The associations of anxiety and depression symptoms with weight change and incident obesity: The HUNT Study

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Abstract

Objective: To investigate the associations of anxiety and depression symptoms with weight change and incident obesity in men and women.

Design: We conducted a prospective cohort study using the Norwegian Nord-Trøndelag Health Study (HUNT).

Subjects: The study cohort included 25 180 men and women, 19-55 years of age from the second survey of the HUNT (1995-1997).

Measurements: Anxiety and depression symptoms were measured using the Hospital Anxiety and Depression Scale. Weight change was determined for the study period of an average 11 years. Incident obesity was new-onset obesity classified as having a body mass index of ≥30.0 kg/m^2 at follow-up. The association of anxiety or depression with weight change in kilograms (kg) was estimated using linear regression models. Risk ratios (RRs) for incident obesity associated with anxiety or depression were estimated using log-binomial regression.

Results: In men, any anxiety or depression was associated with an average 0.81 kg (95% confidence interval [CI] 0.27–1.34) larger weight change after 11 years compared to those without such symptoms (mean weight change: 5.04 kg versus 4.24 kg). Women with any anxiety or depression had an average 0.98 kg (95% CI 0.49–1.47) larger weight change compared to those without such symptoms (mean weight change: 5.02 kg versus 4.04 kg). Participants with any anxiety or depression had a significantly elevated incidence of obesity (men: RR 1.37, 95% CI 1.13–1.65; women: RR 1.18, 95% CI 1.00–1.40).

Conclusion: We found that symptoms of anxiety and depression were associated with larger weight change and an increased incidence of obesity in both men and women.

Keywords - anxiety, depression, mental health, obesity, prospective, weight change.

Introduction

Obesity is quickly becoming one of the most significant causes of poor health worldwide. Latest reports from the World Health Organization (WHO) estimate 30-70% of adults in European Union (EU) countries are overweight or obese and 10-30% are obese.[1] In Norway, the prevalence of obesity has increased steadily since the 1960's and approximately 20 percent of the adult population is obese today.[2] Health risks associated with obesity include heart disease, cancer, type 2 diabetes and stroke.[3]

Psychological disorders have also been common during the past decades. In the EU it is estimated that 38% of the population suffers from psychological disorders, and anxiety disorders and depression account for the majority of cases.[4] In Norway, it is estimated that about 50% of the population will be affected by at least one psychological disorder during their lifetime.[5] Anxiety and depression are two psychological disorders that occur frequently in obese subjects.[6-8] Studies suggest that obese adults have a significantly higher risk for depression than normal weight participants,[6-8] and recent reviews support a prospective association of obesity-to-depression.[9, 10] One possible explanation for why obese people are more prone to anxiety and depression is because of societal attitudes towards obesity and obesity co-morbid medical conditions.[3]

On the other hand, anxiety and depression may also lead to weight gain.[11] Studies investigating whether depression is a risk factor for obesity have observed mixed results.[9] While some studies have found a modest association,[10, 12, 13] others have not observed an association.[14, 15] Several studies have included a measurement of anxiety,[15-20] with varying results. In addition, gender-

related differences in the association have been inconsistent and should be further investigated.[3] This study aimed to prospectively explore the associations of anxiety and depression symptoms with weight change and obesity development in men and women during an average of 11 year follow-up.

Methods

Study population

The Nord-Trøndelag Health Study (HUNT) is a comprehensive population based study having invited the adult part of the population (19 years or older) living in the Nord-Trøndelag County in Norway.[21] The county is located in central Norway and is characterized by several towns with light industrial activity. The population was approximately 127 000 in 1995.[21] Three health surveys, known as HUNT 1 (1984-1987), HUNT 2 (1995-1997) and HUNT 3 (2006-2008) have been conducted.[22] We established a cohort population including participants who attended both HUNT 2 (baseline) and HUNT 3 (follow-up) and were aged <65 years in HUNT 3 (n=25 668) (Figure 1). This cohort was initially designed for studying incident asthma, and the age limit was set to reduce misclassification of asthma and chronic obstructive pulmonary disease. However by restricting the population to those below 65 years of age we also reduced the potential influence from ill health on anxiety and depression, and body mass index (BMI), e.g. age related ill health may lead to reduced BMI and increased anxiety and depression.

