ORIGINAL ARTICLE

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COMPARATIVE ANALYSIS OF ANTHROPOMETRIC PARAMETERS AND THEIR ASSOCIATION WITH METABOLIC HEALTH IN PATIENTS WITH OBESITY

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Abstract. Background and aim: *Body mass index (BMI) is the cornerstone of obesity classification. As the prevalence of obesity as a chronic disease increases, more precise stratification tools are being explored. The aim of this study is to compare established and alternative anthropometric parameters in terms of their association with metabolic health indices in adults with BMI ≥ 25 kg/m^2.* ***Materials and methods:*** *Cross-sectional analysis was performed on first-visit assessment data of 401 individuals from an outpatient endocrinology clinic. Weight, height, and waist circumference values were used to calculate the following anthropometrics: waist-to-height ratio (WHtR), Body Shape Index (ABSI), Body Roundness Index (BRI), Conicity Index (CI), Weight-adjusted Waist Index (WWI), and Relative Fat Mass (RFM). The outcomes of interest were metabolic laboratory indices of glycemia (FPG, HbA1c), lipid metabolism (LDL, TG), and hepatic enzymes (ALT, GGT). After log-transformation, they were standardized and combined into glycemic, lipid, hepatic, and overall metabolic burden composite scores. Hierarchical linear regression models adjusted for age and sex were used to measure the association between each anthropometric index and the composite scores. Incremental explained variance (ΔR2) and regression coefficients were compared.* ***Results:*** *The strongest association with the overall metabolic composite was shown by RFM (β = 0.480, 95% CI = 0.269-0.692; ΔR² = 0.085), followed by WHtR (ΔR² = 0.080), BMI (ΔR² = 0.076), and BRI (ΔR² = 0.076). ABSI, CI, and WWI showed no significant incremental contributions. BMI showed the largest ΔR² for the glycemic composite, whereas RFM ranked highest for lipid and hepatic composites.* ***Conclusion:*** *Waist-based indices – particularly RFM and WHtR – were most consistently associated with composite metabolic laboratory burden in routine outpatient practice.*

Key words: *anthropometrics, obesity, metabolic health, body shape index, body roundness index, conicity index, relative fat mass, weight-adjusted waist index*

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INTRODUCTION

Obesity is a major clinical and public health burden with increasing prevalence. As pharmacological and surgical treatment methods develop rapidly, the need for a more precise risk stratification beyond weight alone receives growing attention [1-3]. Body mass index (BMI), the foundational classification tool, does not capture regional adiposity or body composition accurately, as well as the respective nuance in metabolic risk. Consequently, individuals with a BMI lower than 30 kg/m² may exhibit adverse glycemic, lipid, or hepatic laboratory profiles comparable to or worse than those observed in individuals with a BMI above the classical cut-off for defining obesity [4-6]. This heterogeneity motivates the search for accessible surrogate markers of metabolic risk among adults with BMI ≥ 25 kg/m², as well as the development of alternative staging systems and stratification approaches [1, 2, 7–9].

Anthropometric indicators of central adiposity (e.g., waist-to-height ratio (WHtR) and waist-to-hip ratio (WHR) are increasingly emphasized in clinical guidance due to their closer correspondence with abdominal fat distribution [1, 2, 10, 11]. In parallel, several alternative anthropometric indices have been proposed to characterize body shape and adiposity using simple measurements, including the Body Shape Index (ABSI), Body Roundness Index (BRI), Conicity Index (CI), Weight-Adjusted Waist Index (WWI), and Relative Fat Mass (RFM) [12-17]. However, evidence comparing these indices is heterogeneous across populations and outcomes, and their associations with routinely obtained metabolic laboratory markers remain insufficiently clarified in real-world clinical cohorts of adults with BMI ≥ 25 kg/m² [18-22].

In a cross-sectional outpatient endocrinology cohort, we examined the associations between BMI, WHtR, and selected alternative anthropometric indices (ABSI, BRI, CI, WWI, and RFM) and metabolic laboratory burden summarized using domain-specific (glycemic, lipid, hepatic) and overall composite scores. We further compared indices with respect to incremental explained variance (ΔR^2) beyond age and sex. We hypothesized that indices reflecting central adiposity/body shape would demonstrate stronger associations with composite metabolic burden than BMI alone.

MATERIALS AND METHODS

Study design and setting

This cross-sectional study used routinely collected clinical data from an outpatient endocrinology clinic. Records were extracted for first-visit assessments performed between January 2017 and July 2024.

Participants

Eligible participants were adults (≥ 18 years old) with BMI ≥ 25 kg/m², not pregnant, and with recorded anthropometry, body composition, and biochemical laboratory data. Individuals with common obesity-related comorbidities (e.g., hypertension, type 2 diabetes mellitus) were included to enhance clinical representativeness. A sensitivity analysis restricting to participants without documented comorbidities was performed to evaluate robustness to potential confounding by comorbidity and treatment exposure.

