Cross-sectional and prospective cohort study of serum 25-hydroxyvitamin D level and obesity in adults – the HUNT Study

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Abbreviations:

BMI: body mass index

CI: confidence interval

HUNT: Nord-Trøndelag Health Study

25(OH)D: 25-hydroxyvitamin D

OR: odds ratio

WC: waist circumference

Abstract

Experimental studies suggest that vitamin D modulates the activity of adipocytes. We examined baseline serum 25-hydroxyvitamin D [25(OH)D] level in relation to prevalent and cumulative incident obesity. A cohort of 25,616 adults aged 19-55 years participated in both the second and third survey of the Nord-Trøndelag Health Study (HUNT 2, 1995-97 and HUNT 3, 2006-2008). Serum 25(OH)D levels at baseline and anthropometric measures at both baseline and follow-up were available in a random sample of 2,460 subjects. Overall, 40% of the 2,460 subjects had a serum 25(OH)D level <50.0 nmol/L, and 37% had a level of 50.0-74.9 nmol/L. The prevalence and cumulative incidence of obesity defined by body mass index (BMI) ≥30 kg/m^2 was 12% and 15%. Lower serum 25(OH)D level was associated with higher prevalence of obesity. In the 2,165 subjects with baseline BMI <30 kg/m^2, serum 25(OH)D level <50.0 nmol/L was associated with a significantly increased odds ratio (OR) for incident obesity at follow-up (adjusted OR = 1.73; 95% confidence interval: 1.24, 2.41). When prevalent and incident obesity were classified according to waist circumference, similar results were obtained. In addition to prevalent obesity, serum 25(OH)D level <50.0 nmol/L was significantly associated with new-onset obesity in adults.

Keywords: body mass index, cross-sectional study, obesity, prospective study, the HUNT Study, vitamin D, waist circumference, 25-hydroxyvitamin D

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Both obesity and vitamin D insufficiency are highly prevalent world wide (1-3). They are associated with each other (4-6), but the nature of the association requires clarification. Obesity has been proposed to lead to low body vitamin D status, although this directional association has been investigated in very few prospective epidemiologic studies (5). Due to its hydrophobic characteristics, vitamin D may move out of the circulation into the large amount of adipose tissue in obese subjects, resulting in low serum levels of 25-hydroxyvitamin D [25(OH)D] even if the total body storage of vitamin D might be adequate (7). There is also a possibility that low vitamin D status increases the risk of obesity (8), since vitamin D appears to modulate the catabolic and anabolic activity of adipocytes in experimental studies (9). A recent study of Colombian children demonstrated that low serum 25(OH)D level was associated with a significant increase in adiposity, measured by changes in body mass index (BMI), skin-fold thickness and waist circumference (WC) during a follow-up period of approximately 3 years (10). By contrast, another prospective epidemiologic study from the United States did not observe any significant associations of 25(OH)D with changes in BMI and adipose tissue values in Hispanic and African American adults after a 5-year follow-up (11). In the present study, we used data from the Nord-Trøndelag Health Study in Norway to investigate the cross-sectional and prospective associations of serum 25(OH)D level and obesity in adults, who were followed up for 11 years on average.

MATERIALS AND METHODS

Study population

The Nord-Trøndelag Health Study (HUNT) is the largest and most comprehensive population health survey in Norway. The adult part of the HUNT invited all inhabitants aged 19 years or older in the county of Nord-Trøndelag in the three separate surveys: HUNT 1 (1984-86), HUNT 2 (1995-97) and HUNT 3 (2006-08) (12). Briefly, HUNT 2 invited about 93,000 adults in 1995-97, and 65,215 subjects participated (response rate: 70%). Among them, 57% (n = 37,059) took part in HUNT 3 in 2006-08 (**Figure 1**). We established a cohort that included all subjects who participated in both HUNT 2 and HUNT 3 and were aged <65 years in HUNT 3 (n = 25,616). This cohort was initially designed to study incident asthma. The age limit was set to decrease misclassification of asthma and chronic obstructive pulmonary disease.