Measure of anxiety and depression symptoms

The HUNT surveys included a self-assessment scale for detecting symptoms of anxiety and depression called the Hospital Anxiety and Depression Scale (HADS).[23] The HADS has good psychometric properties and reliable case-finding abilities across various patient samples and settings.[24] The scale was originally designed for a hospital medical outpatient clinic, however many studies have confirmed its validity in a community setting.[25, 26] It included two sets of seven questions about the participants' feelings in the past week, seven for symptoms of anxiety (HADS-A) and seven for symptoms of depression (HADS-D). The participants were instructed to give their immediate reaction to each question answering on a four point ordinal scale describing

symptom severity, e.g. I still enjoy the things I used to enjoy (0: Definitely as much; 1: Not quite so much; 2: Only a little; 3: Hardly at all). A total score in either set of questions from 0-7 indicated no caseness of anxiety or depression (normal), 8-10 indicated borderline caseness of anxiety or depression (borderline), and 11-21 indicated caseness of anxiety or depression (caseness).[25] Any caseness of anxiety or depression was defined as having either anxiety (HADS-A 11-21) or depression (HADS-D 11-21), or both. Likewise any borderline caseness of anxiety or depression was defined as having either borderline anxiety (HADS-A 8-10), depression (HADS-D 8-10) or both. Although these definitions do not correspond to psychiatric diagnoses of anxiety or depression, for the convenience of writing, the terms borderline anxiety or depression, and anxiety or depression were used instead of borderline caseness of anxiety or depression, and caseness of anxiety or depression, respectively in the following text.

Covariables

Important variables at baseline were collected by self-administered questionnaires, including age (19-29, 30-39, 40-49, 50-55 years), current smoker (yes/no, unknown [5.0%]), insomnia symptoms (no, 1, 2, 3 types of symptoms, unknown [14.4%]), alcohol consumption (frequency of alcohol consumption less than monthly, 1-4 times a month, ≥5 times a month, unknown [8.0%]), duration of physical activity (<1, 1-2, ≥3 hours/week, unknown [10%]), years of education (<10, 10-12, ≥13 years, unknown [0.7%]), economic difficulty (yes/no, unknown [14.4%]) and social benefit recipient (yes/no, unknown [19.0%]). Insomnia included cumulative types of insomnia symptoms (do you suffer from non-restorative sleep, difficulty falling asleep in the last month, or often waking up too early and not being able to get back to sleep in the last month). Economic difficulties included participants who had difficulties meeting the cost of food, transport, or housing. Social benefit included participants who received sick pay, rehabilitation benefits,

retraining benefits, disability pension, family income supplement, unemployment benefits, transitional benefits, widow's pension or any other benefits. Each covariable with missing values were defined by an unknown category and included in our analyses.

Weight change and obesity outcome

Height and weight were measured in both surveys by nurses. Height was measured to the nearest centimetre, and weight to the nearest half-kilogram (kg). Weight change (kg) was weight at follow-up minus weight at baseline. To calculate BMI, body weight in kilograms was divided by the squared value of the body height in meters (kg/m^2). We adopted the definition for obesity (BMI $\geq$30.0 kg/m^2) from the World Health Organisation.[27] Incident obesity was defined as those obese at follow-up among non-obese participants at baseline.

Analysis cohorts

We defined two cohorts to study weight change and incident obesity (Figure 1). To study weight change we excluded 488 participants who had missing information on weight in HUNT 2 or HUNT 3, or anxiety or depression in HUNT 2 (n=25 180 [13 910 men and 11 270 women]). To study incident obesity we further excluded 3 117 participants with missing information on height in HUNT 2 or HUNT 3, or obesity in HUNT 2 (n=22 063 [12 178 men and 9 885 women]).

