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Research Article

Effect of Obesity-Linked *FTO* rs9939609 Variant on Physical Activity and Dietary Patterns in Physically Active Men and Women

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Single nucleotide polymorphisms (SNPs) in the fat mass and obesity-associated (*FTO*) locus are associated with obesity, but lifestyle factors may modulate the obesity risk related to *FTO*. This study examined the physical activity and dietary patterns of 528 physically active white men and women (mean (SD): 34.9 (9.5) years, 26.6 (4.3) $kg{\cdot}m^{-2}$) carrying different risk variants of *FTO* SNP rs9939609. Sex, age, and anthropometric measurements (stature, body mass, and waist circumference) were self-reported using an online questionnaire, and body mass index and waist-to-height ratio were calculated. Physical activity and eating behaviour were assessed using the International Physical Activity Questionnaire (IPAQ) and Three-Factor Eating Questionnaire (TFEQ), respectively. Body mass, body mass index (BMI), waist circumference, and waist-to-height ratio were not significantly different between individuals expressing different *FTO* rs9939609 risk variants (all $P \geq 0.66$). The cohort was physically active (4516 (3043) total $MET{\cdot}min{\cdot}week^{-1}$), although homozygous risk allele carriers (AA) displayed higher TFEQ cognitive restraint compared with nonrisk allele carriers (TT) (ES = 0.33 and $P = 0.03$). In conclusion, obesity-related parameters were not different in physically active individuals expressing different risk variants of *FTO* rs9939609, although homozygous risk allele carriers exhibited higher cognitive restraint.

1. Introduction

Obesity is a major risk factor for several chronic diseases and represents a major health and economic burden on society [1]. The aetiology of obesity is multifactorial and is influenced by complex interactions between environmental, lifestyle, and genetic factors [2]. Consequently, it is important to understand the interplay between these factors when designing strategies targeting the prevention of obesity.

The fat mass and obesity-associated gene (*FTO*) was the first common variant identified by genome-wide association studies that influences obesity risk [3]. Single nucleotide polymorphisms (SNPs) in intron 1 of *FTO* have been associated consistently with obesity risk across different ages and populations [4–9]. At *FTO* rs9939609 SNP, individuals homozygous for the risk allele (AA) weigh 3 kg more and have a 1.7-fold higher risk of obesity than those who do not carry a risk allele (TT) [3]. Studies have examined the effect of *FTO* variants on regulators of energy homeostasis to elucidate the mechanisms influencing *FTO*-mediated obesity risk. In this respect, evidence suggests that *FTO* may play a central role in the regulation of food intake [10, 11]. This is supported by studies demonstrating that individuals homozygous for the risk allele exhibit reduced satiety, poor food choices, and increased energy consumption [12–14]. Conversely, *FTO* obesity SNPs have not been related to energy expenditure, with evidence suggesting that those

Table 1: Participant characteristics.

	Total ($n = 528$)	Female ($n = 107$)	Male ($n = 421$)
Age and anthropometric characteristics			
Age (years)	34.9 (9.5)	36.8 (9.1)	34.5 (9.6)*
Stature (cm)	176.5 (9.4)	164.9 (7.1)	179.5 (7.4)*
Body mass (kg)	83.4 (16.5)	69.0 (13.9)	87.0 (15.0)*
Body mass index ($kg \cdot m^{-2}$)	26.6 (4.3)	25.4 (4.8)	27.0 (4.0)*
Waist circumference (cm)	84.6 (10.4)	78.2 (11.8)	86.2 (9.4)*
Waist-to-height ratio	0.48 (0.06)	0.48 (0.07)	0.48 (0.05)
Central obesity[a]	37 (7%)	21 (20%)	16 (4%)**
Physical activity levels[b]			
Vigorous MET ($min \cdot week^{-1}$)	2629 (1908)	2511 (1912)	2659 (1908)
Moderate MET ($min \cdot week^{-1}$)	737 (1026)	716 (874)	743 (1062)
Walking MET ($min \cdot week^{-1}$)	1150 (1269)	1284 (1374)	1115 (1241)
Total MET ($min \cdot week^{-1}$)	4516 (3043)	4511 (3163)	4518 (3017)
Eating behaviour			
Cognitive restraint score	12 (4)	12 (4)	12 (4)
Disinhibition score	6 (4)	7 (4)	6 (3)*
Hunger score	5 (4)	6 (4)	5 (4)
FTO rs99396909 *genotype*			
AA	95 (18%)	18 (17%)	77 (18%)
AT	236 (45%)	40 (37%)	196 (47%)
TT	197 (37%)	49 (46%)	148 (35%)

