




## Article

# Dietary Intakes and Eating Behavior between Metabolically Healthy and Unhealthy Obesity Phenotypes in Asian Children and Adolescents

Delicia Shu Qin Ooi <sup>1,2,\*</sup> , Jia Ying Toh <sup>3</sup> , Lucas Yan Bin Ng <sup>1,4</sup> , Zikang Peng <sup>1,4</sup>, Supeng Yang <sup>1,4</sup>, Nurul Syafiqah Binte Said Abdul Rashid <sup>1,2</sup>, Andrew Anjian Sng <sup>1,2</sup>, Yiong Huak Chan <sup>5</sup>, Mary Foong-Fong Chong <sup>3,6,†</sup> and Yung Seng Lee <sup>1,2,3,†</sup>

<sup>1</sup> Department of Paediatrics, Yong Loo Lin School of Medicine, National University of Singapore, Singapore 117549, Singapore

<sup>2</sup> Khoo Teck Puat-National University Children's Medical Institute, National University Health System, Singapore 119074, Singapore

<sup>3</sup> Singapore Institute for Clinical Sciences, Agency for Science, Technology and Research, Singapore 117609, Singapore

<sup>4</sup> Hwa Chong Institution, Singapore 269734, Singapore

<sup>5</sup> Biostatistics Unit, Yong Loo Lin School Medicine, National University of Singapore, Singapore 117549, Singapore

<sup>6</sup> Saw Swee Hock School of Public Health, National University of Singapore, Singapore 117549, Singapore

\* Correspondence: paeosqd@nus.edu.sg; Tel.: +65-66013316

† Joint last authors.



**Citation:** Ooi, D.S.Q.; Toh, J.Y.; Ng, L.Y.B.; Peng, Z.; Yang, S.; Rashid, N.S.B.S.A.; Sng, A.A.; Chan, Y.H.; Chong, M.F.-F.; Lee, Y.S. Dietary Intakes and Eating Behavior between Metabolically Healthy and Unhealthy Obesity Phenotypes in Asian Children and Adolescents. *Nutrients* **2022**, *14*, 4796. <https://doi.org/10.3390/nu14224796>

Received: 27 September 2022

Accepted: 9 November 2022

Published: 12 November 2022

**Publisher's Note:** MDPI stays neutral with regard to jurisdictional claims in published maps and institutional affiliations.



**Copyright:** © 2022 by the authors. Licensee MDPI, Basel, Switzerland. This article is an open access article distributed under the terms and conditions of the Creative Commons Attribution (CC BY) license (<https://creativecommons.org/licenses/by/4.0/>).

**Abstract:** Diet plays a critical role in the development of obesity and obesity-related morbidities. Our study aimed to evaluate the dietary food groups, nutrient intakes and eating behaviors of metabolically healthy and unhealthy obesity phenotypes in an Asian cohort of children and adolescents. Participants ( $n = 52$ ) were asked to record their diet using a 3-day food diary and intakes were analyzed using a nutrient software. Eating behavior was assessed using a validated questionnaire. Metabolically healthy obesity (MHO) or metabolically unhealthy obesity (MUO) were defined based on criteria of metabolic syndrome. Children/adolescents with MUO consumed fewer whole grains (median: 0.00 (interquartile range: 0.00–0.00 g) vs. 18.5 g (0.00–69.8 g)) and less polyunsaturated fat (6.26% kcal (5.17–7.45% kcal) vs. 6.92% kcal (5.85–9.02% kcal)), and had lower cognitive dietary restraint (15.0 (13.0–17.0) vs. 16.0 (14.0–19.0)) compared to children/adolescents with MHO. Deep fried food, fast food and processed convenience food were positively associated with both systolic ( $\beta$ : 2.84, 95%CI: 0.95–6.62) and diastolic blood pressure ( $\beta$ : 4.83, 95%CI: 0.61–9.04). Higher polyunsaturated fat intake (OR: 0.529, 95%CI: 0.284–0.986) and cognitive dietary restraint (OR: 0.681, 95%CI: 0.472–0.984) were associated with a lower risk of the MUO phenotype. A healthier diet composition and positive eating behavior may contribute to favorable metabolic outcomes in children and adolescents with obesity.

**Keywords:** dietary intakes; eating behavior; metabolically healthy obesity; children with obesity; adolescents with obesity

## 1. Introduction

Childhood obesity is one of the most pertinent public health challenges worldwide [1]. According to the World Health Organization, over 340 million children and adolescents aged 5–19 were overweight or obese in 2016, with childhood obesity levels reaching epidemic levels in developed countries [2]. Children and adolescents with obesity are at higher risks of reduced quality of life and lower life expectancy as excessive adiposity is the root cause of debilitating metabolic diseases including insulin resistance, hypertension and dyslipidemia [3].

Metabolic health in obesity can be heterogeneous, where not all children with obesity manifest adverse metabolic abnormalities. The subset of children with obesity but who do not present with metabolic abnormalities are classified as having “metabolically healthy obesity” (MHO) [4–6]. By definition, MHO presents a more favorable metabolic profile of higher levels of high-density lipoprotein (HDL) cholesterol and lower levels of blood pressure, fasting triglycerides and fasting glucose compared to their counterpart, “metabolically unhealthy obesity” (MUO) [7–9]. In addition, children with MHO have been shown to be younger, and have a lower waist to hip ratio, body mass index (BMI) percentile and body fat percentage [10–13].

Obesity is influenced by genetic, behavioral and obesogenic environmental factors such as an unhealthy diet and lack of physical activity [14]. In particular, diet is considered a major contributor of obesity and its comorbidities, and is often an important target for intervention strategies in the treatment of obesity and obesity-related morbidities [15]. Greater dietary intakes of glucose and trans fat were shown to be associated with increased risk of obesity and metabolic syndrome [16]. Higher total energy and total fat intake were associated with the MUO phenotype [17], while a higher intake of dietary fiber was found to be a predictor of the MHO phenotype in children [18]. The adherence to a Mediterranean diet, characterized by a high intake of vegetables, fruits, nuts, beans, whole grains and fish, was significantly higher in children with MHO compared to children with MUO [19]. In a study by Camhi et al., children with MHO were found to have a higher healthy eating index compared to children with MUO, and the difference was contributed to by a lower consumption of solid fats and added sugars among the children with MHO [20]. Studies have also demonstrated that children with MUO consume more sugar-sweetened beverages (SSB), salty snacks and fast food than children with MHO [21–23].

Apart from diet, eating behaviors are important in influencing energy balance (positive or negative), which is associated with obesity [24]. Rigid control, disinhibition and emotional susceptibility in eating behaviors were shown to be positively correlated to BMI z-scores among adolescents [25]. Moreover, subjects with metabolic syndrome were shown to display poorer eating behaviors including higher motivation to eat, higher emotional eating and a higher perception gap about feelings of fullness and hunger [26]. Adults with MHO were found to have a lower tendency to overeat when stressed compared to adults with MUO [27].

Since diet and eating behavior are modifiable risk factors, the comparison of dietary intakes and eating behavior between MHO and MUO phenotypes may highlight the nutritional components and food approach practices that can be altered to achieve better metabolic health among individuals with obesity. The reported studies on the MHO and MUO phenotypes in children are mainly based on American and European populations [19–23], who have different dietary intakes compared to Asian populations [28]. Hence, this study aimed to compare the intakes of food groups and nutrients, and eating behavior between metabolically healthy and unhealthy obesity phenotypes in an Asian cohort of children and adolescents.

## 2. Materials and Methods

### 2.1. Study Participants

Children and adolescents with obesity ( $n = 52$ ) included in this study were from the OBesity in Singapore Children (OBiSC) study and they were of Chinese, Malay and Indian ethnicity. The participants were recruited from National University Hospital (NUH) and Health Promotion Board (HPB), Singapore. The recruitment criteria for these children and adolescents with obesity, and aged 7 to 19 years old were: (1) obese before age of 10 years, (2) BMI for age  $\geq 97$ th percentile, (3) no syndromic causes of obesity. Medical examinations and history were obtained during study visits. The study was performed in accordance with the Declaration of Helsinki and ethics approval was obtained from Domain Specific Review Board of National Healthcare Group, Singapore (reference number: 2015/00314).

Written informed consent was obtained from all study participants and their parents or legal guardian. The study is registered under clinicaltrials.gov (NCT02418377).

## 2.2. Anthropometric and Biochemical Measurements

Standard anthropometric parameters including weight, height, waist and hip circumference were measured. BMI was calculated as weight in kilograms (kg) divided by the square of height in meters (m). BMI-standard deviation score (SDS) (also known as BMI z-score), which was adjusted for child's age and sex based on local growth chart, was used to interpret physical development and growth in children and adolescents [29]. Body fat percentage was assessed by bioelectrical impedance analysis (BIA) using Tanita body composition analyzer (Model BC-418). Blood pressure was measured using Carescape V100 Dinamap. Fasting blood samples were obtained and assayed for fasting glucose, fasting insulin and lipids. Blood glucose was also measured at 2 h after the subjects underwent an oral glucose tolerance test (OGTT) by consuming a drink consisting of 75 g glucose. Homeostatic model assessment for insulin resistance (HOMA-IR) was calculated as previously described [30].

## 2.3. Assessment of Dietary Intake

Dietary intake was assessed using a 3-day food diary, which is completed by each participant or their caregiver if the child is below the age of 12 years old. The participant or caregiver of the participant was asked to record the participant's intake of meals, snacks, beverages and supplements for a period of three days. The food diary required participants to include the name of the food or beverage consumed, ingredients used in the meal, amount consumed (portion sizes), preparation methods (type of oil, cooking method) and brand name (if available). Pictures illustrating the portion size or amount of food or beverage were included in the food diary to guide the participants in estimating the amount of food or beverage consumed. A study team member would explain to the participant or their caregiver on how to fill up the food diary, which also contained instructions and examples of food records. The participants were asked to send pictures of the food that they consumed to the study team. A well-trained nutritionist reviewed and verified the written 3-day food diaries with the food pictures sent. The intake of energy, macronutrients, micronutrients and food under the different food groups (in terms of quantity/amount) were analyzed using a nutritional analysis program: Dietplan, Forestfield Software, UK (Version 7.00.62), which consists of a local database of energy and nutrient composition of food, as well as food label information of food products obtained from local stores [31]. Macronutrients were energy adjusted using the nutrient density method [32] and expressed as percentage (%) of total energy (kcal). Micronutrients were expressed as per 1000 kcal of energy: (amount of micronutrient/total energy (kcal))\*1000 kcal.