Serum 25(OH)D level

Blood samples were collected from the HUNT 2 participants and stored at -20°C freezers for later use. A 10% random sample of the cohort participants was selected for baseline serum 25(OH)D measurements (n = 2,584) and, of these, 2,505 subjects had sufficient volume of serum. Baseline serum 25(OH)D levels were measured by using LIAISON® 25-OH Vitamin D TOTAL (DiaSorin, Saluggia, Italy), a fully automated antibody-based chemiluminescence assay. The detection range of the assay is 10 to 375 nmol/L. The assay has an intra-assay coefficient of variation of 4% and an inter-assay coefficient of variation of 8%.

Exposure variable and covariates

Baseline serum 25(OH)D levels were classified into three groups: <50.0, 50.0-74.9 and ≥75.0 nmol/L. These are widely used cut-off points in scientific literature (13-15). Other important variables at baseline such as age, smoking habits, education, physical activity, and socioeconomic status were collected in HUNT 2. These covariates were categorized as: age (19-29, 30-39, 40-49, and 50-55 years), daily smoker (yes/no), years of education (<10, 10-12, and ≥13 years), average hours of light physical activity per week (<1, 1-2, and ≥3 hours), social benefit recipient (yes/no), and economic difficulties (yes/no). Social benefit recipients were those who reported receiving any of the public welfare benefits, such as sick pay/rehabilitation/retraining/unemployment/transitional benefits, disability/retirement/widow's pension, family income supplement, and/or other benefits. Subjects who had economic difficulties were identified by their affirmative answer to the question, "During the last year, has it at any time been difficult to meet the costs of food, transportation, housing and such?" People with missing information of smoking, education, physical activity, and socioeconomic status were grouped into an "unknown" category for each variable and included in analyses.

Outcome variables

Body weight, height, and WC were measured by health professionals in both HUNT 2 and HUNT 3 (12). Height and weight were measured with the participants wearing light clothes without shoes; height to the nearest 1.0 cm and weight to the nearest 0.5 kg. Waist circumference was measured horizontally at the height of the umbilicus to the nearest 1.0 cm. Obesity was classified by two definitions in accordance with the recommendations of the World Health Organization and the U.S. National Institutes of Health (1, 16): 1) BMI (weight/height^2) ≥30 kg/m^2 for women and men; and 2) WC ≥88 cm for women and ≥102 cm for men. Prevalent obesity at baseline was classified according to BMI and WC respectively

in HUNT 2. Incident obesity was new-onset obesity during the 11-year follow-up period among subjects who were not obese in HUNT 2.

Statistical analysis

The analysis was based on the random sample of 2,460 subjects who had complete information on serum 25(OH)D levels in HUNT 2, and BMI and WC in both HUNT 2 and HUNT 3 (Figure 1). The cross-sectional association between serum 25(OH)D level and prevalent obesity was evaluated in this 2,460 random sample. The prospective association between serum 25(OH)D level and incident obesity defined by BMI was analyzed in a subsample of 2,165 subject who had BMI <30 kg/ m^2 at baseline. Another subsample of 2,114 subjects who had WC <88 cm for women and <102 cm for men at baseline was used to study the prospective association between 25(OH)D level and incident obesity defined by WC. Baseline characteristics in the 2,460 random sample were compared with the rest of the cohort population (n = 25,616-2,460) using Chi-square tests. We calculated the prevalence of obesity in the cross-sectional analysis and the cumulative incidence of obesity in the prospective analysis according to the categories of serum 25(OH)D levels. Logistic regression models were used to evaluate serum 25(OH)D level in association with prevalent and incident obesity, and crude and adjusted odds ratios (OR) and the 95% confidence intervals (CI) were calculated. The multivariable model included sex, age, smoking, education, physical activity, social benefit, and economic difficulties at baseline as important covariates. The analyses were further stratified by season of blood sample collection (December to May vs. June to November). In addition, 25(OH)D as continuous values in association with BMI, WC and body weight values in HUNT 2, as well as with changes in BMI, WC and body weight between HUNT 3 and HUNT 2 was evaluated in the 2,460 subjects, by using multiple linear

regression analyses (four subjects with 25(OH)D >150 nmol/L were excluded as outliers). All statistical analyses were performed with STATA, release 11.1 (College Station, Texas, USA).