Values for age, anthropometric characteristics, physical activity levels, and eating behaviour are mean (SD) and were analysed between sex using linear mixed models. Values for central obesity and *FTO* rs99396909 genotype are frequency (%) and were analysed between sex using chi-square tests. [a]Central obesity was defined as a waist circumference >88 cm for women and >102 cm for men. [b]Complete physical activity data available for $n = 408$ (female: $n = 83$; male: $n = 325$). *Significant difference between females and males (linear mixed model $P < 0.05$). **Significant difference between females and males (chi-square test $P < 0.05$).

carrying the risk allele do not show reduced basal metabolic rate [15] or physical activity levels [7, 16, 17].

Although the association between *FTO* and obesity risk is well established, lifestyle may modulate obesity risk related to *FTO*. Several studies have suggested that *FTO*-mediated body fatness may be attenuated in physically active individuals [18–20]. Indeed, a meta-analysis concluded that higher physical activity levels attenuate the influence of *FTO* variation on obesity risk by 30% [21], and exercise interventions have demonstrated efficacy in promoting weight loss in *FTO* risk allele carriers [22, 23]. However, it is not well understood how the body mass index- (BMI-) increasing influence of *FTO* is attenuated in physically active individuals. An improved understanding of the differences in dietary and physical activity patterns in variants of *FTO* rs9939609 SNP within a physically active cohort may provide a greater insight into the behaviours that offset *FTO*-mediated obesity. Therefore, the aim of this study was to examine physical activity and dietary habits in a sample of physically active men and women carrying different risk variants of *FTO* rs9939609 SNP.

2. Materials and Methods

2.1. Participants. With the approval of Loughborough University's Ethical Advisory Committee, 708 men and women (age 34.0 (±9.3) years; body mass 82.5 (±16.3) kg; BMI 26.5 (±4.2) $kg \cdot m^{-2}$; waist circumference 84.4 (±11.0) cm; and waist-to-height ratio 0.48 (±0.06)) were recruited between March to November 2015 to participate in this study. Participants were recruited through FitnessGenes Ltd. (Bicester, Oxfordshire, UK—previously MuscleGenes Ltd.) and were predominantly based in European countries or the United States. Full informed consent, via an online consent form, was obtained from participants before the study commenced. Participants were volunteers from a customer base that were known to be participating in a regular fitness program but were not involved in elite sports. Participants classified themselves into one of fifteen ethnic groups. To reduce variability in the cohort, individuals who identified themselves as non-mixed white descent (British, Irish, and other White background) were included in this study (see Supplementary Methods (available here) for ethnic background selection criteria). Of the 708 recruited, 1 had missing genotype data, 17 had missing demographic or anthropometric data, and 162 did not classify themselves as non-mixed white descent. Therefore, results are presented for 528 participants (421 males and 107 females). Participant characteristics are presented in Table 1.

2.2. Genotype Analysis. Participant DNA was obtained from saliva, which was collected via an Oragene DNA self-collection kit (DNA Genotek Inc., Ottawa, ON, Canada)

sent from and returned to FitnessGenes Ltd. by post. DNA was extracted by LGC Genomics (Hertfordshire, UK) and genotyped using a KASP™ genotyping assay for *FTO* rs9939609 SNP. Genotype frequency of *FTO* rs9939609 SNP was assessed using a goodness-of-fit chi-square test and did not deviate from Hardy–Weinberg equilibrium (AA = 95, AT = 236, TT = 197; $P = 0.10$).

2.3. Collection of Demographic and Anthropometric Data. Participants self-reported their sex, age, stature, body mass, waist circumference, country of birth, and ethnicity using an online questionnaire. Sex, age, and ethnicity were confirmed by cross-referencing against customer information held by FitnessGenes Ltd. Participant stature, body mass, and waist circumference were used to calculate BMI and waist-to-height ratio (Table 1). Central obesity was calculated as the percentage of participants that exceeded previously defined thresholds for waist circumference (females > 88 cm and males > 102 cm) [24].

