipes, cigars, or an unknown number of cigarettes) with four dummy variables. A four-graded question concerning weekly frequency of hard physical activity (sweating/feeling out of breath) was entered as a continuous variable. Education was entered as a continuous variable (years of education), and so was alcohol consumption during the last year (in eight categories, the first being “4–7 times per week” and the last being “have never drunk alcohol”). Marital status was entered as married or not married.

We also adjusted for diseases which might influence weight change. This included self-reported information concerning general health at follow-up (“How would you describe your present state of health?”, graded from 1 (poor) to 4 (very good)) and questions on whether the participant suffered or ever had suffered from chronic bronchitis/ emphysema, diabetes mellitus, coronary heart disease (heart infarction or angina pectoris), cerebral stroke, or mental distress for which he had sought help. The reason for adjusting for these factors collected at follow-up was that we thought this would best adjust for possible confounding. However, we also conducted an additional analysis controlling for smoking and physical activity as recorded at baseline.

For each participant, a \( T \) score was calculated accordingly: measured BMD minus mean young adult BMD divided by the standard deviation (SD) of young adult BMD. BMD data for men aged 20–39 years in the GE Lunar database were used as the young adult BMD (13). Osteoporosis was subsequently defined as a \( T \) score less than or equal to \(-2.5\) SDs.

We used results from two large meta-analyses to estimate the impact that differences in mean BMD might have on differences in fracture incidence (14, 15). Given a relative risk of 2.6 for hip fracture per 1-SD decrease in BMD, we log-transformed this relative risk, multiplied it (i.e., the \( \beta \) coefficient) by the differences in BMD (in SDs) between the weight change categories, and recalculated the relative risks. We did the same thing for differences in BMD between BMI categories and the combined BMI and weight change categories.

### RESULTS

Mean BMI was 24.4 (SD, 2.6) at baseline and 26.5 (SD, 3.3) at follow-up. At baseline, 2.2 percent of participants were obese (BMI \( \geq 30 \)), and the corresponding figure at follow-up was 13.9 percent. The mean weight change between the two examinations was 3.8 kg (SD, 7.6), corresponding to 5.2 percent (SD, 10.2).

As can be seen from table 1, participants who gained the most weight had the lowest mean BMI at baseline and the highest mean BMI at follow-up. Weight change was inversely correlated with baseline BMI (\( r = -0.21 \)) and positively correlated with BMI at follow-up (\( r = 0.57 \)). The proportion of ex-smokers at follow-up increased with increasing weight gain, and the highest proportion of current smokers was found in those who had lost the most weight. Men in the stable weight group and men who gained 5–<10 percent of their baseline weight reported the fewest health problems.

Mean total hip BMD was 0.977 g/cm\(^2\) (SD, 0.136), and 5.0 percent of the men (\( n = 74 \)) had osteoporosis at follow-up.

Mean age-adjusted BMD in quarters of baseline BMI increased from 0.993 g/cm\(^2\) in the lowest quarter to 0.968 g/cm\(^2\), 0.984 g/cm\(^2\), and 1.024 g/cm\(^2\) in the following quarters. The corresponding prevalences of osteoporosis were 10.9 percent, 3.5 percent, 4.4 percent, and 1.2 percent after adjustment for age and smoking at follow-up.

BMD at follow-up was lowest in the participants who had lost 10 percent or more of their baseline weight and increased across weight change groups, with the highest level being observed in those who had gained 10 percent or more in weight (table 2). As table 2 shows, adjustment for smoking at follow-up slightly attenuated the association. Additional adjustment for marital status, education, height, physical activity, alcohol consumption, general health status, chronic bronchitis/emphysema, diabetes mellitus, and coronary heart disease did not change the estimates substantially (data not shown). At follow-up the men were also asked about cancer in Tromsø, but not in Oslo. Seventy-six men reported that they had or ever had cancer. However, adjusting for cancer did not affect the relation between weight change and BMD in men from Tromsø.

In an alternative analysis, we adjusted for information concerning smoking and physical activity collected at baseline. This analysis left the BMI- and age-adjusted results presented in table 2 almost unchanged (data not shown).

