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Original Research

Exploring Dietary Intake in Adults with Severe Obesity and Associations with the *FTO* rs9939609 Genotypes

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A B S T R A C T

Background: Few have studied the associations between rs9939609 genotypes in the obesity candidate locus *FTO* and energy and nutrient intakes and meal frequencies in adults with severe obesity. We are unaware of studies that have assessed adherence to key dietary recommendations in this population, at least in Norway. Increased knowledge of genotype associations with dietary factors could improve personalized obesity therapy.

Objectives: The present study aimed to explore how the rs9939609 genotypes associate with dietary variables and adherence to key dietary recommendations in a sample of adults with severe obesity.

Methods: A cross-sectional observation study designed to have similar numbers of participants with genotypes TT, AT, and AA included 100 patients (70% women) with median (25^{th}, 75^{th} percentile) age 42 (32, 50) y and BMI 42.8 (39.5, 46.4) kg/m^2. We assessed intakes of food groups, energy, and macro- and micronutrients from three 24-h dietary recalls and meal frequencies. Genotype associations were analyzed using regression analyses. Reported intakes were evaluated against national diet recommendations.

Results: Using a significance level of 0.01, we found no genotype associations with energy intake, energy density, adherence to recommendations, or meal frequency but tendencies of associations with energy adjusted protein intake (AA > AT, $P = 0.037$; AT > TT, $P = 0.064$), food groups *milk and cream* (AT > TT, $P = 0.029$), and *Mixed dishes* (AA > TT, $P = 0.039$). Few participants complied with recommendations for intakes of whole grains (21%), fruits and vegetables (11%), and fish (37%); however, 67% followed the recommendation to limit added sugar. Less than 20% had recommended intakes of vitamin D and folate.

Conclusions: In our patients with severe obesity, we found tendencies of associations between the *FTO* rs9939609 genotypes and diet but no significant associations at the 0.01 level and below. Few met key food-based diet recommendations, suggesting that the food habits in this population pose an increased risk of nutrient deficiencies. *Curr Dev Nutr* 2023;xx:xx.

Keywords: obesity, energy intake, nutrient intake, food group intake, adherence to dietary guidelines, rs9939609

Introduction

The prevalence of obesity is still on the rise [1], and poor diet and high BMI are among the top risk factors associated with deaths worldwide [2]. Obesity is the result of a sustained positive energy balance, most likely from a higher energy intake (EI) [3]. Energy dense diets are associated with weight gain [4], and health authorities in many countries recommend consuming mainly foods with low to moderate energy density (ED) [5]. In practical terms, this is a diet high in whole grains, vegetables, and fruits and low in ultraprocessed or palatable foods with high saturated fat and added sugar content [5].

Abbreviations: ED, energy density; E%, energy percent; *FTO*, fat mass and obesity-associated gene.

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Eating is a voluntary behavior influenced by energy-homeostatic and non-homeostatic biological systems and non-biological factors. In an environment of high availability of, and easy access to, palatable energy dense foods, the non-homeostatic system easily overrides the homeostatic hunger and satiety system [6]. The dopamine effect associated with the pleasure of eating might lead to overeating and overweight in individuals who meet emotional needs through food rewards [7]. Breakfast skipping, frequent eating occasions as in "grazing," and eating in the evening are associated with a higher EI [8]; however, associations between meal frequency, timing, and body weight are unclear [9,10]. Food insecurity and other forms of social inequality affect dietary choices and health in a negative manner [11,12].

Among the multitude of factors that contribute to obesity is also heredity. Variants in obesity-associated genetic loci act alone and together with environmental/societal contributors in increasing their obesity-promoting risk [13]. Variants in the fat mass and obesity (*FTO*) locus were the first to be associated with obesity [14–16] and remain the genetic variants with the highest impact on BMI [13]. The effect of the *FTO* locus risk alleles on EI and increased body weight has been studied extensively [14,17]. Variants in the *FTO* locus exert their obesity-promoting effects through altered brain food–cue responses, a preference for energy dense foods, lower satiety responsiveness, loss of control overeating, eating (more palatable, energy dense foods) in the absence of homeostatic hunger, and increased EI [17–21]. However, in adults, there is no clear evidence for a genotype effect on diet and EI [17,20,21].Variants in the *FTO* locus have been associated with increased eating occasions [22] and meal skipping [23].

Given the genotypes' association with increased intakes of energy dense foods and energy and its association with hedonic and addictive eating behavior, it is clinically relevant to explore how these factors might play out in patients with severe obesity. Moreover, it is clinically relevant to assess to how well they follow official health-promoting dietary recommendations. The present study aimed to explore how the *FTO* rs9939609 genotypes associate with dietary variables and adherence to key dietary recommendations in a sample of adults with severe obesity.