Ethics

The study was approved by the Regional Committee for Medical Research Ethics. All participants gave their informed written consent.

RESULTS

Table 1 shows the baseline characteristics in the 2,460 random sample and the rest of the cohort population (n = 23,156). There were no significant differences in baseline characteristics between the two groups (for all $P > 0.05$).

The prevalence of BMI-defined obesity was 12% at baseline in the random sample, and the cumulative incidence of obesity during the 11-year follow-up period was 15% in the 2,165 subjects without baseline obesity. Older age and difficult economic situation were significantly associated with an increased OR for prevalent obesity at baseline (**Table 2**)**,** whereas higher education and more hours of light physical activity per week were significantly associated with a reduced OR. With regard to incident obesity, difficult economic situation at baseline was the only statistically significant risk factor (OR = 1.45, 95% CI: 1.12, 1.88).

At baseline, 40% of the 2,460 subjects had a serum 25(OH)D level <50.0 nmol/L, 37% had a level of 50.0-74.9 nmol/L, and the rest had 75.0 nmol/L or higher. Lower serum 25(OH)D level was associated with a higher prevalence of obesity defined by BMI $\geq$30 kg/m^2 in the random sample (**Table 3**). A similar trend was observed for cumulative incidence of obesity in the 2,165 subjects without obesity at baseline. **Figure 2** shows the unadjusted associations between serum 25(OH)D levels and prevalent and incident obesity. The smoothed line shows the probability of prevalent or incident obesity for each observed 25(OH)D level. Serum 25(OH)D levels had an inverse association with both prevalent and incident obesity. After adjustment for sex, age, smoking, education, physical activity, social benefit, and economic difficulties at baseline in multivariable logistic regression analysis, serum 25(OH)D level

<50.0 nmol/L was associated with an increased OR of 3.96 for prevalent obesity and an increased OR of 1.73 for incident obesity (Table 3). Apart from 25(OH)D, economic difficulty was the only significant risk factor for incident obesity in the multivariable model (OR = 1.34; 95% CI: 1.02, 1.76)

When obesity was defined by WC (≥88 cm for women; ≥102 cm for men) the prevalence of obesity was 14% in the random sample, and the cumulative incidence of obesity at 11-year follow-up period was 38% in the 2,114 subjects without baseline obesity (by WC criteria). The associations of baseline serum 25(OH)D level with prevalent and incident obesity defined by WC were similar to those for obesity defined by BMI . In the multivariable logistic regression model, serum 25(OH)D level <50.0 nmol/L was significantly associated with 2.6-time increased odds for prevalent obesity and 1.6-time increased odds for incident obesity (**Table 4**). Besides 25(OH)D, male sex was the only variable that was significantly associated with WC-defined incident obesity in the final model (OR = 0.36; 95% CI: 0.29, 0.43).

Significant associations were found between 25(OH)D <50 nmol/L and BMI-defined incident obesity in subjects whose blood samples were collected in December to May (n = 1207; adjusted OR = 2.78; 95% CI: 1.44, 5.36) and in June to November (n = 958; adjusted OR = 1.81; 95% CI: 1.11, 2.94). The corresponding ORs (95% CIs) for WC-defined incident obesity were 1.41 (0.96-2.07) in December to May and 1.93 (1.31, 2.86) in June to November. The interaction between 25(OH)D <50 nmol/L and season of blood collection was not statistically significant for both BMI- and WC-defined incident obesity.