In a linear regression model entering percent weight change as a continuous variable and adjusting for age, baseline BMI, and smoking at follow-up, total hip BMD increased by 0.029 g/cm\(^2\) (95 percent confidence interval (CI): 0.023, 0.036) per 10 percent of weight gain. After exclusion of 812 men reporting poor or not very good health, chronic
bronchitis/emphysema, diabetes mellitus, coronary heart disease, cerebral stroke, or mental distress at follow-up, the estimate was virtually unchanged (0.030 g/cm², 95 percent CI: 0.020, 0.041).

As can be seen from figure 1, in general BMD increased with increasing BMI at baseline, but in each category of BMI, BMD was lowest in persons with weight loss and highest in persons with weight gain during follow-up. However, there was no difference between men who gained 5–<10 percent and men who gained 10 percent or more, except for those in the second quarter of baseline BMI (difference of 0.044 g/cm²; p = 0.018).

After adjustment for age and smoking status at follow-up, the prevalence of osteoporosis among participants in the lowest quarter of baseline BMI was 31 percent (95 percent CI: 24, 37) in those who lost 5 percent or more in weight and 4 percent (95 percent CI: 1, 7) in those who gained 5 percent or more in weight. Among men in the upper quarter of BMI with stable weight, none had osteoporosis.

For the Tromsø Study, we used data from the first and fifth surveys (Tromsø I and V). However, 807 of the 880 men had participated in all five Tromsø surveys. We were thus able to calculate a systematic change in weight across all surveys by estimating the regression coefficient on weight versus time for each individual. However, this only moderately increased the predictive value of weight change; the coefficient increased from 0.028 g/cm² to 0.033 g/cm² per 10 percent of weight gain (entering percent weight change as a continuous variable and adjusting for age, baseline BMI, and smoking at follow-up).

There was no effect of interaction between BMI and weight change (p = 0.81) or between smoking and weight change (p = 0.53) on the relation with BMD.

When results were adjusted for baseline BMI, participants with weight loss of 10 percent or more had a total hip BMD which was 0.103 g/cm² lower than that of participants who gained 10 percent or more in weight. This corresponds to a 0.76-SD lower BMD and an estimated 106 percent increased risk of future hip fracture. Correspondingly, these participants had an estimated 51 percent increased risk compared with those with stable weight. Taking BMI into account, men in the lowest quarter of baseline BMI who lost 5 percent or more of their baseline weight had a total hip BMD which was 0.146 g/cm² lower than that

### TABLE 1. Background characteristics of men born in 1924–1936 who participated in two consecutive studies, by weight change category, Oslo and Tromsø, Norway, 1972–2001*

<table>
<thead>
<tr>
<th>Variable</th>
<th>Weight change</th>
<th>p value †&lt;br&gt;Loss of ≥10%</th>
<th>Loss of 5–&lt;10%</th>
<th>Loss or gain of &lt;5%</th>
<th>Gain of 5–&lt;10%</th>
<th>Gain of ≥10%</th>
</tr>
</thead>
<tbody>
<tr>
<td>Baseline information</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Mean age (years)</td>
<td>45.6</td>
<td>45.8</td>
<td>45.4</td>
<td>45.0</td>
<td>44.4</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>Mean weight (kg)</td>
<td>79.9</td>
<td>79.0</td>
<td>77.6</td>
<td>75.3</td>
<td>74.6</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>Mean height (cm)</td>
<td>177</td>
<td>177</td>
<td>177</td>
<td>176</td>
<td>177</td>
<td>0.036</td>
</tr>
<tr>
<td>Mean body mass index‡</td>
<td>25.5</td>
<td>25.2</td>
<td>24.6</td>
<td>24.3</td>
<td>23.8</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>Follow-up information</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Mean weight (kg)</td>
<td>68.2</td>
<td>73.3</td>
<td>77.9</td>
<td>80.9</td>
<td>87.6</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>Mean body mass index</td>
<td>22.7</td>
<td>24.3</td>
<td>25.6</td>
<td>26.8</td>
<td>28.8</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>Current smoking (%)</td>
<td>45</td>
<td>28</td>
<td>21</td>
<td>17</td>
<td>10</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>Ex-smoking (%)</td>
<td>40</td>
<td>52</td>
<td>56</td>
<td>57</td>
<td>75</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>Physically active§ (%)</td>
<td>32</td>
<td>35</td>
<td>38</td>
<td>36</td>
<td>33</td>
<td>0.73</td>
</tr>
<tr>
<td>Alcohol consumption¶ (%)</td>
<td>47</td>
<td>43</td>
<td>48</td>
<td>44</td>
<td>48</td>
<td>0.65</td>
</tr>
<tr>
<td>Not in good health# (%)</td>
<td>56</td>
<td>38</td>
<td>34</td>
<td>31</td>
<td>38</td>
<td>0.001</td>
</tr>
<tr>
<td>Diabetes mellitus (%)</td>
<td>7</td>
<td>11</td>
<td>4</td>
<td>5</td>
<td>5</td>
<td>0.037</td>
</tr>
<tr>
<td>Bronchitis/emphysema (%)</td>
<td>11</td>
<td>14</td>
<td>7</td>
<td>4</td>
<td>6</td>
<td>0.002</td>
</tr>
<tr>
<td>Coronary heart disease (%)</td>
<td>24</td>
<td>23</td>
<td>19</td>
<td>23</td>
<td>30</td>
<td>0.005</td>
</tr>
<tr>
<td>Cerebral stroke (%)</td>
<td>14</td>
<td>10</td>
<td>8</td>
<td>6</td>
<td>8</td>
<td>0.25</td>
</tr>
<tr>
<td>Mental distress (%)</td>
<td>1</td>
<td>7</td>
<td>3</td>
<td>4</td>
<td>4</td>
<td>0.40</td>
</tr>
<tr>
<td>Mean education (years)</td>
<td>9.7</td>
<td>10.2</td>
<td>10.7</td>
<td>10.4</td>
<td>10.4</td>
<td>0.17</td>
</tr>
</tbody>
</table>