Outcomes

Primary outcomes were fasting plasma glucose (FPG), HbA1c, LDL-cholesterol (LDL-C), triglycerides (TG), alanine aminotransferase (ALT), and gamma-glutamyl transferase (GGT). Composite scores were calculated to increase statistical power and reduce random measurement noise, as well as false positive rate inflation from multiple testing. From a biological perspective, the composite scores were constructed to summarize correlated biochemical domains and overall metabolic burden. The glycemic composite score reflects insulin resistance and glycemic control (FPG, HbA1c). The lipid composite score reflects atherogenic dyslipidemia (LDL-C, TG). The hepatic composite score reflects hepatocellular injury and cholestatic/oxidative stress pathways often accompanying steatotic liver disease (ALT, GGT). The combination of the three domains (glycemic, lipid, and hepatic score) represents the “metabolic composite score,” which represents general metabolic load as a continuous trait, so that degrees of risk can be captured more precisely. Other scientific teams have applied the same or similar methods, yielding promising results and a clinically potent research gap [23-26].

Exposures

Exposures of interest were BMI, Waist-to-Height Ratio (WHtR), A Body Shape Index (ABSI), Body Roundness Index (BRI), Conicity Index (CI), Weight-Adjusted Waist Index (WWI), and Relative Fat Mass (RFM). The respective formulas used for the calculation of those parameters are presented in Table 1 [13-17]. Height and waist circumference were converted to meters for index computation where required (ABSI, BRI, CI), and unit-consistent ratios were used for WHtR and RFM. WWI was computed as WC (cm) divided by the square root of weight (kg) to preserve comparability with commonly reported implementations.

Table 1. Anthropometric indices

Index	Calculation
Body Mass Index (BMI)	= kg/m^2
Waist-to-Height Ratio (WHtR)	= WC (cm)/height (cm)
Relative Fat Mass (RFM)	= 64 − (20 × (height/waist)) + (12 × sex)
Body Roundness Index (BRI)	= $364.2 - 365.5\sqrt{1 - \frac{\mathrm{WC(m)}}{\pi^2} \mathrm{x\ height\ (m)}^2}$
Weight-adjusted Waist Index (WWI)	= WC (cm)/$\sqrt{\mathrm{weight(kg)}}$
A Body Shape Index (ABSI)	= WC(m)/BMI$^{2/3}$ x height (m)$^{1/2}$
Conicity Index (CI)	= WC (m)/$0.109\sqrt{\mathrm{weight(kg)/height(m)}}$

For RFM, sex is coded 0 for male, 1 for female.

Covariates

Age and sex were pre-specified covariates based on established physiological associations with adiposity distribution and metabolic laboratory parameters.

Data sources and measurement

Weight and height were measured using calibrated scales and a stadiometer. Waist circumference (WC) was measured using a standard tape according to a standardized protocol. Body composition was assessed using bioelectrical impedance analysis (Tanita MC-780), including fat mass, body fat percentage, visceral fat rating, and skeletal muscle mass. Laboratory results were extracted from supplementary medical documentation.

Bias and confounding

Since the cohort consists of a real-world patient sample, estimated associations should be interpreted as age- and sex-adjusted relationships observed in routine practice rather than causal effects. Including participants with hypertension, type 2 diabetes, and other common comorbidities improves clinical representativeness, but introduces potential confounding because both comorbidity status and pharmacotherapy can influence metabolic laboratory markers and may correlate with adiposity distribution. Medication data were incomplete and could not be reliably modelled. Therefore, residual confounding is likely. To examine robustness, we performed sensitivity analyses restricting to participants without documented comorbidities, which reduces (but does not eliminate) the potential influence of disease- and treatment-related confounding. Selection bias may limit generalizability beyond similar outpatient settings. Measurement bias was minimized via standardized protocols and calibrated equipment.

Power analysis

G*Power software was used for post-hoc sensitivity analysis with alpha level set at 0.05 and power 0.80 based on the sample size, choice of test, and number of predictors.

Missing data

Missing value analysis revealed patterns of missingness in data, including Little's test for missing completely at random (MCAR). Given substantial missingness in WC and some laboratory variables (described in Table 2), multiple imputation by chained equations (MICE) with predictive mean matching was performed (m = 200 imputations; 30 iterations). Predictors included age, weight, height, WC, fat mass, skeletal muscle mass, and visceral fat rating, selected a priori based on moderate-to-strong correlations with target variables.

Because of deviation from the normal distribution, laboratory variables were log-transformed before imputation to reduce right-skewness, and MICE was performed on the transformed scale. Log-transformed laboratory variables were then standardized to z-scores using the mean and standard deviation of the full analytic cohort within each imputed dataset. Domain composite scores were computed as the arithmetic mean of the relevant standardized components (glycemic: zFPG and zHbA1c; lipid: zLDL-C and zTG; hepatic: zALT and zGGT), and the overall metabolic score was calculated as the mean of the three domain scores. Anthropometric indices that depend on WC were computed after imputation of WC to preserve deterministic relationships among derived anthropometric and laboratory variables within each imputed dataset. Regression coefficients and model statistics were pooled across imputations using Rubin's rules [27].

