THE ROLE OF FAT MASS AND OBESITY-ASSOCIATED GENE POLYMORPHISM IN OBESE EGYPTIANS

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ABSTRACT

Introduction: Variations in fat mass and obesity associated (FTO) gene increased the risk of obesity in many European populations but with inconclusive results in other populations. There are no previous reports about polymorphism of FTO gene and its association with obesity in Egyptian Arab population.

Aim of the study: this study was designed to investigate the allele frequency and genotype distribution of FTO gene rs9939609 and its association with obesity and insulin resistance (IR).

Subjects and method

102 obese (BMI $\geq$ 30) and 39 non-obese subjects were included. For all subjects rs9939609 of FTO gene was genotyped and other biochemical parameters were measured

Results

The overall minor allele frequency (A) was (0.4). There was significant difference in genotype distribution in obese vs non-obese group under recessive model, with an increased odds ratio (OR) for obesity (OR = 4, P = 0.04)

Conclusions

this study validated that homozygous variant of FTO gene rs9939609 is risk factor for obesity in our female subjects. Hopefully, this study can help to set personalized plans for prevention and treatment of obesity and obesity-related disorders in Egyptian population.

Introduction

Egypt now is one of the top ten countries in the prevalence of obesity and type 2 diabetes mellitus (T2DM) (Martorell et al., 2000; IDF, 2013). About 22% of men and 48% of women are obese according to 2010 WHO estimates. There are many medical complications of obesity as IR, T2DM, fatty liver disease, dyslipidemia, cardiovascular complications, pulmonary diseases and some cancers(Antuna-Puente et al., 2008; Blüher, 2009). It is accepted that many environmental factors play a key role in increasing the susceptibility to obesity (Abelson and Kennedy, 2004; Keith et al., 2006). However, we respond differently indicating that genetic difference affect the outcome (Loos and Bouchard, 2008). From many genes linked to the risk of obesity, the fat mass- and obesity-associated (FTO) gene was one of the robust genes

that has possible link with obesity with genome wide association study based evidence (Frayling et al., 2007) .Human FTO is present on chromosome 16q12.2 and plays a key role in controlling energy homeostasis as being expressed specifically in the hypothalamus(Frayling et al., 2007; Gerken et al., 2007). Based on bioinformatics and in-vitro studies, FTO encodes a 2- oxoglutarate-dependent nucleic acid demethylase (Gerken et al., 2007). Studies have confirmed the FTO gene variants association with obesity in different populations especially in European countries (Frayling et al., 2007; Gonzalez-Sanchez et al., 2009; Zhang et al., 2010; Sentinelli et al., 2012). On the contrary, other studies failed to validate this association (Ohashi et al., 2007; Li et al., 2008; Hennig et al., 2009). To the best of our knowledge, there are no previous reports about polymorphism of FTO gene and its association with obesity in Egyptian Arab population.

Subjects and methods

Study population: a random unrelated 141 female subjects were studied. 102 were obese (BMI ≥ 30 kg/m2) and 39 were non-obese. There was a complete medical evaluation for each case with provided informed consent prior to inclusion in the study. Exclusion criteria included chronic viral hepatitis; malignant disease; acute infections; pituitary, adrenal, thyroid, pancreatic disease, or evidence for any other endocrine disorder; or prolonged use of corticosteroids as well as sex hormones were excluded. The Ethical Committee both of Faculty of Pharmacy/Medicine, Ain Shams University, Cairo, Egypt and the National Diabetes Institute approved this study. Moreover, the study was performed in adherence to the Declaration of Helsinki Guidelines.

Blood sampling

Fasting blood samples were drawn and collected in separate vacutainer tubes. Disodium-ethlenediamine tetra-acetic acid (EDTA) containing vacutainer for whole blood collection required for the genotyping assay. A second vacutainer was fluoride containing one for fasting plasma glucose measuring. The third was plain vacutainer used for measuring serum lipid profile, aminotransferases and fasting insulin.