* Data on all variables (except age) were adjusted for age.
† p value for differences across groups.
‡ Weight (kg)/height (m)².
§ Engaging in 1 hour or more of hard physical activity per week.
¶ Drinking alcohol at least once per week.
# Presently being in poor or not very good health.
of men in the upper quarter with stable weight, corresponding to an estimated 179 percent increased risk of future hip fracture.

DISCUSSION

In this cohort of middle-aged men, low BMI at baseline and weight loss during the following three decades were both strongly and negatively related to total hip BMD. To our knowledge, this is the first study to have explored these long-term relations in men.

The relations between weight (body build) and BMD and hip fracture are well documented, and being underweight is a strong and independent risk factor for osteoporosis and hip fracture in both men and women (1). In our study we found that total hip BMD, a strong determinant of future hip fracture (14, 15), was modified substantially by weight change during nearly three decades from middle age to older age.

Weight change might act on the skeleton through changes in mechanical loading, changes in mechanical muscle stress, changes in hormone regulation of bone metabolism, and changes in intake of nutrients (2). Weight change might also be related to changes in smoking habits and physical activity, and some chronic diseases are associated with unintentional weight loss. In a US study carried out among men aged 67 years or more, weight loss was associated with increased risk of hip fracture while weight gain was associated with decreased risk during follow-up, both in comparison with stable weight (5). Weight change was calculated from self-reported weight at baseline and recalled weight at 50 years of age. The authors, Langlois et al. (5), discussed whether the increased risk in persons who lost weight might be due to frailty, although their findings persisted after results were controlled for health status. There was limited ability to study these aspects further because of somewhat limited study power (72 hip fractures). On the other hand, in another study among US men, Ensrud et al. (2) reported that the relation between weight change and change in total hip BMD over 1.8 years was just as evident in persons with intentional weight loss as in persons with unintentional weight loss. This is in line with our study, since we showed that after exclusion of men with conditions that might affect both weight change and BMD, the relation between weight change and total hip BMD was unchanged. Although weight loss in elderly persons might be an indicator of chronic diseases and frailty, with an associated increased risk of fracture, it seems evident that there is also a direct and strong relation between weight and weight change and the risk of osteoporosis.