Methods

Participants and study design

Between 2013 and 2015, adults without diabetes with BMI $\geq$35 kg/m^2, referred to the university hospital's outpatient obesity clinic, were recruited for a cross-sectional metabolic and genetic study (Figure 1) [24]. The aim was a sample of $\geq$100 individuals in which an equal number of participants had zero, one, or two copies of the *FTO* rs9939609 risk allele in order to test for associations between genotype and metabolic and diet factors.

Patients who participated in an introductory information day reported usual meal frequencies (*breakfast*, *lunch*, *warm dinner*, *evening meal*, *other meal*, and *night-food*, the latter defined as food consumption between midnight and 06:00 h) and habitual vitamin, mineral, and omega-3 containing supplement use as asked in the Trøndelag Health Study, HUNT3 Questionnaire 2 [25]. This information was retrieved from the participants' electronic medical records.

Participants gave three 24-h diet recall interviews to dietitians and student dietitians over a month long period. The two first interviews were conducted in person ~1 wk apart, and the third was a telephone interview. The first recall day was a Sunday; the others were weekdays. The dietitians and participants used a picture booklet [26] with portion sizes as an aid to determine amounts eaten.

Food and nutrient intakes were calculated using database AE-10 in KBS version 7.3 (Kostberegningssystemet KBS, University of Oslo, Norway); for the whole grain database, A-18 was used. From subgroups of food categories, we made new categories to compare participants' intakes with the Norwegian food-based recommendations [5]. The food categories with subgroups are described in Supplementary Material (**Supplemental Methods**), including Supplemental Table 1.

The ED of a food category is energy content (kcal) of the food category/amount consumed (g) $\times$ 100.

EI was compared with the participants' REE measured by indirect calorimetry on the first test day, reported elsewhere [27], and to a sedentary reference person of similar age [5].

Food and nutrient variables represent averages of the available 24-h diet recall interviews for each participant in the analyses. We recoded frequencies and amounts given in the self-administered report, with factors for use in regression analyses as described in [28].

On the test days, participants' height and weight were measured following procedures reported elsewhere [24].

Ethics

The study was approved by the Norwegian Regional Committees for Medical and Health Research (2013/642/REK-midt) and conducted according to the guidelines of the Declaration of Helsinki. All volunteers gave their signed informed consent to participate.

Statistics

Statistical analyses were performed with the STATA package, versions 16 and 17 (StataCorp). We inspected variable distributions by histogram, examined normality with the Shapiro-Wilk test, and present medians, 25th, and 75th percentile values.

For 1-way analysis of variance of the participant characteristics, we used the Kruskal-Wallis method, robust regression ("rreg") [29], and Fisher exact test for biological sex.

Associations between the genotypes and dietary variables were analyzed with robust regression. For "oily fish," we used ordinary least squares regression with robust standard errors because the "rreg" function could not be used due to the small number of observations. Total EI was analyzed with adjustment for body weight and REE in two different models, and micronutrient intakes were analyzed with and without adjustment for total EI. For all regression analyses, genotype had codominant coding, that is, a factor with three levels denoted by the number of minor risk alleles present, thus 0, 1, or 2 for genotypes TT, AT, and AA, respectively. Due to the multiple testing burden, we consider a P value $\leq$ 0.01 as a significant association in this exploratory study.

