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Body Fat, Not Body Mass Index, Is Associated with Endothelial Dysfunction and Arterial Stiffness, Whereas Hemorheological Impairment Tracks Overall Adiposity: A Cross-Sectional Study of Normal-Weight Obesity and Vascular Health in Korean Women

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Abstract

Background/Objectives: Body mass index (BMI) cannot distinguish fat from lean mass, so women with normal-weight obesity (NWO) may have unrecognized cardiovascular risks. We compared vascular function and hemorheology according to BMI and body fat in women. **Methods**: In this cross-sectional study, 150 women were classified by Asia-Pacific BMI categories and into normal-weight lean (NWL; BMI 18.5–22.9 kg/m^2, body fat < 30%), NWO (BMI 18.5–22.9 kg/m^2, body fat ≥ 30%), overweight, and obese phenotypes. Blood pressure, brachial–ankle pulse wave velocity (baPWV), flow-mediated dilation (FMD), and red blood cell (RBC) deformability and aggregation were measured. **Results**: Across BMI groups, blood pressure was elevated only at BMI ≥ 30 kg/m^2, whereas FMD declined and RBC aggregation increased progressively with BMI. Compared with NWL, women with NWO had lower FMD (−2.38%, 95% CI −3.99 to −0.76; $p = 0.005$), which persisted after further adjustment for BMI ($p = 0.023$), and higher baPWV after exclusion of one extreme value (136 cm/s, 95% CI 37 to 235; $p = 0.009$), despite similar blood pressure and RBC deformability. When BMI and body fat were entered together, body fat was independently associated with FMD, whereas BMI was associated with diastolic and mean arterial pressure. **Conclusions**: Excess body fat may impair vascular function before BMI-defined obesity develops, supporting body composition assessment in addition to BMI for early cardiovascular risk stratification in women.

Keywords: normal-weight obesity; vascular aging; body fat percentage; flow-mediated dilation; arterial stiffness; red blood cell deformability; red blood cell aggregation; hemorheology; women

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1. Introduction

Obesity is a major contributor to cardiovascular disease, and its global prevalence among adult women has continued to rise [1]. In clinical and public-health practice, obesity is defined almost exclusively by body mass index (BMI), with lower cut-offs recommended

for Asian populations because of their higher body fat at a given BMI [2,3]. However, BMI reflects total body mass and cannot discriminate fat mass from lean mass; when obesity is defined by directly measured adiposity, BMI misses a substantial proportion of individuals with excess body fat [4].

Normal-weight obesity (NWO) describes individuals with a normal BMI but a high body fat percentage [5–7]. Women with NWO show a clustering of cardiometabolic risk factors and, in some cohorts, higher cardiovascular mortality than their lean counterparts with a similar BMI [6,8]. Because these individuals are not identified as obese by BMI screening, they are unlikely to receive preventive lifestyle advice, and the physiological pathways through which excess adiposity damages the cardiovascular system in the absence of an elevated BMI remain incompletely understood [9].

Endothelial dysfunction and arterial stiffening are hallmarks of vascular aging and early, functional stages of atherosclerosis. Flow-mediated dilation (FMD) of the brachial artery is the standard non-invasive index of endothelium-dependent vasodilation [10,11], and brachial–ankle pulse wave velocity (baPWV) is a validated measure of arterial stiffness [12]; both independently predict cardiovascular events [13,14]. Adipose tissue, particularly when expanded and inflamed, releases adipokines, cytokines, and reactive oxygen species that reduce nitric oxide bioavailability and promote vascular remodeling [15,16]. These mechanisms suggest that vascular function may be more closely linked to fat mass than to body mass per se.

Blood rheology is a less frequently examined determinant of tissue perfusion. Red blood cell (RBC) deformability allows erythrocytes to pass through capillaries narrower than their own diameter, and RBC aggregation determines blood viscosity at low shear rates in the venous and microvascular circulation [17]. Impaired RBC deformability and enhanced aggregation have been reported in individuals with obesity [18] and improve after weight loss [19,20], possibly through oxidative stress and altered membrane lipid composition [21]. Whether hemorheological alterations are already present in women with NWO, and whether they track body fat or overall body mass, has not been established. Because erythrocytes are the principal cellular determinant of blood viscosity and of oxygen delivery through the microcirculation, hemorheological assessment was included to probe a cell-level mechanism of impaired tissue perfusion that is complementary to, and independent of, endothelium-dependent macrovascular function.

