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ORIGINAL ARTICLE

Lipid accumulation product is a powerful index for recognizing insulin resistance in non-diabetic individuals

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BACKGROUND/OBJECTIVES: Lipid accumulation product (LAP) is an index, which combines waist circumference (WC) and triglyceride (TG) reflecting lipid accumulation. The aims of the study were to explore the relationship between LAP and insulin resistance (IR) and to assess whether LAP was superior to WC and body mass index (BMI) in identifying IR.
SUBJECTS/METHODS: The study was cross-sectional and included 2524 non-diabetic subjects from China. The blood pressure (BP), anthropometric measurements, glucose levels, insulin levels and a fasting lipid profile were measured. BMI, the homeostasis model assessment of IR (HOMA-IR) and LAP were calculated.
RESULTS: In both sexes, BP, BMI, total cholesterol (TC), non high-density lipoprotein cholesterol (non-HDL-C), HOMA-IR, fasting and postprandial glucose levels increased across LAP quartiles ($P<0.001$), while HDL cholesterol (HDL-C) levels decreased across LAP quartiles ($P<0.001$). Pearson's correlation analysis demonstrated that HOMA-IR was correlated with LAP, BMI, WC, TG, HDL-C and non-HDL-C in both sexes ($P<0.001$). Multivariate analysis demonstrated that LAP had a greater impact on HOMA-IR than BMI and WC.
CONCLUSIONS: LAP is closely associated with HOMA-IR and is a powerful index that outperforms BMI and WC in identifying IR in non-diabetic individuals.

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Keywords: obesity; insulin resistance; HOMA-IR; lipid accumulation index

INTRODUCTION

Insulin resistance (IR), characterized by the reduced ability of insulin to stimulate glucose utilization and storage, has an important role in the development of cardiovascular diseases (CVD) and metabolic diseases. Although the cause of IR is unknown, obesity exhibits a close correlation with IR.[1] Furthermore, abdominal obesity, especially visceral fat, is correlated with the most severe insulin-resistant state.[2] Two factors may be involved in obesity-related IR: elevations in FFA[3] and adipocytokine secretion by adipose tissue.[4,5] Both factors could impair insulin action and result in IR.

There are many methods used to identify IR. Euglycemic hyperinsulinemic clamping is the gold standard test for estimating IR.[6] However, this test is complex, time-consuming and expensive, and thus, it is not suitable for clinical practice. The homeostasis model assessment of IR (HOMA-IR), is simple, stable and widely used in epidemiological studies.[7] Some antropometric measurements, such as body mass index (BMI), waist circumference (WC), are usually used for identifying IR.[8,9] However, such indexes are not comprehensive in reflecting an individual's obesity and metabolic abnormalities. BMI is easy to measure and is typically used to predict the risk of metabolic abnormalities. However, as BMI is calculated using an individual's height and weight, it cannot distinguish between fat and lean tissues or reflect lipid accumulation and distribution.[2,8] WC was defined by the IDF consensus worldwide as the criteria for abdominal obesity.[10] However, WC cannot distinguish between subcutaneous adipose tissue and visceral adipose tissue, the latter having a more important role in IR.[2,11] Therefore, WC does not sufficiently reflect visceral fat. Imaging methods, such as magnetic resonance imaging (MRI) and computed tomography (CT), can assess lipid accumulation and distribution patterns, but they are expensive and therefore cannot be widely used in clinical practice.

The lipid accumulation product (LAP) was first introduced by Kahn[12] (the study data came from the third National Health and Nutrition Examination Survey, NHANES III). LAP combines waist measurements and fasting triglyceride (TG) levels, reflecting both the anatomic and physiological changes associated with lipid overaccumulation. LAP was closely associated with CVD, diabetes and metabolic syndrome and outperformed BMI for identifying these diseases.[8,12–14] In polycystic ovary syndrome patients, LAP was an effective index with higher sensitivity and specificity than BMI and WC for predicting CVD.[13] Although there were some studies on the correlation between LAP and CVD, diabetes in European subjects, there have been few studies looking at the correlation between LAP and IR in Asian cohort. It is well known that abdominal obesity is a common phenotype of obese Chinese people.[15] The current study aimed to examine the relationship between LAP and IR in Chinese people and explore whether LAP had superior predictability for IR than WC and BMI.