2.4. Physical Activity Levels. Physical activity levels were measured using the validated short format of the International Physical Activity Questionnaire (IPAQ) [25]. The IPAQ assesses the frequency and duration of walking, and moderate- and vigorous-intensity physical activities performed in bouts lasting 10 minutes or more during the previous seven days. Total physical activity (MET (metabolic equivalent), minutes per week) was estimated by multiplying the number of minutes reported in each activity level by the specific MET score for that activity (walking: 3.3, moderate intensity: 4.0, and vigorous intensity: 8.0 METs), and participants were classified in one of the three physical activity levels: low, moderate, or high (http://www.ipaq.ki.se).

2.5. Eating Behaviour. Eating behaviour was assessed using the validated 51-item Three-Factor Eating Questionnaire (TFEQ) to measure the dietary restraint (21 items, Cronbach's α 0.788), disinhibition (16 items, Cronbach's α 0.745), and hunger (14 items, Cronbach's α 0.761) [26]. All TFEQ items were coded with either 0 or 1 point and summed within each domain. Higher scores within each domain were indicative of higher restrained eating, disinhibited eating, or a predisposition to hunger.

2.6. Statistical Analyses. Data were analysed using the IBM SPSS Statistics Software for Windows Version 21 (IBM, New York). Between-sex differences in participant characteristics were examined using linear mixed models with one fixed factor (sex). Linear mixed models, adjusted for age and sex, were used to examine between-genotype differences in obesity-related parameters, physical activity levels, and eating behaviour with one fixed factor (*FTO* rs99396909 genotype). Between-genotype differences in all outcome measures were analysed using the additive genotype model (AA versus AT versus TT). Statistical power for this model was estimated using the CaTS Genome Power Calculator methods [27]. This method calculates the probability of outcomes per genotype, and thus the statistical power of the study based on the disease allele frequencies and prevalence of the disease within a population (i.e., the probability that a randomly sampled individual is obese or has a higher BMI) [27]. The calculation was performed using the genotype relative risk (GRR) for the additive genotype model ($GRR = f_1/f_0$, where f_0 and f_1 are the probabilities of being affected for individuals with 0 or 1 risk allele, resp.). For this study, a power of over 0.97 for a one-stage study was reported with a significance of 0.005. Where significant main effects were found in the additive genotype model, post hoc analysis was performed using Holm–Bonferroni correction for multiple comparisons [28]. Exploratory analysis was also conducted to analyse potential between-genotype differences in all outcome measures using the dominant model (risk allele carriers (AA/AT) versus homozygous nonrisk genotype (TT)) and the recessive model (nonrisk allele carriers (AT/TT) versus homozygous risk genotype (AA)). Differences in categorical variables (central obesity and *FTO* genotype frequency) between sex and/or *FTO* genotype groups were analysed using chi-square tests. Effect sizes are used to supplement important findings. An effect size of 0.2 was considered the minimum important difference in all outcome measures, 0.5 was moderate, and 0.8 was large [29]. Continuous variables are presented as mean (SD) and categorical variables as frequency (%). Statistical significance was accepted as $P < 0.05$.

3. Results

3.1. Participant Characteristics. On average, participants reported engaging in a total of 4516 (3043) MET minutes of activity per week, and the majority of participants were classified in the high physical activity category of the IPAQ (high 75.2%, moderate 19.4%, and low 5.4%), confirming the cohort were physically active (Table 1). Moreover, participants reported engaging in, on average, 3366 MET min·week^{-1} of moderate- to vigorous-intensity physical activity (Table 1), which is considerably higher than the average levels of moderate- to vigorous-intensity physical activity reported in adults across European countries (range 45–960 MET min·week^{-1}) [30]. Participants reported the following reasons for engaging in physical activity: muscle building training $n = 276$ (52%); fat loss training $n = 168$ (32%); strength training $n = 40$ (8%); power training $n = 20$ (4%); endurance training $n = 17$ (3%); and no response $n = 7$ (1%).

Females were significantly older (ES = 0.24, $P = 0.02$), had a higher frequency of central obesity (odds ratio = 6.31, $P < 0.001$), and exhibited higher TFEQ disinhibition scores (ES = 0.42, $P < 0.001$) compared with males (Table 1). Body mass, BMI, and waist circumference were significantly lower in females than males (all ES ≥ 0.38, $P \leq 0.001$), but waist-to-height ratio was similar between the sexes (ES = 0.09, $P = 0.40$) (Table 1). No significant differences were seen between the sexes for any measure of physical activity (all ES ≤ 0.13, $P \geq 0.28$) or *FTO* genotype frequency ($P = 0.12$) (Table 1).