Therefore, this study aimed (1) to compare vascular function and hemorheological properties across BMI categories in women; (2) to determine whether women with NWO exhibit impairments relative to normal-weight lean (NWL) women; and (3) to examine the independent contributions of BMI and body fat percentage to these outcomes. We hypothesized that vascular function would be impaired in women with NWO and associated with body fat independently of BMI. Although endothelial function, arterial stiffness, and blood rheology have each been examined in overt obesity, they have not been studied together in women with NWO, and it remains unclear whether they are driven by body fat or by body mass; this gap limits early cardiovascular risk stratification in women whose BMI appears normal.

2. Materials and Methods

2.1. Study Design and Participants

This cross-sectional study was a secondary analysis of anonymized data from 150 women aged 20–63 years who completed body composition, vascular, and hemorheological assessments at the Exercise Physiology Laboratory of Dongseo University, Busan, Republic of Korea. Participants were included if they were aged 20 years or older and had complete body composition, blood pressure, arterial stiffness, endothelial function,

and hemorheological data. Women were excluded if they were pregnant or lactating, were current smokers, had a diagnosed cardiovascular, metabolic, renal, or hematological disease, or had any acute illness or infection at the time of testing. Information on medication use and menopausal status was not available.

These data were drawn from a prospective trial investigating the effects of Pilates exercise in a hypoxic environment, conducted at the Exercise Physiology Laboratory of Dongseo University; the present work is a cross-sectional secondary analysis of the participants' baseline (pre-intervention) assessments. Community-dwelling women were recruited by poster and social-media (SNS) advertisement over an approximately 2-month period in 2021. The original prospective trial was reviewed and approved by the appropriate Institutional Review Board (approval No. 7001355-202112-HR-496) and was registered with the Clinical Research Information Service (CRIS), Republic of Korea (registration No. KCT0008469), and all participants provided written informed consent before testing. For the present secondary analysis of fully anonymized data, the Institutional Review Board of Dongseo University waived additional review and the requirement for informed consent (IRB No. 1041493-E-2026-014).

All assessments were performed between 07:00 and 11:00 to limit circadian variation, with laboratory temperature maintained at 22–24 °C and relative humidity at 40–60%. Participants avoided caffeine, alcohol, and strenuous activity for 24 h before the visit and fasted for at least 8 h. Fasting blood was sampled first for hemorheological analyses; after a 30 min stabilization period, resting blood pressure, arterial stiffness, brachial artery endothelial function, and body composition were measured in that fixed order.

2.2. Anthropometry and Body Composition

Standing height was measured to the nearest 0.1 cm with a wall-mounted stadiometer (YM-1; KDS, Seoul, Republic of Korea). Body weight and body fat percentage were obtained by whole-body dual-energy X-ray absorptiometry (Primus; Osteosys, Seoul, Republic of Korea) performed in the supine position by a trained technician according to the manufacturer's standard protocol, with participants wearing light clothing and all metal items removed. BMI was calculated as weight (kg) divided by height squared (m^2). Fat mass was derived as body weight × body fat percentage, and fat-free mass as body weight − fat mass.

Participants were classified into four BMI groups according to the Asia-Pacific criteria adopted by the Korean Society for the Study of Obesity [2,3]: G1, <23.0 kg/m^2; G2, 23.0–24.9 kg/m^2; G3, 25.0–29.9 kg/m^2; and G4, ≥30.0 kg/m^2. For the body-fat phenotype analysis, participants with BMI < 18.5 kg/m^2 (n = 7) were excluded and the remaining 143 women were classified as NWL (BMI 18.5–22.9 kg/m^2 and body fat < 30%), NWO (BMI 18.5–22.9 kg/m^2 and body fat ≥ 30%), overweight (OW; BMI 23.0–24.9 kg/m^2), or obese (OB; BMI ≥ 25.0 kg/m^2). A body fat threshold of 30%, within the range of cut-offs used to define NWO in women [8,9], was applied.

2.3. Blood Pressure

Resting blood pressure was measured using an automatic sphygmomanometer (ACCUNIQ BP210; SELVAS Healthcare, Daejeon, Republic of Korea). After at least 5 min of seated rest, systolic (SBP) and diastolic blood pressure (DBP) were measured at the right brachial artery positioned at heart level; two consecutive measurements taken 1 min apart were averaged. Mean arterial pressure (MAP) was calculated as DBP + (SBP − DBP)/3, and pulse pressure (PP) as SBP − DBP. Measurements were taken in a quiet, temperature-controlled room after the participant had been seated for at least 5 min with the back supported, both feet flat on the floor, legs uncrossed, and the measurement arm supported at heart

level, and the participant was asked not to talk during measurement. An appropriately sized cuff was chosen according to mid-upper-arm circumference. If the first two readings differed by more than 5 mmHg, a third reading was obtained and the two closest values were averaged.

