Contents available at ScienceDirect

Diabetes Research and Clinical Practice

journal homepage: www.elsevier.com/locate/diabres

International Diabetes Federation

The impact of body weight gain on nonalcoholic fatty liver disease and metabolic syndrome during earlier and later adulthood

Shanshan Du [a,1], Cheng Wang [b,1], Wei Jiang [c], Chunlong Li [d], Yanchuan Li [a], Rennan Feng [a,*], Changhao Sun [a,*]

[a] National Key Discipline, Department of Nutrition and Food Hygiene, School of Public Health, Harbin Medical University, Harbin, China
[b] Department of Environmental Hygiene, School of Public Health, Harbin Medical University, Harbin, China
[c] Physical Examination Center, Second Affiliated Hospital of Harbin Medical University, Harbin, China
[d] Department of General Surgery, The Second Affiliated Hospital of Harbin Medical University, Harbin, China

ARTICLE INFO

Article history:
Received 12 October 2015
Received in revised form
12 April 2016
Accepted 24 April 2016
Available online 30 April 2016

Keywords:
Body weight gain
Non-alcoholic fatty liver disease
Metabolic syndrome
Adulthood
Insulin

ABSTRACT

Aim: Body weight gain adds risk for metabolic disorders and there are different metabolic changes in earlier and later adulthood. However, its impact on non-alcoholic fatty liver disease (NAFLD) was indeterminate. The aim of current study was to evaluate the impact of body weight gain on NAFLD and metabolic syndrome (MetS) during overall, earlier (25–40 y) and later (over 40 y) adulthood.

Methods: 1119 subjects were selected to calculate changes in body weight (ΔBW), body mass index (BMI) (ΔBMI) and bodyweight per year (ΔBW/y) to analysis their impact on NAFLD and MetS in multi-variable regression models, and explored the potential mediators that associated ΔBMI with NAFLD by mediation analysis.

Results: ΔBMI, ΔBW and ΔBW/y in whole adulthood were all positively associated with NAFLD and MetS. Body weight gain during earlier adulthood was more strongly associated with NAFLD than those during later adulthood. In NAFLD, the ORs of ΔBMI (third trisection), ΔBW and ΔBW/y were 3.86 (2.25, 6.57), 1.05 (1.02, 1.09) and 2.05 (1.29, 3.24) during earlier adulthood, and 1.47 (1.09, 2.02), 1.02 (1.00, 1.06), and 1.04 (.99, 1.13) over 40 y. Insulin and HOMA-IR were important intermediates that associated ΔBMI with NAFLD. ΔBMI in earlier adulthood increased higher insulin and insulin resistance (IR) than later adulthood.

Conclusions: Body weight gain in adulthood was positively associated with NAFLD and MetS, and the association was stronger in earlier than later adulthood. Insulin and IR were important mediators that contributed to the association.

* *Corresponding authors*. Tel.: +86 0451 87502801; fax: +86 0451 87502885.
E-mail addresses: fengrennan@163.com (R. Feng), changhao2002sun@gmail.com (C. Sun).
[1] These authors contributed equally to the study.
http://dx.doi.org/10.1016/j.diabres.2016.04.047
0168-8227/

1. Introduction

The prevalence of overweight and obesity are increasing dramatically around the world [1]. About 1.46 billion adults had body mass index (BMI) higher than 25 kg/m^2 and 205 million men and 297 million women were obese in 2008 [2], causing estimated 3.4 million deaths and 3.9% of years of life lost worldwide in 2010 [3]. As overweight/obesity is a current condition, weight gain histories may have more vicious impact on individuals' health [4]. For example, adult weight gain is positively associated with cancers in breast, colon, thyroid and endometrium [5–8]. It is also an independent risk factor for high blood glucose [9], blood pressure [10] and morbidity of cardiovascular disease [11]. Furthermore, the maximum body weight during adulthood also has adverse impact on microvascular complications in patients with type 2 diabetes (T2DM) [12], and a greater rate of weight gain in early adulthood may increase more risk for subclinical coronary artery disease in diabetics [13]. However, there are still limited published reports regarding weight histories and non-alcoholic fatty liver disease (NAFLD).