Statistical analysis

The statistical software used for analysis was IBM SPSS Statistics v.26.0. Data distribution of each variable was assessed using visual inspection of Q-Q plots and the Kolmogorov-Smirnov test. Associations be-

tween anthropometric indices and composite outcomes were examined using hierarchical linear regression models: Block 1 included age and sex, while Block 2 added one anthropometric index (separate models per index). Comparative performance was summarized using incremental explained variance attributable to the anthropometric index (mean ΔR^2 – summarized across imputations for descriptive comparison of indices) and the respective pooled β-indices. Analyses were conducted for the overall metabolic score and each domain composite score. Sensitivity analyses repeated the same modelling framework in participants without documented comorbidities. Statistical significance was accepted at the $p < 0.05$ level.

RESULTS

Participants

A total of 401 participants (242 females, 159 males) met the eligibility criteria and were included in the analytic cohort. Documented comorbidities included hypertension (n = 152), type 2 diabetes mellitus (n = 76), Hashimoto thyroiditis (n = 91), and polycystic ovary syndrome (n = 33). Baseline characteristics are presented in Table 2.

Given the sample size (n = 401), alpha and power levels set at 0.05 and 0.80, respectively, multivariable linear regression with 3 predictors, post-hoc sensitivity analysis with G*Power revealed the minimum detectable effect size was f2 = 0.03 (a small effect size according to Cohen's criteria).

Missing data and imputation

Little's MCAR test was non-significant (χ^2 (266) = 291.699, p = 0.134), supporting compatibility with an MCAR mechanism, but not excluding MAR. Data missingness ranged from 16% to 78.8%. The proportion of missingness by variable is reported in Table 2. Multiple imputation by chained equations (MICE) with predictive mean matching was therefore applied (m = 200; 30 iterations), using highly correlated variables – age, weight, height, WC, fat mass, skeletal muscle mass, and visceral fat rating as predictors. All regression estimates were pooled across imputations using Rubin's rules.

Table 2. Descriptive statistics of the whole sample

Index	Mean ± SD/Median (IQR)	Missing values	
		N	Percent
Age (years)	39.68 ± 10.11	0	0%
Weight (kg)	104.71 ± 24.82	0	0%
BMI (kg/m^2)	35.10 ± 6.80	0	0%
WC (cm)	110.28 ± 14.90	316	78.8%
Body fat (%)	36.10 ± 7.46	0	0%
VFR	11.85 ± 6.32	0	0%
FPG (mmol/l)	5.56 (5.10-6.10)	65	16.2%
HbA1c (%)	5.54 (5.20-6.10)	219	54.6%
LDL (mmol/l)	3.47 (2.30-4.00)	192	47.9%
TG (mmol/l)	1.68 (1.10 -2.50)	229	57.1%
ALT (U/l)	30 (19-48)	210	52.4%
GGT (U/l)	31 (20-53)	243	60.6%
Values are presented as mean ± SD or median (IQR), depending on the normality of distribution.			

Associations with overall metabolic burden (metabolic score)

Associations between each anthropometric index and the metabolic composite score are summarized in Table 3 and graphically represented in Fig. 1. After adjustment for age and sex, RFM demonstrated the largest incremental explained variance (ΔR^2) in the metabolic score, followed by WHtR, BMI, and BRI. In contrast, ABSI, CI, and WWI did not show statistically significant incremental contributions in this modelling framework.

For RFM, the pooled Block 2 coefficient was β = 0.480 (95% CI [0.269-0.692]), $p < 0.001$, with total $R^2 = 0.285$ and $\Delta R^2 = 0.085$ attributable to RFM. For WHtR, β = 0.289 (95% CI [0.164-0.414]), $p < 0.001$, $R^2 = 0.280$, and $\Delta R^2 = 0.080$, while BMI had a β = 0.277 (95% CI [0.175-0.379]), $p < 0.001$, with total $R^2 = 0.276$ and $\Delta R^2 = 0.076$.

In the sensitivity subsample excluding participants with documented comorbidities (n = 136), the ranking of indices by ΔR^2 had a similar pattern (Fig. 2), but estimates were less precise and fewer models reached statistical significance (presented in Table 4). This phenomenon is likely due to reduced power and/or restricted variance in metabolic burden within the healthier subgroup, rather than clear evidence of a lack of effect.

Table 3. Metabolic score regression models (pooled)

Index	β	95% CI		p-value	R^2 (total)	ΔR^2
		Lower bound	Upper bound			
BMI	0.277	0.175	0.379	0.000	0.276	0.076
WHtR	0.289	0.164	0.414	0.000	0.280	0.080
ABSI	-0.019	-0.210	0.171	0.843	0.205	0.005
BRI	0.280	0.157	0.404	0.000	0.275	0.076
CI	0.133	-0.063	0.329	0.184	0.218	0.018
WWI	0.156	-0.023	0.335	0.087	0.225	0.025
RFM	0.480	0.269	0.692	0.000	0.285	0.085