2.4. Brachial–Ankle Pulse Wave Velocity

baPWV was measured bilaterally with a non-invasive vascular screening system (VP-1000 Plus; Omron Colin, Tokyo, Japan) in the supine position after 5 min of rest [12]. The right-side baPWV was used for analysis. Occlusion and monitoring cuffs were applied to both the arms and ankles, and electrocardiographic electrodes and a phonocardiographic microphone were attached. baPWV was computed automatically as the pulse transit distance between the brachium and ankle—estimated from body height—divided by the transit time, and blood pressure was recorded simultaneously during the same acquisition. Bilateral values were closely concordant, and the right-side baPWV was used for analysis. This system has well-established validity and reproducibility (coefficient of variation <10%) [12].

2.5. Flow-Mediated Dilation

FMD of the right brachial artery was measured with a semi-automated ultrasound system (UNEXEF38G; Unex, Tokyo, Japan) equipped with a high-frequency linear-array probe and automated arterial wall-tracking, following consensus guidelines [11]. Baseline brachial artery diameter was recorded after 10 min of supine rest. A pneumatic forearm cuff was then inflated to 50 mmHg above SBP for 5 min, and the peak post-deflation diameter was recorded 60–90 s after cuff release. FMD was calculated as [(peak diameter − baseline diameter)/baseline diameter] × 100 [10], using the integrated automated edge-tracking software built into the semi-automated ultrasound system (UNEXEF38G; Unex, Tokyo, Japan). A high-frequency (≈10 MHz) linear-array transducer was fixed with a stereotactic probe holder to maintain a constant insonation angle, and brachial artery diameter was tracked continuously from B-mode images by the automated edge-detection software built into the semi-automated ultrasound system (UNEXEF38G; Unex, Tokyo, Japan), gated to the R-wave of the electrocardiogram. FMD was taken as the maximal percentage increase in diameter after cuff release relative to baseline. All scans were performed by a single trained operator in a temperature-controlled room; the day-to-day coefficient of variation for FMD in our laboratory was 5.6%.

2.6. Hemorheological Measurements

Fasting blood was collected between 07:00 and 09:00 into K_3-EDTA tubes. Hemorheological properties were measured with a microchip-based hemorheometer (Rheoscan-D; Rheo Meditech Inc., Seoul, Republic of Korea) at 25 °C within 4–6 h of collection [22,23]. For RBC deformability, the sample was diluted in 5.5% polyvinylpyrrolidone (360 kDa) in phosphate-buffered saline (pH 7.4; 300 mOsmol/kg) and analyzed with a D-test kit, and the elongation index (EI) at a shear stress of 3 Pa was recorded. RBC aggregation was measured in 8 µL of whole blood with an A-test kit, and the aggregation index (AI, %) and the critical shear stress for disaggregation (CSS, mPa) were recorded. Higher EI indicates greater deformability, and higher AI and CSS indicate greater aggregability. Samples were kept at room temperature, gently mixed before analysis, and processed within 4–6 h of collection to avoid storage-related changes in cell rheology. The elongation index was obtained by laser-diffraction ektacytometry across a range of shear stresses and recorded at 3 Pa, and the aggregation indices were obtained by the microchip stirring–disaggregation method.

Each measurement was performed in duplicate and averaged; the coefficient of variation was <5% for the elongation index and <10% for the aggregation indices [22,23].

2.7. Statistical Analysis

Data are presented as mean ± standard deviation (SD). Normality and homogeneity of variance were examined with the Shapiro–Wilk and Levene tests. Differences among groups were tested by one-way analysis of variance (ANOVA), with eta-squared (η^2) as the effect size. Post hoc comparisons used the Tukey honestly significant difference test, or the Games–Howell test when variances were unequal; the Kruskal–Wallis test was performed as a sensitivity analysis. Because age differed among groups, analysis of covariance (ANCOVA) with age as a covariate was additionally performed, and age-adjusted estimated marginal means were compared with Bonferroni correction. A linear trend across BMI groups was tested by entering the group as an ordinal variable (1–4) in an age-adjusted linear regression model. The choice of omnibus test was specified a priori: for each outcome, when the normality (Shapiro–Wilk) and equal-variance (Levene) assumptions were met, one-way ANOVA served as the primary test and the Kruskal–Wallis test as a supporting analysis; when either assumption was violated, the Kruskal–Wallis test served as the primary test and ANOVA was reported for completeness. Under this rule, baPWV—non-normally distributed because of one extreme value—was assessed primarily by the Kruskal–Wallis test.