The acceptable macronutrient distribution range (AMDR) for carbohydrates (%kcal), total fat (%kcal), saturated fat (%kcal), protein (%kcal) and recommended dietary allowance (RDA) for dietary fiber (g/1000 kcal) were obtained from Dietary Guidelines for Americans 2020–2025 [33]. The RDA for calcium (mcg), iron (mcg) and vitamin A (mcg) were obtained from Health Promotion Board Singapore dietary guidelines [34]. Table S1 showed the AMDR for macronutrients and RDA for micronutrients for the various age groups.

The reported foods consumed by the participants were categorized into nine main food groups: deep fried food, fast food and processed convenience food, fish, fruits, savory snacks, sugar-sweetened beverage (SSB), sweet snacks, vegetables and whole grains. Table S2 lists the food items under each of the food groups.

## 2.4. Evaluation of Eating Behavior

The Three-Factor Eating Questionnaire—Revised 18-item version (TFEQ-R18) is a validated questionnaire comprised of three subscales: cognitive dietary restraint, emotional eating and uncontrolled eating [35,36]. The 18 items are on a 4-point response scale, e.g., definitely true/mostly true/mostly false/definitely false, and a score between 1 and 4

is assigned to each response. Item scores are summated into the three subscales and higher scores in the respective subscales are indicative of greater cognitive dietary restraint, emotional or uncontrolled eating.

### 2.5. Classification of MHO and MUO Children/Adolescents

There is currently a lack of consensus on the definition of MHO [37]. However, most studies have defined MHO by either absence of metabolic syndrome (having  $\leq 2$  criteria) or total absence of metabolic abnormalities [37,38]. Children/adolescents with obesity were classified as having MHO or MUO according to two different definitions, metabolic syndrome (MS) and metabolic health (MH), in accordance with a standard protocol [39]. The criteria for both MS and MH definitions are adapted and modified from the International Diabetes Federation (IDF) consensus definition of metabolic syndrome in children and adolescents [40]: (1) hypertriglyceridemia: fasting triglycerides  $\geq 1.7$  mmol/L or on hyperlipidemia medication, (2) dyslipidemia: high-density lipoprotein (HDL) cholesterol  $< 1.03$  mmol/L for children under age of 16, HDL  $< 1.03$  mmol/L for male  $\geq 16$  years old and HDL  $< 1.29$  mmol/L for female  $\geq 16$  years old, (3) abnormal glucose tolerance: fasting glucose  $\geq 5.6$  mmol/L or glucose at 2 h OGTT  $\geq 7.8$  mmol/L or on diabetic medication, (4) elevated blood pressure: blood pressure  $\geq 90$ th percentile based on age, sex and height or on hypertensive medication [41]. In this study, for the MS definition, MHO was considered as being obese with fewer than two of the criteria. For the MH definition, MHO was considered as being obese without any of the criteria.

### 2.6. Statistical Analysis

All analyses were performed using SPSS 27.0 and STATA 17.0 with level of significance set at 2-sided  $p < 0.05$ . Our data did not follow a normal distribution so non-parametric statistical methods were used for data analyses. Descriptive statistics for numerical and categorical variables were presented as median (interquartile range: 25th percentile–75th percentile) and proportion (%), respectively. Differences in clinical characteristics between children/adolescents with MHO and children/adolescents with MUO were analyzed by Mann–Whitney U test for continuous parameters and Chi-square for categorical parameters. Kruskal–Wallis H test was used to analyze the difference in food groups, nutrient intakes and eating behavior across the 3 ethnic groups. Quantile regression was performed to model median differences in food groups, nutrient intakes and eating behavior between children/adolescents with MHO and children/adolescents with MUO, and to analyze the association between food groups/nutrient intake/eating behavior and continuous metabolic parameters with adjustment for age, sex, race and BMI-SDS. Logistic regression was performed to identify the factors (food groups/nutrients/eating behavior) associated with various metabolic conditions and MUO phenotype, with adjustment for age, sex, race and BMI-SDS.

## 3. Results

### 3.1. Clinical Characteristics of Participants

There were no significant differences in demographics data such as age, sex, race and monthly household income, and adiposity outcomes between children/adolescents with MHO and children/adolescents with MUO for both the MS and MH definitions (Table 1).

With regards to the MS definition, children/adolescents with MUO had significantly higher systolic blood pressure (median: 130 mmHg (interquartile range: 124–134 mmHg) vs. 118 mmHg (111–126 mmHg),  $p = 0.003$ ), triglycerides levels (1.55 mmol/L (1.10–2.17 mmHg) vs. 1.01 mmol/L (0.89–1.25 mmHg),  $p = 0.014$ ) and lower HDL cholesterol (1.01 mmol/L (0.89–1.17 mmol/L) vs. 1.17 mmol/L (0.99–1.25 mmol/L),  $p = 0.048$ ) compared to children/adolescents with MHO.

**Table 1.** Clinical characteristics between children/adolescents with MHO and children/adolescents with MUO classified by MS and MH definitions.

Parameter	All (n = 52)	MHO (n = 42)	MUO (n = 10)	p	MHO (n = 12)	MUO (n = 40)	p
Age (years)	14.1 (12.3–16.1)	14.1 (11.8–16.1)	14.1 (13.8–16.3)	0.531	12.6 (8.59–17.0)	14.6 (13.1–16.1)	0.182
Sex (%Male/%Female)	59.6/40.4	60/40	60/40	1.000	83/17	52.5/47.5	0.093
Race (%Chinese/%Malay/%Indian)	40.4/53.8/5.8	38/55/7	50/50/0	0.597	25/67/8	45/50/5	0.457
Monthly household income < SGD 2000 (%)	15.6	15	17	1.000	10	18	1.000
BMI (kg/m <sup>2</sup> )	35.9 (32.1–41.3)	35.9 (31.8–40.9)	36.4 (32.3–44.0)	0.763	33.6 (30.1–39.4)	36.5 (32.4–42.2)	0.152
BMI-SDS	2.43 (2.16–2.64)	2.44 (2.17–2.63)	2.28 (2.05–2.79)	0.781	2.36 (1.99–2.55)	2.44 (2.19–2.68)	0.422
Waist to hip ratio	0.98 (0.93–1.02)	0.98 (0.95–1.02)	0.95 (0.91–1.00)	0.189	1.01 (0.94–1.03)	0.98 (0.93–1.00)	0.142
Body fat percentage (%)	48.2 (39.4–55.4)	47.9 (39.8–55.7)	50.3 (35.5–54.8)	0.952	51.5 (37.1–62.6)	48.0 (40.1–54.3)	0.558
Systolic blood pressure (mmHg)	120 (111–130)	118 (110–126)	130 (124–134)	0.003 *	113 (108–118)	124 (115–132)	0.004 *
Diastolic blood pressure (mmHg)	66 (59–72)	64 (57–72)	70 (62–73)	0.197	58 (55–72)	67 (60–73)	0.080
Total cholesterol (mmol/L)	4.49 (3.92–5.04)	4.52 (4.03–5.03)	4.18 (3.54–5.17)	0.493	4.61 (3.77–5.05)	4.37 (3.98–5.04)	0.991
Triglycerides (mmol/L)	1.12 (0.94–1.35)	1.01 (0.86–1.24)	1.55 (1.10–2.17)	0.014 *	0.88 (0.69–1.24)	1.13 (0.95–1.42)	0.059
HDL cholesterol (mmol/L)	1.11 (0.98–1.24)	1.17 (0.99–1.25)	1.01 (0.89–1.17)	0.048 *	1.24 (1.19–1.29)	1.06 (0.95–1.19)	0.002 *
LDL cholesterol (mmol/L)	2.84 (2.33–3.17)	2.95 (2.40–3.38)	2.52 (2.06–2.95)	0.099	2.96 (2.32–3.17)	2.80 (2.33–3.32)	0.871
Fasting glucose (mmol/L)	4.75 (4.60–5.10)	4.70 (4.60–5.00)	5.00 (4.58–5.20)	0.434	4.65 (4.50–4.78)	4.85 (4.60–5.20)	0.027 *
Glucose at 2 h of OGTT (mmol/L)	5.50 (5.00–6.20)	5.30 (4.88–6.10)	5.85 (5.23–8.55)	0.140	5.30 (5.10–5.68)	5.55 (4.85–6.38)	0.535
Fasting insulin (mU/L)	22.6 (14.3–30.7)	21.4 (14.1–30.4)	25.0 (19.1–38.5)	0.403	17.4 (11.4–25.1)	25.0 (17.2–32.5)	0.039 *
HOMA-IR	4.85 (3.14–6.43)	4.48 (3.09–6.29)	5.41 (3.76–8.84)	0.410	3.67 (2.30–5.22)	5.15 (3.35–7.70)	0.019 *

Data were presented as median (interquartile range: 25th–75th percentile) and percentage (%) for continuous and categorical variables, respectively. Differences in continuous variables between groups were analyzed using Mann–Whitney U test, while differences in categorical variables between groups were analyzed using Chi-square test. Asterisk \* denotes significance of  $p < 0.05$ .

With regards to the MH definition, children/adolescents with MUO had higher systolic blood pressure (125 mmHg (115–132 mmHg) vs. 114 mmHg (108–118 mmHg),  $p = 0.004$ ), fasting glucose (4.90 mmol/L (4.60–5.20 mmHg) vs. 4.70 mmHg (4.50–4.80 mmHg),  $p = 0.027$ ), fasting insulin (24.7 mU/L (16.9–33.0 mU/L) vs. 19.7 mU/L (13.0–26.3 mU/L),  $p = 0.039$ ), HOMA-IR (5.15 (3.30–7.73) vs. 4.26 (2.72–5.49),  $p = 0.019$ ) and lower HDL cholesterol (1.06 mmol/L (0.95–1.19 mmol/L) vs. 1.24 mmol/L (1.19–1.29 mmol/L),  $p = 0.002$ ) levels compared to children/adolescents with MHO.

### 3.2. Food Groups, Nutrient Intakes and Eating Behavior between Children/adolescents with MHO and Children/Adolescents with MUO

With regards to the MH definition, children/adolescents with MUO were found to consume a significantly lower amount of whole grains (0.00 (0.00–0.00 g) vs. 18.5 g (0.00–69.8 g),  $p = 0.027$ ) and polyunsaturated fat (6.26% kcal (5.17–7.45% kcal) vs. 6.92% kcal (5.85–9.02% kcal),  $p = 0.027$ ), and displayed lower cognitive restraint in eating (15.0 (13.0–17.0) vs. 16.0 (14.0–19.0),  $p = 0.009$ ) compared to children/adolescents with MHO (Table 2).