Statistical methods

We evaluated the associations in men and women separately, as gender might be an important modifier.[3] In the analysis of the cohort of weight change (n=25 180), we studied the associations of baseline characteristics with the prevalence of anxiety or depression. We used linear regression analysis to estimate the mean difference in weight change in kilograms among participants with anxiety or depression compared with those without these symptoms. The models were adjusted

for potential confounding factors such as age, smoking, insomnia, alcohol consumption, physical activity, education, economic difficulty and social benefit. We used cumulative incidence instead of incidence rate to measure obesity development because BMI was only measured at baseline and follow-up. Risk ratio (RR) instead of odds ratio was used to measure the associations of anxiety or depression with incident obesity and was estimated by log-binomial regression in STATA (StataCorp LP, College Station, Texas). We used log-binomial regression due to the tendency of logistic regression and odds ratios to overestimate relative risk when the incidence of an outcome is common (i.e. cumulative incidence of obesity approximately 14%).[28] To evaluate the potential influence of baseline BMI, the analyses were repeated in subgroups stratified by BMI at baseline (normal weight <25.0 kg/m^2, overweight 25.0-29.9 kg/m^2, and obese ≥30.0 kg/m^2) in the linear regression models, and in normal weight and overweight subgroups in log-binomial regression model of incident obesity. Further to this we performed two sensitivity analyses 1) Re-categorization of the exposure by including participants who reported using antidepressant medication at baseline (2.5%) in the category of any anxiety or depression; 2) Excluding those with comorbidities (yes/no) at baseline. Comorbidity included any long-term (at least one year) illness or injury that impaired functioning in everyday life (excluding impairment due to mental health problems). We used STATA 12.0 for windows, for all statistical analyses.

Ethics

The project was approved by the Regional Committee for Ethics in Medical Research. All participants signed informed consent for participation and the use of data in research.

Results

At baseline the prevalence of anxiety was lower in men than in women (3.9% versus 5.8%, p=0.001), but the prevalence of depression was similar between genders (Table 1). HADS scores for anxiety and depression were moderately correlated. Pearson correlation coefficient between anxiety and depression was 0.32 in both men and women. In both genders the prevalence of anxiety and depression was higher in those reporting smoking, insomnia symptoms, fewer hours of physical activity, low education, economic difficulties and those receiving social benefit. The prevalence of anxiety and depression was also higher in older age groups compared to younger age groups. In women, the prevalence of anxiety and depression was U-shaped with alcohol consumption. In men, depression was inversely related to alcohol consumption.

In the analysis of weight change, men with anxiety had an average 0.95 kg (95% CI 0.34–1.55) larger weight change than men without anxiety (Table 2). The means of weight change in men with and without anxiety were 5.19 kg and 4.24 kg, respectively, after an average 11 years follow-up. Women with anxiety had an average 1.12 kg (95% CI 0.60–1.64) larger weight change than women without anxiety. The means of weight change in women with and without anxiety were 5.16 kg and 4.04 kg, respectively. Depression had a slightly weaker influence on weight change than anxiety, in both men and women (Table 2). Any anxiety or depression was associated with an average 0.81 kg (95% CI 0.27–1.34) larger weight change in men compared to men without any anxiety or depression (Table 2). Likewise, women with any anxiety or depression had an average 0.98 kg (95% CI 0.49–1.47) larger weight change than women without any anxiety or depression. While we observed a significant larger weight change (0.60 kg, 95% CI 0.24–0.95) in men with any borderline anxiety or depression, we did not observe a

corresponding change (0.01 kg, 95% CI -0.34–0.37) in women. The p-value for interaction between any borderline anxiety or depression and gender was 0.03. In the sensitivity analyses when participants who used antidepressant medication were included in the category of any anxiety or depression, we found similar results to the main analyses (data not shown). Furthermore, excluding those with comorbidities at baseline did not alter our estimates; the mean weight changes in men and women with any anxiety or depression were 0.82 kg and 0.92 kg, respectively, compared to those without any anxiety or depression.

