Combined effect of body mass index and waist-height ratio on incident diabetes; a population based cohort study

Kazuteru Mitsuhashi,1 Yoshitaka Hashimoto,1 Muhei Tanaka,1 Hitoshi Toda,2 Shinobu Matsumoto,1 Emi Ushigome,1 Mai Asano,1 Masahiro Yamazaki,1 Yohei Oda1 and Michiaki Fukui1,*

1Department of Endocrinology and Metabolism, Kyoto Prefectural University of Medicine, Graduate School of Medical Science, 465 Kajii-cho, Kawaramachi-Hirokoji, Kamigyo-ku, Kyoto 602-8566, Japan
2Department of Internal Medicine, Oike Clinic, 11 Shimoai-cho, Nishinokyo, Nakagyo-ku, Kyoto, 604-8436, Japan

(Received 7 December, 2016; Accepted 26 December, 2016; Published online 28 July, 2017)

We investigated the impact of combined effect of body mass index and waist-to-height ratio on risk of diabetes. Overweight and abdominal obesity were defined as body mass index ≥23 kg/m² and waist-to-height ratio ≥0.5, respectively. We divided participants into four groups according to presence of overweight and/or abdominal obesity. About 20% individuals with overweight did not complicated with an abdominal obesity. Among 3,737 participants, 286 participants had diabetes at baseline-examination. Adjusted odds ratios for prevalence of diabetes compared with non-overweight participants without abdominal obesity were as follow: 1.87 (95% confidence interval 1.09–3.14, p = 0.024) in non-overweight participants with abdominal obesity, 1.51 (0.87–2.55, p = 0.141) in overweight participants without abdominal obesity and 3.25 (2.37–4.52, p<0.001) in overweight participants with abdominal obesity. In the follow-up examination, 86 participants were diagnosed as diabetes among 2,263 participants. Adjusted odds ratios for incident diabetes were as follow: 2.59 (0.98–6.44, p = 0.056) in non-overweight participants with abdominal obesity, 1.65 (0.64–4.00, p = 0.288) in overweight participants without abdominal obesity and 2.77 (1.55–5.15, p<0.001) in overweight participants with abdominal obesity. Non-overweight individuals with abdominal obesity as well as overweight individuals with abdominal obesity was associated with diabetes compared with non-overweight individuals without abdominal obesity.

Key Words: obesity, body mass index, type 2 diabetes, abdominal obesity, waist circumference.

Obesity, which is a major public health problem worldwide,(1) is known as a risk of incident type 2 diabetes.(2–5) Body mass index (BMI) has been used as a proxy for obesity, because it is the most economical and practical approach in both clinical and epidemiologic settings.(6) On the other hand, waist circumference is strongly correlated with abdominal fat measures from advanced imaging techniques, and thought to represent fat stored in visceral depots.(7) In addition, it has been reported that waist-to-height ratio (WHR) is useful for detecting abdominal obesity(8) and that WHR is a simple and rapid screening tool, including its ability to identify health risks in both men and women, in different ethnic groups, and in all age groups.(9,10) Previous studies showed that both BMI(2–4) and WHR(5,10–12) are associated with incident diabetes. However, the impact of combined effect of BMI and WHR on incident diabetes remains to be elucidated. Therefore, we investigated the association between the combined effect of BMI and WHR, and prevalence or incident type 2 diabetes in this population based study.

Materials and Methods

Participants and study design. We designed a cross-sectional study and a 5-years follow-up cohort study to investigate the impact of combined effect of BMI and WHR on prevalence or incident diabetes in participants who received a medical health checkup program at Oike Clinic, Kyoto, Japan. The Oike Health Survey is an ongoing cohort investigation of risk factors for chronic diseases including hypertension, diabetes and chronic kidney disease.(13) In Japan, yearly routine examination for employees is legally mandated and all or most of the costs for the health check-up are usually paid by their employers. The Oike Clinic provides regular health check-up for the employees of various companies. Participants, who received health check-up at Oike Clinic in 2009 were included in this study. We excluded the participants with missing data of body weight or waist circumstance and the participants who did not have a data for diabetes in the cross-sectional study. Then, we excluded the participants who were diabetes at baseline examination and the participants who did not received follow-up examination in 2014 in the retrospective cohort study. The study was conducted in accordance with Declaration of Helsinki and approval for the study was obtained from the Ethical Committee of Oike Clinic. Informed consent was obtained from each participant.