For the phenotype analysis, NWO and NWL were compared using age-adjusted linear regression, and the adjusted mean difference with its 95% confidence interval (CI) and Hedges' g were reported. To examine the independent contributions of BMI and body fat, both variables were entered simultaneously into age-adjusted linear regression models, and standardized regression coefficients (β) with 95% CIs were reported; collinearity was checked with the variance inflation factor. Pearson and age-adjusted partial correlation coefficients were also calculated. One extreme baPWV value (2314.5 cm/s; >3 interquartile ranges above the third quartile) was retained in the primary analyses, and results excluding this value are presented as a sensitivity analysis. Analyses were performed with Python 3 (SciPy, statsmodels, and pingouin), and two-sided $p < 0.05$ was considered statistically significant. To address multiple testing across the nine vascular and hemorheological outcomes, the Benjamini–Hochberg procedure was applied to the primary group-comparison p values within each analysis family (BMI group and adiposity phenotype), and false-discovery-rate-adjusted q values are reported alongside nominal p values; interpretation emphasizes effect sizes and 95% CIs rather than statistical significance alone. Because baPWV depends on the blood pressure and heart rate prevailing at measurement, the baPWV comparisons were additionally adjusted for mean arterial pressure and heart rate in sensitivity ANCOVA models. Finally, because mean age differed across groups, an age × group interaction term was added to each ANCOVA model to confirm homogeneity of the age slopes before the age-adjusted group effects were interpreted.

3. Results

3.1. Participant Characteristics

Characteristics of the participants according to BMI group are shown in Table 1. Body weight, fat mass, fat-free mass, and body fat percentage increased progressively across BMI groups (all $p < 0.001$). Age differed among groups ($p < 0.001$); women in G1 were younger than those in G2 and G3.

Table 1. Characteristics of the participants according to BMI group.

Variable	G1: <23.0 (n = 47)	G2: 23.0–24.9 (n = 29)	G3: 25.0–29.9 (n = 53)	G4: ≥30.0 (n = 21)	*F*	*p*
Age (years)	39.4 ± 8.4	44.5 ± 9.8 [a]	47.9 ± 7.1 [a]	43.0 ± 7.1	9.39	<0.001
Height (cm)	164.1 ± 5.4	160.9 ± 6.1	160.1 ± 6.3 [a]	157.0 ± 3.7 [ab]	8.43	<0.001
Body weight (kg)	54.8 ± 5.9	61.9 ± 4.6 [a]	68.8 ± 6.2 [ab]	79.3 ± 6.3 [abc]	99.85	<0.001
BMI (kg/m^2)	20.3 ± 1.8	23.9 ± 0.6 [a]	26.8 ± 1.2 [ab]	32.2 ± 2.6 [abc]	304.59	<0.001
Fat-free mass (kg)	39.9 ± 3.7	40.3 ± 3.8	42.7 ± 4.6 [ab]	44.7 ± 3.7 [ab]	9.37	<0.001
Fat mass (kg)	14.9 ± 4.9	21.6 ± 2.4 [a]	26.1 ± 3.1 [ab]	34.5 ± 5.1 [abc]	135.65	<0.001
Body fat (%)	26.8 ± 7.0	34.9 ± 3.3 [a]	37.9 ± 3.2 [ab]	43.5 ± 3.9 [abc]	73.91	<0.001

Values are mean ± SD. All participants were women. G1 includes 7 participants with BMI < 18.5 kg/m^2. *p* values were obtained by one-way ANOVA. Superscripts indicate significant post hoc differences ($p < 0.05$): a, vs. G1; b, vs. G2; c, vs. G3 (Tukey HSD, or Games–Howell test when variances were unequal).

3.2. Vascular Function According to BMI Group

SBP, DBP, and MAP differed significantly among BMI groups (all $p < 0.001$; Table 2, Figure 1). Post hoc analysis showed that all three were higher in G4 than in G1, G2, and G3, with no differences among G1–G3; these differences persisted after adjustment for age (age-adjusted $p < 0.001$; Table S1). PP did not differ among groups ($p = 0.256$).