Obesity and weight gain impose increased risk for most chronic diseases (16, 17), whereas an opposite effect is seen for osteoporosis and fracture (1). Although weight gain and

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**TABLE 2.** Mean total hip bone mineral density among men born in 1924–1936 who participated in two consecutive studies, by weight change category, Oslo and Tromsø, Norway, 1972–2001

| Weight change category | BMD* (g/cm²)† | BMD (g/cm²)‡ | Osteoporosis‡ (%)
<table>
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<tr>
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</thead>
<tbody>
<tr>
<td></td>
<td>Mean 95% CI*</td>
<td>Mean 95% CI‡</td>
<td></td>
</tr>
<tr>
<td>Loss of ≥10%</td>
<td>0.899 0.870, 0.927</td>
<td>0.908 0.880, 0.937</td>
<td>15.1</td>
</tr>
<tr>
<td>Loss of 5–&lt;10%</td>
<td>0.927 0.905, 0.949</td>
<td>0.930 0.908, 0.952</td>
<td>14.1</td>
</tr>
<tr>
<td>Loss or gain of &lt;5%</td>
<td>0.963 0.953, 0.973</td>
<td>0.963 0.953, 0.973</td>
<td>6.2</td>
</tr>
<tr>
<td>Gain of 5–&lt;10%</td>
<td>0.999 0.985, 1.013</td>
<td>0.997 0.983, 1.012</td>
<td>2.6</td>
</tr>
<tr>
<td>Gain of ≥10%</td>
<td>1.009 0.996, 1.021</td>
<td>1.007 0.995, 1.019</td>
<td>0.6</td>
</tr>
</tbody>
</table>

* BMD, bone mineral density; CI, confidence interval.
† Results were adjusted for age and baseline body mass index (weight (kg)/height (m)²).
‡ Results were adjusted for age and baseline body mass index and for smoking status at follow-up.

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**FIGURE 1.** Mean total hip bone mineral density (BMD) at follow-up according to body mass index (BMI; weight (kg)/height (m)²) at baseline and weight change among men born in 1924–1936 who participated in two consecutive studies, Oslo and Tromsø, Norway, 1972–2001. Results were adjusted for age and smoking status at follow-up.
high body weight might be beneficial for the skeleton, a stable, healthy weight is recommended for overall purposes (16, 17). However, when considering weight loss interventions, the effect on osteoporosis and fracture should also be included and, if possible, counteracted. Our study also shows that weight in middle-aged men and future weight change are important in the assessment of future risk of osteoporosis.

In our cohort—men with a mean BMI of 24.4 at baseline—average weight change over three decades was a modest gain of 3.9 kg or 5.3 percent, and we found that weight gain of 10 percent or more added little additional BMD than a weight gain of 5–10 percent. The prevalence of obesity was lower in our study population than in, for example, the US population (18). However, we also found an effect of weight loss in the higher quarters of baseline BMI, and there was no effect of an interaction between BMI and weight change on BMD. On the other hand, since BMI is important in itself, the effect of weight loss implies a greater risk in slim men. Concerning hip fracture, increased risk is first and foremost seen in persons with BMI less than 25 (1).

A strength of this study was that weights were measured and thus not subjected to information bias. We were also able to control for a number of possibly confounding factors, including comorbid conditions. A limitation of our study was that we were not able to study the direct relation between change in weight and change in BMD, as this information was available only at follow-up. There were also factors we were not able to control for, such as family history of fracture. In the calculation of which impact the differences in BMD might have on risk of future hip fractures, it is important to note that these are merely estimates based on results from other studies. It was a limitation of our study that we did not have data on fracture endpoints with which to directly assess the risk of fracture. Whereas there were only small differences in background characteristics between persons who underwent BMD measurement and those who did not in Tromsø, the differences were larger in Oslo. In spite of that, the relations between weight change and BMD were similar in the two subpopulations, suggesting that the substantial loss of participants to follow-up in Oslo had no major impact on the results.

In summary, we found that low BMI in middle-aged men was related to the risk of osteoporosis three decades later and that this risk was modulated considerably by later weight change. Weight gain reduced the risk, whereas weight loss increased the risk considerably. Studies on the long-term effects of weight and weight change on the risk of hip fracture are warranted.

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H. E. M., A. J. S., and N. E. planned the study. H. E. M. performed the analyses and wrote the manuscript. All of the authors participated in data collection, reviewed the article critically, and approved the final version.

Members of the NOREPOS Core Research Group: Nina Emaus, Guri Grimes (Tromsø), Haakon E. Meyer, Anne Johanne Søgaard (Oslo), Berit Schei, Siri Forsmo (Nord-Trøndelag), Grethe S. Tell, and Clara Gram Gjesdal (Bergen). Conflict of interest: none declared.

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