Date collection and measurements. All participants provided details of their demographics. We classified the participants as non-smokers, ex-smokers or current-smokers according to a self-administered questionnaire. Habit of exercise was defined as performing any kind of sports at least once a week. Habit of alcohol was defined as daily alcohol consumption. Body mass index was calculated as body weight in kilograms divided by the square of the participant’s height in meters. Waist-to-height ratio was calculated by dividing the waist circumstance by the participant’s height. After an overnight fast, venous blood was collected for the measurement of the levels of various factors, including fasting plasma glucose (FPG), triglycerides and high-density lipoprotein (HDL) cholesterol. Hemoglobin Alc (HbAlc) was assayed using high-performance liquid chromatography. The value for HbAlc (%) was estimated as the National Glycohemoglobin Standardization Program value (%) calculated by the formula HbAlc (%) = HbAlc (Japan Diabetes Society) (%) × 1.02 + 0.25%.(14) Diagnosis of type 2 diabetes was made according to the American Diabetes Association (ADA) criteria of a FPG level of ≥7.0 mM, self-reported clinician-diagnosed diabetes or

*To whom correspondence should be addressed. E-mail: sayarinapm@hotmail.com

doi: 10.3164/jcbn.16-116
©2017 JCBN

J. Clin. Biochem. Nutr. | September 2017 | vol. 61 | no. 2 | 118–122
HbA1c ≥6.5% (48 mmol/mol; National Glycohemoglobin Standardization Program, NGSP). Participants with fasting plasma glucose ≥5.6 mM were considered to have impaired fasting glucose.

**Definition of overweight and abdominal obesity.** Body mass index ≥23.0 kg/m², which has been proposed as a cut-off for the diagnosis of overweight in Asian people, was defined as overweight. This definition of overweight has often been used in Japanese population. In addition, Hsu et al. recommended a cut-off point of BMI 23 for the Asian American population, because this population was susceptible to overweight on incident type 2 diabetes. Abdominal obesity was defined as WHtR ≥0.5, according to the recommended criteria for diabetes. Then, participants were categorized at the baseline examination into four groups: 1) non-overweight participants without abdominal obesity group, 2) non-overweight participants with abdominal obesity group, 3) overweight participants without abdominal obesity group or 4) overweight participants with abdominal obesity group.

**Statistical analysis.** Continuous variables were expressed as mean (SD) and categorical variables were expressed as number. Student t tests or χ² tests was conducted to assess the statistical significance of differences between participants without diabetes and participants with diabetes. In addition, the study participants were divided into four groups based on the presence of overweight and/or abdominal obesity and baseline characteristics of four groups were compared. The analysis of continuous variables to assess differences was determined by Tukey HSD test. The analysis of categorical variables to assess differences was determined by the χ² test. Odds ratio (OR) of the four groups for prevalence or incident type 2 diabetes was calculated by logistic regression analysis. The following variables were analyzed as potential covariates: age, sex, smoking status, habit of exercise, habit of alcohol and family history of diabetes in the cross-sectional study. The following variables were analyzed as potential covariates: age, sex, smoking status, habit of exercise, habit of alcohol, family history of diabetes and impaired fasting glucose at baseline examination in the cohort study. The statistical analyses were performed using the JMP software version 10.0 software (SAS Institute Inc., Cary, NC) and p value <0.05 was considered to represent a statistically significant difference.

**Results**

**Cross-sectional study.** In 2009, we enrolled 3,924 participants (Fig. 1). Among them, 187 participants were excluded. Thus, 3,737 participants were eligible for the cross-sectional study. About 20% of participants with overweight did not complicated with an abdominal obesity. Moreover about 20% participants without overweight complicated with an abdominal obesity.

