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Original Article

Relations of major dietary patterns and metabolically unhealthy overweight/obesity phenotypes among Iranian women

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ABSTRACT

Objective: No studies have examined the contribution of major dietary patterns to MUH phenotypes in obese and overweight people based on Karelis criteria. This study was conducted to evaluate the association of major dietary patterns with MUHOW/O and MHOW/O phenotypes.

Methods: This cross-sectional study was conducted on 290 overweight and obese women aged 18–50 (BMI≥25 kg/m^2). Anthropometric measurements were assessed in all participants. The MH phenotype was defined according to the Karelis criteria. Major dietary patterns were determined using factor analysis of 21 foods groups using a valid and reliable FFQ containing 147 items. Participants' body composition was assessed by BIA. Serum HDL, LDL, TG, insulin, and hs-CRP levels were quantified by ELISA.

Results: By the use of factor analysis, 3 major dietary patterns were extracted: healthy dietary pattern (HDP), western dietary pattern (WDP) and unhealthy dietary pattern (UNHDP). Binary logistic analysis showed that participants in the in the upper category of WDP had greater odds of MUH phenotype (OR = 2.33, 95%CI = 1.11–4.91, P = 0.02), after confounder factor control. Individuals with high adherence to the UNHDP score had high odds of MUH phenotype (OR = 1.75, 95%CI = 0.98–3.10, P = 0.05), after adjustment for BMI, age, and total EI, compared to those with low adherence. A positive relation was observed between WDP and levels of hs-CRP, HOMA-IR (OR = 1.94, 95%CI = 0.91–4.10, P = 0.05 and OR = 2.53, 95%CI = 1.26–5.11, P = 0.009) as well as a positive association between UHDP and plasma level of LDL (OR = 1.90, 95%CI = 1.04–3.47, P = 0.03), but an inverse association between HDP and hs-CRP level (OR = 0.56, 95%CI = 0.29–0.92, P = 0.03).

Conclusions: The present evidence indicates various significant associations among major dietary patterns and MUHOW/O phenotypes.

1. Introduction

The prevalence of obesity is increasing in most parts of the world, and the International Obesity Task Force (IOF) estimates that at least 1.1 billion adults are overweight worldwide, including 312 million who are obese [1]. Obesity often clusters with risk factors of cardiovascular disease (CVDs) and type 2 diabetes (T2D) that comprise metabolic syndrome (MetS), including insulin resistance (IR), hypertension, dyslipidemia, and low levels of high density lipoprotein-cholesterol (HDL-C) [2].

Intriguingly, obesity is not synonymous with metabolic disease, as people who are overweight or obese do not always have metabolic dysfunction, especially cardio-metabolic dysfunction [3]. These populations have been termed as having metabolically healthy obesity (MHO) [4]. MHO describes [5] the absence of any overt cardio-metabolic disease, along with a favorable metabolic profile and inflammation profile, characterized by high levels of insulin sensitivity, a lower proportion of visceral fat, less liver fat, low prevalence of hypertension and components in an individual with a body mass index (BMI) ≥30 kg/m^2. The diagnostic criteria

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List of abbreviations

ATPIII	Adult Treatment Panel
BMI	Body mass index
BIA	Bioelectrical impedance analysis
BP	blood pressure
BF %	body fat percentage
CVDs	cardiovascular diseases
CHOD-PAP	cholesterol oxidase Phenol 4-Aminoantipyrine Peroxidase
ELISA	enzyme-linked immunosorbent assay
FBS	Fasting Blood Glucose
EI	energy intake
FFQ	food frequency questionnaire
FPG	fasting plasma glucose
FBS	fasting blood sugar
FFM	Fat Free Mass
FM	Fat Mass
GOD-PAP	Glucose Oxidase Phenol 4-Aminoantipyrine Peroxidase
GPO-PAP	Glycerol-3-phosphate oxidase Phenol 4-Aminoantipyrine Peroxidase
hs-CRP	Serum hyper sensitive C-reactive protein
HDP	healthy dietary pattern
HOMA-IR	Homeostatic model assessment insulin resistance
HDL-C	High density lipoprotein cholesterol
IR	insulin resistance
IOF	International Obesity Task Force
IDF	International Diabetes Federation
IPAQ	International Physical Activity Questionnaire
IL6	interlukin6
KMO	Kaiser-Meyer-Olkin
LDL	Low density lipoprotein cholesterol
MetS	metabolic syndrome
MH	Metabolically healthy
MUH	Metabolically unhealthy
MHO	Metabolically healthy obesity
MUHO	Metabolically unhealthy obesity
NC	Neck circumference
NAFLD	Non-alcoholic fatty liver disease
OR	odds ratio
PA	physical activity
PCA	Principal component analysis
PCO	Polycystic Ovary Syndrome
T2D	type 2 diabetes
TBW	Total Body Water
Total-Chol	Total cholesterol
TG	Triglyceride
TNF α	Tumor necrosis factor alpha
TUMS	Tehran University of Medical Sciences
UNHDP	unhealthy dietary pattern
USDA	United States Department of Agriculture
WC	waist circumference
WHR	waist to hip ratio
WDP	western dietary pattern