SUBJECTS AND METHODS

The study was approved by the local ethical committee. All subjects were healthy employees recruited from Chongqing University, in urban area of Chongqing city, which is a big city in Southwestern China. Informed consent was obtained from all participants before participation in the study. Subjects were included if they were 20– 80 years old, not pregnant, and non-smokers or seldom smokers. Subjects were excluded if they met

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any of the following criteria: fasting plasma glucose (FPG) $\geqslant$ 7.0 mmol/l, 2-h plasma glucose (PPG) following 75 g oral glucose load (2 h-PPG) $\geqslant$ 11.1 mmol/l, diagnosis of diabetes, cancer, acute or chronic inflammatory disease, current renal disease, liver disease or rheumatic disease, the use of steroids or other drugs affecting lipid metabolism within three months. Subjects were also excluded if they did not have sufficient information. The study included a total of 2524 apparently healthy adults, of which 1510 were males (average age: 57.5 ± 12.8 years) and 1014 were females (average age: 55.9 ± 12.2 years).

Medical examinations and measurements

Anthropometry indexes, such as weight, height and WC, were measured with all subjects. WC was measured at the midpoint between the lower edge of the costal arch and the top of the iliac crest. BMI was calculated as the ratio of weight in kilograms to height squared. Blood pressure (BP) was measured in the sitting position.

Routine biochemical analyses were performed on all participants after 10 h of fasting. The 75 g oral glucose tolerance test was performed in subjects older than 30 years. Plasma glucose was measured using hexokinase assays (Olympus Diagnostics, Tokyo, Japan). Fasting serum insulin (FIns) was measured using a chemiluminescence assay (Roche Diagnostics, Basel, Switzerland). Total cholesterol (TC), total TGs, high-density lipoprotein cholesterol (HDL-C) and low-density lipoprotein cholesterol (LDL-C) were measured with an enzymology assay (Wako Diagnostics, Tokyo, Japan). Non high-density lipoprotein cholesterol (Non-HDL-C) was calculated by subtracting HDL-C from the TC level. IR was estimated using HOMA-IR, which was calculated with the following formula: FIns (μU/ml) × FPG (mmol/l)/22.5. LAP was calculated using the formula previously reported:[12] male LAP = [waist (cm) − 65] × TG concentration (mmol/l) and female LAP = [waist (cm) − 58] × TG concentration (mmol/l).

Statistical analysis

All statistical analyses were conducted using the statistical software SPSS 11.5. Normal distribution data were expressed as the mean ± s.d. Skewed distribution data were expressed as the median and interquartile ranges and were log transformed (base 10) before analysis. Independent sample *t* tests were used for the comparisons between the two groups. Mean levels of variables were compared across quartiles of LAP by one-way analysis of variance. For post-hoc analysis, homogeneity of variance was tested using the SNK test, and the Games–Howell test was utilized to identify any heterogeneity of variance. The association between HOMA-IR and other variables was examined by Pearson's correlation analysis. Multivariable, forward stepwise analysis was used to analyze the correlated variables of HOMA-IR. A *P*-value < 0.05 (two-tailed) was considered significant.

RESULTS

General characteristics

Our study included 2524 subjects, 1510 males and 1014 females. Overall, males had a higher average age, systolic BP (SBP), diastolic BP (DBP), WC, BMI and TG compared with females ($P < 0.05$–0.001). HDL-C levels were lower in males than in females ($P < 0.001$), while females had a higher TC levels compared with males ($P < 0.001$). There was no significant difference between males and females for the following variables: LDL-C, non-HDL-C, FPG, 2 h-PPG, FIns, HOMA-IR and LAP ($P > 0.05$) (Table1). According to the diagnostic criteria of metabolic syndrome (MS),[10] of all the 2524 subjects, 406 subjects met the criteria for the diagnosis of MS, and the prevalence of MS in our study was 16.1%. Overall, 32.2% of subjects had abdominal obesity (WC $\geqslant$ 90 cm in males or $\geqslant$ 80 cm in females), 29.2% of subjects had hypertriglceridemia (TG $\geqslant$ 1.7 mmol/l), 13.0% of subjects had low level of HDL-C (HDL: male < 1.0 mmol/l, female < 1.3 mmol/l), 49.8% of subjects had hypertension (either a history of hypertension or a current SBP $\geqslant$ 130 mm Hg or DBP $\geqslant$ 85 mm Hg) and 9.9% of subjects had a current FPG $\geqslant$ 5.6 mmol/l.