Anthropometric and biochemical measurements

Body mass index (BMI) (Kg/m^2) and waist circumference (WC) (cm) were measured for all subjects. Biochemical measurements included plasma glucose, serum total cholesterol (TC), triacylglycerol (TAG), high-density lipoprotein (HDL) cholesterol, aspartate aminotransferase (AST) and alanine aminotransferase (ALT) were determined by enzymatic methods using autoanalyser (Beckman synchron cx systems) and Dimension RxL analyzer (Dade Behring, Newark, DE). Low-density lipoprotein (LDL) cholesterol was calculated by the Friedewald formula(Friedewald et al., 1972). Serum insulin was measured using commercially available ELISA kit (Immunospec® kit, provided from Immunospec Corporation, Canoga Park, CA, USA). Homeostasis model assessment (HOMA-IR) calculated as: [(FBG X fasting insulin)/405](Matthews et al., 1985) and Quantitative insulin sensitivity check index (QUICKI) calculated as : 1/[log FI (μIU/ml) + log FBG (mg/dl)](Katz et al., 2000) were used as a measure of IR.

Genotyping

Genomic DNA was extracted from blood using QIAamp DNA Mini Kit protocol (QIAGEN, Santa Clarita, CA).

FTO gene genotyping rs9939609 was performed by TaqMan SNP Genotyping Assays using Assays-by-Design supplied by Applied Biosystems International (ABI; Applied Biosystems, Foster City, CA) and using The Applied Biosystems® Step One Plus™

Real-Time PCR System

Statistical Analysis

Data were collected, recorded and tabulated. Afterwards, statistical analyses were performed using windows based Statistical Package for Social Sciences (SPSS®)

Table 1. Characteristics of studied groups

group/parameter	Non-obese (n=39)	Obese (n=102)	P-value
Age(years) Weight (kg)	41.1 ± 5.4 65.2 ± 8.9	44.1 ± 10.2 95.2 ± 15.7	0.086 <0.001
Height (cm)	163.6 ± 8.6	159.1 ± 6.9	0.002
BMI (kg/m^2)	24.3 ± 1.8	37.6 ± 5.5	<0.001
WC(Cm)	84.3 ± 4.2	119.1 ± 10.9	<0.001
TC (mg/dL)	156 ± 19	181 ± 41	<0.001
TG (mg/dL)	107 ± 20	164 ± 78	<0.001
HDL-C (mg/dL)	45 ± 7	38 ± 9	<0.001
LDL-C (mg/dL)	90 ± 18	111 ± 38	0.002
FPG (mg/dL)	87 (71-110)	158.5 (60-361)	<0.001
FSI (□IU/mL)	10.2 (1 -18)	12.5(2.9-30.3)	0.003
HOMA-IR	1.9 (0.2 - 4.8)	4.2 (0.4 - 23.9)	<0.001
QUICKI	0.35 ± 0.06	0.31 ± 0.04	<0.001
AST (U/L)	8(4 – 12)	19(10-60)	<0.001
ALT (U/L)	7 (4 -12)	18.5 (6 -58)	<0.001

computer database version 17.0 and Microsoft Excel® version 2010 Data were described as mean and standard deviation for numeric variables, or median and range for numeric nonparametric variables, and number and percentage (%) for categorical

variables The difference between 2 groups was analyzed using an independent Student's t-test (for numeric parametric variables), Mann-Whitney's U-test (for numeric nonparametric variables) or Chi-squared ($\chi 2$) test (for categorical variables). The Chi-squared ($\chi 2$) test was utilized to test deviation or accordance with the Hardy–Weinberg Equilibrium (HWE) using an online calculator(Rodriguez et al., 2009) (The online encyclopedia for genetic human epidemiology studies, 2008). The same statistical test was used to estimate the genetic association of the investigated SNP with obesity and diabetes by estimating the differences in genotype distributions between case and control groups For estimating the association between measured variables binary logistic regression (using 95% confidence interval) analyses; were performed Significance level was set at 0.05.

RESULTS

As shown in table (1), the anthropometric and biochemical measurements differed significantly between the cases and controls The distribution of genotype for rs 9939609 variant in the whole population and in the study groups came in agreement with Hardy–Weinberg equilibrium ($P > 0.05$). The minor allelic frequency (MAF) of the risk allele A was (0.4). The genotype distribution differed significantly between

obese and non-obese individuals under the recessive model with increased significant odds ratio with obesity (OR = 4.2, P = 0.04) table (2).