Subgroup analyses for weight change were also completed after stratification by three categories of baseline BMI (normal, overweight and obese). In both men and women, we found similar patterns in participants with any anxiety or depression compared to those without any anxiety or depression in the normal weight and overweight subgroups. However, in the obese subgroups the coefficients were towards negative but non-significant in both men and women (Supplementary Table 1).

Regarding incident obesity, men with anxiety (RR 1.29, 95% CI 1.04–1.61) and depression (RR 1.39, 95% CI 1.07–1.80) had increased odds of incident obesity compared to men without anxiety or depression (Table 3). A similar although non-significant association of anxiety (RR 1.15, 95% CI 0.97–1.38) and depression (RR 1.20, 95% CI 0.91–1.59) with incident obesity was observed in women. Any anxiety or depression was associated with incident obesity in both men (RR 1.37, 95% CI 1.13–1.65) and women (RR 1.18, 95% CI 1.00–1.40). While the association was also observed in men with any borderline anxiety or depression (RR 1.23, 95% CI 1.07–1.42), it was not observed in women (RR 0.95, 95% CI 0.83–1.09), and the p-value for interaction between any borderline anxiety or depression and gender was 0.01. In the sensitivity analyses when participants who used antidepressant medication were included in the category of

any anxiety or depression, we found similar results (Supplementary Figure 1). Excluding those with comorbidities at baseline did not substantially change the estimates (data not shown).

Subgroup analyses for incident obesity, were completed in participants with baseline normal weight and overweight (Supplementary Table 2). Among men, the RRs for incident obesity associated with any anxiety or depression were 1.89 (95% CI 0.75–4.75, n=4 153) and 1.34 (95% CI 1.12–1.60, n=5 732) in normal weight and overweight participants, respectively. Among women the RRs were 1.50 (95% CI 0.91–2.49) in the normal weight (n=7 457) and 1.21 (95% CI 1.04–1.41) in the overweight (n=4 721) groups.

Discussion

In the present study we observed an association of anxiety or depression with both weight change and risk of incident obesity in men and women.

Previous studies have observed mixed results on the association of anxiety and depression with weight change. In a longitudinal study, high anxiety scores were associated with 1-year weight gain in men,[15] while another study suggested that anxiety and depression were associated with a higher BMI change in women.[16] DiPietro et al. observed that depressed male adults (<55 years of age) gained nearly 3 kg more over a 8 year follow-up period compared to those not depressed, whereas depressed women of the same age group gained less weight than non-depressed.[12] In addition, several other studies have not observed any association of anxiety and depression with body weight change.[14, 15, 29] Our study extends previous observations by investigating the association of anxiety and depression with weight change over an 11 year follow-up period in a large population. We found that those who had any anxiety or depression had a significantly larger weight change than those without these symptoms in both men and women. The lack of consistency and association in the previous studies may be due to methodological variations, such as adolescent and elderly samples, small study size, shorter follow-up duration and varying measures of anxiety and depression. In our study we also found that the association was marked in normal weight and overweight subgroups but weakened in those who were obese at baseline. This loss of association in obese participants may be in part due to intervention (e.g. medical or diet), regression to the mean, and low participation rate.