NAFLD also has a rapid increase worldwide, paralleling with obesity [14]. It was estimated to affect 20–30% of the general population in Western countries, and 12– 24% in Asians [15,16]. NAFLD is associated with hypertension, T2DM and cardiovascular diseases [17–19], and is thought to be a hepatic manifestation of metabolic syndrome (MetS) [20]. Body weight gain has been found as a good predictor for MetS [21] and individuals with higher body weight increase history are more likely to suffer from MetS [22]. However, whether body weight gain or body weight gain rate could add risk for NAFLD has not been affirmed by available evidence yet. Additionally, Chinese people has relative lower BMI, but comparable prevalence of metabolic disorders with the Western people [23]. Whether body weight gain could also increase the risk of MetS in Chinese people was unsure.

Considering the lack of knowledge about the impact of body weight gain on NAFLD and MetS in Chinese area, we conducted present study to explore whether body weight gains of overall, earlier and later adulthood were positively associated with NAFLD and MetS in the cross-sectional study.

2. Methods

2.1. The study population

A cross-sectional study was performed at Physical Examination Center of the Second Affiliated Hospital of Harbin Medical University from February to September in 2013. This study was approved by the ethics committee of Harbin Medical University. Written informed consent was obtained from each participant.

1/3 subjects from all participants were randomly selected every day to complete a questionnaire privately by trained interviewers ($n = 4715$). This questionnaire included name, age, gender, education level, work units, telephone number, history of disease, drug or tobacco use, alcohol consumption and physical activities. Smoking was defined as never, ⩽1 cigarettes/day, ⩽10 cigarettes/day, ⩽20 cigarettes/day, and >20 cigarettes/day; physical activity was categorized into three groups: none, those without any regular hard physical activities; moderate, those had hard physical activities at least once a week regularly; vigorous, those who had hard physical activities (leisure time or occupational) at least three times a week. Alcohol consumption was calculated by the amount of alcohol drinks (alcohol concentration) multiplied by the frequency, history/current of excessive alcohol intake was defined as alcohol consumption ⩾140 g/week for male adults and ⩾70 g/week for female adults.

The questionnaire also included weight history questions as follows: (1) how much was your current weight and height; (2) did you ever try to lose weight or have a weight control history; (3) how much was your maximum weight during the adulthood and when; (4) how much was your minimum weight during the adulthood and when; (5) how much did you weigh when you were 25 y; If the subject is elder than 40 y, we further asked; (6) How much did you weigh when you were 40 y. Mirrors and pictures of different height and weight as references were also shown to help the participants recall their weight and height histories.

After questionnaire, a well-trained examiner in another examination room conducted physical check-ups. Participants' current weight, height and fat mass (FM) were measured using the electric impedance method with a body fat mass analyzer (ioi 353; Janex Medical, Seoul, Korea) with minimally clothes and no socks. Weight and FM was recorded to the nearest .1 kg and height to the nearest .1 cm. BMI was calculated as weight in kilograms divided by height in meters squared (kg/m^2). Waist circumference (WC) was measured at the umbilical level, using a un-stretchable tape meter, and recorded to the nearest .1 cm. Blood pressure was measured twice in a seated position after 15 min rest by a qualified physician, using a standard mercury sphygmomanometer and the mean of two measurements was recorded as the participant's blood pressure. A fasting blood sample was taken from all participants after >10 h overnight fasting for measurement of biochemical indexes according to the standard protocol.

2.2. Laboratory analysis

All blood samples were centrifuged immediately at 2500×g for 15 min to obtain serum, which was immediately cooled, stored in refrigerator at −80 °C, and thawed only once for measurement of fasting blood glucose (FBG), total cholesterol (TC), high-density lipoprotein cholesterol (HDL-C), low-density lipoprotein cholesterol (LDL-C), triglycerides (TG), aspartate aminotransferase (AST), alanine aminotransferase (ALT), γ-glutamyl transferase (GGT), alkaline phosphatase (ALP), serum creatinine (CRE), blood urea nitrogen (BUN) and serum uric acid (UA). All of these variables were determined using a ROCHE Modular P800 Automatic Biochemical Analyzer (Roche Diagnostics, Mannheim, Germany). Serum fasting insulin concentration was measured by ROCHE Elecsys 2010 Chemiluminescence Immune Analyzer (Roche Diagnostics). The homeostasis model assessment for insulin resistance index (HOMA-IR) was calculated as previously described [24].