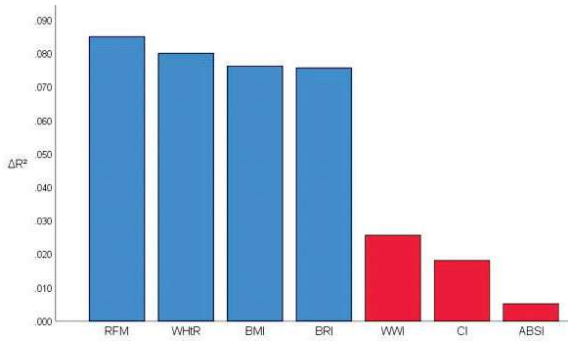

Fig. 1. Comparison of the incremental variance explained (ΔR^2) by each anthropometric index regarding the metabolic composite score. Blue = p < 0.001, red = p > 0.05

Table 4. Metabolic score regression models (sensitivity analysis)

Index	β	95% CI		p-value	R^2 (total)	ΔR^2
		Lower bound	Upper bound			
BMI	0.350	0.120	0.580	0.003	0.220	0.074
WHtR	0.309	0.074	0.544	0.010	0.218	0.071
ABSI	-0.013	-0.314	0.288	0.932	0.157	0.010
BRI	0.313	0.070	0.557	0.012	0.214	0.068
CI	0.116	-0.183	0.414	0.447	0.165	0.019
WWI	0.157	-0.117	0.431	0.260	0.175	0.029
RFM	0.482	0.121	0.842	0.009	0.223	0.076

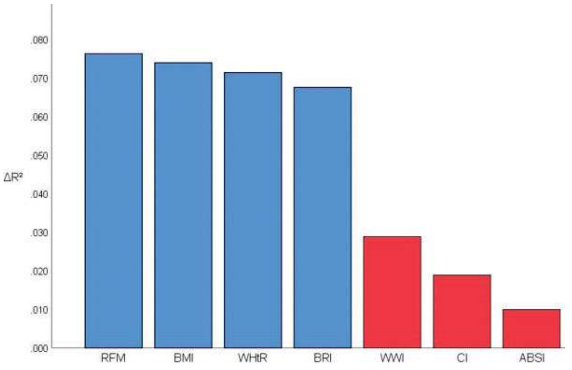

Fig. 2. Comparison of the incremental variance explained (ΔR^2) by each anthropometric index regarding the metabolic composite score (sensitivity analysis). Blue = p < 0.001, red = p > 0.05

Table 5. Glycemic score regression models (pooled)

Index	β	95% CI		p-value	R^2 (total)	ΔR^2
		Lower bound	Upper bound			
BMI	0.170	0.074	0.265	0.001	0.236	0.029
WHtR	0.143	0.032	0.253	0.012	0.229	0.020
ABSI	-0.027	-0.182	0.128	0.735	0.212	0.003
BRI	0.144	0.033	0.255	0.011	0.229	0.020
CI	0.058	-0.099	0.215	0.466	0.214	0.005
WWI	0.039	-0.107	0.185	0.601	0.213	0.004
RFM	0.219	0.038	0.400	0.018	0.227	0.018

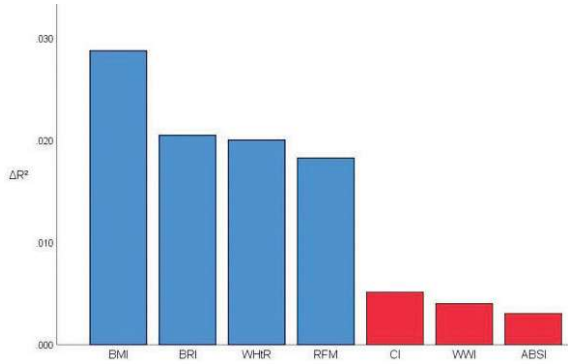

Fig. 3. Comparison of the incremental variance explained (ΔR^2) by each anthropometric index regarding the glycemic composite score. Blue = p < 0.001, red = p > 0.05

Table 6. Glycemic score regression models (sensitivity analysis)

Index	β	95% CI		p-value	R^2 (total)	ΔR^2
		Lower bound	Upper bound			
BMI	0.050	-0.083	0.182	0.461	0.172	0.006
WHtR	0.012	-0.120	0.143	0.861	0.170	0.003
ABSI	-0.036	-0.188	0.117	0.646	0.175	0.008
BRI	0.013	-0.124	0.149	0.857	0.170	0.003
CI	-0.008	-0.159	0.142	0.913	0.173	0.005
WWI	-0.028	-0.168	0.111	0.692	0.174	0.007
RFM	0.017	-0.182	0.215	0.870	0.170	0.003

Associations with glycemic score

Results for the glycemic composite are presented in Table 5 and visualized in Fig. 3. BMI demonstrated the largest ΔR^2 for the glycemic score, followed by BRI, WHtR, and RFM. In the no-comorbidity sensitivity subsample, none of the models reached statistical significance, with wider confidence intervals consistent with reduced power (Table 6).