In general, 25(OH)D levels as continuous values were inversely and significantly associated with the values of BMI, WC and body weight at baseline. For follow-up, each 25 nmol/L

decrease of 25(OH)D level was associated with +0.14 kg/m^2 BMI change (P = 0.042) and +0.4 kg body weight change (P = 0.037) in subjects who had normal baseline BMI (<25 kg/m^2); Each 25 nmol/L decrease of 25(OH)D level was associated with +0.5 cm WC change (P = 0.001) in subjects who had normal baseline WC (<80 for women; <94 for men). Among subjects who were overweight or obese at baseline, however, 25(OH)D levels were not significantly associated with changes in BMI, body weight or WC.

DISCUSSION

Our population-based study of Norwegian adults demonstrated that serum 25(OH)D level has a cross-sectional association with prevalent obesity and also a prospective association with incident obesity during a 11-year follow-up period. The cross-sectional association is well-established (4-6), and thought to be due to obesity-related decreases in circulating levels of 25(OH)D. By contrast, our study is one of the few prospective cohort studies to investigate the possible effect of low vitamin D status on change in adiposity and development of obesity. We found a consistent inverse association between baseline 25(OH)D levels and incident obesity defined by either BMI or WC after 11 years of follow-up, and this inverse association was not modified by season of blood sample collection. We also observed inverse associations between 25(OH)D values and changes in BMI, WC and body weight in subjects who had normal baseline BMI or WC.

Our results are in consistence with the longitudinal study of school-age children in Colombia (10) which found that a plasma 25(OH)D level <50 nmol/L was associated with a significant increase in BMI and WC values during an approximate 3-year follow-up period. In addition, both our study and this pediatric study suggest that lower 25(OH)D levels are not only a risk factor for overall obesity defined by BMI but also for central obesity defined by WC. By contrast, the aforementioned prospective epidemiologic study in adults does not support an association between 25(OH)D and change in adiposity measured by the changes in BMI and adipose tissue values after a 5-year follow up (11). Difference in follow-up duration may explain the discrepancy between our current study and this previous adult study. Our findings of a different pattern with change in BMI among baseline BMI subgroups may also explain some of the inconsistent results. A larger change in BMI on average in the normal BMI group

than in the overweight or obese group was a possible reason for this observed difference in our study.

Findings from clinical trials – in terms of the effect of vitamin D supplement on weight control – also are mixed. A Norwegian randomized clinical trial showed no effect of vitamin D supplementation, with 20,000 or 40,000 IU cholecalciferol per week vs. placebo, on weight loss in 334 healthy overweight or obese individuals after 12 months (17). Another trial with supplementation of vitamin D and calcium vs. placebo during a 3-month low caloric diet failed to reduce additional weight in obese women (18). In contrast, another clinical study reported that overweight or obese women responded more positively to hypocaloric diets and lost more body fat if they had a better vitamin D status (19). In addition, in a large trial in the Women's Health Initiative study women who received 1000 mg calcium plus 400 IU vitamin D per day vs. a placebo had significant lower gain in BMI and WC over a 7-year follow-up (20). Taken together, vitamin D, or vitamin D together with calcium supplement, seemed to have an effect on restriction of weight gain in general population, but their effect on weight reduction in subjects who had already become obese may be limited. However, we should bear in mind that control of energy balance is the predominant factor to achieve a weight reduction in obese subjects (21). If energy balance was not equally controlled in the trial groups, the effect of vitamin D could be masked. In addition to this, the relatively short follow-up periods may help explain the observed lack of effect of vitamin D supplement on weight reduction in already obese people.