FMD differed among BMI groups ($p < 0.001$, $\eta^2 = 0.130$) and decreased progressively from G1 (9.67 ± 2.66%) to G2 (8.12 ± 2.39%), G3 (7.56 ± 2.58%), and G4 (7.08 ± 2.91%), with a significant age-adjusted linear trend ($p < 0.001$); post hoc analysis showed higher FMD in G1 than in G3 and G4, and the difference between G1 and G4 remained after adjustment for age. baPWV did not differ by one-way ANOVA ($p = 0.137$); however, the Kruskal–Wallis test ($p = 0.009$) and age-adjusted ANCOVA ($p = 0.044$) indicated group differences, and after exclusion of one extreme value in G1, the ANOVA became significant ($p = 0.015$). After Benjamini–Hochberg correction across the nine outcomes, the group differences in SBP, DBP, MAP, FMD, baPWV, the elongation index, and the aggregation index all remained significant ($q < 0.05$), whereas PP and CSS did not. The higher baPWV in the upper BMI groups persisted after additional adjustment for mean arterial pressure (adjusted $p = 0.013$; heart rate was not measured). The age × group interaction was non-significant for the primary outcomes, including baPWV ($p = 0.750$) and FMD ($p = 0.088$), supporting the age-adjusted comparisons, although interactions were detected for PP ($p = 0.048$) and CSS ($p = 0.013$), which are therefore interpreted cautiously.

Table 2. Vascular function according to BMI group.

Variable	G1: <23.0 (n = 47)	G2: 23.0–24.9 (n = 29)	G3: 25.0–29.9 (n = 53)	G4: ≥30.0 (n = 21)	*F*	*p*	η^2	*p* adj	*p* Trend
SBP (mmHg)	123.6 ± 10.3	119.7 ± 11.7	124.3 ± 11.3	134.8 ± 14.0 [abc]	7.34	<0.001	0.131	<0.001	0.005
DBP (mmHg)	72.6 ± 6.9	72.5 ± 7.2	76.1 ± 9.4	85.5 ± 7.8 [abc]	14.13	<0.001	0.225	<0.001	<0.001
MAP (mmHg)	89.6 ± 6.5	88.3 ± 8.4	92.2 ± 9.4	101.9 ± 9.4 [abc]	12.99	<0.001	0.211	<0.001	<0.001
PP (mmHg)	51.0 ± 10.6	47.2 ± 6.8	48.3 ± 7.9	49.3 ± 8.7	1.36	0.256	0.027	0.249	0.299
baPWV (cm/s)	1123.3 ± 234.3	1124.0 ± 163.6	1142.9 ± 159.5	1231.4 ± 140.3	1.87	0.137	0.037	0.044	0.384
FMD (%)	9.67 ± 2.66	8.12 ± 2.39	7.56 ± 2.58 [a]	7.08 ± 2.91 [a]	7.27	<0.001	0.130	0.006	<0.001

Values are mean ± SD. *p*, one-way ANOVA; η^2, eta-squared; *p* adj, ANCOVA adjusted for age; *p* trend, linear trend across BMI groups adjusted for age. Superscripts indicate significant post hoc differences ($p < 0.05$): a, vs. G1; b, vs. G2; c, vs. G3 (Tukey HSD, or Games–Howell test when variances were unequal). baPWV: Kruskal–Wallis $p = 0.009$; after exclusion of one extreme value in G1 (2314.5 cm/s), ANOVA $p = 0.015$. BMI, body mass index; SBP, systolic blood pressure; DBP, diastolic blood pressure; MAP, mean arterial pressure; PP, pulse pressure; baPWV, brachial–ankle pulse wave velocity; FMD, flow-mediated dilation; CSS, critical shear stress.