Our results for the association between depression and incident obesity support a previous meta-analysis of 10 longitudinal studies showing that depression increased the odds of developing obesity (OR 1.58, 95% CI 1.33–1.87).[10] In another review paper including 15 prospective studies, six of the studies found that depression was a predictor of obesity.[9] However, fewer studies have evaluated the impact of anxiety on development of obesity.[15-20] Our findings for the association between anxiety and incident obesity are supported by results from two studies.[17, 20]

In our study the associations of anxiety and depression symptoms with weight change and incident obesity in general did not differ by gender. However, we did observe that the association in men occurred at a lower cut-point of anxiety or depression symptoms than in women. This may be because of reporting biases i.e. men may be less likely to report symptoms compared to women.[30] It may also suggest that a gender-difference in the threshold limit is present in the association of anxiety and depression symptoms with weight change and incident obesity. Previous studies have found mixed evidence for gender modification, however they generally find a stronger association between depression and obesity in women when compared to men.[10]

Our study is one of the largest and longest prospective studies investigating anxiety and depression associated with weight change and obesity. The study design allowed for several key strengths. Using a large prospective cohort, the study had the capability to evaluate the possible gender-specificity of the association, and the associations in subgroups stratified by baseline BMI. The similar results in normal weight and overweight subgroups indicate that the influences by baseline BMI were minimal. Previous research also suggests that time may play a role in the association between anxiety and depression, and obesity.[10] Our study had an average 11 year follow-up period, and a long follow-up period increases the study power to detect an association. In self-reported surveys, measures of weight and height are commonly under and over-reported,[9]

while in the present study we used standardized measures of height and weight recorded at a medical examination to strengthen our outcome. In addition, the use of HADS as a measure of symptoms of anxiety and depression instead of a clinical diagnosis has allowed us to study the possible effect of mild mental distress on weight change and obesity, which may have a large public health impact. Finally, we were also able to exclude participants with comorbidities (e.g. asthma, cancer, heart disease) that might influence anxiety and depression, as well as body weight.

Meanwhile, there are several weaknesses in the present study. Firstly, although we controlled for many covariates there might be some residual confounding, e.g. we did not have detailed information on dietary habits. However, studies suggest that diet is more likely a mediating factor in the depression-obesity association.[9, 10] Secondly, our measures of anxiety and depression symptoms were gauged using questionnaires as opposed to clinical interviews which have greater diagnostic specificity.[9] Furthermore, the ability of the HADS to separate anxiety and depression is uncertain. Recent reviews suggest that the individual HADS subscales should be interpreted with caution, and suggest that the HADS should be used as an overall measure of emotional distress.[31, 32] The suboptimal diagnostic specificity of the HADS may have added misclassification error to our study; however we would expect that this would most likely weaken the associations. To capture those with a clinical diagnosis, we incorporated the use of antidepressant medication in the sensitivity analyses, and the results were similar to the original analysis. Our study may also be subject to selection bias, as participants with anxiety, depression or obesity at baseline were less likely to present at follow-up, potentially weakening the association we observed. Finally, while our study design indicated the direction of anxiety/depression → obesity association, in light of a potential bidirectional association of the

two chronic diseases,[9, 10] future research is warranted to address the bidirectional relationship concurrently.

Our observations that anxiety and depression may increase weight change and the risk of obesity have several possible explanations. These may include over-activity of the hypothalamic-pituitary-adrenocortical (HPA) axis, changes in dietary habits and behaviors, and use of antidepressant medication.[9, 10, 33] One of the important roles of the HPA is to link the nervous system to the endocrine system. It has been suggested that long term activation of the HPA axis by stress (e.g. anxiety and depression) may result in endocrine abnormalities. Such abnormalities include elevated cortisol production which deregulates metabolism promoting accumulation of fat.[34] Changes in diets and behaviors may also account for some of the observed association. Overeating, unhealthy diet preferences, insufficient physical activity and insomnia may be patterns adopted by people with high levels of anxiety and depression,[9, 10, 17] which may lead to subsequent obesity. Some of these factors can be both mediators and confounders of the association we observed. After adjustment for a range of these factors, the independent association between anxiety or depression and weight change and incident obesity remained. Medications such as antidepressants are associated with excessive weight gain.[9] However, our main results were not significantly altered after we excluded those using anti-depressant medication at baseline in our models. This makes us speculate that activation of the HPA may play an important role in long term weight gain and the presentation of obesity found in participants with symptoms of anxiety and depression. Further prospective studies are needed to explore potential mechanisms of this association.