The male and female groups were divided into four subgroups based on LAP quartiles, respectively. 45 men with WC $\leqslant$ 65 cm were considered the WC value of 66 cm, and 5 women with WC $\leqslant$ 58 cm were considered the WC value of 59 cm. The male LAP quartile groups were defined as follows: Q1: LAP < 13.65 cm × mmol/l; Q2: LAP (14.65–25.75) cm × mmol/l; Q3: LAP (25.76–41.67) cm × mmol/l; and Q4: LAP > 41.70 cm × mmol/l. The female LAP quartile groups were defined as follows: Q1: LAP < 12.80 cm × mmol/l; Q2: LAP (12.80–23.76) cm × mmol/l; Q3: LAP (23.79–40.14) cm × mmol/l; Q4: LAP > 40.14 cm × mmol/l. Age, SBP, DBP, BMI, TC, HDL-C, LDL-C, non-HDL-C, FPG, 2 h-PPG, FIns and HOMA-IR for male and female groups were compared in Table 2.

Table 1. The general characteristics between males and females

Variable	*Male* (n = *1510*)	*Female* (n = *1014*)	P
Age (years)	57.5 ± 12.8	55.9 ± 12.2	0.002
SBP (mm Hg)	127.6 ± 19.4	124.7 ± 21.5	< 0.001
DBP (mm Hg)	80.3 ± 10.0	77.2 ± 9.9	< 0.001
WC (cm)	84.5 ± 8.5	77.8 ± 9.2	< 0.001
BMI (kg/m^2)	24.2 ± 3.2	23.3 ± 3.4	< 0.001
TC (mmol/l)	5.09 ± 0.91	5.28 ± 1.00	< 0.001
TG[a] (mmol/l)	1.34 (0.95–1.84)	1.25 (0.91–1.77)	0.022
HDL-C (mmol/l)	1.40 ± 0.35	1.63 ± 0.39	< 0.001
LDL-C (mmol/l)	2.98 ± 0.83	2.98 ± 0.90	0.971
non-HDL-C (mmol/l)	3.68 ± 0.89	3.65 ± 0.96	0.371
FPG (mmol/l)	4.93 ± 0.51	4.90 ± 0.48	0.059
2h-PPG[b] (mmol/l)	6.19 ± 1.70	6.15 ± 1.63	0.635
Fins[a] (μU/ml)	6.18 (3.94–9.02)	6.07 (4.05–8.77)	0.217
HOMA-IR[a]	1.34 (0.85–2.00)	1.31 (0.86–1.90)	0.146
LAP[a] (cm × mmol/l)	25.96 (14.2–41.97)	23.99 (13.09–40.12)	0.074

Abbreviations: BMI, body mass index; DBP, diastolic blood pressure; FIns, fasting serum insulin; FPG, fasting plasma glucose; HDL-C, high-density lipoprotein cholesterol; HOMA-IR, homeostasis model assessment of insulin resistance; LAP, lipid accumulation product; LDL-C, low-density lipoprotein cholesterol; non-HDL-C, non high-density lipoprotein cholesterol; SBP, systolic blood pressure; TC, total cholesterol; TG, total triglycerides; WC, waist circumference; 2h-PPG, 2-h plasma glucose following 75 g oral glucose load. Data were expressed as mean ± s.d. [a]Skewed distribution data were expressed as median and interquartile ranges, and log transformed (base 10) before statistic analysis. [b]2h-PPG had taken in subjects older than 30 years old.

The results suggested that in both sexes, the levels of SBP, DBP, BMI, TC, non-HDL-C, HOMA-IR, FPG and 2 h-PPG increased as LAP increased ($P < 0.001$), whereas the level of HDL-C decreased as LAP increased ($P < 0.001$). In males, neither age nor LDL-C increased across LAP quartiles, whereas in females, the two variables increased across LAP quartiles ($P < 0.001$).