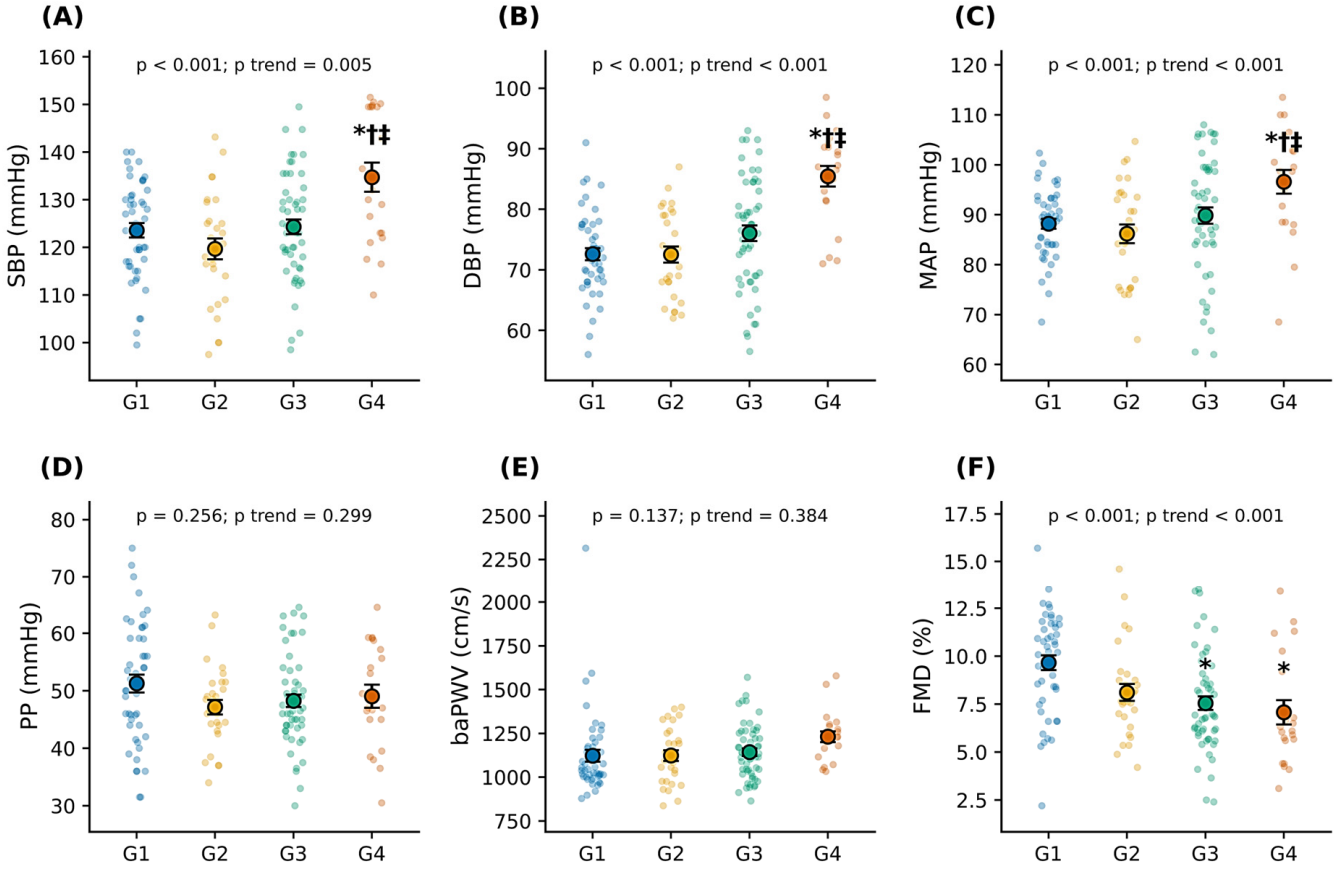


Figure 1. Vascular function according to BMI group. (**A**) SBP, (**B**) DBP, (**C**) MAP, (**D**) PP, (**E**) baPWV, and (**F**) FMD. Small symbols represent individual values; large symbols with error bars represent the mean ± standard error. *p* values are from one-way ANOVA and the age-adjusted linear trend test. * $p < 0.05$ vs. G1; † $p < 0.05$ vs. G2; ‡ $p < 0.05$ vs. G3 (post hoc test). For clarity, panels share a common x-axis order (G1–G4), each y-axis is labeled with its unit, and group sample sizes are those given in Table 2. A high-resolution (600 dpi) version is provided for production.

3.3. Hemorheological Properties According to BMI Group

RBC deformability differed among BMI groups ($p = 0.012$; Table 3, Figure 2): the EI was lower in G2 (0.324 ± 0.020), G3 (0.322 ± 0.020), and G4 (0.322 ± 0.014) than in G1 (0.335 ± 0.025), with a significant age-adjusted linear trend ($p = 0.003$); in post hoc analysis, the difference between G1 and G3 was significant before and after adjustment for age. The AI increased progressively from G1 (36.6 ± 6.9%) to G4 (40.4 ± 5.4%) ($p = 0.020$; age-adjusted p for trend = 0.006), although no individual pairwise comparison was significant. CSS did not differ among groups ($p = 0.179$).

Table 3. Hemorheological properties according to BMI group.