At baseline examination, 286 participants have diabetes. The prevalence rate of diabetes was 3.2% (case/n = 58/1,795) in non-overweight participants without abdominal obesity group, 7.2% (25/345) in non-overweight participants with abdominal obesity, 5.9% (21/355) in overweight participants without abdominal obesity and 14.7% (182/1,242) in overweight participants with abdominal obesity. The adjusted ORs for prevalence of diabetes compared with non-overweight participants without abdominal obesity were as follow: 1.87 (95% confidence interval (CI) 1.09–3.14, p = 0.024) in non-overweight participants with abdominal obesity, 1.51 (95% CI 0.87–2.55, p = 0.141) in overweight participants without abdominal obesity and 3.25 (95% CI 2.37–4.52, p<0.001) in overweight participants with abdominal obesity (Table 2).

**Retrospective cohort study.** In the cohort study, we excluded the participants who were diabetes at baseline examination and the participants who did not received follow-up examination, which performed in 2014 (Fig. 1). Thus, 2,263 participants were eligible for the cohort study. Baseline characteristics of participants of cohort study according to four groups are shown in Table 3. Participants with abdominal obesity had higher blood pressure, FPG and triglycerides compared with participants without abdominal obesity. In addition, overweight participants with abdominal obesity had higher blood pressure, FPG and triglycerides, and lower HDL cholesterol compared with non-overweight participants with abdominal obesity.
### Table 1. Characteristics of study participants of cross-sectional study

|                        | Participants without diabetes | Participants with diabetes | p value |
|------------------------|------------------------------|---------------------------|---------|
| N                      | 3,432                        | 286                       | —       |
| Age (years)            | 55.1 (10.2)                  | 63.2 (9.0)                | <0.001  |
| Sex (male/female)      | 2,016/1,416                  | 243/43                    | <0.001  |
| Body mass index (kg/m²)| 22.5 (3.0)                   | 24.9 (3.4)                | <0.001  |
| Waist circumference (cm)| 80.6 (9.0)               | 88.1 (8.8)                | <0.001  |
| Systolic blood pressure (mmHg) | 121.0 (15.3)   | 129.3 (14.1)              | <0.001  |
| Diastolic blood pressure (mmHg) | 75.7 (10.4)   | 79.4 (9.5)                | <0.001  |
| Fasting plasma glucose (mM) | 5.1 (0.5)               | 7.6 (1.7)                 | <0.001  |
| HbA1c (%)              | 5.4 (0.4)                    | 6.6 (0.9)                 | <0.001  |
| HDL cholesterol (mM)   | 1.7 (0.5)                    | 1.4 (0.4)                 | <0.001  |
| Smoking (non-/ex-/current-) | 2,444/439/548             | 181/50/55                 | 0.014   |
| Habit of alcohol (+/-) | 2,158/1,272                  | 177/109                   | 0.73    |
| Habit of exercise (+/-)| 1,398/2,033                  | 97/189                    | 0.024   |
| Family history of diabetes (+/-) | 2,773/639               | 164/122                   | <0.001  |
| Overweight/abdominal obesity (+/-) | 1,727/319/333/1,053 | 58/25/21/182              | <0.001  |

HDL; high-density lipoprotein. Data are number or mean (standard deviation). Student t tests or \( \chi^2 \) tests was conducted to assess the statistical significance of differences between participants without diabetes and participants with diabetes.

### Table 2. Odds ratios for prevalence of type 2 diabetes at baseline examination according to presence of overweight and/or abdominal obesity