for MHO are controversial, therefore the prevalence of MHO is difficult to quantify, and varies according to the definition used [6]. The prevalence of MHO has been reported as being 10%–25% [7]. However, factors such as race, age, physical activity (PA) have effects on the prevalence of MHO [8]. The underlying mechanisms which cause the MHO phenotype among obese people are multiple and complex. This phenotype is the result of complex interactions between genetic, environmental, and behavioral factors [9]. The absence of genetic traits associated with fat distribution, reduced visceral fat mass and ectopic fat accumulation in liver, muscle, and pancreatic beta cells, and low systemic inflammation all contribute to MHO [10]. Metabolically unhealthy obesity (MUHO), in contrast to the MHO group, displays the typical obesity-related metabolic disturbances of IR, hypertriglyceridemia and possibly elevated risk of developing T2D and CVDs, which could be due to higher fat mass as well as higher visceral fat and liver fat content [11].

Although diet is a modifiable risk factor, controversy remains surrounding the relation between diet and obesity, particularly regarding the role of fat intake [12]. Due to the possibility of many undiscovered compounds in foods, the enormity of interactions among nutrients and foods, and the collinearity among food and nutrient intakes, using a multivariate approach like dietary patterns has the potential to resolve concerns about confounding factors and interactions of foods and nutrients [13]. Furthermore, a dietary pattern approach reflects individuals' dietary behaviors and therefore can provide more detailed information about the nutritional etiology of chronic disease [14]. Several studies have reported the associations of major dietary patterns with obesity and central adiposity [15,16].

However, no study is currently available evaluating major dietary patterns in metabolic healthy (MH)/metabolic unhealthy (MUH) individual based on Karelis criteria. It was therefore decided to test whether differences in major dietary patterns in MUHOW/O and MHOW/O phenotypes can lead to differences in metabolic status.

2. Methods and materials

2.1. Participants

This cross-sectional study was conducted among a representative sample of Tehrani overweight/obese females aged 18–50, selected by a multistage cluster random sampling method. Registered patients in the health center were enrolled in the study, according to inclusion and exclusion criteria. Individuals were included if they met following criteria: age 18–50 years; no current weight loss program; no use of weight loss supplements. Participants with a history of T2D, CVDs, polycystic ovary syndrome (PCO), stroke, non-alcoholic fatty liver disease (NAFLD), inflammatory disease, hypertension, cancer, thyroid diseases, or who were currently pregnant were excluded because of possible changes in diet. Women were also excluded who did not respond to more than 70 food items in the food frequency questionnaire (FFQ), or who reported a total daily energy intake (EI) outside the range of 800–4200 kcal (3344–17556 kJ), as well as those taking medication that could affect plasma lipoproteins (Atorvastatin, Cholestyramine, etc.), blood pressure (Captopril, etc.) and carbohydrate metabolism (Metformin, etc.). After these exclusions, 290 women remained in the current analysis. Each participant was informed completely regarding the study protocol and provided a written and informed consent form before taking part in the study. The study protocol was approved by the ethics committee of Tehran University of Medical Sciences (TUMS) with the following identification IR.TUMS.VCR.REC.1395.1234.