In conclusion, our study suggests that symptoms of anxiety and depression are risk factors for larger weight change and the development of obesity in both men and women.

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Conflict of interest

The authors declare no conflict of interest.

Supplementary information

Supplementary information is available at the *International Journal of Obesity*'s website.

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Table 1. Prevalence of anxiety and depression in relation to baseline characteristics in a cohort population of the Nord-Trøndelag Health Study (HUNT), Norway (n=25 180).

Baseline characteristics	Prevalence of anxiety at baseline[a]				Prevalence of depression at baseline[a]			
Men (n= 11 270)	Men		Women		Men		Women	
Women (n= 13 910)	%	P value[c]	%	P value[c]	%	P value[c]	%	P value[c]
Overall	3.9		5.8		2.3		2.1	
Age (years)		0.46		0.03		<0.001		<0.001
19-29	3.3		4.9		0.9		1.0	
30-39	3.9		5.5		1.7		1.5	
40-49	4.2		6.5		2.7		2.4	
50-55	3.7		6.1		3.9		2.8	
Smoking		<0.001		<0.001		0.15		<0.001
Yes	5.5		8.9		2.6		3.0	
No	3.3		4.4		2.2		1.6	
Unknown	4.0		5.3		2.6		2.6	
Insomnia (types of symptoms)		<0.001		<0.001		<0.001		<0.001
No	2.3		3.6		1.4		1.1	
1	7.6		12.6		4.4		4.0	
2	19.0		20.9		10.5		8.6	
3	33.1		31.7		19.4		16.0	
Unknown	4.1		5.6		2.6		1.8	
Alcohol consumption (frequency)		0.58		<0.001		<0.001		0.01
Less than monthly	4.2		7.0		3.4		2.6	
1-4 times a month	3.7		5.1		2.1		1.8	
≥5 times a month	4.1		6.9		1.7		2.0	
Unknown	4.4		5.5		3.8		2.4	
Physical activity (hrs/wk)		0.009		<0.001		<0.001		<0.001
<1	4.8		8.1		4.3		3.8	
1-2	3.3		5.4		1.6		1.8	
≥3	3.7		4.8		1.6		1.4	
Unknown	4.1		6.4		1.8		1.6	
Education (years)		<0.001		<0.001		<0.001		<0.001
<10	5.3		8.7		4.1		3.3	
10-12	3.7		5.7		2.1		1.9	
≥13	3.1		3.7		1.2		1.4	
Unknown	7.8		13.7		6.3		4.9	
Economic difficulties		<0.001		<0.001		<0.001		<0.001
Yes	6.7		10.1		3.7		3.2	
No	2.5		3.6		1.6		1.5	
Unknown	4.0		5.9		2.4		1.9	
Social benefit		<0.001		<0.001		<0.001		<0.001
Recipient	9.6		10.1		6.0		3.7	
Non-Recipient	2.9		4.1		1.7		1.4	
Unknown	4.1		5.9		2.4		2.1	

[a] Hospital Anxiety and Depression Scale (HADS 11-21)

[c] P value between categories (excluding the unknown category).

Table 2.Difference in weight change (kg) between participants with anxiety or depression and those without, over the follow-up period in the Nord-Trøndelag Health Study (HUNT), Norway (n=25 180).