With regards to the MS definition, there were no significant differences in food groups, nutrients intake, eating behavior and percentage of participants meeting the AMDR and RDA of nutrients between children/adolescents with MHO and children/adolescents with MUO (Table S3).

**Table 2.** Food groups, nutrients intakes and eating behavior between children/adolescents with MHO and children/adolescents with MUO by MH definition.

	MH Definition		
	MHO (n = 12)	MUO (n = 40)	p
<b>Food groups (continuous variables)</b>			
Deep fried food (g)	76.6 (19.0–136)	55.1 (39.3–129)	0.558
Fast food and processed convenience food (g)	121 (16.1–152)	54.1 (0.00–135)	0.502
Fish (g)	0.00 (0.00–0.00)	0.00 (0.00–66.7)	0.788
Fruits (g)	17.7 (0.00–44.4)	0.00 (0.00–39.8)	0.721
Savory snacks (g)	1.22 (0.00–21.3)	5.00 (0.00–34.5)	0.965
Sugar-sweetened beverage, SSB (ml)	342 (163–421)	278 (161–464)	0.417
Sweet snacks (g)	52.0 (6.25–118)	23.5 (0.00–58.2)	0.171
Vegetables (g)	73.5 (43.7–113)	82.3 (37.5–140)	0.430
Whole grains (g)	18.5 (0.00–69.8)	0.00 (0.00–0.00)	0.027 *



Table 2. Cont.

	MH Definition		
	MHO ( <i>n</i> = 12)	MUO ( <i>n</i> = 40)	<i>p</i>
Nutrients (continuous variables)			
Total energy (kcal)	1856 (1670–2470)	1855 (1730–2260)	0.239
Carbohydrates (%kcal)	49.1 (46.8–52.1)	45.3 (40.2–51.9)	0.539
Protein (%kcal)	16.4 (14.7–17.6)	17.7 (15.4–21.4)	0.536
Total fat (%kcal)	34.4 (32.8–37.2)	36.0 (31.8–39.5)	0.918
Saturated fat (%kcal)	12.6 (10.1–14.2)	12.3 (11.1–14.3)	0.319
Monounsaturated fat (%kcal)	11.7 (10.4–12.5)	13.5 (11.4–14.9)	0.851
Polyunsaturated fat (%kcal)	6.92 (5.85–9.02)	6.26 (5.17–7.45)	0.027 *
Beta-carotene (mcg per 1000 kcal)	5.16 (0.00–50.1)	0.19 (0.00–3.83)	0.655
Calcium (mg per 1000 kcal)	304 (183–368)	252 (209–298)	0.166
Cholesterol (mg per 1000 kcal)	172 (101–229)	198 (149–228)	0.160
Dietary fiber (g per 1000 kcal)	6.73 (5.85–7.59)	6.60 (5.98–7.82)	0.955
Iron (mg per 1000 kcal)	6.54 (5.02–7.21)	6.03 (5.24–7.09)	0.719
Sodium (mg per 1000 kcal)	1550 (1410–1840)	1780 (1360–2050)	0.839
Vitamin A (mcg per 1000 kcal)	248 (114–338)	258 (187–363)	0.797
% of participants meeting AMDR/RDA of nutrients			
Carbohydrates † (AMDR)	91.7	50	0.186
Total fat † (AMDR)	58.3	42.5	0.924
Saturated fat † (AMDR)	16.7	17.5	0.689
Protein † (AMDR)	100	100	1.000
Calcium ‡ (RDA)	8.3	5	0.498
Dietary fiber † (RDA)	8.3	5	0.891
Iron ‡ (RDA)	83.3	50	0.290
Vitamin A ‡ (RDA)	50	20	0.072
Eating behavior (continuous variables)			
Cognitive dietary restraint	16.0 (14.0–19.0)	15.0 (13.0–17.0)	0.009 *
Emotional eating	6.00 (3.25–6.00)	6.00 (4.00–8.00)	1.000
Uncontrolled eating	22.0 (18.0–24.5)	21.0 (19.0–24.0)	0.766

Data were presented as median (interquartile range: 25th–75th percentile) and percentage (%) for continuous and categorical variables, respectively. Differences in continuous variables between groups were analyzed using quantile regression with adjustment for age, sex, race and BMI-SDS, while differences in categorical variables between groups were analyzed using logistic regression with adjustment for age, sex, race and BMI-SDS. Asterisk \* denotes significance of  $p < 0.05$ . † AMDR and RDA of nutrients were according to Dietary Guidelines for Americans 2020–2025, ‡ RDA of nutrients were according to dietary guidelines by Health Promotion Board, Singapore.

### 3.3. Food Groups, Nutrient Intakes and Eating Behavior between Children/Adolescents with MHO and Children/Adolescents with MUO Stratified by Sex or Race

Gender differences [42,43] and ethnicity [44,45] have been reported to influence dietary intakes. Hence, we further stratified the children/adolescents with obesity by sex or race, and examined the nutrient intakes between children/adolescents with MHO and children/adolescents with MUO.

With regards to the MH definition, male children/adolescents with MUO consumed a significantly lower amount of fruits (0.00 g (0.00–17.9 g) vs. 23.3 g (0.00–48.9 g),  $p = 0.010$ ) and reported lower cognitive dietary restraint (14.0 (13.0–16.0) vs. 15.5 (13.8–19.0),  $p = 0.031$ ) compared to male children/adolescents with MHO. Female children/adolescents with MUO were found to consume a significantly lower amount of whole grains (0.00 g (0.00–0.00 g) vs. 133 g,  $p < 0.001$ ), and exhibited higher emotional (7.00 (5.00–8.00) vs. 3.00,  $p = 0.043$ ) and uncontrolled eating (21.0 (18.0–23.0) vs. 17.0,  $p = 0.027$ ) compared to female children/adolescents with MHO (Table 3).

With regards to the MS definition, there were no significant differences in food groups, nutrients intake and eating behavior between children/adolescents with MHO and children/adolescents with MUO stratified by sex (Table S4).

There were significant differences in vegetables, carbohydrates and protein between the three ethnic groups. However, due to the small sample size of Indian children/adolescents ( $n = 3$ ) within the cohort, comparisons were made between Chinese and Malay children/adolescents. There were significant differences in vegetable, carbohydrate, protein, total fat and saturated fat intakes between Chinese and Malay children/adolescents (Table S5).

**Table 3.** Food groups, nutrient intakes and eating behavior between children/adolescents with MHO and children/adolescents with MUO (MH definition) stratified by sex.

	MH Definition					
	Male			Female		
	MHO (n = 10)	MUO (n = 21)	p	MHO (n = 2)	MUO (n = 19)	p
<b>Food groups</b>						
Deep fried food (g)	53.4 (7.18–110)	80.9 (43.3–138)	0.997	204	43.8 (23.3–106)	0.401
Fast food and processed convenience food (g)	121 (19.5–150)	75.0 (0.00–151)	0.855	107	50.0 (0.00–133)	0.182
Fish (g)	0.00 (0.00–0.00)	14.0 (0.00–95.5)	0.577	0.00	0.00 (0.00–47.7)	0.774
Fruits (g)	23.3 (0.00–48.9)	0.00 (0.00–17.9)	0.010 *	13.1	4.00 (0.00–60.0)	0.970
Savory snacks (g)	1.22 (0.00–19.8)	0.00 (0.00–33.1)	0.940	16.7	12.8 (0.00–44.5)	0.742
Sugar-sweetened beverage, SSB (ml)	355 (209–472)	257 (129–472)	0.163	182	313 (207–444)	0.212
Sweet snacks (g)	52.0 (18.8–121)	16.7 (0.00–50.8)	0.097	61.8	26.7 (7.67–66.7)	0.202
Vegetables (g)	66.1 (39.8–118)	71.3 (35.9–112)	0.725	86.3	85.3 (36.7–159)	0.555
Whole grains (g)	17.2 (0.00–38.6)	0.00 (0.00–42.3)	0.933	133	0.00 (0.00–0.00)	<0.001 *
<b>Nutrients</b>						
Total energy (kcal)	1860 (1670–2460)	2110 (1790–2420)	0.253	2080	1780 (1680–1900)	0.432
Carbohydrates (% kcal)	49.1 (47.1–52.7)	49.0 (38.6–52.8)	0.900	43.1	44.0 (41.1–50.6)	0.052
Protein (% kcal)	16.0 (14.5–17.3)	17.6 (15.9–21.5)	0.987	19.4	19.7 (14.0–21.4)	0.396
Total fat (% kcal)	34.4 (32.9–36.4)	34.7 (30.8–39.7)	0.409	37.5	36.2 (32.7–39.6)	0.971
Saturated fat (% kcal)	12.3 (10.0–13.7)	11.6 (9.93–13.8)	0.371	14.3	12.7 (12.1–14.9)	0.386
Monounsaturated fat (% kcal)	11.7 (10.0–12.2)	13.5 (11.4–15.1)	0.786	13.5	13.4 (9.77–14.5)	0.244
Polyunsaturated fat (% kcal)	7.47 (5.46–9.25)	6.09 (5.11–7.80)	0.268	6.82	6.56 (5.67–7.24)	0.746
Beta-carotene (mcg per 1000 kcal)	3.39 (0.00–59.3)	0.00 (0.00–1.32)	0.548	15.4	0.81 (0.00–27.6)	0.801
Calcium (mg per 1000 kcal)	298 (158–318)	225 (195–284)	0.367	383	277 (221–359)	0.282
Cholesterol (mg per 1000 kcal)	161 (83.7–185)	208 (160–254)	0.097	305	190 (136–219)	0.096
Dietary fiber (g per 1000 kcal)	6.73 (5.72–7.80)	6.67 (5.85–8.14)	0.397	6.93	6.44 (5.97–7.53)	0.982
Iron (mg per 1000 kcal)	6.54 (4.94–7.14)	6.01 (5.24–7.29)	0.367	7.12	6.55 (5.23–6.92)	0.540
Sodium (mg per 1000 kcal)	1520 (1290–1840)	1760 (1480–2050)	0.795	1680	1890 (1340–2060)	0.957
Vitamin A (mcg per 1000 kcal)	188 (108–300)	219 (123–353)	0.275	395	314 (227–402)	0.348
<b>Eating behavior</b>						
Cognitive dietary restraint	15.5 (13.8–19.0)	14.0 (13.0–16.0)	0.031 *	17.5	17.0 (14.0–18.0)	0.999
Emotional eating	6.00 (5.50–6.50)	5.00 (3.50–6.00)	0.490	3.00	7.00 (5.00–8.00)	0.043 *
Uncontrolled eating	23.0 (18.0–25.0)	21.0 (19.0–24.0)	0.772	17.0	21.0 (18.0–23.0)	0.027 *

Data were presented as median (interquartile range: 25th–75th percentile). Due to small sample size for female MHO children/adolescents ( $n = 2$ ), no interquartile range is available. Differences in continuous variables between groups were analyzed using quantile regression with adjustment for age, race and BMI-SDS. Asterisk \* denotes significance of  $p < 0.05$ .