Although the mechanism for how low serum 25(OH)D level might increase the incidence of obesity is not well understood, our findings have biologic plausibility. *In vitro*, experimental studies suggest that 1,25-dihydroxyvitamin D [$1,25(OH)_2D$] favors lipogenesis and inhibits

lipolysis, and it also modulates the distribution of fat (9, 22, 23). Subjects with clinically important low vitamin D status [defined as serum 25(OH)D level <50 nmol/L] often have secondary hyperparathyroidism and elevated levels of parathyroid hormone and $1,25(OH)_2D$ (15, 24, 25). Parathyroid hormone itself has also been suggested to play a role in fat accumulation by increasing the risk of insulin resistance and inhibiting lipolysis, and it may be mediated by $1,25(OH)_2D$ (8, 26). This might help to explain why serum 25(OH)D levels <50 nmol/L were most strongly associated with cumulative incident obesity in our study and with greater increase in adiposity values in the Colombian pediatric study (10).

Our large population-based study has several strengths. We did a cross-sectional analysis to confirm the previously suggested association between serum 25(OH)D level and prevalent obesity, and we also did a prospective analysis to elucidate the role of body vitamin D status in the development of obesity. Moreover, baseline serum 25(OH)D levels were measured in a large random sample which was highly representative of the cohort population, and there was large variation in the serum 25(OH)D levels (e.g., 10^{th} and 90^{th} percentiles were 31.6 and 89.8 nmol/L, respectively). The mean value (SD) of 25(OH)D in our study [58.8 (23.1) nmol/L] is compatible with those from another Norwegian study and studies from the Scandinavian countries in general (27, 28). In addition, a previous study provided some evidence for a small variation of 25(OH)D levels overtime in adults after taking season into consideration (27). Body weight, height and waist circumference were objectively measured and obesity was classified by standard definitions. The prevalence of obesity in present study was in line with that of in the general population in Norway (29).

Although the independent association between serum 25(OH)D level and incident obesity remained after adjustment for several important covariates, there is a possibility of additional

confounding. For example, the measurement of some covariates, such as physical activity, is not perfect and we also lacked data on energy intake and dietary intake in general. Socio-economic status can be used as a proxy measure for dietary factors since low social status is associated with diet insecurity (30). Our study demonstrated that the association of 25(OH)D with obesity development was independent of low socio-economic status. This confounding, if existed, would need to be quite large, however, to explain the observed ORs. Furthermore, our finding may have limited generalizability to the entire adult population due to the age limit at enrollment. Studies on association between vitamin D and obesity development in elderly people are called for.

To summarize, obesity is widely-recognized to lead to lower vitamin D status in the body due to the fat soluble property of vitamin D and other factors (7). This inference was mainly made from cross-sectional associations (4-6) and a few prospective observations and clinical trials (5, 31, 32). Our study confirmed the cross-sectional association, but also provided new evidence that lower 25(OH)D levels may contribute to new-onset obesity in adults. We also found that central obesity increased more rapidly than overall obesity (38% vs. 15%) and lower serum 25(OH)D level was a risk factor for both overall and central obesity. The detrimental effects of overall obesity have been shown world-wide (33-35). Central obesity is an important component and a risk factor of metabolic syndrome (36). Recent research has also provided evidence for the multiple adverse effects of central abdominal obesity (37-39). Thus, the implication can be profound if improvement of vitamin D status could reduce both overall and central obesity. We suggest that there might be a harmful cycle (i.e., low vitamin D → obesity → low vitamin D) that complicates obesity prevention and treatment efforts. This possibility merits further research in well-designed large prospective studies.