Men (n=11 270) Women (n=13 910)	No.	Age adjusted difference in weight change (95% CI)	Adjusted[a] difference in weight change (95% CI)	Fully adjusted[b] difference in weight change (95% CI)
HADS-Anxiety[c]				
Men				
Normal	9 800	Ref.	Ref.	Ref.
Borderline	1 033	0.94 (0.55–1.33)	0.84 (0.44–1.23)	0.79 (0.39–1.18)
Caseness	437	1.27 (0.69–1.86)	1.04 (0.43–1.64)	0.95 (0.34–1.55)
P-value for trend		<0.001	<0.001	<0.001
Women				
Normal	11 511	Ref.	Ref.	Ref.
Borderline	1 587	0.13 (-0.23–0.50)	0.08 (-0.29–0.46)	0.03 (-0.34–0.40)
Caseness	812	1.35 (0.85–1.85)	1.24 (0.73–1.76)	1.12 (0.60–1.64)
P-value for trend		<0.001	<0.001	0.001
HADS-Depression[c]				
Men				
Normal	10 324	Ref.	Ref.	Ref.
Borderline	686	0.39 (-0.08–0.87)	0.26 (-0.22–0.73)	0.19 (-0.28–0.67)
Caseness	260	1.03 (0.27–1.78)	0.79 (0.03–1.56)	0.68 (-0.09–1.45)
P-value for trend		0.002	0.03	0.07
Women				
Normal	12 860	Ref.	Ref.	Ref.
Borderline	763	0.60 (0.08–1.11)	0.49 (-0.03–1.01)	0.41 (-0.11–0.93)
Caseness	287	0.87 (0.05–1.69)	0.74 (-0.10–1.58)	0.66 (-0.17–1.50)
P-value for trend		0.003	0.02	0.04
HADS-Any anxiety or depression[c]				
Men				
Normal	9 384	Ref.	Ref.	Ref.
Borderline	1 310	0.74 (0.39–1.09)	0.64 (0.29–1.00)	0.60 (0.24–0.95)
Caseness	576	1.11 (0.60–1.62)	0.90 (0.37–1.43)	0.81 (0.27–1.34)
P-value for trend		<0.001	<0.001	<0.001
Women				
Normal	11 187	Ref.	Ref.	Ref.
Borderline	1 793	0.13 (-0.22–0.48)	0.08 (-0.28–0.43)	0.01 (-0.34–0.37)
Caseness	930	1.19 (0.73–1.66)	1.09 (0.61–1.58)	0.98 (0.49–1.47)
P-value for trend		<0.001	<0.001	0.001

Abbreviations: CI, confidence interval; HADS, Hospital Anxiety and Depression Scale; Ref, reference.

[a] Adjusted for age, smoking, insomnia, alcohol consumption, and physical activity.

[b] Adjusted for age, smoking, insomnia, alcohol consumption, physical activity, education, economic difficulties, and social benefit at baseline.

[c] Normal HADS<8, borderline HADS 8-10, caseness HADS 11-21.

Table 3. Anxiety and depression at baseline in association with incident obesity in the Nord-Trøndelag Health Study (HUNT), Norway (n=22 063).