Associations with lipid score

For the lipid composite, RFM again showed the largest incremental explained variance, followed by WHtR, BRI, and BMI, while ABSI, CI, and WWI did not contribute significantly (Table 7, Figure 4). The sensitivity analysis exhibited the same rank-order pattern, although statistical significance was attenuated (Table 8).

Table 7. Lipid score regression models (pooled)

Index	β	95% CI		p-value	R^2 (total)	ΔR^2
		Lower bound	Upper bound			
BMI	0.151	0.027	0.275	0.017	0.108	0.024
WHtR	0.173	0.027	0.319	0.020	0.115	0.031
ABSI	0.028	-0.168	0.224	0.778	0.090	0.006
BRI	0.166	0.022	0.310	0.024	0.113	0.029
CI	0.115	-0.087	0.317	0.263	0.099	0.015
WWI	0.113	-0.071	0.298	0.229	0.100	0.017
RFM	0.294	0.051	0.536	0.018	0.118	0.034

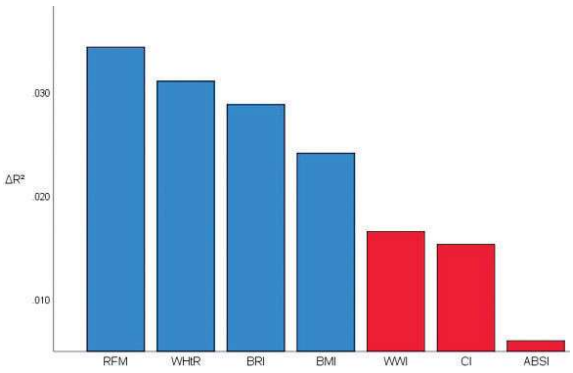

Fig. 4. Comparison of the incremental variance explained (ΔR^2) by each anthropometric index regarding the lipid composite score. Blue = $p < 0.001$, red = $p > 0.05$

Table 8. Lipid score regression models (sensitivity analysis)

Index	β	95% CI		p-value	R^2 (total)	ΔR^2
		Lower bound	Upper bound			
BMI	0.199	-0.076	0.474	0.156	0.092	0.023
WHtR	0.196	-0.073	0.465	0.154	0.098	0.029
ABSI	0.044	-0.282	0.370	0.791	0.080	0.011
BRI	0.190	-0.089	0.469	0.183	0.095	0.026
CI	0.119	-0.198	0.437	0.460	0.087	0.018
WWI	0.124	-0.170	0.419	0.828	0.089	0.020
RFM	0.329	-0.081	0.739	0.116	0.104	0.035

Associations with hepatic score

For the hepatic composite score, the pattern mirrored the lipid and overall metabolic composites: RFM showed the largest ΔR^2, followed by WHtR, BMI, and BRI, while ABSI, CI, and WWI were not significant contributors (Table 9, Figure 5). Similar tendencies were observed in the no-comorbidity sensitivity analysis (Table 10).

Table 9. Hepatic score regression models (pooled)

Index	β	95% CI		p-value	R^2 (total)	ΔR^2
		Lower bound	Upper bound			
BMI	0.251	0.145	0.357	0.000	0.227	0.063
WHtR	0.261	0.130	0.391	0.000	0.230	0.066
ABSI	-0.038	-0.234	0.159	0.708	0.170	0.006
BRI	0.253	0.124	0.382	0.000	0.226	0.062
CI	0.100	-0.103	0.302	0.333	0.177	0.013
WWI	0.138	-0.046	0.322	0.142	0.185	0.022
RFM	0.434	0.216	0.652	0.000	0.234	0.070

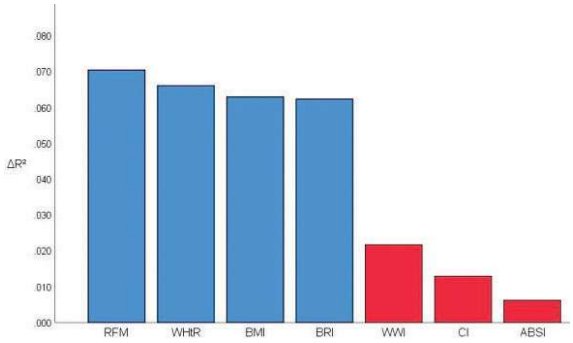

Fig. 5. Comparison of the incremental variance explained (ΔR^2) by each anthropometric index regarding the hepatic composite score. Blue = $p < 0.001$, red = $p > 0.05$

Table 10. Hepatic score regression models (sensitivity analysis)

Index	β	95% CI		p-value	R^2 (total)	ΔR^2
		Lower bound	Upper bound			
BMI	0.353	0.102	0.604	0.006	0.215	0.065
WHtR	0.307	0.046	0.569	0.021	0.214	0.062
ABSI	-0.037	-0.369	0.296	0.829	0.163	0.011
BRI	0.317	0.045	0.588	0.022	0.213	0.060
CI	0.089	-0.241	0.420	0.595	0.167	0.015
WWI	0.149	-0.154	0.453	0.333	0.177	0.025
RFM	0.465	0.063	0.866	0.023	0.214	0.062

Given the clinic-based design and incomplete medication information, these findings should be interpreted as comparative age- and sex-adjusted associations in routine practice rather than causal effects of adiposity distribution on laboratory outcomes.