With regards to the MH definition, among the Chinese participants, children/adolescents with MUO had a significantly lower SSB intake (237 mL (108–336 mL) vs. 333 mL,  $p = 0.020$ ) and higher monounsaturated fat intake (14.1% kcal (13.2–15.1% kcal) vs. 9.88% kcal,  $p = 0.002$ ) than children/adolescents with MHO. Among the Malay participants, children/adolescents with MUO had lower polyunsaturated fat intake (5.84% kcal (5.17–6.92% kcal) vs. 8.38% kcal (6.03–9.35% kcal),  $p = 0.039$ ), and reported higher cognitive dietary restraint (16.0 (13.0–18.0) vs. 15.5 (14.0–19.0),  $p = 0.016$ ) compared to children/adolescents with MHO (Table 4).

With regards to the MS definition, there were no significant differences in food groups, nutrients intake and eating behavior between children/adolescents with MHO and children/adolescents with MUO stratified by race (Table S6).

### 3.4. Association between Food Groups/Nutrients/Eating Behavior and Risk Factors of Metabolic Syndrome

Iron intake was found to be negatively associated with HDL cholesterol ( $\beta$ : −2.34, 95%CI: −4.65– −0.04) and glucose level at 2 h OGTT ( $\beta$ : −0.37, 95%CI: −0.67– −0.07), while deep fried food, processed food and convenience food intakes were positively associated with both systolic ( $\beta$ : 2.84, 95%CI: 0.95–6.62) and diastolic ( $\beta$ : 4.83, 95%CI: 0.61–9.04) blood pressure (Table 5).

We also examined the association between food groups/nutrients/eating behavior and metabolic conditions. Protein (OR= 0.791, 95%CI: 0.642–0.974), calcium (OR= 0.991, 95%CI: 0.982–1.000) and iron (OR= 0.527, 95%CI: 0.309–0.899) intakes, and cognitive dietary restraint (OR: 0.711, 95%CI: 0.523–0.966) were associated with a lower risk of elevated blood pressure. Iron intake was associated with a higher risk of dyslipidemia in HDL cholesterol (OR= 2.363, 95%CI: 1.258–4.437) but a lower risk of abnormal glucose tolerance (OR= 0.349,

95%CI: 0.134–0.908). Polyunsaturated fat intake (OR= 0.529, 95%CI: 0.284–0.986) and cognitive dietary restraint (OR: 0.681, 95%CI: 0.472–0.984) were associated with a lower risk of MUO by the MH definition (Table 6).

**Table 4.** Food groups, nutrient intakes and eating behavior between children/adolescents with MHO and children/adolescents with MUO (MH definition) stratified by race.

	MH Definition					
	Chinese			Malay		
	MHO ( <i>n</i> = 3)	MUO ( <i>n</i> = 18)	<i>p</i>	MHO ( <i>n</i> = 8)	MUO ( <i>n</i> = 20)	<i>p</i>
<b>Food groups</b>						
Deep fried food (g)	47.3	78.8 (42.3–161)	0.972	78.3 (19.2–151)	48.8 (23.5–119)	0.274
Fast food and processed convenience food (g)	0.00	54.1 (0.00–123)	0.389	129 (35.7–198)	75.3 (0.00–170)	0.296
Fish (g)	0.00	0.00 (0.00–105)	1.000	0.00 (0.00–0.00)	5.00 (0.00–66.7)	0.771
Fruits (g)	26.1	7.07 (0.00–62.1)	0.963	4.58 (0.00–52.2)	0.00 (0.00–29.5)	1.000
Savory snacks (g)	0.00	6.42 (0.00–30.2)	0.551	15.8 (0.61–30.5)	0.00 (0.00–44.9)	0.683
Sugar-sweetened beverage, SSB (ml)	333	237 (108–336)	0.020 *	291 (57.5–378)	370 (219–588)	0.194
Sweet snacks (g)	124	23.5 (5.75–63.7)	0.750	34.3 (6.25–90.7)	22.5 (0.00–48.9)	0.422
Vegetables (g)	70.5	125 (53.7–169)	0.387	66.1 (43.7–101)	50.1 (29.9–92.0)	0.466
Whole grains (g)	19.0	0.00 (0.00–0.00)	0.680	17.2 (0.00–69.8)	0.00 (0.00–15.9)	0.209
<b>Nutrients</b>						
Total energy (kcal)	1670	1860 (1770–2300)	0.830	1860 (1680–2400)	1820 (1680–2220)	0.358
Carbohydrates (% kcal)	52.6	40.5 (36.3–43.9)	0.051	47.6 (45.7–49.5)	50.6 (46.0–53.2)	0.076
Protein (% kcal)	14.6	21.3 (17.1–23.2)	0.224	16.9 (15.8–18.7)	15.8 (14.0–19.6)	0.211
Total fat (% kcal)	32.8	39.1 (35.7–40.7)	0.058	35.7 (34.3–37.9)	32.6 (30.6–36.2)	0.076
Saturated fat (% kcal)	13.0	12.4 (11.9–14.9)	0.922	10.9 (9.84–14.9)	11.8 (10.1–13.2)	0.226
Monounsaturated fat (% kcal)	9.88	14.1 (13.2–15.1)	0.002 *	12.0 (11.6–14.3)	12.1 (9.90–14.2)	0.991
Polyunsaturated fat (% kcal)	6.46	6.83 (6.00–7.96)	0.215	8.38 (6.03–9.35)	5.84 (5.17–6.92)	0.039 *
Beta-carotene (mcg per 1000 kcal)	24.9	0.09 (0.00–1.50)	0.066	3.39 (0.00–45.3)	0.27 (0.00–4.86)	0.872
Calcium (mg per 1000 kcal)	316	233 (199–288)	0.585	298 (151–368)	259 (205–299)	0.332
Cholesterol (mg per 1000 kcal)	85.7	213 (179–248)	0.236	176 (158–229)	198 (116–222)	0.484
Dietary fiber (g per 1000 kcal)	7.39	6.22 (5.83–8.10)	0.712	6.67 (5.56–7.49)	7.08 (6.03–7.71)	0.148
Iron (mg per 1000 kcal)	6.50	5.83 (5.24–6.70)	0.735	6.84 (5.26–7.21)	6.72 (5.06–7.34)	0.365
Sodium (mg per 1000 kcal)	964	1860 (1370–2560)	0.352	1630 (1440–1840)	1700 (1390–2050)	0.834
Vitamin A (mcg per 1000 kcal)	286	288 (195–403)	0.997	248 (121–338)	250 (155–325)	0.468
<b>Eating behavior</b>						
Cognitive dietary restraint	15.0	14.0 (13.0–17.0)	0.612	15.5 (14.0–19.0)	16.0 (13.0–18.0)	0.016 *
Emotional eating	3.00	5.50 (3.75–8.00)	0.085	6.00 (6.00–7.50)	6.00 (4.00–7.75)	0.398
Uncontrolled eating	19.0	21.0 (18.0–25.0)	0.110	23.0 (18.8–25.0)	21.0 (19.0–23.0)	0.549

Data were presented as median (interquartile range: 25th–75th percentile). Due to small sample size for Chinese MHO children/adolescents (*n* = 3), no interquartile range is available. Differences in continuous variables between groups were analyzed using quantile regression with adjustment for age, sex and BMI-SDS. Asterisk \* denotes significance of *p* < 0.05.