Values are x ± SD for parametric variables or median (range) for nonparametric variables.

BMI: body mass index, WC: waist circumference, T.C: total cholesterol, TAG: triacylglycerol, HDL-C: high density lipoprotein-cholesterol, LDL-C: low density lipoprotein-cholesterol, FPG: fasting plasma glucose, FSI: fasting serum insulin, AST: aspartate aminotransferase, ALT: alanine aminotransferase.

Table 2: Genotype distribution of the rs9939609 variant of FTO gene and its risk with obesity.

model	Genotype n (%)	Non-obese (n= 39)	Obese (n= 102)	(χ2)/P/OR
Additive model	(TT)	16(41%)	33(32.4%)	4/0.12/----
	(TA)	21(53.8%)	50(49%)	
	(AA)	2(5.1%)	19(18.6%)	
Dominant model	(TT)	16(41%)	33(32.4%)	0.9/0.3/1.5
	(TA+AA)	23(59%)	69(67.6%)	
Recessive model	(TT+TA)	37(94.9%)	83(81.4%)	4.1/0.04/4
	(AA)	2(5.1%)	19(18.6%)	

Pa : p- value adjusted for age, OR : odds ratio

There was no significant difference in BMI, WC and other biochemical measurements among the different genotypes (data not shown).

Discussion

Regarding our subjects in the current study, we aimed to investigate the genetic risk specifically in cohort of female subjects. The reason for that is the high prevalence of obesity in Egyptian female population that exceeded the males and reaching alarming level (Badran and Laher, 2011; Ng et al., 2014). Regarding the allele frequency and genotype distribution in the current study, this is the first study to investigate the allele frequency and genotype distribution of FTO gene rs9939609 in Egyptian population. In the current study, the overall MAF was (0.4). In comparison with HapMap data (http://hapmap.org), it comes in agreement with the allele frequency of CEPH Europeans (Utah residents with ancestry from northern and western Europe) (0·45), higher than in the HapMap CHB (Han Chinese in Beijing, China) (0·12) and JPT (Japanese in Tokyo, Japan) samples (0·17), but lower than in HapMap YRI (Yoruba in Ibadan, Nigeria) population (0·49).

The genotypes distribution with obesity was unique. The association of the FTO variant with obesity in our population fitted more to the recessive model. In the context of FTO association with obesity, this is the first study that investigated the association between FTO and obesity in Egyptian population. In the current study the homozygous variant of FTO increases the risk for obesity under the recessive model. This came in agreement with other studies in different populations mainly of European descent (Scuteri et al., 2007; Peeters et al., 2008; Gonzalez-Sanchez et al.,

2009; Zhang et al., 2010; Sentinelli et al., 2012). Of particular interest, this is in contrast with other studies in which the FTO variants do not seem to affect BMI or the risk of obesity consistently as in African Americans (Scuteri et al., 2007), Chinese Hans (Li et al., 2008), Japanese (Horikoshi et al., 2007) or Oceanic populations (Ohashi et al., 2007). This difference may be due to ethnic difference and different study design. Many possible mechanisms may be possible for FTO gene association with obesity as in our results. Subjects homozygous for the A risk allele of rs9939609 eat significantly more (Speakman et al., 2008; Wardle et al., 2009), have reduced satiety (den Hoed et al., 2009; Tanofsky-Kraff et al., 2009; Wardle et al., 2009), prefer higher caloric food and have a higher fat mass (Cecil et al., 2008; Timpson et al., 2008) than subjects homozygous for the T allele. The association of FTO SNPs with energy intake is seen even in small sample sizes (Speakman et al., 2008). Therefore, FTO risk alleles are unequivocally associated with increased food intake and appetitive behavior (Cheung and Yeo, 2011), which may be responsible for association with obesity in our results.

Conclusions

This study validated the association of the homozygous mutant (AA) of FTO gene rs9939609 with increased risk for obesity in Egyptians. Hopefully, this study may help to set personalized treatment for obesity in our population Conflict of Interests; the authors declare that there is no conflict of interests regarding this study Acknowledgment: We want to express our deep appreciation to all members of national institute of diabetes and endocrinology, for their kind help in samples collection.