DISCUSSION

This cross-sectional analysis on a real-world outpatient endocrinology cohort of adults with BMI

≥ 25 kg/m^2 compared the association of several anthropometric parameters with composite measures of metabolic health. Our main finding was that RFM showed the largest contribution to explained variance (ΔR^2) for the general metabolic score when adjusted for age and sex. The pattern was similar, with reduced precision in the comorbidity-free subsample as a sensitivity check. Other indices – WHtR, BMI, and BRI, closely followed RFM in the strength of association with the composite laboratory indices. A few of them – ABSI, CI, WWI – did not show statistically significant contributions in this modelling framework. Domain-specific analyses further indicated heterogeneity by metabolic pathway: BMI demonstrated the largest ΔR^2 for the glycemic composite, whereas RFM most consistently ranked highest for lipid and hepatic composites, paralleling the pattern observed for overall metabolic burden. Similar results were provided by the sensitivity analysis in each domain.

A biologically plausible explanation for the superior performance of RFM and WHtR is that both indices directly reflect the relationship between waist circumference and stature, thereby capturing aspects of central adiposity that are more closely linked to insulin resistance [28, 29], atherogenic dyslipidemia [30], and hepatic metabolic dysfunction [29] than total mass alone, which is the main weakness of BMI. RFM is explicitly constructed to approximate adiposity using height-to-waist geometry, including the impact of gender, which may increase its ability to track adiposity-related biochemical changes in mixed-sex clinical samples [15, 31, 32]. Similarly, WHtR is an established measure of abdominal size relative to body frame and may act as a pragmatic surrogate of visceral adiposity [11, 12, 18, 20, 33]. In contrast, indices such as ABSI and CI were designed to normalize waist circumference by body size in a manner that may attenuate associations with metabolic biomarkers in certain populations, particularly when the sample spans wide ranges of age, adiposity, and treatment exposure [18, 20, 32-34]. The comparatively stronger association of BMI with the glycemic composite score may reflect a closer coupling of total mass with insulin resistance–related glycaemia in this clinic population. However, glycemic markers are also particularly sensitive to pharmacotherapy and disease duration, which could influence observed associations in patients with comorbidities.

Our findings align with the broader concept that anthropometric indices emphasizing central adiposity often demonstrate stronger associations with cardiometabolic risk markers than BMI alone [10, 35], although the literature is heterogeneous with respect to which “beyond BMI” index performs best and in which populations [12, 33-35]. Head-to-head comparisons remain limited, and differences in covariate adjustment, outcome definitions, and sampling frames complicate direct comparison across studies [36, 37]. The present analysis contributes to this evidence base by evaluating multiple indices within a single analytic framework, using standardized domain composites to reflect glycemic, lipid, and hepatic laboratory burden, and by incorporating a sensitivity analysis in a subgroup without documented comorbidities.

From a clinical perspective, these results suggest that simple indices incorporating waist circumference – particularly RFM and WHtR – may provide a practical complement to BMI when clinicians seek anthropometric correlates of overall metabolic laboratory burden in adults with BMI ≥25 kg/m² seen in routine endocrinology practice. More importantly, the present study does not establish causal effects of adiposity distribution on laboratory outcomes and was not designed to assess out-of-sample predictive performance. Rather, the observed ΔR^2 rankings should be interpreted as comparative explanatory contributions within age- and sex-adjusted models. The composite-score approach may also be clinically informative because it represents metabolic burden as a continuous trait across biologically grounded domains, potentially capturing gradations of risk that are not well represented by binary diagnostic thresholds.

There are several strengths that support the validity of the study. Firstly, we used a real-world clinical sample that included individuals with different obesity-related comorbidities. That reflects the regular context where anthropometric stratification is most frequently applied. Second, multiple indices were compared directly in a single analytical framework using hierarchical regression models and biologically anchored metabolic composite scores, reducing multiplicity and increasing statistical power. Another characteristic of the analysis that contributes to sensitivity is the continuous nature of the composite scores – by eliminating dichotomous thresholds, they align closer to biology, accenting different degrees of metabolic burden. Third, missing data were addressed using multiple imputation with predictive mean matching by incorporating only highly correlated predictors to ensure robustness, along with the large number of imputations.

The limitations of this study should also be emphasized – first, the cross-sectional design that cannot capture causality and a clear direction of association. The single-center outpatient setting also limits generalizability to broader populations or other clinical cohorts. Since some important factors like medication (because of missing documentation), physical activ-

ity or dietary patterns were not adjusted for, residual confounding is likely. Although sensitivity analyses excluding participants with documented comorbidities reduce this concern, they do not eliminate confounding by unmeasured treatment exposure or subclinical disease and may introduce selection effects. Another important limitation worth mentioning and considering in the results interpretation is the missing data issue. Missingness in waist circumference was substantial (up to 79%), while multiple imputation can reduce bias and improve precision under plausible assumptions, the MAR assumption is not empirically verifiable, and misspecification of the imputation model could influence derived indices. Therefore, we recommend careful interpretation of the results, given that the main exposures include waist circumference data that is mostly imputed. Comparison with results from established and complete datasets is desirable in order to verify the validity of our imputation-based analysis. Finally, the composite scores apply equal weighting to standardized laboratory components. Alternative weighting schemes or inclusion of additional biomarkers (e.g., HDL, blood pressure, uric acid, inflammatory markers) could yield different domain structures and should be explored.