| Case of diabetes                                      | Model 1  | p value     | Model 2  | p value     |
|------------------------------------------------------|----------|-------------|----------|-------------|
| Non-overweight without abdominal obesity              | 58/1,795 | 1 (Reference) | 1 (Reference) | —           |
| Non-overweight with abdominal obesity                 | 25/345   | 1.93 (1.14–3.19) | 0.015    | 1.87 (1.09–3.14) | 0.024 |
| Overweight without abdominal obesity                  | 21/355   | 1.65 (0.95–2.74) | 0.072    | 1.51 (0.87–2.55) | 0.141 |
| Overweight with abdominal obesity                     | 182/1,242| 3.33 (2.44–4.60) | <0.001   | 3.25 (2.37–4.52) | <0.001 |
| Age (per one year)                                    | —        | 1.07 (1.05–1.08) | <0.001   | 1.08 (1.06–1.10) | <0.001 |
| Men                                                   | —        | 3.26 (2.32–4.68) | <0.001   | 3.87 (2.67–5.70) | <0.001 |
| Habit of alcohol                                      | —        | —            | —        | 1.07 (0.81–1.42) | 0.656 |
| Ex-smoker                                            | —        | —            | —        | 1.18 (0.81–1.69) | 0.387 |
| Current smoker                                        | —        | —            | —        | 1.45 (1.01–2.07) | 0.044 |
| Family history of diabetes                            | —        | —            | —        | 4.31 (3.26–5.70) | <0.001 |

Model 1 adjusted for age and sex. Model 2 adjusted for Model 1 and habit of alcohol, habit of exercise, smoking status, and family history of diabetes.

### Table 3. Characteristics of study participants of cohort study at the baseline examination

|                        | Non-overweight without abdominal obesity | Non-overweight with abdominal obesity | Overweight without abdominal obesity | Overweight with abdominal obesity |
|------------------------|------------------------------------------|---------------------------------------|--------------------------------------|-----------------------------------|
| N                      | 1,138                                    | 183                                   | 235                                  | 707                               |
| Age (years)            | 52.8 (9.3)                               | 58.8 (9.7)*                           | 51.0 (8.9)*                          | 57.1 (10.4)*                       |
| Sex (male/female)      | 556/582                                  | 44/139                                | 203/32                               | 514/193                           |
| Body mass index (kg/m²)| 20.3 (1.7)                               | 21.8 (1.0)*                           | 24.0 (0.8)*                          | 25.9 (2.2)*                        |
| Waist circumference (cm)| 74.1 (5.9)                              | 82.4 (3.9)*                           | 82.1 (3.7)*                          | 90.0 (5.9)*                        |
| Systolic blood pressure (mmHg) | 115.0 (14.4)     | 121.1 (14.7)*                         | 122.5 (12.3)*                        | 128.9 (13.8)*                      |
| Diastolic blood pressure (mmHg) | 72.0 (10.1)    | 75.0 (10.6)*                           | 77.5 (8.5)*                          | 80.8 (9.2)*                        |
| Fasting plasma glucose (mM) | 5.0 (0.5)                           | 5.1 (0.5)*                            | 5.1 (0.5)*                           | 5.3 (0.5)*                         |
| HbA1c (%)              | 5.3 (0.3)                                | 5.4 (0.4)*                            | 5.3 (0.4)                            | 5.4 (0.4)*                         |
| HbA1c (mmol/mol)       | 34 (4)                                   | 36 (4)*                               | 34 (5)                               | 36 (4)*                            |
| Total cholesterol (mM) | 5.3 (0.9)                                | 5.5 (0.8)                             | 5.3 (0.8)                            | 5.3 (0.8)                          |
| Triglycerides (mM)     | 1.0 (0.6)                                | 1.2 (0.6)*                            | 1.3 (0.7)*                           | 1.5 (1.0)*                         |
| HDL cholesterol (mM)   | 1.9 (0.5)                                | 1.8 (0.4)                             | 1.6 (0.4)*                           | 1.5 (0.4)*                         |
| Smoking (non-/ex-/current-) | 861/115/161 | 158/10/15                           | 135/53/47                            | 473/116/118                        |
| Habit of alcohol (+/-) | 754/383                                  | 128/55                                | 137/98                               | 419/287                           |
| Habit of exercise (+/-) | 449/688                                  | 82/101                                | 68/167                               | 287/420                           |
| Family history of diabetes (+/-) | 931/207                        | 153/30                                | 187/48                               | 556/151                           |