Bivariate analysis

Pearson's correlation analysis showed that, in males, HOMA-IR correlated positively with SBP ($r = 0.128$, $P < 0.001$), DBP ($r = 0.141$, $P < 0.001$), WC ($r = 0.347$, $P < 0.001$), BMI ($r = 0.346$, $P < 0.001$), LAP ($r = 0.361$, $P < 0.001$), TG ($r = 0.323$, $P < 0.001$), and non-HDL-C ($r = 0.133$, $P < 0.001$) and negatively with HDL ($r = -0.241$, $P < 0.001$). HOMA-IR was not associated with age ($r = 0.020$, $P = 0.443$), LDL-C ($r = -0.009$, $P = 0.734$) or TC ($r = 0.038$, $P = 0.145$). In females, HOMA-IR correlated positively with age ($r = 0.077$, $P = 0.014$), SBP ($r = 0.136$, $P < 0.001$), DBP ($r = 0.149$, $P < 0.001$), WC ($r = 0.31$, $P < 0.001$), BMI ($r = 0.309$, $P < 0.001$), LAP ($r = 0.35$, $P < 0.001$), TG ($r = 0.293$, $P < 0.001$), TC ($r = 0.080$, $P = 0.011$), and non-HDL-C ($r = 0.170$, $P < 0.001$) and negatively with HDL ($r = -0.211$, $P < 0.001$). No associations were observed between HOMA-IR and LDL-C ($r = 0.059$, $P = 0.059$).

Multivariate analysis

Multivariate, stepwise, forward regression analyses with a model using HOMA-IR as the dependent variable, WC, BMI, LAP, HDL-C

Table 2. Comparison of variables in quartiles of LAP

Variable	*Quartiles of LAP*			
	Q1	*Q2*	*Q3*	*Q4*
Male				
Age (years)	58.0 ± 12.9	58.0 ± 12.7	57.8 ± 12.8	56.4 ± 12.5
SBP (mm Hg)	123.1 ± 19.3	127.5 ± 19.0*	130.6 ± 20.6*	129.4 ± 17.9*
DBP (mm Hg)	76.9 ± 9.6	79.8 ± 9.4*	81.7 ± 10.5*,#	82.8 ± 9.4*
BMI (kg/m^2)	21.4 ± 2.6	23.6 ± 2.2*	25.0 ± 2.3*,#	26.7 ± 2.5*,§
TC (mmol/l)	4.82 ± 0.91	5.06 ± 0.85*	5.18 ± 0.91*	5.28 ± 0.89*
HDL-C (mmol/l)	1.66 ± 0.38	1.45 ± 0.28*	1.35 ± 0.29*,#	1.15 ± 0.23*,§
LDL-C (mmol/l)	2.77 ± 0.77	3.12 ± 0.84*	3.09 ± 0.82*	2.93 ± 0.84*,§
non-HDL-C (mmol/l)	3.16 ± 0.81	3.63 ± 0.77*	3.84 ± 0.83*,#	4.13 ± 0.85*,§
FPG (mmol/l)	4.80 ± 0.44	4.89 ± 0.49*	4.99 ± 0.52*,#	5.05 ± 0.55*
2h-PPG (mmol/l)	5.66 ± 1.60	6.10 ± 1.74*	6.35 ± 1.61*,#	6.65 ± 1.70*,§
FIns[a] (μU/ml)	5.24 ± 5.14	6.00 ± 3.33*	10.51 ± 46.82*,#	10.06 ± 6.41*,§
HOMA-IR[a]	1.12 ± 1.16	1.31 ± 0.78*	2.35 ± 10.61*,#	2.28 ± 1.53*,§
Female				
Age (years)	47.1 ± 12.9	54.8 ± 11.8*	59.1 ± 9.7*,#	62.4 ± 8.2*,§
SBP (mm Hg)	113.3 ± 17.8	122.1 ± 20.1*	128.3 ± 20.4*,#	134.9 ± 21.7*,§
DBP (mm Hg)	73.4 ± 9.3	76.3 ± 9.9*	78.7 ± 9.5*,#	80.2 ± 9.7*
BMI (kg/m^2)	20.8 ± 2.8	22.5 ± 2.5*	24.1 ± 2.5*,#	25.9 ± 3.2*,§
TC (mmol/l)	4.86 ± 1.01	5.16 ± 0.98*	5.43 ± 0.86*,#	5.68 ± 0.97*,§
HDL-C (mmol/)	1.85 ± 0.38	1.69 ± 0.34*	1.59 ± 0.34*,#	1.39 ± 0.35*,§
LDL-C (mmol/l)	2.62 ± 0.80	2.94 ± 0.84*	3.16 ± 0.74*,#	3.20 ± 1.06*
non-HDL-C (mmol/l)	3.01 ± 0.83	3.47 ± 0.84*	3.84 ± 0.77*,#	4.29 ± 0.89*,§
FPG (mmol/l)	4.65 ± 0.44	4.79 ± 0.41*	5.04 ± 0.47*,#	5.10 ± 0.48*
2h-PPG[b] (mmol/l)	5.31 ± 1.29	5.86 ± 1.50*	6.48 ± 1.51*,#	6.98 ± 1.71*,§
FIns[a] (μU/ml)	5.82 ± 9.78	6.34 ± 4.73*	7.04 ± 5.04*	8.94 ± 5.21*,§
HOMA-IR[a]	1.21 ± 2.13	1.35 ± 1.01*	1.58 ± 1.16*,#	2.04 ± 1.22*,§