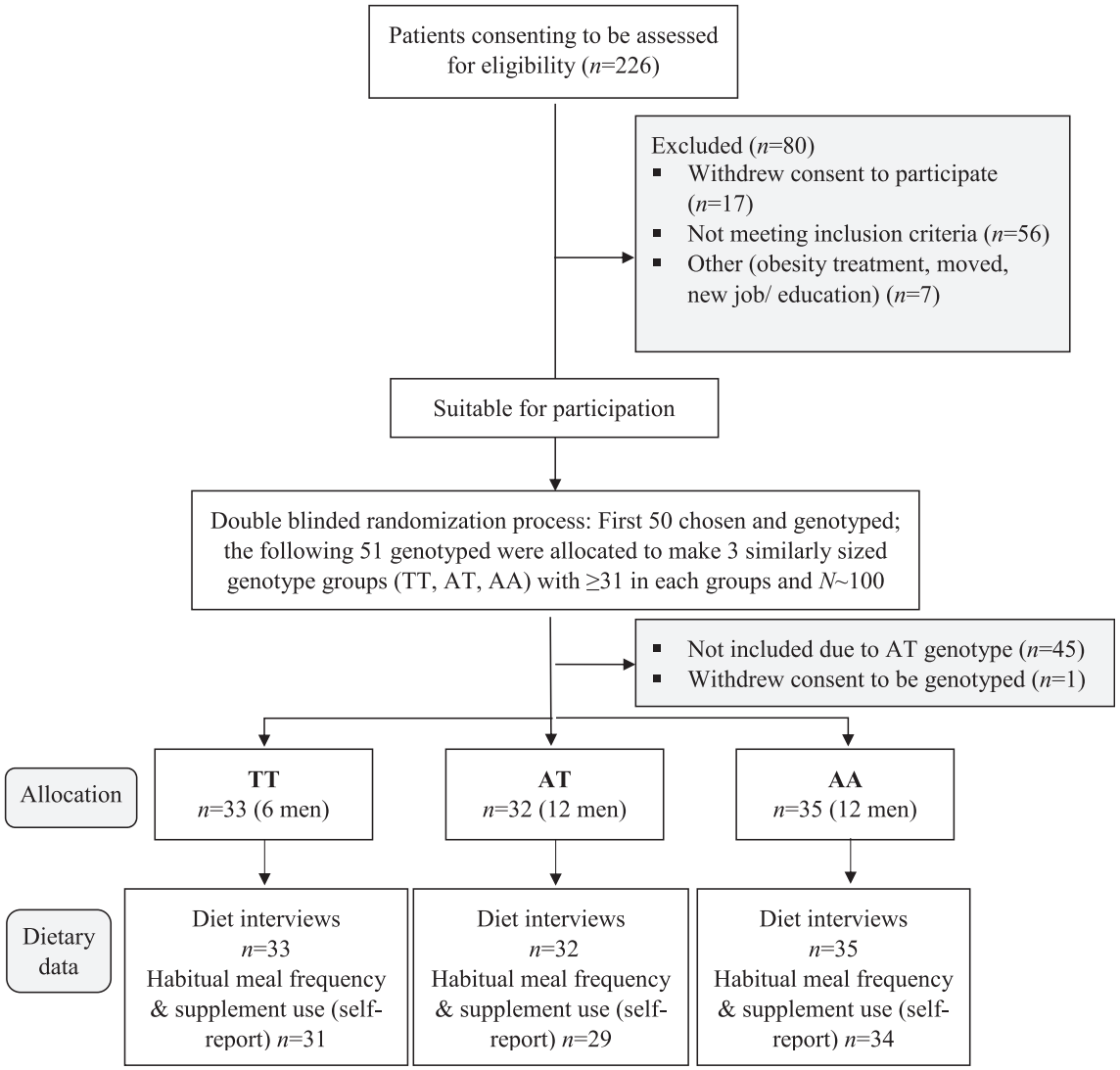

FIGURE 1. Flow diagram, participant selection, and group allocation.

Results

Participants

All 100 participants completed at least one 24-h diet recall interview, 97 completed 2, and 89 all 3. Participant characteristics were the same in all genotype groups (Table 1) (*P* values not shown).

Energy and nutrient intake

Median EI was 81% of the reference value for daily EI [5] and 20% above participants' REE (14%, 25%, and 22% for genotype groups TT, AT, and AA, respectively) (Table 2). Median energy percent (E%) for protein and fat were in the higher end of the recommended ranges (Table 2). For one-third of the participants, E% for fat exceeded the recommended maximum (Figure 2A). Eight participants got >50% of their EI from fat, and 93 did not meet the recommendation to limit saturated fat to <10 E%. Median 43 (38, 47) E% for carbohydrate was below the recommended range. About one-third of participants had carbohydrate intakes within the recommended E% range. The majority met the recommendation of <10 E% added sugar, whereas less than one in five met the minimum recommendation for fiber (Figure 2A).

TABLE 1
Characteristics of study population, by genotype and for the whole sample

Females/males, *n*	All *n* = 100 70/30	TT *n* = 33 27/6	AT *n* = 32 20/12	AA *n* = 35 23/12
		Median (25th, 75th percentile)		
Age, y	42 (32, 50)	39 (30, 46)	44 (37, 51)	44 (31, 53)
Height, cm	170.0 (164.5, 177.0)	169.0 (166.0, 175.0)	173.0 (164.5, 180.0)	169.0 (164.0, 178.0)
Weight, kg	122.4 (109.9, 141.2)	119.2 (106.7, 140.0)	124.0 (110.5, 140.7)	126.6 (115.4, 142.4)
BMI, kg/m^2	42.8 (39.5, 46.4)	41.7 (38.5, 46.5)	41.0 (38.0, 45.4)	43.2 (40.5, 47.4)
REE[1], kcal/d	1609 (1454, 1806)	1600 (1454, 1725)	1587 (1442, 1904)	1635 (1492, 1805)

REE: resting energy expenditure.
[1] Measured by indirect calorimetry, *n* = 98.