**Table 5.** Association between food groups/nutrients/eating behavior and continuous metabolic parameters.

	BMI-SDS		Triglycerides		HDL Cholesterol		Fasting Glucose		Glucose at 2 h OGTT		Systolic Blood Pressure		Diastolic Blood Pressure	
	$\beta$	95% CI	$\beta$	95% CI	$\beta$	95% CI	$\beta$	95% CI	$\beta$	95% CI	$\beta$	95% CI	$\beta$	95% CI
<b>Food groups</b>														
Deep fried food (g)	6.01	−83.5–95.5	−0.85	−47.1–45.4	82.0	−55.6–220	−7.16	−60.6–46.3	−0.16	−17.0–16.7	2.84 *	0.95–6.62 *	0.84	−2.93–4.61
Fast food and processed convenience food (g)	−17.0	−140–106	−31.8	−92.9–29.3	−35.5	−248–177	7.25	−67.8–82.3	1.86	−21.2–25.0	1.02	−2.89–4.92	4.83*	0.61–9.04 *
Fish (g)	0.00	−52.3–52.3	0.00	−28.9–28.9	−23.5	−114–67.1	0.00	−32.1–32.1	7.39	−2.32–17.1	0.00	−1.78–1.78	0.00	−2.05–2.05
Fruits (g)	7.72	−24.1–39.6	0.22	−15.9–16.3	6.00	−47.2–59.2	−6.83	−24.7–11.0	−0.80	−6.97–5.37	−0.05	−1.13–1.02	0.19	−1.05–1.42
Savory snacks (g)	16.6	−12.8–46.0	−0.50	−16.4–15.4	−14.7	−69.0–39.6	−9.14	−28.0–9.72	−1.81	−7.61–3.99	−0.04	−1.05–0.97	0.95	−0.25–2.15
Sugar-sweetened beverage, SSB (ml)	−79.8	−322–163	−28.5	−164–107	133	−370–635	31.5	−112–175	−25.9	−72.8–21.1	−1.33	−9.85–7.19	−3.73	−12.9–5.44
Sweet snacks (g)	7.37	−43.7–58.3	0.27	−26.6–27.1	24.9	−59.9–110	0.35	−30.9–31.6	2.02	−7.74–11.8	−0.02	−1.61–1.57	−0.02	−1.86–1.82
Vegetables (g)	38.3	−38.4–115	−9.54	−48.7–29.6	−89.9	−212–32.0	5.74	−40.1–51.6	15.6	−2.98–28.1	−1.31	−3.65–1.03	−1.02	−4.00–1.95
Whole grains (g)	0.00	−14.4–14.4	0.00	−7.64–7.64	0.00	−45.8–45.8	0.00	−6.94–6.94	0.00	−2.64–2.64	0.00	−0.83–0.83	0.00	−1.02–1.02
<b>Macronutrients</b>														
Carbohydrates (% kcal)	−2.09	−9.80–5.63	1.58	−2.22–5.39	9.57	−3.09–16.06	2.42	−2.18–7.01	0.91	−0.42–2.23	0.15	−0.12–0.42	0.09	−0.19–0.37
Protein (% kcal)	2.19	−2.44–6.82	−0.73	−3.08–1.62	−7.25	−14.8–0.31	−1.31	−4.12–1.51	0.38	−0.50–1.26	−0.08	−0.23–0.07	−0.03	−0.20–0.15
Total fat (% kcal)	2.48	−3.65–8.62	−1.39	−4.67–1.88	−1.49	−11.6–8.64	0.06	−3.60–3.72	−0.41	−1.52–0.69	0.01	−0.17–0.20	0.01	−0.21–0.23
Saturated fat (% kcal)	0.39	−2.66–3.44	−0.48	−2.17–1.22	−1.51	−6.10–3.07	0.15	−1.76–2.06	−0.20	−0.73–0.33	−0.01	−0.11–0.08	−0.02	−0.13–0.09
Monounsaturated fat (% kcal)	1.29	−1.77–4.35	1.26	−0.35–2.86	−0.90	−6.01–4.22	1.11	−0.61–2.83	−0.28	−0.91–0.36	−0.05	−0.16–0.06	−0.02	−0.14–0.10
Polyunsaturated fat (% kcal)	−0.078	−1.89–1.73	−0.57	−1.53–0.39	2.81	−0.74–6.35	−0.19	−1.29–0.91	−0.05	−0.44–0.33	−0.00	−0.06–0.06	0.05	−0.02–0.12
<b>Micronutrients</b>														
Beta-carotene (mcg per 1000 kcal)	−0.00	−16.2–16.2	−0.41	−8.86–8.03	0.00	−27.7–27.7	−1.72	−11.4–7.99	−0.32	−3.50–2.86	−0.01	−0.65–0.64	0.00	−0.83–0.83
Calcium (mg per 1000 kcal)	−10.6	−116–94.7	−9.17	−64.0–45.6	127	−36.1–290	−6.82	−71.2–57.5	−10.7	−31.1–9.69	−2.40	−5.98–1.18	−1.08	−5.03–2.87
Cholesterol (mg per 1000 kcal)	−10.0	−91.2–71.1	−10.4	−52.9–32.2	−62.7	−213–87.2	−17.4	−68.1–33.3	−6.91	−23.1–9.32	0.94	−2.07–3.95	−0.30	−3.37–2.78
Dietary fiber (g per 1000 kcal)	−0.33	−2.36–1.69	0.06	−0.87–0.98	−0.87	−4.32–2.59	−0.12	−1.30–1.07	−0.09	−0.45–0.26	−0.01	−0.08–0.06	0.02	−0.07–0.10
Iron (mg per 1000 kcal)	−0.89	−2.09–0.91	0.36	−0.37–1.09	−2.34 *	−4.65–−0.04 *	−0.17	−1.09–0.76	−0.37 *	−0.67–−0.07 *	−0.04	−0.08–0.01	−0.02	−0.07–0.04
Sodium (mg per 1000 kcal)	207	−507–921	−34.7	−372–303	576	−519–1672	165	−272–601	−59.7	−196–77	−7.49	−31.4–16.4	−2.69	−27.5–22.1
Vitamin A (mcg per 1000 kcal)	−60.1	−210–90.3	−43.8	−120–32.7	−62.8	−328–202	−58.1	−142–25.7	−13.2	−40.0–13.6	−0.80	−6.10–4.51	−1.48	−7.40–4.44
<b>Eating behavior</b>														
Cognitive dietary restraint	−0.63	−3.87–2.61	0.15	−1.65–1.95	1.29	−3.91–6.49	−0.06	−2.06–1.95	0.09	−0.53–0.71	−0.06	−0.17–0.04	0.02	−0.11–0.15
Emotional eating	−0.00	−2.54–2.54	0.00	−1.43–1.43	−0.00	−4.44–4.44	0.00	−1.51–1.51	0.00	−0.48–0.48	−0.00	−0.09–0.09	0.05	−0.05–0.15
Uncontrolled eating	1.62	−2.55–5.78	−0.98	−3.11–1.15	4.15	−3.09–11.4	−0.74	−3.20–1.71	−0.32	−1.07–0.44	−0.07	−0.19–0.06	0.05	−0.11–0.21

Data for the total sample size ( $n = 52$ ) were presented as  $\beta$ , 95% confidence interval (CI). Association between food groups/nutrients/eating behavior and metabolic parameters (except BMI-SDS) was analyzed using quantile regression with adjustment for age, sex, race and BMI-SDS (for BMI-SDS, analysis was adjusted for age, sex and race only). Asterisk \* denotes significance of  $p < 0.05$ .

**Table 6.** Nutritional factors predictive of metabolic conditions.

	Elevated Blood Pressure		Hypertriglyceridemia		Dyslipidemia (HDL)		Abnormal Glucose Tolerance		MUO (MS Definition)		MUO (MH Definition)	
	OR	95% CI	OR	95% CI	OR	95% CI	OR	95% CI	OR	95% CI	OR	95% CI
<b>Food groups</b>												
Deep fried food (g)	1.005	0.997–1.013	1.003	0.993–1.014	0.997	0.989–1.005	0.994	0.980–1.007	1.000	0.991–1.009	0.996	0.986–1.006
Fast food and processed convenience food (g)	1.002	0.995–1.008	0.992	0.980–1.005	0.999	0.993–1.006	0.994	0.981–1.007	0.998	0.989–1.006	0.997	0.989–1.005
Fish (g)	1.007	0.994–1.020	1.010	0.992–1.029	0.998	0.984–1.013	1.013	0.996–1.031	1.009	0.994–1.023	1.028	0.999–1.059
Fruits (g)	1.007	0.992–1.021	1.001	0.978–1.024	0.992	0.977–1.007	0.993	0.973–1.013	1.001	0.985–1.018	1.004	0.982–1.026
Savory snacks (g)	1.010	0.992–1.028	0.991	0.959–1.024	1.003	0.984–1.022	1.002	0.976–1.029	1.014	0.993–1.034	1.019	0.980–1.059
Sugar-sweetened beverage, SSB (ml)	1.002	1.000–1.005	1.001	0.998–1.003	1.000	0.997–1.002	0.994	0.987–1.001	1.002	0.999–1.004	1.001	0.998–1.004
Sweet snacks (g)	0.997	0.986–1.009	0.994	0.975–1.014	0.996	0.983–1.009	0.986	0.960–1.014	0.995	0.978–1.011	0.988	0.974–1.002
Vegetables (g)	0.997	0.990–1.004	0.988	0.970–1.007	1.001	0.994–1.008	1.004	0.997–1.012	1.001	0.993–1.008	0.998	0.990–1.007
Whole grains (g)	0.991	0.975–1.007	1.006	0.990–1.023	1.004	0.990–1.018	0.996	0.976–1.015	1.006	0.992–1.021	0.993	0.978–1.008
<b>Macronutrients</b>												
Carbohydrates (% kcal)	1.100	0.982–1.232	1.022	0.880–1.187	0.913	0.811–1.028	0.994	(0.864–1.142)	1.020	0.905–1.150	0.988	0.861–1.134
Protein (% kcal)	0.791 *	0.642–0.974 *	0.980	0.741–1.297	1.115	0.929–1.340	1.213	(0.955–1.541)	0.942	0.764–1.160	1.074	0.830–1.391
Total fat (% kcal)	0.976	0.848–1.122	0.972	0.799–1.182	1.083	0.935–1.253	0.871	(0.706–1.074)	1.002	0.852–1.179	0.983	0.814–1.188
Saturated fat (% kcal)	1.057	0.795–1.405	0.918	0.618–1.365	0.986	0.725–1.341	0.903	(0.581–1.403)	1.125	0.807–1.570	0.799	0.555–1.153
Monounsaturated fat (% kcal)	0.981	0.772–1.247	1.353	0.909–2.012	1.004	0.780–1.291	1.009	(0.700–1.455)	1.146	0.843–1.558	1.133	0.841–1.526
Polyunsaturated fat (% kcal)	0.870	0.598–1.265	0.957	0.584–1.568	0.914	0.613–1.363	0.623	(0.318–1.222)	0.803	0.502–1.284	0.529 *	0.284–0.986 *
<b>Micronutrients</b>												
Beta-carotene (mcg per 1000 kcal)	0.977	0.939–1.017	0.360	0.070–1.859	1.000	0.995–1.005	0.984	(0.924–1.048)	0.982	0.925–1.042	0.995	0.989–1.001
Calcium (mg per 1000 kcal)	0.991 *	0.982–1.000 *	0.997	0.987–1.007	1.002	0.995–1.009	0.999	(0.989–1.009)	0.992	0.981–1.003	0.997	0.989–1.005
Cholesterol (mg per 1000 kcal)	1.001	0.992–1.010	1.009	0.995–1.022	1.007	0.997–1.017	1.006	(0.994–1.018)	1.005	0.994–1.015	1.003	0.992–1.014
Dietary fiber (g per 1000 kcal)	0.714	0.473–1.079	0.967	0.584–1.601	1.188	0.863–1.635	0.747	(0.416–1.340)	0.655	0.365–1.175	1.015	0.702–1.468
Iron (mg per 1000 kcal)	0.527 *	0.309–0.899 *	1.033	0.583–1.832	2.363 *	1.258–4.437 *	0.349 *	(0.134–0.908) *	0.716	0.416–1.231	1.154	0.643–2.071
Sodium (mg per 1000 kcal)	1.000	0.999–1.001	1.000	0.999–1.002	0.999	0.998–1.000	1.000	(0.999–1.002)	0.999	0.998–1.001	1.001	0.999–1.003
Vitamin A (mcg per 1000 kcal)	0.998	0.993–1.002	0.996	0.989–1.003	1.001	0.997–1.005	1.002	(0.996–1.008)	0.998	0.992–1.003	1.000	0.995–1.004
<b>Eating behavior</b>												
Cognitive dietary restraint	0.711 *	0.523–0.966 *	0.987	0.690–1.413	0.747	0.527–1.059	1.259	0.853–1.859	0.797	0.557–1.139	0.681 *	0.472–0.984 *
Emotional eating	0.866	0.641–1.172	1.017	0.695–1.488	0.958	0.698–1.314	0.968	0.650–1.442	0.877	0.617–1.246	1.084	0.756–1.553
Uncontrolled eating	0.972	0.805–1.174	0.987	0.763–1.275	1.027	0.840–1.256	0.934	0.720–1.213	0.925	0.745–1.148	1.109	0.873–1.410

Data for the total sample size ( $n = 52$ ) were presented odds ratio (OR), 95%CI. Association between food groups/nutrients/eating behavior and metabolic abnormalities was analyzed using logistic regression with adjustment for age, sex, race and BMI-SDS. Asterisk \* denotes significance of  $p < 0.05$ .