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Table 1. Baseline Characteristics in the Random Sample and Rest of the Cohort Population, the HUNT Study, 1995-97 to 2006-08

	Random sample n = 2,460		Rest of the cohort n = 23,156	
	No.	%	No.	%
Age (yrs)				
19-29	363	15	3,675	16
30-39	729	30	6,887	30
40-49	978	40	8,953	39
50-55	390	16	3,641	16
Sex				
Female	1,342	55	12,851	56
Male	1,118	45	10,305	45
Smoking				
No	1,638	67	15,283	66
Yes	685	28	6,701	29
Unknown	137	6	1,172	5
Education (yrs)				
<10	483	20	4,624	20
10-12	1,327	54	12,307	53
≥13	628	26	6,021	26
Unknown	22	<0.1	204	<0.1
Physical activity (hrs/week)				
<1	556	23	5,270	23
1-2	847	34	8,371	36
≥3	762	31	7,169	31
Unknown	295	12	2,346	10
Social benefit				
Non-recipient	1,585	64	14,464	63
Recipient	446	18	4,161	18
Unknown	429	18	4,531	20
Economic difficulties				
No	1,452	59	13,214	57
Yes	695	28	6,461	28
Unknown	313	13	3,481	15
BMI [kg/m^2, mean (SD)]	25.8 (3.7)		25.8 (3.8)	
WC [cm, mean (SD)]	84.1 (11.2)		84.0 (11.2)	

Abbreviations: BMI, body mass index; HUNT, Nord-Trøndelag Health Study; WC, waist circumference.

Table 2. Baseline Characteristics in Association With Prevalent Obesity at Baseline and Incident Obesity at Follow-up, With Obesity Defined by Body Mass Index ≥30 kg/m^2, the HUNT Study, 1995-97 to 2006-08

	Prevalent obesity at baseline n = 2,460		Incident obesity at follow-up n = 2,165	
	Crude OR	95% CI	Crude OR	95% CI
Age (yrs)				
19-29	1.00	Referent	1.00	Referent
30-39	1.39	0.89, 2.15	0.92	0.64, 1.32
40-49	1.49	0.98, 2.28	0.86	0.61, 1.21
50-55	2.34	1.49, 3.70	0.86	0.57, 1.32
Sex				
Female	1.00	Referent	1.00	Referent
Male	0.95	0.75, 1.22	1.14	0.90, 1.44
Smoking				
No	1.00	Referent	1.00	Referent
Yes	0.91	0.69, 1.20	1.18	0.91, 1.53
Education (yrs)				
<10	1.00	Referent	1.00	Referent
10-12	0.68	0.50, 0.91	0.88	0.65, 1.20
≥13	0.57	0.40, 0.81	0.82	0.58, 1.17
Physical activity (hrs/week)				
<1	1.00	Referent	1.00	Referent
1-2	0.50	0.37, 0.68	0.85	0.62, 1.17
≥3	0.46	0.33, 0.63	0.77	0.56, 1.07
Social benefit				
Non-recipient	1.00	Referent	1.00	Referent
Recipient	1.25	0.92, 1.71	1.21	0.89, 1.65
Economic difficulties				
No	1.00	Referent	1.00	Referent
Yes	1.33	1.01, 1.75	1.45	1.12, 1.88

Abbreviations: CI, confidence interval; HUNT, Nord-Trøndelag Health Study; OR, odds ratio.

Table 3. Baseline Serum 25(OH)D Level in Association With Prevalent Obesity at Baseline and Incident Obesity at Follow-up, With Obesity Defined by Body Mass Index ≥30 kg/m^2, the HUNT Study, 1995-97 to 2006-08

25(OH)D (nmol/L)	No.	Cases	%	Crude OR	95% CI	Adjusted[a] OR	95% CI
Prevalent obesity at baseline (n = 2,460)							
≥75.0	565	27	4.8	1.00	Referent	1.00	Referent
50.0-74.9	922	97	10.5	2.34	1.51, 3.64	2.19	1.40, 3.41
<50.0	973	171	17.6	4.25	2.79, 6.47	3.96	2.58, 6.08
Incident obesity at follow-up (n = 2,165)							
≥75.0	538	58	10.8	1.00	Referent	1.00	Referent
50.0-74.9	825	122	14.8	1.44	1.03, 2.00	1.38	0.99, 1.94
<50.0	802	147	18.3	1.86	1.34, 2.57	1.73	1.24, 2.41

Abbreviations: CI, confidence interval; HUNT, Nord-Trøndelag Health Study; 25(OH)D, 25-hydroxyvitamin D; OR, odds ratio.