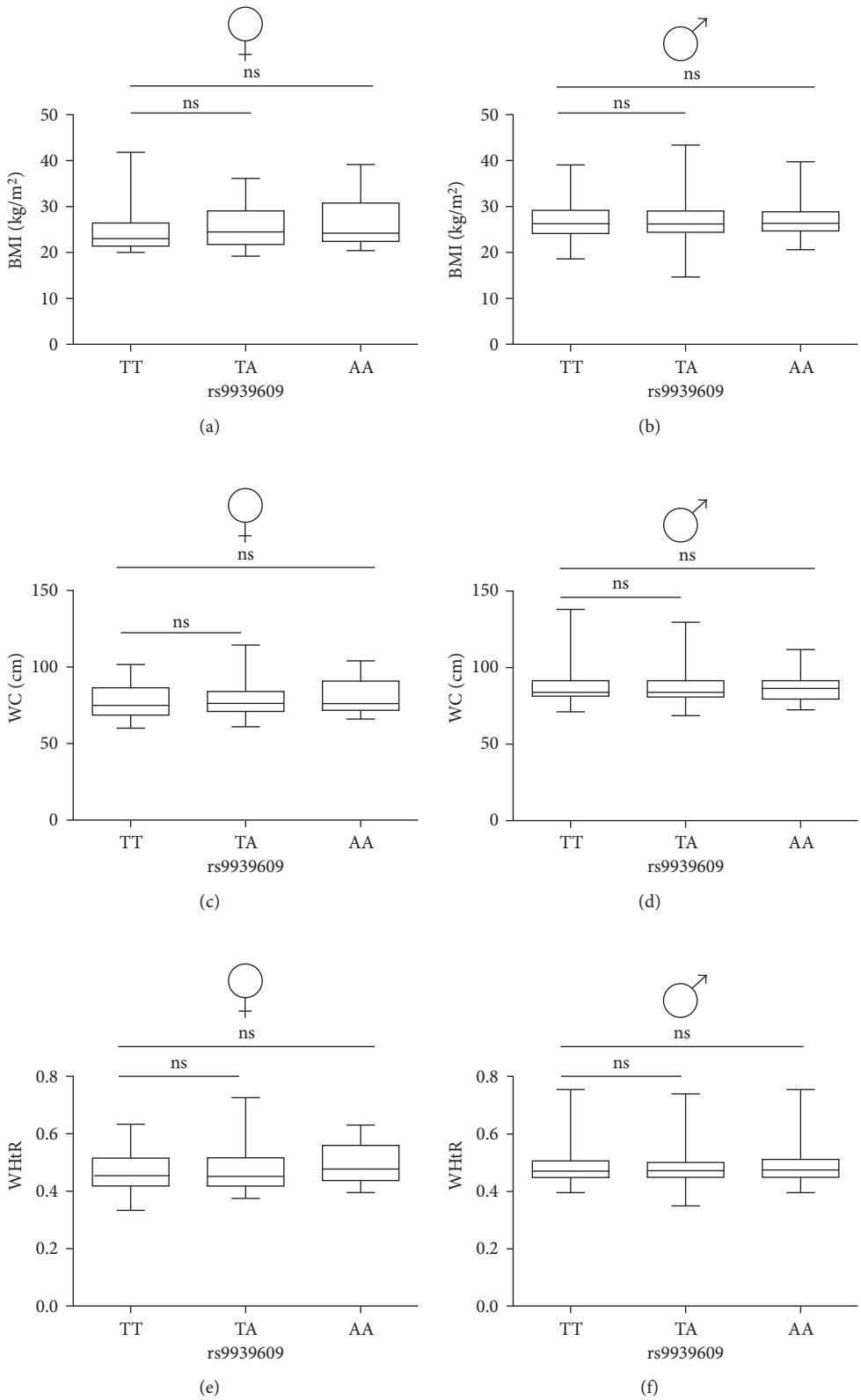

Figure 1: Box plots and analysis of the effect of *FTO* rs9939609 gene variant on obesity-related parameters. (a, b) Body mass index (BMI); (c, d) waist circumference (WC); (e, f) waist-to-height ratio (WHtR). (a, c, e) female; (b, d, f) male. Females: $n = 107$ (AA = 18, AT = 40, and TT = 49). Males: $n = 421$ (AA = 77, AT = 196, and TT = 148). The line within box represents the median, while the lower and upper box lines represent the interquartile range (1st and 3rd quartiles). The whiskers represent the minimum and maximum observations. Linear mixed models, adjusted for age and sex, were used to examine between-genotype differences. No significant differences were seen in obesity-related parameters between *FTO* rs9939609 genotypes.