Variable	G1: <23.0 (n = 47)	G2: 23.0–24.9 (n = 29)	G3: 25.0–29.9 (n = 53)	G4: ≥30.0 (n = 21)	*F*	*p*	η^2	*p* adj	*p* Trend
Elongation index	0.335 ± 0.025	0.324 ± 0.020	0.322 ± 0.020 [a]	0.322 ± 0.014	3.78	0.012	0.072	0.011	0.003
CSS (mPa)	397.9 ± 119.1	390.4 ± 149.7	442.1 ± 130.6	439.4 ± 107.3	1.66	0.179	0.033	0.232	0.087
Aggregation index (%)	36.6 ± 6.9	36.1 ± 7.4	39.4 ± 5.8	40.4 ± 5.4	3.37	0.020	0.065	0.023	0.006

Values are mean ± SD. *p*, one-way ANOVA; η^2, eta-squared; *p* adj, ANCOVA adjusted for age; *p* trend, linear trend across BMI groups adjusted for age. Superscripts indicate significant post hoc differences ($p < 0.05$): a, vs. G1 (Tukey HSD, or Games–Howell test when variances were unequal). Elongation index, RBC deformability; CSS and aggregation index, RBC aggregation.

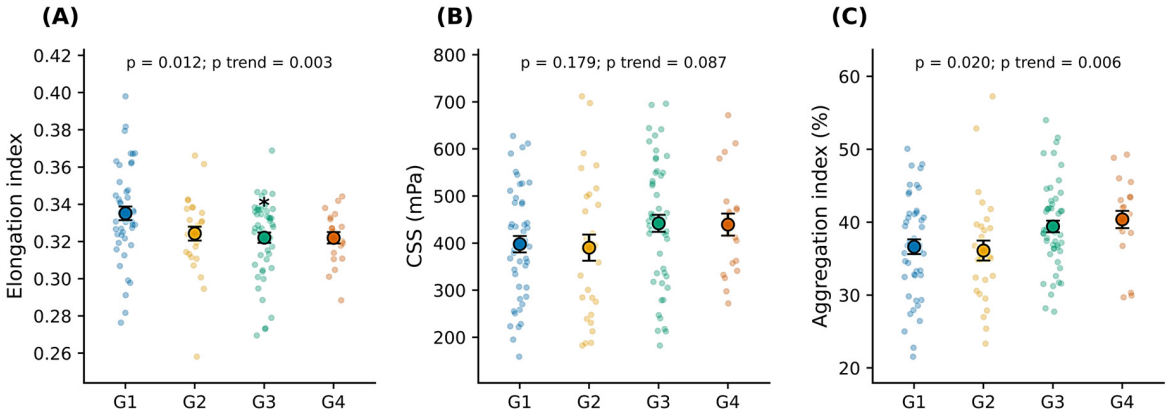


Figure 2. Hemorheological properties according to BMI group. (**A**) Elongation index (RBC deformability); (**B**) critical shear stress (CSS); and (**C**) aggregation index. Small symbols represent individual values; large symbols with error bars represent the mean ± standard error. *p* values are from one-way ANOVA and the age-adjusted linear trend test. * $p < 0.05$ vs. G1 (post hoc test). For clarity, each y-axis is labeled with its unit and group sample sizes match Table 3; a high-resolution (600 dpi) version is provided for production.

3.4. Normal-Weight Obesity Phenotype

Of the 40 women with BMI 18.5–22.9 kg/m^2, 20 (50.0%) met the criterion for NWO (Table 4). Compared with NWL women, women with NWO had a slightly higher BMI within the normal range (21.7 ± 0.9 vs. 20.0 ± 1.1 kg/m^2), markedly higher body fat (33.8 ± 2.9% vs. 21.9 ± 2.8%), and lower fat-free mass (39.0 ± 4.1 vs. 41.8 ± 2.1 kg; independent t test $p = 0.009$), and they were older (43.4 ± 8.7 vs. 36.7 ± 5.8 years).

Table 4. Body composition, vascular function, and hemorheological properties according to adiposity phenotype.