Abbreviations: BMI, body mass index; DBP, diastolic blood pressure; FIns, fasting serum insulin; FPG, fasting plasma glucose; HDL-C, high-density lipoprotein cholesterol; HOMA-IR, homeostasis model assessment of insulin resistance; LAP, lipid accumulation product; LDL-C, low-density lipoprotein cholesterol; non-HDL-C, non high-density lipoprotein cholesterol; SBP, systolic blood pressure; TC, total cholesterol; TG, total triglycerides; WC, waist circumference; 2h-PPG, 2-h plasma glucose following 75 g oral glucose load. Data were expressed as mean ± s.d. *$P<0.05$, Q2, Q3, Q4 vs Q1, respectively. #$P<0.05$, Q3 vs Q2. §$P<0.05$, Q4 vs Q3. [a]Skewed distribution data were log transformed (base 10) before statistic analysis. [b]2h-PPG had taken in subjects older than 30 years old.

Table 3. Multivariate stepwise regression analysis the impact of variables on HOMA-IR

Dependent variable	*Independent variables*	*Absolute value of standardized coefficients*	P
Male			
HOMA-IR[a]	LAP[a]	0.22	<0.001
	BMI	0.20	<0.001
Female			
HOMA-IR[a]	LAP[a]	0.24	<0.001
	BMI	0.15	<0.001
	HDL-C	0.07	0.041

Abbreviations: BMI, body mass index; HDL-C, non high-density lipoprotein cholesterol; LAP, lipid accumulation product. [a]Log transformed (base 10) before statistic analysis.

and non-HDL-C as independent variables showed that in the male group, LAP (standardized coefficient $\beta = 0.22$, $P<0.001$) was superior to BMI (standardized coefficient $\beta = 0.20$, $P<0.001$) on HOMA-IR. WC, HDL-C and non-HDL-C were not included in the regression model. In the female group, LAP (standardized coefficient $\beta = 0.24$, $P<0.001$) had greater impact than BMI (standardized coefficient $\beta = 0.15$, $P<0.001$) on HOMA-IR. WC and non-HDL-C were not included in the regression model. These data suggested that in both males and females, LAP had a greater impact on HOMA-IR (Table 3).

DISCUSSION

IR with resultant hyperinsulinemia is an independent risk factor for type 2 diabetes and CVD. It has been found that most individuals with CVD present early with IR.[16] Moreover, early recognition of IR is important to predict the development of CVD, fatty liver disease and metabolic diseases.[17] Obesity is one of the risk factors for CVD. It has been shown that obesity, especially visceral fat, strongly correlated to IR. Visceral fat is characterized by a hyperlipolytic state with a high level of FFA, which can infuse into the liver and impair the insulin signal, leading to IR.[2,4] Additionally, as an endocrine organ, adipose tissue can secrete adipocytokines such as TNF-α, leptin in obesity, which can interfere with the insulin signal and induce IR.[4,5] Individuals with different degree visceral fat have different levels of IR.[18] The index that could reflect visceral fat may be more suitable for assessing IR and predicting CVD and metabolic diseases.