Table 2: Obesity-related parameters in men and women carrying different risk variants of the *FTO* rs9939609 single nucleotide polymorphism.

Characteristic	*FTO* rs9939609 genotype			Model 1[a]: genotype *P* value
	AA	AT	TT	
Body mass (kg)	83.7 (14.7)	84.4 (16.5)	82.0 (17.2)	0.69
Body mass index (kg·m^{-2})	26.8 (3.9)	26.8 (4.3)	26.4 (4.3)	0.66
Waist circumference (cm)	85.2 (9.4)	84.9 (10.4)	84.1 (11.0)	0.87
Waist-to-height ratio	0.48 (0.05)	0.48 (0.06)	0.48 (0.06)	0.85
Central obesity[b]	6 (7%)	16 (7%)	15 (8%)	0.92

Values for body mass, body mass index, waist circumference, and waist-to-height ratio represent mean (SD) and were analysed using linear mixed models adjusted for age and sex. Values for central obesity represent frequency (%) and were analysed using chi-square tests. ES, effect size (body mass, body mass index, waist circumference, and waist-to-height ratio); OR, odds ratio (central obesity). [a]Model 1: additive genotype model (AA versus AT versus TT). [b]Central obesity was defined as a waist circumference > 88 cm for women and > 102 cm for men.

Table 3: Physical activity levels in men and women carrying different risk variants of the *FTO* rs9939609 single nucleotide polymorphism.

Characteristic	*FTO* rs9939609 genotype			Model 1[a]: genotype *P* value
	AA ($n = 74$)	AT ($n = 177$)	TT ($n = 157$)	
Vigorous MET (min·week^{-1})	2808 (1966)	2751 (1903)	2407 (1876)	0.19
Moderate MET (min·week^{-1})	726 (1069)	816 (1127)	654 (874)	0.41
Walking MET (min·week^{-1})	1240 (1242)	1207 (1345)	1043 (1192)	0.34
Total MET (min·week^{-1})	4774 (3125)	4774 (3193)	4104 (2795)	0.10

Values are mean (SD) for $n = 408$. Comparisons were made using linear mixed models adjusted for age and sex. ES, effect size. [a]Model 1: additive genotype model (AA versus AT versus TT).

Table 4: Eating behaviour in men and women carrying different risk variants of the *FTO* rs9939609 single nucleotide polymorphism.

Characteristic	*FTO* rs9939609 genotype			Model 1[a]: genotype *P* value
	AA ($n = 95$)	AT ($n = 236$)	TT ($n = 197$)	
Cognitive restraint score	13 (4)	12 (4)	11 (4)	0.03*
Disinhibition score	6 (3)	7 (4)	6 (4)	0.38
Hunger score	5 (3)	5 (4)	5 (4)	0.92

Values are mean (SD) for $n = 528$. Comparisons were made using linear mixed models adjusted for age and sex. ES, effect size. [a]Model 1: additive genotype model (AA versus AT versus TT). *Significant difference between AA and TT *FTO* rs9939609 genotype (linear mixed model $P < 0.05$ after Holm–Bonferroni correction).

3.2. FTO rs9939609 Genotype and Obesity-Related Parameters. Obesity-related parameters in men and women carrying different risk variants of *FTO* rs9939609 SNP are displayed in Figure 1 and Table 2. Linear mixed models for the additive genotype model revealed no significant differences in body mass, BMI, waist circumference, or waist-to-height ratio across *FTO* rs9939609 genotype groups (all $P \geq 0.66$; Table 2). The minor allele frequency is analogous with that expected (0.4, A), suggesting this is not due to an unrepresentative population. Exploratory analysis revealed no significant differences in obesity-related parameters when analysed in dominant (all ES ≤ 0.13, $P \geq 0.37$) or recessive (all ES ≤ 0.08, $P \geq 0.68$) allele models (Supplementary Table 1).