TABLE 2
Intake per day of energy and macronutrients[1] for all participants and genotype groups

	Recommendation[2]	All $n = 100$	TT $n = 33$	AT $n = 32$	AA $n = 35$
		Median (25^{th}, 75^{th} percentile)			
Energy, kcal	—	1955 (1549, 2337)	1896 (1386, 2209)	1948 (1665, 2180)	1963 (1541, 2499)
Protein, g	—	86 (68, 104)	79[3] (60, 99)	93[3,4] (74, 110)	87[4] (66, 104)
Total fat, g	—	82 (61, 103)	83 (60, 94)	79 (67, 109)	82 (60, 108)
SFA, g	—	31 (23, 40)	31 (19,36)	34 (24, 40)	30 (23, 45)
MUFA, g	—	29 (21, 40)	28 (21, 34)	28 (22, 42)	31 (21, 40)
PUFA, g	—	12 (9, 16)	12 (9, 16)	13 (10, 16)	12 (8, 15)
ω-3 PUFA, g	—	2.1 (1.6, 2.9)	2.0 (1.6, 3.0)	2.1 (1.8, 2.7)	2.2 (1.5, 3.1)
CHO, g	—	191 (146, 249)	192 (136, 245)	188 (152, 230)	203 (152, 267)
Added sugar, g	—	34 (15, 60)	38 (16, 62)	29 (14, 46)	37 (14, 86)
Fiber, g	≥25	18 (15, 23)	18 (14, 22)	20 (16, 24)	18 (14, 22)
Protein, E%	10–20	18 (15, 21)	18 (15, 20)	19 (16, 22)	17 (15, 21)
Total fat, E%	25–40	36 (33,42)	36 (33, 42)	36 (33, 42)	37 (32, 42)
SFA, E%	<10	14 (12, 16)	14 (11, 15)	15 (13, 16)	14 (12, 16)
MUFA, E%	10–20	13 (11, 15)	13 (12, 17)	13 (11, 15)	13 (11, 15)
PUFA, E%	5–10	5 (4, 7)	6 (5, 7)	5 (4, 7)	5 (4, 7)
ω-3 PUFA, E%	≥1	1.0 (0.7, 1.3)	1.1 (0.7, 1.5)	1.0 (0.7, 1.2)	0.9 (0.7, 1.3)
CHO, E%	45–60	43 (38, 47)	43 (38, 49)	43 (37, 46)	43 (36, 48)
Added sugar, E%	<10	7 (4, 11)	8 (4, 11)	6 (4, 10)	7 (4, 12)
Fiber, E%	—	1.8 (1.5, 2.4)	1.8 (1.5, 2.4)	2.0 (1.6, 2.4)	1.8 (1.4, 2.3)

E%, energy percent. [1]Intakes from supplements are included. [2]Nordic nutrition recommendations, 2012 (40). [3]$P = 0.059$ for AT compared with TT, and $P = 0.064$ with adjustment for total energy intake (EI) in pairwise comparisons of genotype effect, analyzed with robust regression. [4]$P = 0.281$ for AA compared with AT, and $P = 0.037$ with adjustment for total EI in pairwise comparisons of genotype effect, analyzed with robust regression.

Using a significance level of 0.01, there was a tendency toward a higher protein intake in the AT genotype compared with the other groups, but we did not find significant associations between genotype and total energy or macronutrient intake or energy distribution (Table 2). Controlling for sex did not change the results. For micronutrients, genotype AT was associated with significantly higher intake of vitamin D, riboflavin, and magnesium relative to genotype AA and a higher phosphorus intake than both AA and TT (Supplemental Table 2).

Food intake and ED

There were tendencies for a higher intake of the food categories *milk and cream* among the AT compared with TT participants and of *Mixed dishes* among AA compared with TT participants (Table 3). The AA participants tended to consume *milk and cream* with a higher ED than the AT participants (82 kcal/100 g compared with 61 kcal/100 g, $P = 0.031$), and likewise for *vegetables* (38 kcal/100 g compared with 34 kcal/100 g, $P = 0.050$). The AT participants tended to consume *grains* with a higher ED relative to participants with genotype TT ($P = 0.079$). Controlling for sex did not change the results. Sugar-sweetened beverages accounted for one-fifth of the intake of added sugar, but intake did not differ between the genotype groups. A quarter of participants reported habitual daily or occasional use of cod liver oil, ω-3 capsules, vitamins, or mineral supplements, and 80% never used an ω-3 supplement (Supplemental Figure 1).