HDL; high-density lipoprotein. Data are number or mean (standard deviation). The analyses of continuous among four groups were performed by Tukey HSD test. \(^*\) vs Non-overweight without abdominal obesity, \(^*\) vs Non-overweight with abdominal obesity, \(^*\) vs Overweight without abdominal obesity. The analyses of categorical variables among four groups were performed by \( \chi^2 \) test. \(^p<0.05\)
At follow-up examination, 86 participants have diabetes. The incident rate of diabetes was 1.5% (case/n = 17/1,138) in non-overweight participants without abdominal obesity, 4.4% (8/183) in non-overweight participants with abdominal obesity, 3.4% (8/235) in overweight participants without abdominal obesity and 7.5% (53/707) in overweight participants with abdominal obesity. The adjusted ORs for incident diabetes compared with non-overweight participants without abdominal obesity were as follow: 2.59 (95% CI 0.98–6.44, \( p = 0.056 \)) in non-overweight participants with abdominal obesity, 1.65 (95% CI 0.64–4.00, \( p = 0.283 \)) in overweight participants without abdominal obesity and 2.77 (95% CI 1.55–5.15, \( p = 0.001 \)) in overweight participants with abdominal obesity (Table 4).

Discussion

In this study, we showed that overweight individuals with abdominal obesity was associated with higher risk of prevalence or incident diabetes compared with non-overweight individuals without abdominal obesity. Furthermore, non-overweight individuals with abdominal obesity was also associated with higher risk of prevalence or incident diabetes compared with non-overweight individuals without abdominal obesity in our study population. These results revealed that abdominal obesity, an important component of metabolic syndrome, may be considered high risk obesity for diabetes.

Possible explanations for the association between combination of overweight and abdominal obesity and risk of diabetes are as follows. It is well known that obesity is associated with insulin resistance,\(^{19}\) B-cell exhaustion due to continuous insulin resistance leads to decline of insulin secretion.\(^{20}\) Because East Asian people have a limited innate capacity of insulin secretion,\(^{12}\) even a small increase of insulin resistance might lead to incident type 2 diabetes. On the other hand, it has been reported that visceral adiposity, which has closely association with abdominal obesity,\(^{22}\) is the independent risk factor for insulin resistance.\(^{23}\) It has been reported that advanced glycation end products, which has a close association with visceral adiposity, is associated with diabetes and diabetes complications.\(^{24}\) In addition, abdominal obesity is more related to diabetes than overall obesity.\(^{10,11}\) Recent studies revealed that a subset of individuals with obesity who have a low burden of adiposity-related metabolic abnormalities compared with individuals with ‘at risk’ obesity, the so-called ‘metabolically healthy obesity’ (MHO) phenotype.\(^{25–27}\) Visceral adiposity of MHO is lower than metabolically abnormal obesity.\(^{23}\) Furthermore, we previously reported that overweight individuals with non-alcoholic fatty liver disease (NAFLD) and non-overweight individuals with NAFLD is associated with incident diabetes.\(^{17}\) NAFLD is caused by ectopic fat accumulation in the liver.\(^{28}\) Ectopic fat accumulation, which has a close association with visceral adiposity, is reported to be strongly correlated with insulin resistance.\(^{30}\) Unfortunately, we did not have data of the presence of NAFLD in this study population. Thus, the concept of metabolically abnormal or presence of NAFLD is almost same as presence of abdominal obesity. In fact, the result of this study was the same as past studies of MHO phenotype\(^{26,27}\) or presence of NAFLD.\(^{17}\) Taking these findings together, non-overweight individuals with abdominal obesity as well as overweight individuals with abdominal obesity was associated with higher risk of diabetes compared with non-overweight individuals without abdominal obesity.

Dietary modification is an important for the prevention of incident type 2 diabetes. In fact, it has been reported that supplementation of soy isoflavones is effective for improving lipid profiles and apolipoprotein levels in patients with type 2 diabetes.\(^{21}\)

Some limitations of our study should be noted. First, body fat distribution was assessed based on anthropometric indicators alone in this study. However, WHR has been reported to be useful for central fat distribution.\(^{32}\) Second, we did not have a data of insulin, thus, we could not assess the insulin resistance. Finally, the study population consisted of Japanese men and women, therefore, it is uncertain whether these findings are generalized in other ethnic groups.