#### 4. Discussion

Our findings demonstrated variations in dietary intakes between children/adolescents with MHO and children/adolescents with MUO for different MHO definitions. Significant differences in dietary factors between children/adolescents with MHO and children/adolescents with MUO were reported with the more stringent MH definition [39]. The lack of significant differences for the MS definition may be due to the heterogeneity of the definition as some children/adolescents with MHO under the MS definition would have one metabolic abnormality that may overall be similar to the MUO phenotype [37]. Hence, our results highlighted the importance of establishing a consensus definition for MHO [37,38,46]. Children/adolescents with MHO were found to consume more whole grains and polyunsaturated fats in their diet, and had higher cognitive dietary restraint than children/adolescents with MUO. Whole grains are rich in dietary fiber, vitamins, minerals and beneficial phytochemicals from plants [47], and they are recommended in dietary guidelines for healthy eating [48,49]. An increased consumption of whole grains was associated with a lower risk of metabolic syndrome [50], and subjects with MHO were found to have a higher intake of whole grains compared to subjects with MUO [20]. Polyunsaturated fats are considered the good fats, which help to reduce bad cholesterol (LDL) in the blood and lower cardiovascular risk [51], and they were shown to ameliorate obesity and obesity-induced metabolic syndrome [52]. Telle-Hansen et al. also reported lower levels of total polyunsaturated fatty acid in subjects with MUO compared to subjects with MHO [53]. There were no significant differences in the proportion of participants meeting the recommended intake of macronutrients and micronutrients between children/adolescents with MHO and children/adolescents with MUO. Our cohort of children/adolescents with obesity had a higher intake of saturated fat and lower intake of micronutrients including calcium, dietary fiber, iron and vitamin A than recommended amounts [33]. Cognitive dietary restraint is the perceived effort to limit dietary intake [54] and higher cognitive dietary restraint is associated with a reduction in adiposity outcomes [55]. Our observation of higher cognitive dietary restraint among children/adolescents with MHO may indicate an intentional dietary restriction to improve metabolic health outcomes. We did not find significant differences in energy intake and other macro- and micronutrient intakes between children/adolescents with MHO and children/adolescents with MUO. Although this lack of significant differences was also observed in other studies [56,57], our small sample size of children/adolescents with MHO and MUO phenotypes may have contributed to the lack of statistical significant difference in the dietary intakes.

We found that dietary intakes of fruits and whole grains, and eating behavior were significantly different between children/adolescents with MHO and children/adolescents with MUO stratified by sex, and our findings are consistent with other studies that reported gender difference in dietary intakes and eating behavior [58,59]. There were also significant differences in SSB, monounsaturated fat and polyunsaturated fat intakes as well as cognitive dietary restraint between children/adolescents with MHO and children/adolescents with MUO stratified by race. This supports our aim to investigate dietary intakes and eating behavior between MHO and MUO phenotypes in our local cohort of children/adolescents due to the differing diets between different ethnic groups [44,45]. However, the stratification analyses by sex and race were based on a small sample size of children/adolescents with MHO and MUO phenotypes.

Similar to previous reports [60–64], we found that deep fried food, fast food and processed convenience food intakes were positively associated with blood pressure [60–62], while the intake of micronutrients such as calcium and iron was negatively associated with metabolic outcomes [63,64]. Protein intake was also found to be associated with a lower risk of hypertension [65]. However, only polyunsaturated fat intake and cognitive dietary constraint were shown to be associated with metabolic health in our cohort of children/adolescents with obesity.

The susceptibility to obesity-related comorbidities among individuals with obesity who are exposed to the same obesogenic environment (i.e., our cohort of children/adolescents

with MHO and children/adolescents with MUO reported no significant differences in most food groups and nutrient intakes except whole grains and polyunsaturated fat intake) might be attributed to the interaction between genetic variants and diet (also known as nutrigenetics), which affects the body's response to specific nutrients [66]. Significant interactions between genetic variants of lipid metabolism genes and dietary intakes of fat were found to be associated with blood lipid profiles in adults with overweight and obesity [67]. The greater risk of developing metabolic syndrome conferred by the GG genotype of the leptin receptor genetic variant (rs3790433) was shown to be abrogated among individuals with a high intake of polyunsaturated fatty acids [68]. In addition, there are other aspects of diet and nutrition that have not been investigated in our study. The eating habits such as tendency to snack, eating in the absence of hunger, and the number, duration and regularity of meals have been found to be associated with metabolic health in individuals with obesity [27]. Accumulating evidence has indicated a role of diet in regulating the gut microbiome, which has been proposed as an underlying mechanism in obesity and metabolic diseases [69]. A Western-style diet that is high in fat and refined carbohydrates may promote pro-inflammatory intestinal bacteria that are linked to obesity and metabolic diseases [70]. Hence, to better elucidate the impact of diet on obesity and its associated metabolic health, it is imperative to examine the other factors that interact with dietary intake and response.

There are severable limitations in this study. Firstly, our sample size is small and the study may be underpowered to establish significant differences between children/adolescents with MHO and children/adolescents with MUO. Secondly, our dataset may not have included all the important micronutrients, e.g., polyphenols, which may influence the metabolic phenotype of individuals with obesity [71]. Thirdly, a longitudinal study is required to establish the effect of polyunsaturated fat intake and cognitive dietary restraint on the metabolic health of individuals with obesity.

Despite the caveats, our study had several strengths. We had a well-phenotyped cohort of children/adolescents with obesity and this allowed us to clearly categorize the cohort into the MHO and MUO phenotypes. The dietary intakes of the participants were collected in the form of a comprehensive 3-day food diary, which was reported to have better agreement with observed food intakes [72]. Children/adolescents with MHO were fairly well matched with children/adolescents with MUO in terms of demographics such as age, sex, race, household income and adiposity measures such as BMI, BMI-SDS, waist to hip ratio and body fat percentage, which were confounding variables of metabolic health in obesity. Hence, this allowed a fair comparison of dietary intakes between children/adolescents with MHO and children/adolescents with MUO.

## 5. Conclusions

In conclusion, our study demonstrated that a healthier diet composition and positive eating behavior may contribute to favorable metabolic outcomes in children/adolescents with obesity. Interventions targeting the dietary intake of polyunsaturated fats and eating behavior such as cognitive dietary restraint may improve metabolic health in children/adolescents with obesity.

**Supplementary Materials:** The following supporting information can be downloaded at: <https://www.mdpi.com/article/10.3390/nu14224796/s1>, Table S1: Acceptable macronutrient distribution range (AMDR) and recommended dietary allowance (RDA) for individuals of different age groups; Table S2: Food items in the different food groups; Table S3: Food groups, nutrients intakes and eating behavior between children/adolescents with MHO and children/adolescents with MUO by MS definition; Table S4: Food groups, nutrient intakes and eating behavior between children/adolescents with MHO and children/adolescents with MUO (MS definition) stratified by sex; Table S5: Food groups, nutrient intakes and eating behavior between the 3 ethnic groups; Table S6: Food groups, nutrient intakes and eating behavior between children/adolescents with MHO and children/adolescents with MUO (MS definition) stratified by race.

**Author Contributions:** Conceptualization, D.S.Q.O., J.Y.T., M.F.-F.C. and Y.S.L.; methodology, D.S.Q.O., J.Y.T., M.F.-F.C. and Y.S.L.; formal analysis, D.S.Q.O., J.Y.T., L.Y.B.N., Z.P., S.Y., N.S.B.S.A.R. and Y.H.C.; investigation, D.S.Q.O., A.A.S. and Y.S.L.; resources, M.F.-F.C. and Y.S.L.; data curation, D.S.Q.O., J.Y.T. and N.S.B.S.A.R.; writing—original draft preparation, D.S.Q.O., L.Y.B.N., Z.P. and S.Y.; writing—review and editing, D.S.Q.O., J.Y.T., L.Y.B.N., Z.P., S.Y., A.A.S., Y.H.C., M.F.-F.C. and Y.S.L.; supervision, D.S.Q.O., J.Y.T., M.F.-F.C. and Y.S.L.; project administration, D.S.Q.O. and N.S.B.S.A.R.; funding acquisition, Y.S.L. All authors have read and agreed to the published version of the manuscript.

**Funding:** This research was funded by the Ministry of Health’s National Medical Research Council (NMRC), Singapore-NMRC/CIRG/1407/2014.

**Institutional Review Board Statement:** The study was conducted in accordance with the Declaration of Helsinki, and approved by the Domain Specific Review Board of National Healthcare Group, Singapore (Reference number: 2015/00314, approval date: 5 June 2015).

**Informed Consent Statement:** Informed consent was obtained from all subjects involved in the study.

**Data Availability Statement:** The data presented in this study are available within the manuscript and Supplementary Material.

**Acknowledgments:** We are grateful for the administrative help provided by Chan Fong Yee in this project.

**Conflicts of Interest:** The authors declare no conflict of interest. The funders had no role in the design of the study; in the collection, analyses, or interpretation of data; in the writing of the manuscript, or in the decision to publish the results.