3.3. FTO rs9939609 Genotype and Physical Activity Levels. Complete physical activity data were available for 408 participants (AA $n = 74$, AT $n = 177$, TT $n = 157$; female: $n = 83$ and male: $n = 325$). The physical activity levels in men and women carrying different risk variants of *FTO* rs9939609 SNP are displayed in Table 3. Linear mixed models for the additive genotype model revealed no significant differences in physical activity levels across *FTO* rs9939609 genotype groups (all $P \geq 0.10$). In the exploratory analysis, the dominant allele model revealed total MET minutes per week (ES = 0.22, $P = 0.03$, Supplementary Table 2) and a tendency for vigorous MET minutes per week (ES = 0.19, $P = 0.08$) to be higher in A allele carriers (AA/AT) than nonrisk allele carriers (TT). No significant differences in moderate or walking MET minutes per week were identified in the dominant allele model (AA/AT versus TT) (all ES ≤ 0.13, $P \geq 0.17$; Supplementary Table 2). The recessive allele model (AT/TT versus AA) revealed no significant differences in physical activity levels between *FTO* genotype groups (all ES ≤ 0.11, $P \geq 0.28$; Supplementary Table 2).

3.4. FTO rs9939609 Genotype and Eating Behaviours. Eating behaviours in men and women carrying different risk variants of *FTO* rs9939609 SNP are displayed in Table 4. Linear

mixed models for the additive genotype model identified a significant main effect for cognitive restraint score across *FTO* genotype groups ($P = 0.03$). Post hoc analysis of between-group differences revealed that the cognitive restraint score was higher in homozygous A allele carriers than AT (ES = 0.25, $P = 0.07$) and TT (ES = 0.33, $P = 0.03$) genotypes; AT and TT genotypes were similar (ES = 0.06, $P = 0.49$). No differences in disinhibition or hunger scores were seen across *FTO* genotype groups (both $P \geq 0.38$).

In the exploratory analysis, the dominant allele model (AA/AT versus TT) revealed no significant differences in cognitive restraint, disinhibition, or hunger scores between *FTO* genotype groups (all ES ≤ 0.14, $P \geq 0.12$). The recessive allele model was consistent with that seen in the additive genotype model, in that the cognitive restraint score was higher in homozygous A allele carriers (AA) than T allele carriers (AT/TT) (ES = 0.28, $P = 0.01$; Supplementary Table 3). No significant differences in disinhibition or hunger scores were identified in the recessive allele model (all ES ≤ 0.08, $P \geq 0.60$; Supplementary Table 3).

4. Discussion

The primary finding from the present study was that obesity-related parameters were not different in physically active individuals carrying different risk variants of *FTO* rs9939609 SNP. Furthermore, *FTO* rs9939609 homozygous A allele carriers exhibited higher cognitive restraint than nonrisk allele carriers, and exploratory analysis also showed higher levels of physical activity in A allele carriers compared with nonrisk allele carriers.

The consensus of evidence suggests that *FTO* risk alleles are associated with elevated body weight across different ages and populations, with each minor risk allele increasing BMI and obesity risk by 0.25–0.39 kg·m^{-2} and 1.18–1.27 fold, respectively [9]. However, the most extensively studied rs9939609 SNP had no influence on obesity-related parameters in the current study. The participants in our cohort were physically active, and the average levels of reported total and moderate- to vigorous-intensity physical activity were considerably higher in comparison with both the general population [30] and individuals in previous *FTO* studies [19–23]. Several studies have demonstrated, through self-reported questionnaires [31, 32] and objective physical activity devices [19, 33], that obesity-related traits associated with *FTO* risk alleles are attenuated in individuals with higher physical activity levels. Therefore, it is possible that the high physical activity levels within our cohort may have diminished any differences in obesity-related parameters between *FTO* rs9939609 genotypes. Intriguingly, despite being a highly active group when taken as a whole, the exploratory analysis using the dominant model demonstrated that individuals with the A allele reported higher total and vigorous physical activity levels compared with TT individuals. It is possible, therefore, that this difference in physical activity patterns between *FTO* genotypes may further offset the adiposity-increasing effect of *FTO* within this cohort, but additional work is required to examine this systematically. Nonetheless, this elevation in activity was small, and these differences in physical activity levels between *FTO* genotypes conflict with previous studies suggesting that *FTO* genotype does not impact on physical activity levels [7, 31]. Contradictions between the current study and other evidence may be due to the high levels of intensive physical activity of individuals within the current study. Although Berentzen et al. [16] reported no effect of the *FTO* variant on physical activity in individuals classified by the authors as highly active (more than 4 h moderate physical activity a week), there is a lack of studies that have examined the effect of *FTO* genotype on activity levels in cohorts with even greater levels of physical activity. Consequently, further studies, including longitudinal investigations, are required to examine *FTO*-mediated differences in physical activity among individuals with varying activity status. In addition, given that much of the data on *FTO*-mediated differences in physical activity levels are reliant on self-reported questionnaire data, additional scientific enquiry using objective physical activity measures, such as accelerometers, is required.