Participants' adherence to nutrient and food intake recommendations

The intake of foods or food groups in which the Norwegian guidelines quantify a recommended intake did not differ between genotypes (Table 4). Median daily intake of "*5-a-day*" (fruits and vegetables) was about half of that recommended (Table 4). Eleven percent (nine women and two men) met the recommendation of "*5-a-day,*" and 21% (18 women and three men) met the *whole grain* recommendation (Figure 2B).

Median daily intake from the major food category *fish and seafood* was 17 g, which, converted to weekly intake, is ~117 g. Median intake of oily fish was 0 g, mean (standard deviation) intake was 9 (28) g. Less than half achieved the minimum recommended fish intake, and 1 in 10 consumed the recommended amount of oily fish (Figure 2B).

Median daily intake of *meat and meat products* was 132 g (Table 3) of which 88 g were red and processed meats (Table 4), which is above the maximum recommended intake. Less than half of participants met the recommendation of limiting red and processed meats to <500 g/wk (Figure 2B).

Meal frequency

Participants had an average of 3.3 meals/d, and the number was the same for all genotype groups. Sixteen percent answered that they rarely/never ate *breakfast*, and slightly more than half reported eating *breakfast* every day (Figure 3). Nearly 20% reported *night-food* ≥1–2 d/wk. Almost half rarely or never had in-between meals (*other meal*). There were no differences between genotype groups with regard to the average weekly frequencies of the different meals.

Discussion

In the present study of patients with severe obesity selected for equal representation of the three rs9939609 genotypes, the obesity-related risk allele (A) was not associated with energy, fat or carbohydrate intake, or energy dense foods. It was associated with a higher intake of a few food categories. The AT variant had a weak positive association with protein intake. As a group, the participants did not adhere well with national dietary recommendation.