Men (n=9 885) Women (n=12 178)	No.	Obesity	%	Age adjusted RR (95% CI)	Adjusted[a] RR (95% CI)	Fully adjusted[b] RR (95% CI)
HADS-Anxiety[c]						
Men						
Normal	8 600	1 188	13.8	1.00	1.00	1.00
Borderline	908	160	17.6	1.28 (1.10–1.49)	1.24 (1.07–1.45)	1.21 (1.04–1.41)
Caseness	377	76	20.2	1.46 (1.19–1.80)	1.36 (1.09–1.68)	1.29 (1.04–1.61)
P-value for trend				<0.001	<0.001	0.002
Women						
Normal	10 106	1 352	13.4	1.00	1.00	1.00
Borderline	1 386	187	13.5	1.01 (0.87–1.16)	0.99 (0.85–1.14)	0.94 (0.81–1.09)
Caseness	686	123	17.9	1.34 (1.13–1.58)	1.27 (1.06–1.51)	1.15 (0.97–1.38)
P-vale for trend				0.005	0.04	0.37
HADS-Depression[c]						
Men						
Normal	9 085	1 276	14.1	1.00	1.00	1.00
Borderline	579	98	16.9	1.23 (1.02–1.49)	1.17 (0.96–1.41)	1.14 (0.94–1.38)
Caseness	221	50	22.6	1.65 (1.28–2.12)	1.61 (1.26–2.07)	1.39 (1.07–1.80)
P-value for trend				<0.001	<0.002	0.008
Women						
Normal	11 313	1 517	13.4	1.00	1.00	1.00
Borderline	637	102	16.0	1.19 (0.99–1.43)	1.13 (0.94–1.36)	1.06 (0.88–1.28)
Caseness	228	43	18.9	1.40 (1.06–1.84)	1.29 (0.98–1.70)	1.20 (0.91–1.59)
P-vale for trend				0.003	0.04	0.18
HADS-Any anxiety or depression[c]						
Men						
Normal	8 253	1 123	13.6	1.00	1.00	1.00
Borderline	1 133	197	17.4	1.29 (1.13–1.49)	1.26 (1.09–1.45)	1.23 (1.07–1.42)
Caseness	499	104	20.8	1.54 (1.29–1.85)	1.43 (1.19–1.73)	1.37 (1.13–1.65)
P-value for trend				<0.001	<0.001	0.001
Women						
Normal	9 847	1 310	13.3	1.00	1.00	1.00
Borderline	1 552	211	13.6	1.02 (0.89–1.17)	1.00 (0.87–1.15)	0.95 (0.83–1.09)
Caseness	779	141	18.1	1.36 (1.16–1.59)	1.29 (1.09–1.52)	1.18 (1.00–1.40)
P-vale for trend				0.001	0.02	0.20

Abbreviations: CI, confidence interval; HADS, Hospital Anxiety and Depression Scale; RR, risk ratio.

[a] Adjusted for age, smoking, insomnia, alcohol consumption, physical activity and education.

[b] Adjusted for age, smoking, insomnia, alcohol consumption, physical activity, education, economic difficulties, and social benefit at baseline.

[c] Normal HADS<8, borderline HADS 8-10, caseness HADS 11-21.

462 Figure 1. Flow chart of study cohort.
463

464 Abbreviations: HADS, Hospital Anxiety and Depression Scale; HUNT: Nord-Trøndelag Health
465 Study; n, number of observations.

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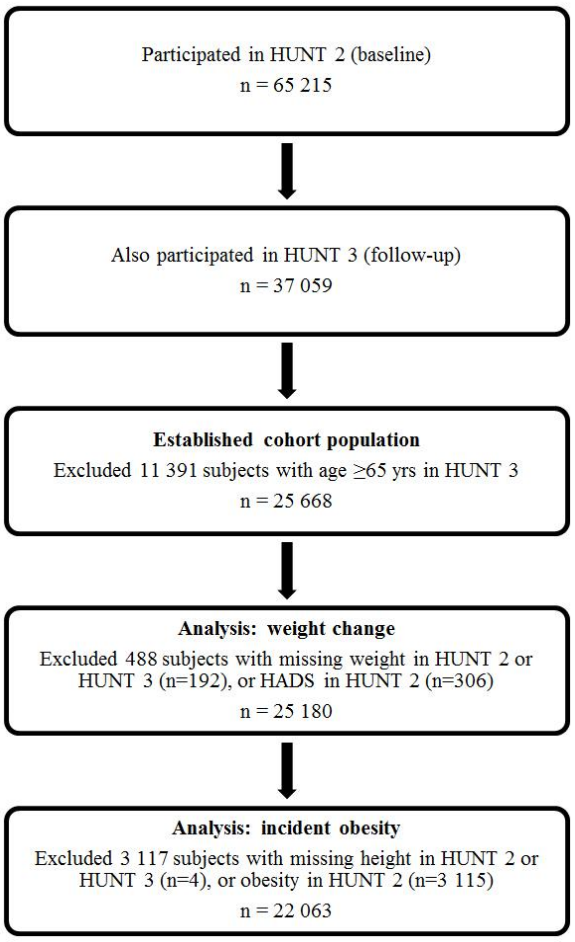

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