## References

- Wang, Y.; Lim, H. The Global Childhood Obesity Epidemic and the Association between Socio-Economic Status and Childhood Obesity. *Int. Rev. Psychiatry* **2012**, *24*, 176–188. [\[CrossRef\]](#)
- World Health Organization: Obesity and Overweight. Available online: <https://www.who.int/news-room/fact-sheets/detail/obesity-and-overweight> (accessed on 5 August 2022).
- Sahoo, K.; Sahoo, B.; Choudhury, A.K.; Sofi, N.Y.; Kumar, R.; Bhadoria, A.S. Childhood obesity: Causes and consequences. *J. Fam. Med. Prim. Care* **2015**, *4*, 187–192. [\[CrossRef\]](#)
- Hsueh, Y.W.; Yeh, T.L.; Lin, C.Y.; Tsai, S.Y.; Liu, S.J.; Lin, C.M.; Chen, H.H. Association of metabolically healthy obesity and elevated risk of coronary artery calcification: A systematic review and meta-analysis. *PeerJ* **2020**, *8*, e8815. [\[CrossRef\]](#)
- Smith, G.I.; Mittendorfer, B.; Klein, S. Metabolically healthy obesity: Facts and fantasies. *J. Clin. Invest.* **2019**, *129*, 3978–3989. [\[CrossRef\]](#)
- Blüher, M. Metabolically Healthy Obesity. *Endocr. Rev.* **2020**, *41*(3), bnaa004. [\[CrossRef\]](#)
- Hinnouho, G.M.; Czernichow, S.; Dugravot, A.; Nabi, H.; Brunner, E.J.; Kivimäki, M.; Singh-Manoux, A. Metabolically healthy obesity and the risk of cardiovascular disease and type 2 diabetes: The Whitehall II cohort study. *Eur. Heart J.* **2015**, *36*, 551–559. [\[CrossRef\]](#)
- Ding, W.Q.; Yan, Y.K.; Zhang, M.X.; Cheng, H.; Zhao, X.Y.; Hou, D.Q.; Mi, J. Hypertension outcomes in metabolically unhealthy normal-weight and metabolically healthy obese children and adolescents. *J. Hum. Hypertens* **2015**, *29*, 548–554. [\[CrossRef\]](#)
- Yu, X.; Wang, L.; Zhang, W.; Ming, J.; Jia, A.; Xu, S.; Li, Q.; Ji, Q. Fasting triglycerides and glucose index is more suitable for the identification of metabolically unhealthy individuals in the Chinese adult population: A nationwide study. *J. Diabetes Investig.* **2019**, *10*, 1050–1058. [\[CrossRef\]](#)
- Khokhar, A.; Chin, V.; Perez-Colon, S.; Farook, T.; Bansal, S.; Kochummen, E.; Umpaichitra, V. Differences between Metabolically Healthy vs Unhealthy Obese Children and Adolescents. *J. Natl. Med. Assoc.* **2017**, *109*, 203–210. [\[CrossRef\]](#)
- Genovesi, S.; Antolini, L.; Orlando, A.; Gilardini, L.; Bertoli, S.; Giussani, M.; Invitti, C.; Nava, E.; Battaglini, M.G.; Leone, A.; et al. Cardiovascular Risk Factors Associated With the Metabolically Healthy Obese (MHO) Phenotype Compared to the Metabolically Unhealthy Obese (MUO) Phenotype in Children. *Front. Endocrinol.* **2020**, *11*, 27. [\[CrossRef\]](#)
- Yoon, D.Y.; Lee, Y.A.; Lee, J.; Kim, J.H.; Shin, C.H.; Yang, S.W. Prevalence and Clinical Characteristics of Metabolically Healthy Obesity in Korean Children and Adolescents: Data from the Korea National Health and Nutrition Examination Survey. *J. Korean Med. Sci.* **2017**, *32*, 1840–1847. [\[CrossRef\]](#) [\[PubMed\]](#)
- Li, L.; Yin, J.; Cheng, H.; Wang, Y.; Gao, S.; Li, M.; Grant, S.F.; Li, C.; Mi, J.; Li, M. Identification of Genetic and Environmental Factors Predicting Metabolically Healthy Obesity in Children: Data From the BCAMS Study. *J. Clin. Endocrinol. Metab* **2016**, *101*, 1816–1825. [\[CrossRef\]](#) [\[PubMed\]](#)
- Albuquerque, D.; Nobrega, C.; Manco, L.; Padez, C. The contribution of genetics and environment to obesity. *Br. Med. Bull.* **2017**, *123*, 159–173. [\[CrossRef\]](#)



15. McAllister, E.J.; Dhurandhar, N.V.; Keith, S.W.; Aronne, L.J.; Barger, J.; Baskin, M.; Benca, R.M.; Biggio, J.; Boggiano, M.M.; Eisenmann, J.C.; et al. Ten putative contributors to the obesity epidemic. *Crit. Rev. Food Sci. Nutr.* **2009**, *49*, 868–913. [CrossRef] [PubMed]
16. Gregory, J.W. Prevention of Obesity and Metabolic Syndrome in Children. *Front Endocrinol* **2019**, *10*, 669. [CrossRef] [PubMed]
17. Prince, R.L.; Kuk, J.L.; Ambler, K.A.; Dhaliwal, J.; Ball, G.D. Predictors of metabolically healthy obesity in children. *Diabetes Care* **2014**, *37*, 1462–1468. [CrossRef]
18. Kranz, S.; Brauchla, M.; Slavin, J.L.; Miller, K.B. What do we know about dietary fiber intake in children and health? The effects of fiber intake on constipation, obesity, and diabetes in children. *Adv. Nutr.* **2012**, *3*, 47–53. [CrossRef]
19. Arenaza, L.; Huybrechts, I.; Ortega, F.B.; Ruiz, J.R.; De Henauw, S.; Manios, Y.; Marcos, A.; Julian, C.; Widhalm, K.; Bueno, G.; et al. Adherence to the Mediterranean diet in metabolically healthy and unhealthy overweight and obese European adolescents: The HELENA study. *Eur. J. Nutr.* **2019**, *58*, 2615–2623. [CrossRef]
20. Camhi, S.M.; Whitney Evans, E.; Hayman, L.L.; Lichtenstein, A.H.; Must, A. Healthy eating index and metabolically healthy obesity in U.S. adolescents and adults. *Prev. Med.* **2015**, *77*, 23–27. [CrossRef]
21. Roberge, J.B.; Van Hulst, A.; Barnett, T.A.; Drapeau, V.; Benedetti, A.; Tremblay, A.; Henderson, M. Lifestyle Habits, Dietary Factors, and the Metabolically Unhealthy Obese Phenotype in Youth. *J. Pediatr.* **2019**, *204*, 46–52.e41. [CrossRef]
22. Qorbani, M.; Khashayar, P.; Rastad, H.; Ejtahed, H.-S.; Shahrestanaki, E.; Seif, E.; Daniali, S.S.; Goudarzi, M.; Motlagh, M.E.; Khodaparast, Z.; et al. Association of dietary behaviors, biochemical, and lifestyle factors with metabolic phenotypes of obesity in children and adolescents. *Diabetol. Metab. Syndr.* **2020**, *12*, 108. [CrossRef] [PubMed]
23. Elmaogullari, S.; Demirel, F.; Hatipoglu, N. Risk factors that affect metabolic health status in obese children. *J. Pediatr. Endocrinol. Metab* **2017**, *30*, 49–55. [CrossRef] [PubMed]
24. Denney-Wilson, E.; Campbell, K.J. Eating behaviour and obesity. *BMJ* **2008**, *337*, a1926. [CrossRef]
25. Gallant, A.R.; Tremblay, A.; Pêrusse, L.; Bouchard, C.; Després, J.P.; Drapeau, V. The Three-Factor Eating Questionnaire and BMI in adolescents: Results from the Québec Family Study. *Br. J. Nutr.* **2010**, *104*, 1074–1079. [CrossRef] [PubMed]
26. Morita, A.; Aiba, N.; Miyachi, M.; Watanabe, S.; the Saku Cohort Study Group. The associations of eating behavior and dietary intake with metabolic syndrome in Japanese: Saku cohort baseline study. *J. Physiol. Anthropol.* **2020**, *39*, 40. [CrossRef] [PubMed]
27. Torres-Castillo, N.; Martinez-Lopez, E.; Vizmanos-Lamotte, B.; Garaulet, M. Healthy Obese Subjects Differ in Chronotype, Sleep Habits, and Adipose Tissue Fatty Acid Composition from Their Non-Healthy Counterparts. *Nutrients* **2020**, *13*, 119. [CrossRef]
28. Henry, C.J.; Kaur, B.; Quek, R.Y.C. Are Asian foods as “fattening” as western-styled fast foods? *Eur. J. Clin. Nutr.* **2020**, *74*, 348–350. [CrossRef]
29. Health Promotion Board, Singapore: Singapore Children Growth Charts. Available online: <https://www.healthhub.sg/live-healthy/2016/child-bigger-than-average-but-active-should-i-be-worried> (accessed on 14 September 2022).
30. Wallace, T.M.; Levy, J.C.; Matthews, D.R. Use and abuse of HOMA modeling. *Diabetes Care* **2004**, *27*, 1487–1495. [CrossRef]
31. Health Promotion Board, Singapore Energy and Nutrient Composition of Food (2011). Available online: <http://focos.hpb.gov.sg/eservices/ENCF/> (accessed on 21 October 2020).
32. Willett, W.C.; Howe, G.R.; Kushi, L.H. Adjustment for total energy intake in epidemiologic studies. *Am. J. Clin. Nutr.* **1997**, *65*, 1220S–1228S; discussion 1229S–1231S. [CrossRef]
33. Dietary Guidelines for Americans 2020–2025. Available online: <https://www.dietaryguidelines.gov/resources/2020-2025-dietary-guidelines-online-materials>. (accessed on 10 August 2022).
34. Health Promotion Board Dietary Guidelines. Available online: [https://www.healthhub.sg/live-healthy/192/recommended\\_dietary\\_allowances](https://www.healthhub.sg/live-healthy/192/recommended_dietary_allowances). (accessed on 10 August 2022).
35. de Lauzon, B.; Romon, M.; Deschamps, V.; Lafay, L.; Borys, J.M.; Karlsson, J.; Ducimetière, P.; Charles, M.A. The Three-Factor Eating Questionnaire-R18 is able to distinguish among different eating patterns in a general population. *J. Nutr.* **2004**, *134*, 2372–2380. [CrossRef]
36. Chong, M.F.; Ayob, M.N.; Chong, K.J.; Tai, E.S.; Khoo, C.M.; Leow, M.K.; Lee, Y.S.; Tham, K.W.; Venkataraman, K.; Meaney, M.J.; et al. Psychometric analysis of an eating behaviour questionnaire for an overweight and obese Chinese population in Singapore. *Appetite* **2016**, *101*, 119–124. [CrossRef] [PubMed]
37. Tsatsoulis, A.; Paschou, S.A. Metabolically Healthy Obesity: Criteria, Epidemiology, Controversies, and Consequences. *Curr. Obes. Rep.* **2020**, *9*, 109–120. [CrossRef] [PubMed]
38. April-Sanders, A.K.; Rodriguez, C.J. Metabolically Healthy Obesity Redefined. *JAMA Netw. Open* **2021**, *4*, e218860. [CrossRef] [PubMed]
39. Ooi, D.S.Q.; Ong, S.G.; Lee, O.M.H.; Chan, Y.H.; Lim, Y.Y.; Ho, C.W.L.; Tay, V.; Vijaya, K.; Loke, K.Y.; Sng, A.A.; et al. Prevalence and predictors of metabolically healthy obesity in severely obese Asian children. *Pediatr. Res.* **2022**. [CrossRef] [PubMed]
40. Zimmet, P.; Alberti, K.G.; Kaufman, F.; Tajima, N.; Silink, M.; Arslanian, S.; Wong, G.; Bennett, P.; Shaw, J.; Caprio, S.; et al. The metabolic syndrome in children and adolescents—An IDF consensus report. *Pediatr Diabetes* **2007**, *8*, 299–306. [CrossRef] [PubMed]
41. National High Blood Pressure Education Program Working Group on High Blood Pressure in Children and Adolescents. The fourth report on the diagnosis, evaluation, and treatment of high blood pressure in children and adolescents. *Pediatrics* **2004**, *114*, 555–576. [CrossRef]