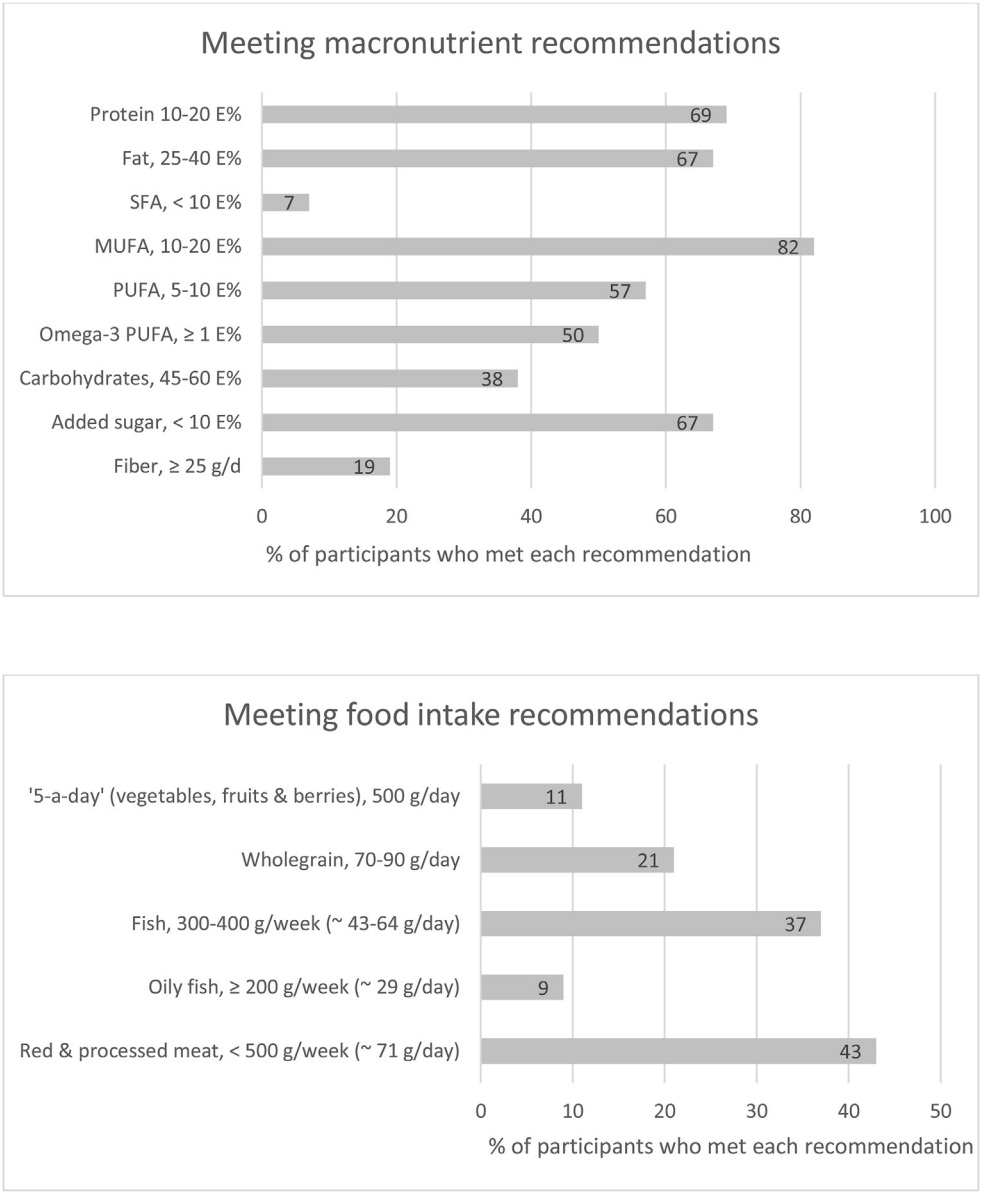

FIGURE 2. Proportion of participants that met the recommended percent energy (E%) distribution of macronutrients (A), and recommended amounts of whole grain and selected food categories (B).

A limitation of our study is that the intra- and interindividual variations in food intake require a larger sample than ours, and we cannot rule out the possibility of type II statistical errors. It was therefore a strength that for each participant we estimated EI from 3 24-h recalls from nonconsecutive days of the week, which has been found to reduce the error in estimating EI [30]. Nearly 90% completed all three 24-h recall interviews. Moreover, we reported our data for each genotype of the *FTO* variant rs9939609, a practice recommended by others [31]. Another strength of our study was the context of evaluating genotype associations in individuals who had already developed severe obesity.

Our findings on EI, macronutrient intakes, and composition of the diet are similar to what some have reported [31–34], though, in contrast to studies among nonobese adults [35], adults with type 2 diabetes and overweight/obesity [22] and children and adolescents [36] found positive associations between the risk allele (A) and EI. Among physically active adults, AA carriers had significantly higher cognitive restraint scores than TT carriers [37], and this and other compensatory behaviors could potentially attenuate or negate any weight-increasing dietary behavior associated with the risk allele, thus partially explaining the weak negative association between EI and *FTO* rs9939609 risk allele obtained in two systematic reviews and meta-analyses [21,38].

Our positive finding of higher intakes of riboflavin, phosphorus, and magnesium among AT participants likely reflected that they tended to consume more milk products as well as grains with a higher ED. The non-findings as to genotype associations with food intake are similar to those of Hasselbalch et al. [39] and are supported by a prospective Swedish cohort of adults >44 y of age [33].

Given the high response rate in our study, it is worthwhile to note the small proportion that seemed to follow national dietary guidelines and met national diet recommendations for which we

TABLE 3
Intakes (g/d) from main food categories, for all participants and by genotype

	All $n = 100$	TT $n = 33$	AT $n = 32$	AA $n = 35$
	Median (25th, 75th percentile)			
Total, g	2927 (2464, 3347)	2888 (2459, 3463)	2931 (2396, 3817)	2942 (2678, 3194)
Bread, g	125 (88, 171)	126 (91, 154)	128 (88, 191)	122 (75, 167)
Grains, g	32 (0, 83)	30 (0, 53)	25 (0, 106)	35 (0, 90)
Cakes, g	13 (0,38)	14 (0, 43)	14 (0, 46)	8 (0, 27)
Potato, g	37 (0, 75)	20 (0, 73)	38 (14, 66)	52 (0, 83)
Vegetables, g	129 (70, 204)	134 (62, 206)	109 (70, 174)	132 (72, 227)
Fruit and berries, g	110 (21, 217)	108 (22, 293)	123 (54, 217)	87 (13, 167)
Meat and meat products, g	132 (84, 184)	133 (88, 165)	137 (83, 178)	130 (83, 207)
Fish and seafood, g	17 (0, 66)	23 (0, 62)	24 (1, 72)	5 (0, 63)
Egg, g	0 (0, 26)	0 (0, 30)	4 (0, 43)	0 (0, 17)
Milk and cream, g	246(87, 395)	157[1] (69, 307)	322[1](112, 495)	230 (118, 311)
Cheese, g	32 (12, 62)	35 (12, 47)	33 (12, 76)	27 (13, 67)
Fats, g	17 (7, 31)	19 (7, 31)	19 (9, 32)	14 (7, 22)
Sugar and sweets, g	7 (0, 33)	6 (0, 30)	5 (0, 21)	10 (1, 50)
Beverages, g	1572 (1257, 2049)	1580 (1303, 2160)	1471 (1060, 2135)	1643 (1339, 2014)
Various, g	24 (2, 51)	24 (3, 57)	23 (5, 39)	23 (0, 61)
Mixed dishes, g	0 (0, 100)	0[2] (0, 58)	4 (0, 97)	48[2] (0, 100)
Supplements, g	19 (9, 27)	12 (7, 22)	23 (15, 30)	19 (11, 26)