Future research should focus on including more complete covariate and medication data across age and sex strata, as well as different obesity phenotypes. Prospective designs could reveal temporal relationships and predictive capabilities of the anthropometric indices of interest, along with their association with treatment modalities.

CONCLUSION

In conclusion, among adults with BMI ≥25 kg/m² attending an outpatient endocrinology clinic, RFM showed the most consistent and largest incremental association with overall metabolic laboratory burden, with WHtR, BMI, and BRI also demonstrating meaningful associations, whereas ABSI, CI, and WWI contributed little in this setting. These findings support the pragmatic clinical value of waist-based indices, particularly RFM and WHtR, as complements to BMI for describing metabolic laboratory burden in routine practice.

Statement of Ethics: *This study did not require ethical approval, as it involved the use of routinely collected, completely anonymized data and complied with the local regulations governing non-interventional research. Written informed consent was not required for this study, as all patients had previously provided general consent for the use of their anonymized medical data for scientific research during their admission in the outpatient clinic.*

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REFERENCES

1. Busetto L, Dicker D, Frühbeck G, et al. A new framework for the diagnosis, staging and management of obesity in adults. Nat Med. 2024 July 5;1–5.
2. Rubino F, Cummings DE, Eckel RH, et al. Definition and diagnostic criteria of clinical obesity. Lancet Diabetes Endocrinol. 2025 Jan; S2213858724003164.
3. Yao Z, Dardari ZA, Razavi AC, et al. Prevalence of clinical obesity versus BMI-defined obesity among US adults: a cohort study. Lancet Diabetes Endocrinol. 2025 June; S2213858725001597.
4. Blundell JE, Dulloo AG, Salvador J, Frühbeck G, on behalf of the EASO SAB Working Group on BMI G. Beyond BMI – Phenotyping the Obesities. Obes Facts. 2014 Oct 23;7(5):322–8.
5. Romero-Corral A, Somers VK, Sierra-Johnson J, et al. Normal weight obesity: a risk factor for cardiometabolic dysregulation and cardiovascular mortality. Eur Heart J. 2010 Mar;31(6):737–46.
6. Ahmed KY, Aychiluhm SB, Thapa S, et al. Cardiometabolic Outcomes Among Adults With Abdominal Obesity and Normal Body Mass Index. JAMA Netw Open. 2025 Oct 17;8(10):e2537942.
7. Padwal RS, Pajewski NM, Allison DB, Sharma AM. Using the Edmonton obesity staging system to predict mortality in a population-representative cohort of people with overweight and obesity. CMAJ. 2011 Oct 4;183(14):E1059–66.
8. Dulloo AG, Jacquet J, Solinas G, et al. Body composition phenotypes in pathways to obesity and the metabolic syndrome. Int J Obes. 2010 Dec;34(S2):S4–17.
9. Goossens GH. The Metabolic Phenotype in Obesity: Fat Mass, Body Fat Distribution, and Adipose Tissue Function. Obes Facts. 2017 July;10(3):207–15.
10. 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: Waist-to-height ratio as a screening tool. Obes Rev. 2012 Mar;13(3):275–86.
11. Zazai R, Wilms B, Ernst B, et al. Waist Circumference and Related Anthropometric Indices Are Associated with Metabolic Traits in Severely Obese Subjects. Obes Surg. 2014 May;24(5):777–82.
12. Piqueras P, Ballester A, Durá-Gil JV, et al. Anthropometric Indicators as a Tool for Diagnosis of Obesity and Other Health Risk Factors: A Literature Review. Front Psychol [Internet]. 2021 July 9 [cited 2025 Jan 4];12. Available from: https://www.frontiersin.org/journals/psychology/articles/10.3389/fpsyg.2021.631179/full