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الملخص العربي

دور تعدد الأشكال الجينية للجين المرتبط بالسمنة وكتلة الدهون فى المصريين البدناء

للسادة الدكاترة

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تعد السمنة من المخاطر الصحية المتزايدةالتيوصلتإلىمستوياتخطيرةوتساهمفيزيادةمعدلاتالاعتلالوالوفياتفيجميعأنحاءالعالم. وتعتبر مصر الآن واحدة من أكبر عشر دول في انتشار السمنة. تعد السمنة هى الاساس للعديد من المضاعفات الطبية وخاصة متلازمة التمثيل الغذائي(متلازمة الأيض) ومضاعفات اعتلاالقلب والأوعية الدموية. ان العديد من العوامل البيئية تعد اساس الزيادة العرضة للاصابة بالسمنة،ومع ذلك ونظرا لأن كل منا له خلفية وراثية مختلفة تكون استجابتنا لتلك العوامل البيئية مختلفة. هذا يؤكد أنا لاختلافات الجينية تؤثرعلى النتيجة النهائية.لقد أرتبطت اختلافات الأشكال الجينية للجين المرتبط بالسمنة وكتلة الدهون (FTO gene) بزيادة خطورة الأصابة بمرض السمنة فى العديد من الشعوب الاوروبية، لكن مع نتائج غير حاسمة في الشعوب الأخرى. الى حد الان،لاتوجد تقارير سابقة عن تعدد الأشكال الجينية للجين المرتبط بالسمنة وكتلة الدهون (FTO gene)وارتباطه بالسمنة في الشعب المصري.

و لذلك استهدفنا من خلال هذه الدراسة إلى دراسة توزيع الأليلو النمط الجيني للجين المرتبط بالسمنة وكتلة الدهون (FTO gene rs9939609في المصريات. بالاضافة الى المقارنة بغير هم من الشعوب التى تم دراستها سابقا. وأخيرا،دراسة مدى ارتباط هذا التعدد الشكلى الجينى بالسمنة ومقاومة الأنسولين.

و لتحقيق هذه الأهداف فقد اشتملت هذه الدراسة على 141شخصا من الاناثتم تقسيمهم على النحو التالي:

المجموعة الضابطة: و تتكون من 39 من الاناث المتطوعات الغير بدناء

مجموعة الأشخاص البدناء: و تتكون من 102من الاناث البدناء

وقد خضع كل المشاركين بالدراسة لقياس عدد من المؤشرات المرتبطة بالسمنة. هذا بالاضافة إلى قياس مستوى كل من الأنسولين، وتحديد النمط الجينى للجين المرتبط بالسمنة وكتلة الدهون (FTO gene rs9939609)

ويمكن تلخيص نتائج الدراسة كما يلي:

أظهرت النتائج وجود زيادات ذات دلالة إحصائية في مستوى كل من السكر، الأنسولين في مصل الدم، مؤشر مقاومة هرمون الأنسولين، كما حدث اضطراب بالدهون، بالاضافة إلى وجود انخفاض بمؤشر قياس الحساسية للأنسولين في مجموعة البدناء مقارنة بالمجموعة الضابطة.

كان تردد الأليلالثانو يالعام (A) هو 0.4 جاء هذا التردد للأليل مماثلا لتوزيع هاردى وينبرج. ان هناك اختلاف ذو دلالة احصائية في التوزيع الوراثي(توزيع الانماط الجينية) في السمنة مقابل مجموعة غير البدين ات فيطار النموذج المتنحي ،حيث كان النمط الجيني AA بشكل ملحوظ أكثر تواترا في السمنة من الأفراد غير البدناء.

أظهر النمط الجينيAA زيادة كبيرة في نسبة الأرجحية (odds ratio) للسمنة في إطار النموذج المتنحي.

في اطار ماسبق يمكن القول أن البحث قد خلص إلى أن النمط الجيني (AA) للجين المرتبط بالسمنة وكتلة الدهون (FTO gene rs9939609 هو أحد عوامل الخطر للاصابة بالسمنة في الإناث المصريات. نأمل،أن تبنى خطط شخصية مستقبلية للوقاية والعلاج من السمنة وغيرها من الأمراض المرتبطة بالسمنة في المجتمع المصري على أساس هذه الدراسة.