In conclusion, our study firstly showed an evidence that the risk of incident diabetes in overweight individuals with abdominal obesity was significantly higher than that in non-overweight individuals without abdominal obesity. In addition, the risk of incident diabetes in non-overweight individuals with abdominal obesity was also higher than that in non-overweight individuals without abdominal obesity.

Acknowledgments

We thank all of the staff members of Oike Clinic.

Abbreviations

| Abbreviation | Description |
|--------------|-------------|
| BMI          | body mass index |
| FPG          | fasting plasma glucose |
| HbA1c        | hemoglobin A1c |
| HDL          | high-density lipoprotein |
| NAFLD        | non-alcoholic fatty liver disease |
| MHO          | metabolically healthy obesity |
| WHtR         | waist-to-height ratio |

Table 4. Odds ratios for incident type 2 diabetes at 5 years after the baseline examination according to presence of overweight and/or abdominal obesity

| Case of diabetes                  | Model 1  | \( p \) value | Model 2  | \( p \) value | Model 3  | \( p \) value |
|-----------------------------------|----------|---------------|----------|---------------|----------|---------------|
| Non-overweight without abdominal obesity | 17/1,138 | 1 (Reference) | —        | 1 (Reference) | —        | —             |
| Non-overweight with abdominal obesity | 8/183    | 3.48 (1.37–8.21) | 0.01     | 3.60 (1.41–8.52) | 0.009    | 2.59 (0.98–6.44) | 0.056 |
| Overweight without abdominal obesity | 8/235    | 1.82 (0.73–4.20) | 0.192    | 1.72 (0.68–3.99) | 0.239    | 1.65 (0.64–4.00) | 0.288 |
| Overweight with abdominal obesity | 53/707   | 4.13 (2.38–7.49) | <0.001   | 3.95 (2.28–7.17) | <0.001   | 2.77 (1.55–5.15) | <0.001 |
| Age (per one year) | 1.02 (1.00–1.04) | 0.097 | 1.03 (1.01–1.05) | 0.01 | 1.01 (0.99–1.04) | 0.335 |
| Men | 2.54 (1.47–4.66) | <0.001 | 2.59 (1.43–4.90) | 0.001 | 1.69 (0.91–3.26) | 0.1 |
| Habit of alcohol | — | — | — | 0.64 (0.39–1.03) | 0.068 | 0.58 (0.34–0.96) | 0.033 |
| Habit of exercise | — | — | — | 0.90 (0.57–1.44) | 0.659 | 0.90 (0.55–1.48) | 0.664 |
| Ex-smoker | — | — | — | 1.66 (0.89–3.00) | 0.111 | 1.61 (0.82–3.05) | 0.164 |
| Current smoker | — | — | — | 2.30 (1.29–4.02) | 0.005 | 2.29 (1.22–4.23) | 0.01 |
| Family history of diabetes | — | — | — | 1.95 (1.16–3.19) | 0.013 | 1.40 (0.80–2.38) | 0.233 |
| Impaired fasting glucose | — | — | — | — | — | 20.0 (11.4–37.2) | <0.001 |

Model 1 adjusted for age and sex. Model 2 adjusted for Model 1 and habit of alcohol, habit of exercise, smoking status, and family history of diabetes. Model 3 adjusted for Model 2 and impaired fasting glucose at baseline examination.
Conflict of Interest

Mai Asano, Masahiro Yamazaki and Michiaki Fukui have received grants, honoraria and research supports from AstraZeneca plc, Astellas Pharma Inc., Nippon Boehringer Ingelheim Co., Ltd., Daiichi Sankyo Co., Ltd., Eli Lilly Japan K.K., Kyowa Hakko Kirin Company Ltd., Kissei Pharmaceutical Co., Ltd., MSD K.K., Mitsubishi Tanabe Pharma Corporation, Novo Nordisk Pharma Ltd., Sanwa Kagaku Kenkyusho Co., Ltd., Sanofi K.K., Ono Pharmaceutical Co., Ltd., and Takeda Pharmaceutical Co., Ltd. The sponsors were not involved in the study design; in the collection, analysis, interpretation of data; in the writing of this manuscript; or in the decision to submit the article for publication. The authors, their immediate families, and any research foundations with which they are affiliated have not received any financial payments or other benefits from any commercial entity related to the subject of this article. The authors declare that although they are affiliated with a department that is supported financially by a pharmaceutical company, the authors received no current funding for this study and this does not alter their adherence to all the journal policies on sharing data and materials. The other authors have nothing to disclose.