13. Park Y, Kim NH, Kwon TY, Kim SG. A novel adiposity index as an integrated predictor of cardiometabolic disease morbidity and mortality. Sci Rep. 2018 Nov 13;8(1):16753.
14. Thomas DM, Bredlau C, Bosy-Westphal A, et al. Relationships between body roundness with body fat and visceral adipose tissue emerging from a new geometrical model. Obesity. 2013;21(11):2264–71.
15. Woolcott OO, Bergman RN. Relative fat mass (RFM) as a new estimator of whole-body fat percentage — A cross-sectional study in American adult individuals. Sci Rep. 2018 July 20;8:10980.
16. Krakauer NY, Krakauer JC. A New Body Shape Index Predicts Mortality Hazard Independently of Body Mass Index. Li S, editor. PLoS ONE. 2012 July 18;7(7):e39504.
17. Valdez R, Seidell JC, Ahn YI, Weiss KM. A new index of abdominal adiposity as an indicator of risk for cardiovascular disease. A cross-population study. Int J Obes Relat Metab Disord. 1993 Feb 1;17(2):77–82.
18. Wu Q, Qin K, Wang Y, Nie H. Anthropometric indices and their predictive ability on metabolic syndrome in west China. Int J Diabetes Dev Ctries. 2022 Oct 1;42(4):666–82.
19. Wu L, Zhu W, Qiao Q, et al. Novel and traditional anthropometric indices for identifying metabolic syndrome in non-overweight/obese adults. Nutr Metab. 2021 Jan 6;18:3.
20. Yin T, Wang J, Lan X, et al. Different obesity indicators and their correlation with hypertension, diabetes, and dyslipidemia in 35–74 years rural residents in Northwest China. Front Endocrinol [Internet]. 2025 June 9 [cited 2025 Nov 21];16. Available from: https://www.frontiersin.org/journals/endocrinology/articles/10.3389/fendo.2025.1346193/full
21. Al-Shami I, Al-Dalaeen A, Agraib L, Alkhatib B. Obesity indices may affect and reflect the blood glucose and lipid profile values. Healthc Low-Resour Settings. 2024 Dec 4; DOI:10.4081/hls.2024.12865
22. Fahami M, Hojati A, Farhangi MA. Body shape index (ABSI), body roundness index (BRI) and risk factors of metabolic syndrome among overweight and obese adults: a cross-sectional study. BMC Endocr Disord. 2024 Oct 28;24(1):230.
23. Eisenmann JC. On the use of a continuous metabolic syndrome score in pediatric research. Cardiovasc Diabetol. 2008 June 5;7(1):17.
24. Okosun IS, Boltri JM, Lyn R, Davis-Smith M. Continuous Metabolic Syndrome Risk Score, Body Mass Index Percentile, and Leisure Time Physical Activity in American Children. J Clin Hypertens. 2010;12(8):636–44.
25. Shafiee G, Kelishadi R, Heshmat R, et al. First report on the validity of a continuous Metabolic Syndrome score as an indicator for Metabolic Syndrome in a national sample of paediatric population – the CASPIAN-III study. Endokrynol Pol. 2013;64(4):278–84.
26. DeBoer MD, Gurka MJ. Clinical utility of metabolic syndrome severity scores: considerations for practitioners. Diabetes Metab Syndr Obes. 2017 Feb 20;10:65–72.
27. Murray JS. Multiple Imputation: A Review of Practical and Theoretical Findings. Stat Sci. 2018 May;33(2):142–59.
28. Zatterale F, Longo M, Naderi J, et al. Chronic Adipose Tissue Inflammation Linking Obesity to Insulin Resistance and Type 2 Diabetes. Front Physiol. 2020 Jan 29; 10:1607.
29. Finer N. Medical consequences of obesity. Medicine (Baltimore). 2015 Feb; 43(2):88–93.
30. Vekic J, Stefanovic A, Zeljkovic A. Obesity and Dyslipidemia: A Review of Current Evidence. Curr Obes Rep. 2023 Sept 1; 12(3):207–22.
31. Hosseini SA, Alipour M, Sarvandian S, et al. Assessment of the appropriate cutoff points for anthropometric indices and their relationship with cardio-metabolic indices to predict the risk of metabolic associated fatty liver disease. BMC Endocr Disord. 2024 June 4;24(1):79.
32. Chaquila JA, Ramirez-Jeri G, Miranda-Torvisco F, et al. Predictive ability of anthropometric indices for risk of developing metabolic syndrome: a cross-sectional study. J Int Med Res. 2024 Nov 1;52(11):03000605241300017.
33. Mahdavi-Roshan M, Rezazadeh A, Joukar F, et al. Comparison of anthropometric indices as predictors of the risk factors for cardiovascular disease in Iran: The PERSIAN Guilan Cohort Study. Anatol J Cardiol. 2021;25(2):120.
34. Yang H, Zhang M, Nie J, et al. Associations of obesity-related indices with prediabetes regression to normoglycemia among Chinese middle-aged and older adults: a prospective study. Front Nutr [Internet]. 2023 May 19 [cited 2025 Nov 19];10. Available from: https://www.frontiersin.org/journals/nutrition/articles/10.3389/fnut.2023.1075225/full
35. Lee BJ, Yim MH. Comparison of anthropometric and body composition indices in the identification of metabolic risk factors. Sci Rep. 2021 May 11;11(1):9931.
36. Gavrailova M, Elenkova A, Robeva R. Differences in Metabolic Parameters in Patients with Different Degrees of Obesity. Acta Medica Bulg. 2026 Jun 16;53(2):22–8. doi:10.2478/amb-2026-0055
37. Stoynova M, Lilova L, Petrova D, et al. Prevalence of Metabolic Syndrome and its Components in Patients with Controlled Graves' Disease. Acta Medica Bulg. 2024 Mar 16;51(1):17–24. doi:10.2478/amb-2024-0003