42. Wansink, B.; Cheney, M.M.; Chan, N. Exploring comfort food preferences across age and gender. *Physiol. Behav.* **2003**, *79*, 739–747. [CrossRef]
43. Wardle, J.; Haase, A.M.; Steptoe, A.; Nillapun, M.; Jonwutiwes, K.; Bellisle, F. Gender differences in food choice: The contribution of health beliefs and dieting. *Ann. Behav. Med. A Publ. Soc. Behav. Med.* **2004**, *27*, 107–116. [CrossRef]
44. Reddy, G.; van Dam, R.M. Food, culture, and identity in multicultural societies: Insights from Singapore. *Appetite* **2020**, *149*, 104633. [CrossRef]
45. Tan, Y.W.B.; Lau, J.H.; AshaRani, P.V.; Roystonn, K.; Devi, F.; Lee, Y.Y.; Whitton, C.; Wang, P.; Shafie, S.; Chang, S.; et al. Dietary patterns of persons with chronic conditions within a multi-ethnic population: Results from the nationwide Knowledge, Attitudes and Practices survey on diabetes in Singapore. *Arch. Public Health* **2022**, *80*, 62. [CrossRef]
46. Phillips, C.M. Metabolically healthy obesity: Definitions, determinants and clinical implications. *Rev. Endocr. Metab. Disord.* **2013**, *14*, 219–227. [CrossRef] [PubMed]
47. Slavin, J. Whole grains and human health. *Nutr. Res. Rev.* **2004**, *17*, 99–110. [CrossRef] [PubMed]
48. Drewnowski, A.; McKeown, N.; Kisseck, K.; Beck, E.; Mejbourn, H.; Vieux, F.; Smith, J.; Masset, G.; Seal, C.J. Perspective: Why Whole Grains Should Be Incorporated into Nutrient-Profile Models to Better Capture Nutrient Density. *Adv. Nutr.* **2021**, *12*, 600–608. [CrossRef] [PubMed]
49. Singapore Dietary Guidelines. Available online: [https://www.healthhub.sg/live-healthy/111/living\\_with\\_health\\_8\\_sets\\_diet\\_guidelines](https://www.healthhub.sg/live-healthy/111/living_with_health_8_sets_diet_guidelines). (accessed on 27 September 2022).
50. Esmailzadeh, A.; Mirmiran, P.; Azizi, F. Whole-grain consumption and the metabolic syndrome: A favorable association in Tehranian adults. *Eur. J. Clin. Nutr.* **2005**, *59*, 353–362. [CrossRef] [PubMed]
51. Ander, B.P.; Dupasquier, C.M.; Prociuk, M.A.; Pierce, G.N. Polyunsaturated fatty acids and their effects on cardiovascular disease. *Exp. Clin. Cardiol.* **2003**, *8*, 164–172.
52. Huang, C.W.; Chien, Y.S.; Chen, Y.J.; Ajuwon, K.M.; Mersmann, H.M.; Ding, S.T. Role of n-3 Polyunsaturated Fatty Acids in Ameliorating the Obesity-Induced Metabolic Syndrome in Animal Models and Humans. *Int. J. Mol. Sci.* **2016**, *17*, 1689. [CrossRef]
53. Telle-Hansen, V.H.; Christensen, J.J.; Formo, G.A.; Holven, K.B.; Ulven, S.M. A comprehensive metabolic profiling of the metabolically healthy obesity phenotype. *Lipids Health Dis.* **2020**, *19*, 90. [CrossRef]
54. Meiselman, H.L.; Bell, R. EATING HABITS. In *Encyclopedia of Food Sciences and Nutrition*, 2nd ed.; Caballero, B., Ed.; Academic Press: Oxford, UK, 2003; pp. 1963–1968.
55. Urbanek, J.K.; Metzgar, C.J.; Hsiao, P.Y.; Piehowski, K.E.; Nickols-Richardson, S.M. Increase in cognitive eating restraint predicts weight loss and change in other anthropometric measurements in overweight/obese premenopausal women. *Appetite* **2015**, *87*, 244–250. [CrossRef]
56. Phillips, C.M.; Dillon, C.; Harrington, J.M.; McCarthy, V.J.; Kearney, P.M.; Fitzgerald, A.P.; Perry, I.J. Defining metabolically healthy obesity: Role of dietary and lifestyle factors. *PLoS ONE* **2013**, *8*, e76188. [CrossRef]
57. Kim, H.N.; Song, S.W. Associations between Macronutrient Intakes and Obesity/Metabolic Risk Phenotypes: Findings of the Korean National Health and Nutrition Examination Survey. *Nutrients* **2019**, *11*, 628. [CrossRef]
58. Rolls, B.J.; Fedoroff, I.C.; Guthrie, J.F. Gender differences in eating behavior and body weight regulation. *Health Psychol.* **1991**, *10*, 133–142. [CrossRef] [PubMed]
59. Leblanc, V.; Bégin, C.; Corneau, L.; Dodin, S.; Lemieux, S. Gender differences in dietary intakes: What is the contribution of motivational variables? *J. Hum. Nutr. Diet* **2015**, *28*, 37–46. [CrossRef]
60. Kang, Y.; Kim, J. Association between fried food consumption and hypertension in Korean adults. *Br. J. Nutr.* **2016**, *115*, 87–94. [CrossRef]
61. Alsabieh, M.; Alqahtani, M.; Altamimi, A.; Albasha, A.; Alsulaiman, A.; Alkhamshi, A.; Habib, S.S.; Bashir, S. Fast food consumption and its associations with heart rate, blood pressure, cognitive function and quality of life. Pilot study. *Heliyon* **2019**, *5*, e01566. [CrossRef] [PubMed]
62. Scaranni, P.; Cardoso, L.O.; Chor, D.; Melo, E.C.P.; Matos, S.M.A.; Giatti, L.; Barreto, S.M.; da Fonseca, M.J.M. Ultra-processed foods, changes in blood pressure and incidence of hypertension: The Brazilian Longitudinal Study of Adult Health (ELSA-Brasil). *Public Health Nutr.* **2021**, *24*, 3352–3360. [CrossRef] [PubMed]
63. Shenkin, A. Micronutrients in health and disease. *Postgrad. Med. J.* **2006**, *82*, 559–567. [CrossRef]
64. van den Broek, T.J.; Kremer, B.H.A.; Marcondes Rezende, M.; Hoevenaars, F.P.M.; Weber, P.; Hoeller, U.; van Ommen, B.; Wopereis, S. The impact of micronutrient status on health: Correlation network analysis to understand the role of micronutrients in metabolic-inflammatory processes regulating homeostasis and phenotypic flexibility. *Genes Nutr.* **2017**, *12*, 5. [CrossRef]
65. Appel, L.J. The effects of protein intake on blood pressure and cardiovascular disease. *Curr. Opin. Lipidol.* **2003**, *14*, 55–59. [CrossRef]
66. Barrea, L.; Annunziata, G.; Bordini, L.; Muscogiuri, G.; Colao, A.; Savastano, S.; On behalf of Obesity Programs of Nutrition, Education, Research and Assessment (OPERA) Group. Nutrigenetics—Personalized nutrition in obesity and cardiovascular diseases. *Int. J. Obes. Suppl.* **2020**, *10*, 1–13. [CrossRef]
67. Hannon, B.A.; Edwards, C.G.; Thompson, S.V.; Burke, S.K.; Burd, N.A.; Holscher, H.D.; Teran-Garcia, M.; Khan, N.A. Genetic Variants in Lipid Metabolism Pathways Interact with Diet to Influence Blood Lipid Concentrations in Adults with Overweight and Obesity. *Lifestyle Genom.* **2020**, *13*, 155–163. [CrossRef]

- 
68. Phillips, C.M.; Goumidi, L.; Bertrais, S.; Field, M.R.; Ordovas, J.M.; Cupples, L.A.; Defoort, C.; Lovegrove, J.A.; Drevon, C.A.; Blaak, E.E.; et al. Leptin Receptor Polymorphisms Interact with Polyunsaturated Fatty Acids to Augment Risk of Insulin Resistance and Metabolic Syndrome in Adults. *J. Nutr.* **2009**, *140*, 238–244. [[CrossRef](#)] [[PubMed](#)]
  69. Fan, Y.; Pedersen, O. Gut microbiota in human metabolic health and disease. *Nat. Rev. Microbiol.* **2021**, *19*, 55–71. [[CrossRef](#)] [[PubMed](#)]
  70. Malesza, I.J.; Malesza, M.; Walkowiak, J.; Mussin, N.; Walkowiak, D.; Aringazina, R.; Bartkowiak-Wieczorek, J.; Mądry, E. High-Fat, Western-Style Diet, Systemic Inflammation, and Gut Microbiota: A Narrative Review. *Cells* **2021**, *10*, 3164. [[CrossRef](#)] [[PubMed](#)]
  71. Guasch-Ferre, M.; Merino, J.; Sun, Q.; Fito, M.; Salas-Salvado, J. Dietary Polyphenols, Mediterranean Diet, Prediabetes, and Type 2 Diabetes: A Narrative Review of the Evidence. *Oxid. Med. Cell Longev.* **2017**, *2017*, 6723931. [[CrossRef](#)]
  72. Crawford, P.B.; Obarzanek, E.; Morrison, J.; Sabry, Z.I. Comparative advantage of 3-day food records over 24-hour recall and 5-day food frequency validated by observation of 9- and 10-year-old girls. *J. Am. Diet Assoc.* **1994**, *94*, 626–630. [[CrossRef](#)]