[1] $P = 0.029$ for AT compared with TT in pairwise comparisons of genotype association, analyzed with robust regression. Result did not change with adjustment for sex. [2] $P = 0.039$ for AA compared with TT in pairwise comparisons of genotype association, analyzed with robust regression. Result did not change with adjustment for sex.

have quantifiable recommendations. The data was collected only a few years after the revised Norwegian dietary guidelines had been published [5]. This also happened to be toward the later part of the "high-protein, low-carb" diet trend in the country. That diet got heavy publicity in Norwegian media, and much less attention was given to the new food-based dietary guidelines. As such, it is not surprising that the carbohydrate intake was rather low, resulting in a low intake of whole grains, fiber, and folate, and that the intakes of protein and fat were proportionally higher in our sample. The participants chose red and processed meats rather than fish and seafood as major protein sources, which contributed to a fat intake that is contrary to recommendations (particularly with regard to fat type). This is particularly worrisome because it likely translates into a low intake of not only ω-3 fat but also vitamin D. In Norway, dietary supplements of ω-3 fat and vitamin D are recommended if intake of oily fish and/or vitamin D-fortified foods is low. It did not seem as though the participants compensated for the low intake by using dietary supplements, as the great majority never took any, as also indicated by the reported vitamin D intake. Vitamin D intake in our participants was similar to that reported among adults in other Nordic countries who did not use vitamin D supplements, including cod liver oil [40]. Although we cannot compare directly, our results are in stark contrast to what adult Norwegians self-reported in a 2020 national survey [41]. In that survey, 79% took some kind of dietary supplement, and 63% took cod liver oil or ω-3.

The low compliance with following the "*5-a-day*" recommendations in our participants is of concern, as those food groups are not only good, low energy sources of dietary fiber, but are the major or even sole diet sources of several micronutrients in Norway. The intake of non-vegetarian sources of folic acid was also low. As a group, the participants likely had a low to marginal intake of several essential nutrients. The findings highlight the need to assess reasons for the dietary choices, to provide sound nutrition guidance to the patients, and to target this population group in particular in nutrition campaigns. Our findings on median intakes of "*5-a-day*" are in contrast with studies among

TABLE 4
Daily intake of "5-a-day," whole grain, fish, and red and processed meat for all participants and genotype groups, along with recommended intake

Food group (g/d)	Recommended intake[1]	All $n = 100$	TT $n = 33$	AT $n = 32$	AA $n = 35$
		Median (25th, 75th percentile)			
"*5-a-day*" (vegetables, fruits, and berries)[2]	≥ 500 g/d	261 (134, 384)	317 (109, 388)	261 (134, 415)	213 (161, 333)
Vegetables[3]	≥ 250 g/d	124 (70, 204)	134 (62, 206)	109 (70, 174)	130 (72, 227)
Fruits and berries[4]	250 g/d	81 (7, 192)	67 (7, 260)	103 (42, 194)	80 (0, 160)
Whole grain	70–90 g/d	48 (32, 70)	48 (25, 67)	60 (36, 95)	42 (29, 69)
Fish[5,6]	300–450 g/wk (~43–64 g/d)	13 (0, 182)	23 (0, 157)	19 (0, 150)	0 (0, 208)
Oily fish[6]	≥200 g/wk (~29 g/d)	0 (0, 75)	0 (0, 50)	0 (0, 100)	0 (0, 142)
Red and processed meat	< 500 g/wk (~71 g/d)	88 (31, 124)	82 (29, 112)	90 (53, 129)	92 (29, 124)

[1] Norwegian dietary recommendations (2011).
[2] "*5-a-day*" is the sum of *vegetables* and *fruits and berries*.
[3] *Vegetables* not including dry legumes and potatoes.
[4] *Fruit and berries* includes juice.
[5] *Fish* includes fish and fish products but not shellfish and innards.
[6] 5th to 95th percentile.