Author Contribution

M.K. originated and designed the study, researched data and wrote manuscript. Y.H. originated and designed the study, researched data and reviewed the manuscript. M.T. originated the study, researched data, contributed to discussion and reviewed the manuscript. H.T., S.M., E.U., M.A. and M.Y. researched data and contributed to discussion. M.F. researched data and reviewed and edited the manuscript. M.F. is the guarantor of this work and, as such, had full access to all the data in the study and takes responsibility for the integrity of the data and the accuracy of the data analysis. All authors critically reviewed the article and approved the final version of the manuscript.

References

1. Funacane MM, Stevens GA, Cowan MJ, et al. National, regional, and global trends in body-mass index since 1980: systematic analysis of health examination surveys and epidemiological studies with 960 country-years and 9.1 million participants. Lancet 2011; 377: 557–567.
2. Chan JM, Rimm EB, Colditz GA, Stampfer MJ, Willett WC. Obesity, fat distribution, and weight gain as risk factors for clinical diabetes in men. Diabetes Care 1994; 17: 961–969.
3. Colditz GA, Willett WC, Rotnitzky A, Manson JE. Weight gain as a risk factor for clinical diabetes mellitus in women. Ann Intern Med 1995; 122: 481–486.
4. Schienkiewitz A, Schulze MB, Hoffmann K, Kroke A, Boeing H. Body mass index history and risk of type 2 diabetes: results from the European Prospective Investigation into Cancer and Nutrition (EPIC)-Potsdam Study. Am J Clin Nutr 2006; 84: 427–433.
5. Browning LM, Hsieh SD, Ashwell M. A systematic review of waist-to-height ratio as a screening tool for the prediction of cardiovascular disease and diabetes: 0.5 could be a suitable global boundary value. Nutr Res Rev 2010; 23: 247–269.
6. World Health Organization. Obesity: Preventing and Managing the Global Epidemic: Report of a WHO Consultation (WHO Technical Report Series 894). Geneva: World Health Organization, 2000.
7. Yusuf S, Hawken S, Ounpuu S, et al. Effect of potentially modifiable risk factors associated with myocardial infarction in 52 countries (the INTERHEART study): case-control study. Lancet 2004; 364: 937–952.
8. Ashwell M, Lejeune S, Mephserson K. Ratio of waist circumference to height may be better indicator of need for weight management. BMJ 1996; 312: 377.
9. Ashwell M, Hsieh SD. Six reasons why the waist-to-height ratio is a rapid and effective global indicator for health risks of obesity and how its use could simplify the international public health message on obesity. Int J Food Sci Nutr 2005; 56: 303–307.
10. Xu Z, Qi X, Dahl AK, Xu W. Waist-to-height ratio is the best indicator for undiagnosed type 2 diabetes. Diabet Med 2013; 30: e201–e207.
11. Hadaegh F, Zabetian A, Harati H, Aizizi F. Waist-to-height ratio as a better predictor of type 2 diabetes compared to body mass index in Tehranian adult men—a 3.6-year prospective study. Exp Clin Endocrinol Diabetes 2006; 114: 310–315.
12. Ashwell M, Gunn P, Gibson S. Waist-to-height ratio is a better screening tool than waist circumference and BMI for adult cardiometabolic risk factors: systematic review and meta-analysis. Obes Rev 2012; 13: 275–286.
13. Hashimoto Y, Tanaka M, Kimura T, et al. Hemoglobin concentration and incident metabolic syndrome: a population-based large-scale cohort study. Endocrine 2015; 50: 390–396.