It has been postulated that *FTO*-mediated predisposition to weight gain and obesity may be modified by dietary behaviours. Research suggests that *FTO* may play a significant role in the regulation of satiety and food intake [10, 11]. Furthermore, it has been reported in previous observational studies that the risk variant of *FTO* is associated with lower cognitive restraint and higher disinhibition and hunger, which may be indicative of poorer eating behaviours [34, 35]. The present study extends these findings by demonstrating that physically active individuals homozygous for *FTO* rs9939609 risk allele display higher cognitive restraint but similar disinhibition and hunger scores compared with nonrisk allele carriers. The potential benefit of cognitive restraint on the propensity to body weight changes is highlighted by studies demonstrating a negative association between cognitive restraint and markers of adiposity (e.g., body mass and BMI) [36, 37]. However, such associations are not reported universally [38], and cognitive restraint tends to be higher in overweight than in healthy weight individuals [39]. Additionally, disinhibition has been proposed as a stronger predictor of BMI [40]. Nevertheless, increased dietary restraint during weight loss has been identified as a significant predictor of successful weight loss maintenance [41]. Therefore, it is possible that the higher cognitive restraint observed in *FTO* risk allele carriers in the present study may offset *FTO*-mediated obesity risk in physically active individuals; but further work is required to confirm this chronically.

The current study is not without limitations. The observations in the present study are limited by the cross-sectional design, which precludes the ability to establish causality. Future longitudinal studies are required to establish systematically whether higher physical activity levels and cognitive restraint may diminish *FTO*-mediated predisposition to weight gain. In addition, the anthropometric, physical activity, and eating behaviour data were self-measured and self-reported using an online survey, which may have led to problems with participant measurement error, recall, and bias. Additional work is therefore required

using more direct and objective measures. Furthermore, one of the primary measures of body fatness in the present study is BMI. Despite its frequent use in large-scale population studies, BMI does not take into account differences between fat and fat-free mass such as muscle and bone [42]. Given the current cohort reported high levels of total activity, with the majority indicating that their primary fitness goal was muscle building (52%), it is possible that variations in body mass (and hence BMI) could be largely due to higher muscle mass. This may explain why the average BMI among individuals in the current data set falls within the overweight category. Consequently, future studies using more direct measures of body adiposity are required to replicate the current study's findings. Finally, statistical power was limited in the dominant and recessive models analysed in this paper. However, the results from these exploratory models allow for preliminary inferences that may guide a larger population-based study.

5. Conclusion

Within a physically active cohort, risk allele carriers of *FTO* rs9939609 exhibited greater physical activity levels and cognitive restraint than nonrisk allele carriers despite demonstrating similar adiposity-related measures. Future studies with repeated and objective measurements are required to further investigate physical activity and dietary behaviours that underscore the effects of *FTO* on obesity risk.

Conflicts of Interest

Nathan R. West, Nicola C. Hanson, Samantha E. Decombel, and Stuart J. Grice work for FitnessGenes Ltd.

Authors' Contributions

Nathan R. West and James Dorling contributed equally to this work.

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Supplementary Materials

Supplementary Methods: ethnicity information collection. Supplementary Table 1: obesity-related parameters in men and women carrying different risk variants of the *FTO* rs9939609 single nucleotide polymorphism. Supplementary Table 2: physical activity levels in men and women carrying different risk variants of the *FTO* rs9939609 single nucleotide polymorphism. Supplementary Table 3: eating behaviour in men and women carrying different risk variants of the *FTO* rs9939609 single nucleotide polymorphism. (*Supplementary Materials*)

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