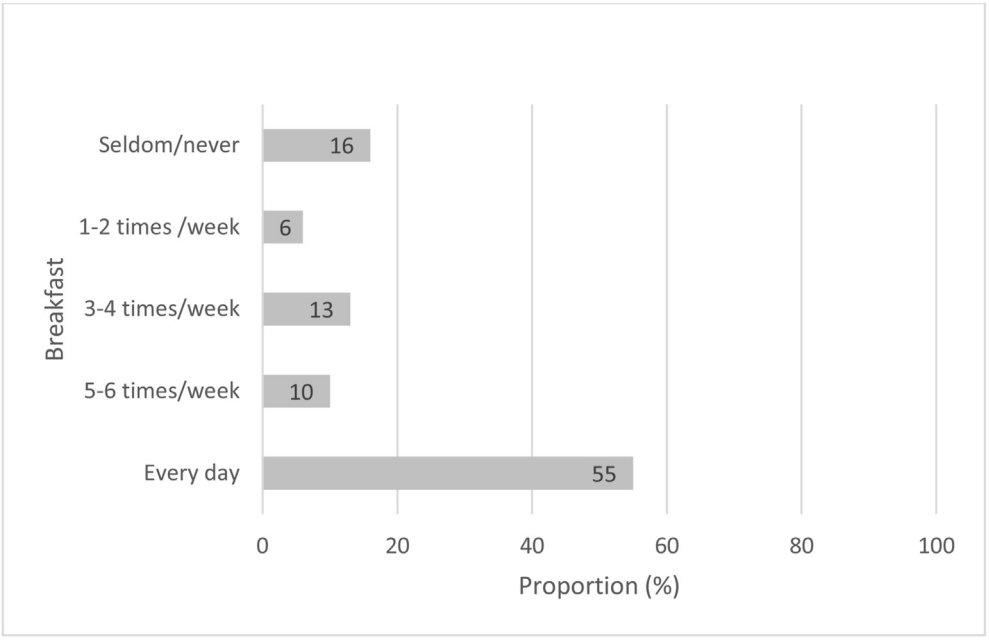

FIGURE 3. Breakfast consumption. Frequency of eating breakfast as reported by participants, as percent of total ($n = 94$).

patients in two obesity clinics in Norway that, using food frequency questionnaires, reported a mean fruit and vegetable intake in line with national recommendations, resulting in a higher fiber intake [42,43]. Two larger studies in Norway [44, 45] also reported higher fiber intake than we did, whereas the macronutrient composition of the diet in the latter [45] was similar to ours.

Meal timing and frequency might be altered among people with obesity [10,46,47], exemplified by the substantial number of our participants that answered they rarely/never ate breakfast. Contrary to others [22,23], we did not find a genotype association with meal frequency. Ours was a comparatively small sample and participants were asked to answer on prespecified meals and in-between meals. Unfortunately, there was no option to indicate more than one daily *other meal* (that is, in-between meal or snack), and this could have made it difficult for participants to report correctly. A more neutral terminology and the possibility to let the respondent fill in the number of such intake occasions would have been useful. Such an approach would be able to describe eating patterns better [8].

Our observation of a weak nonadditive association with the rs9939609 risk allele, for example, genotype associations with dietary intake were for genotype AT, illustrates the importance of collecting and reporting results on each genotype. Having a sample of patients newly referred to the obesity clinic allowed us to study their current dietary intake and determine that the genotype risk has little (if any) association with current dietary choices. This is an important finding on its own as it can steer research and weight control interventions toward a greater focus on the hedonic and behavioral aspects associated with dietary intake.

In our study, the low reported EI was likely a combination of underreporting (given that all participants had obesity and the majority were women) and a reflection of their status as patients wanting weight control [48–50]. Although they had not initiated any formal obesity treatment, they might have exerted a greater effort to control their food intake, thus precluding us of finding a genotype association with EI. It has been suggested that AA carriers underreport their EI more than TT carriers [32]; however, our participants had similar BMI and we have no indication to say that AA carriers misreported more than the others.

In summary, in a sample of men and women with severe obesity, we revealed no strong genotype associations between *FTO* rs9939609 and food, energy, or nutrient intake using a stringent significance level. Negative findings such as ours can help clinicians gain more knowledge in managing patients directly and can add evidence for future strategies to prevent and treat obesity. Our results reiterate the importance of nutrition assessment and guidance to assist a greater proportion of our patients to follow healthy diet recommendations.

Author disclosures

The authors report no conflicts of interest.

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Data Availability

Data described in the manuscript, code book, and analytic code will be made available upon request pending Norwegian law and approval by the regional ethics committee (REK-midt).

Appendix A. Supplementary data

Supplementary data to this article can be found online at https://doi.org/10.1016/j.cdnut.2023.100032.

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