14. Kashiwagi A, Kasuga M, Araki E, et al. International clinical harmonization of glycated hemoglobin in Japan: from Japan Diabetes Society to National Glycohemoglobin Standardization Program values. J Diabetes Investig 2012; 3: 39–40.
15. American Diabetes Association. Diagnosis and classification of diabetes mellitus. Diabetes Care 2010; 33 (Suppl 1): S62–569.
16. Weisell RC. Body mass index as an indicator of obesity. Asia Pac J Clin Nutr 2002; 11: 5681–5684.
17. Fukuda T, Hamaguchi M, Kojima T, et al. The impact of non-alcoholic fatty liver disease on incident type 2 diabetes mellitus in non-overweight individuals. Liver Int 2016; 36: 275–283.
18. Hsu WC, Araneta MR, Kanaya AM, Chiang JL, Fujimoto W. BMI cut points to identify at-risk Asian Americans for type 2 diabetes screening. Diabetes Care 2015; 38: 150–158.
19. Kahn SE, Hull RL,utzschneider KM. Mechanisms linking obesity to insulin resistance and type 2 diabetes. Nature 2006; 444: 840–846.
20. Kodama K, Tojjar D, Yamada S, Toda K, Patel CJ, Butte AJ. Ethnic differences in the relationship between insulin sensitivity and insulin response: a systematic review and meta-analysis. Diabetes Care 2013; 36: 1789–1796.
21. Fujimoto WY. Overview of non-insulin-dependent diabetes mellitus (NIDDM) in different population groups. Diabet Med 1996; 13 (9 Suppl 6): S7–510.
22. Bays H. Central obesity as a clinical marker of adiposopathy; increased visceral adiposity as a surrogate marker for global fat dysfunction. Curr Opin Endocrinol Diabetes Obes 2014; 21: 345–351.
23. Pouliot MC, Despré JP, Nadeau A, et al. Visceral obesity in men. Associations with glucose tolerance, plasma insulin, and lipoprotein levels. Diabetes 1992; 41: 826–834.
24. Yamanaka M, Matsumura T, Ohno R, et al. Non-invasive measurement of skin autofluorescence to evaluate diabetic complications. J Clin Biochem Nutr 2016; 58: 135–140.
25. Hashimoto Y, Tanaka M, Okada H, et al. Metabolically healthy obesity and risk of incident CKD. Clin J Am Soc Nephrol 2015; 10: 578–583.
26. Hashimoto Y, Tanaka M, Semmaru T, et al. Maintaining metabolically healthy overweight and risk of incident type 2 diabetes or cardiovascular disease. Int J Diabetes Vasc Dis Res 2015; 53(901): 1–6.
27. Bell JA, Kivimaki M, Hamer M. Metabolically healthy obesity and risk of incident type 2 diabetes: a meta-analysis of prospective cohort studies. Obes Rev 2014; 15: 504–515.
28. Koster A, Stenhofm S, Alley DE, et al. Body fat distribution and inflammation among obese older adults with and without metabolic syndrome. Obesity (Silver Spring) 2010; 18: 2354–2361.
29. Byrne CD. Ectopic fat, insulin resistance and non-alcoholic fatty liver disease. Proc Nutr Soc 2013; 72: 412–419.
30. Yoshimura E, Kumahara H, Tobima T, et al. Relationships between fat deposition in the liver and skeletal muscle and insulin sensitivity in Japanese individuals: a pilot study. Diabetes Metab Syndr Obes 2011; 4: 35–43.
31. Chi XX, Zhang T, Zhang DJ, Yu W, Wang QY, Zhen JL. Effects of isoflavones on lipid and apolipoprotein levels in patients with type 2 diabetes in Heilongjiang Province in China. J Clin Biochem Nutr 2016; 59: 134–138.
32. Hsieh SD, Yoshinaga H, Muto T. Waist-to-height ratio, a simple and practical index for assessing central fat distribution and metabolic risk in Japanese men and women. Int J Obes Relat Metab Disord 2003; 27: 610–616.

Funding

None.