[a] Multivariable logistic regression model included sex, age, smoking, education, physical activity, social benefit, and economic difficulties at baseline.

Table 4. Baseline Serum 25(OH)D Level in Association With Prevalent Obesity at Baseline and Incident Obesity at Follow-up, With Obesity Defined by Waist Circumference (≥88 cm for Women; ≥102 cm for Men), the HUNT Study, 1995-97 to 2006-08

25(OH)D (nmol/L)	No.	Cases	%	Crude OR	95% CI	Adjusted[a] OR	95% CI
Prevalent obesity at baseline (n = 2,460)							
≥75.0	565	45	8.0	1.00	Referent	1.00	Referent
50.0-74.9	922	112	12.2	1.60	1.11, 2.30	1.43	0.99, 2.08
<50.0	973	189	19.4	2.79	1.98, 3.93	2.63	1.84, 3.76
Incident obesity at follow-up (n =2,114)							
≥75.0	520	170	32.7	1.00	Referent	1.00	Referent
50.0-74.9	810	298	36.8	1.20	0.95, 1.51	1.13	0.89, 1.44
<50.0	784	340	43.4	1.58	1.25, 1.99	1.56	1.22, 1.99

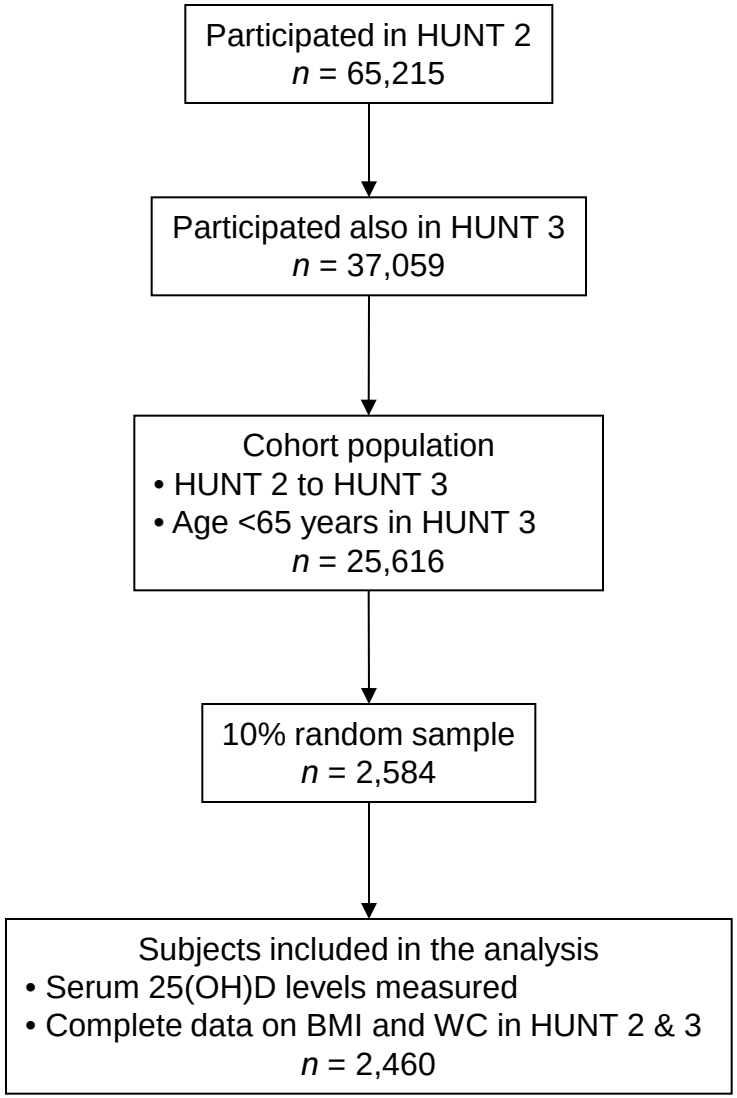
Abbreviations: CI, confidence interval; HUNT, Nord-Trøndelag Health Study; 25(OH)D, 25-hydroxyvitamin D; OR, odds ratio.