Our data showed that in Chinese non-diabetic subjects, LAP was closely associated with IR and exhibited stronger predictability of IR than WC and BMI. Pearson's correlation analysis showed that LAP positively correlated with HOMA-IR. Multivariate regression analysis suggested that LAP had a greater impact on HOMA-IR than did BMI and WC. Compared with LAP, BMI reflects only excess weight. Individuals with various risk levels of CVD and diabetes may have a similar BMI, while their WC and metabolic risk profiles may be different.[2] Although WC is a common index for obesity, it cannot distinguish between subcutaneous and visceral adipose tissue. Therefore, an increased WC cannot always reflect high-risk visceral fat.[2] Our results were in accordance with the Despres' 'hypertriglyceridemic waist phenotype'

(Quebec Cardiovascular Study). Such phenotype was defined as increased WC and TG levels in indivduals, and was considered as a marker of atherogenic metabolic triad and a predictor of hyperinsulinemia.[18–20] However, hypertriglyceridemic waist is a dichotomous marker, and LAP is a continuous variable. The latter may be more suitable for comparison between populations.

In our study, we found the prevalence of the MS was 16.1%, which was similar to the Gu *et al*.[21,22] study in Chinese. As the component of MS, the evaluated TG levels have closed relationship with IR.[23] We found that, in both sexes, TG was positively associated with HOMA-IR (male: $r = 0.323$, female: $r = 0.293$, both $P < 0.001$). Individuals with hypertriglyceridemia are usually accompanied with TG accumulation in muscle and liver, indicating lipid ectopic deposition.[23] The metabolites of TG (fatty acyl CoA, diacylglycerol and ceramides) accumulates in the hepatocytes and skeletal myocytes, that can lead to an impaired insulin signal, suppressing insulin-induced activation of glycogen synthase and insulin receptor substrate-1-associated PI 3-kinase.[24,25] Lipids accumulation in the body are accompanied with both increased level of TG and evaluated WC levels.[23] Therefore, increased LAP may indicate ectopic lipid deposition and reflect lipid overaccumulation.[12,14]

It suggested that in both the cross-sectional study and the prospective study, LAP was an effective index for assessing the risk of CVD and diabetes. Previous studies found that LAP outperformed BMI and WC in identifying metabolic disease. Kahn[12,26] found that LAP outperformed BMI in recognizing cardiovascular risk and diabetes. In polycystic ovary syndrome patients,[13] LAP positively correlated with HOMA-IR, receiver operating characteristic curve analysis suggested that a LAP index of 34.5 (sensitivity: 84%; specificity 79%) had the highest area under the curve and thus demonstrated superior performance in identifying IR than did BMI or WC. Additionally, a 6-year, follow-up, prospective study found LAP was superior to BMI for identifying prevalent diabetes and predicting incident diabetes in young men (<50 years old).[8]

Our study had several limitations. First, because it is a cross-sectional study, we only analyzed the correlation between HOMA-IR and the other variables without attempting to identify the causality or mechanisms. Second, the gold standard for diagnosing IR is euglycemic hyperinsulinemic clamping, which is not available in our study for the large sample size. Therefore, we used HOMA-IR for assessing IR. However, HOMA-IR estimates hepatic IR, and is a poor estimator of IR in skeletal muscle or the whole body. Third, because the subjects were selected by random sampling from Chongqing University, a community with a superior living standard than other areas, our data does not reflect the general population of China.

CONCLUSIONS

In conclusion, our study indicates that LAP is strongly correlated with IR, and outperforms BMI and WC in recognizing IR. LAP was an inexpensive and easy index that reflected both the anatomic and physiological changes associated with lipid overaccumulation and that therefore can be used as a powerful index for recognizing IR in large populations.

CONFLICT OF INTEREST

The authors declare no conflict of interest.

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