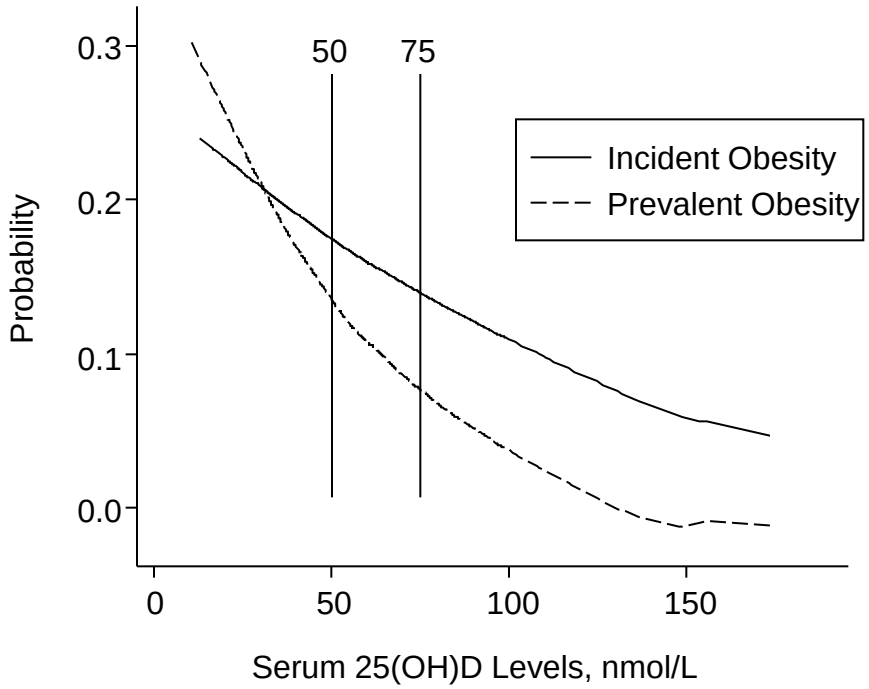
[a] Multivariable logistic regression model included sex, age, smoking, education, physical activity, social benefit, and economic difficulties at baseline.

Figure legends

Figure 1. Flow chart of the study population, the HUNT Study, 1995-97 to 2006-08. Abbreviations: BMI, body mass index; HUNT, Nord-Trøndelag Health Study; 25(OH)D, 25-hydroxyvitamin D; WC: waist circumference.

Figure 2. Unadjusted associations of serum 25(OH)D levels with probabilities of prevalent obesity and cumulative incident obesity defined by BMI ≥30 kg/m^2, the HUNT Study, 1995-97 to 2006-08. The graph was smoothed by using Lowess smoothing method. The vertical lines indicate 25(OH)D cut-off points. Four subjects with 25(OH)D >150 nmol/L were excluded. Abbreviations: BMI, body mass index; HUNT, Nord-Trøndelag Health Study; 25(OH)D, 25-hydroxyvitamin D.


Participated in HUNT 2
n = 65,215
Participated also in HUNT 3
n = 37,059
Cohort population
• HUNT 2 to HUNT 3
• Age <65 years in HUNT 3
n = 25,616
10% random sample
n = 2,584
Subjects included in the analysis
• Serum 25(OH)D levels measured
• Complete data on BMI and WC in HUNT 2 & 3
n = 2,460


0.3
0.2
0.1
0.0
Probability
50
75
Incident Obesity
Prevalent Obesity
0
50
100
150
Serum 25(OH)D Levels, nmol/L