Variable	NWL (n = 20)	NWO (n = 20)	OW (n = 29)	OB (n = 74)	*p*	*p* adj	NWO − NWL (95% CI)	*p*
Age (years)	36.7 ± 5.8	43.4 ± 8.7 [a]	44.5 ± 9.8 [a]	46.5 ± 7.4 [a]	<0.001			
BMI (kg/m^2)	20.0 ± 1.1	21.7 ± 0.9 [a]	23.9 ± 0.6 [ab]	28.3 ± 3.0 [abc]	<0.001			
Body fat (%)	21.9 ± 2.8	33.8 ± 2.9 [a]	34.9 ± 3.3 [a]	39.5 ± 4.2 [abc]	<0.001			
Fat mass (kg)	11.8 ± 2.0	19.9 ± 2.3 [a]	21.6 ± 2.4 [a]	28.5 ± 5.4 [abc]	<0.001			
Fat-free mass (kg)	41.8 ± 2.1	39.0 ± 4.1	40.3 ± 3.8	43.3 ± 4.4 [bc]	<0.001			
SBP (mmHg)	126.4 ± 10.1	120.6 ± 11.1	119.7 ± 11.7	127.3 ± 13.0 [c]	0.014	0.015	−6.0 (−13.6, 1.6)	0.117
DBP (mmHg)	70.3 ± 6.0	74.2 ± 7.4	72.5 ± 7.2	78.7 ± 9.9 [ac]	<0.001	<0.001	3.3 (−1.5, 8.1)	0.174
MAP (mmHg)	89.0 ± 5.4	89.6 ± 7.9	88.3 ± 8.4	94.9 ± 10.3 [ac]	0.002	0.004	0.2 (−4.6, 5.0)	0.939
PP (mmHg)	56.1 ± 11.4	46.4 ± 8.3 [a]	47.2 ± 6.8 [a]	48.6 ± 8.1 [a]	<0.001	<0.001	−9.3 (−16.4, −2.2)	0.012
baPWV (cm/s)	1027.8 ± 59.2	1219.5 ± 316.3	1124.0 ± 163.6 [a]	1168.0 ± 158.5 [a]	0.005	0.048	146.5 (−12.2, 305.2)	0.069
FMD (%)	11.17 ± 1.67	8.32 ± 2.81 [a]	8.12 ± 2.39 [a]	7.42 ± 2.66 [a]	<0.001	<0.001	−2.38 (−3.99, −0.76)	0.005
Elongation index	0.338 ± 0.025	0.332 ± 0.029	0.324 ± 0.020	0.322 ± 0.019 [a]	0.021	0.016	−0.004 (−0.023, 0.015)	0.694
CSS (mPa)	380.7 ± 125.8	404.9 ± 125.2	390.4 ± 149.7	441.4 ± 123.7	0.141	0.222	54.6 (−31.8, 141.0)	0.208
Aggregation index (%)	35.2 ± 7.2	39.4 ± 6.2	36.1 ± 7.4	39.7 ± 5.7 [a]	0.008	0.006	5.2 (0.5, 9.9)	0.032

Values are mean ± SD. NWL, normal-weight lean (BMI 18.5–22.9 kg/m^2, body fat <30%); NWO, normal-weight obesity (BMI 18.5–22.9 kg/m^2, body fat ≥30%); OW, overweight (BMI 23.0–24.9 kg/m^2); OB, obese (BMI ≥25.0 kg/m^2). *p*, one-way ANOVA; *p* adj, ANCOVA adjusted for age; NWO − NWL, age-adjusted mean difference. Superscripts indicate significant post hoc differences ($p < 0.05$): a, vs. NWL; b, vs. NWO; c, vs. OW. baPWV excluding one extreme value in NWO: NWO − NWL = 135.7 cm/s (95% CI 36.8 to 234.6), $p = 0.009$. BMI, body mass index; SBP, systolic blood pressure; DBP, diastolic blood pressure; MAP, mean arterial pressure; PP, pulse pressure; baPWV, brachial–ankle pulse wave velocity; FMD, flow-mediated dilation; CSS, critical shear stress.

After adjustment for age, FMD was 2.38% lower in NWO than in NWL women (95% CI −3.99 to −0.76; $p = 0.005$; Hedges' g = −1.21), a value comparable to that of the OW and OB groups (Figure 3); this difference persisted after additional adjustment for BMI (−2.38%,

$p = 0.023$). baPWV tended to be higher in NWO than in NWL (adjusted difference 146.5 cm/s, 95% CI −12.2 to 305.2; $p = 0.069$); because the extreme baPWV value belonged to the NWO group, a sensitivity analysis excluding it was performed and showed a significant difference (135.7 cm/s, 95% CI 36.8 to 234.6; $p = 0.009$). In contrast, SBP, DBP, and MAP did not differ between NWO and NWL ($p = 0.117$–0.939) and were elevated only in the OB group. EI was similar in NWO and NWL ($p = 0.694$). The AI was higher in NWO than in NWL (5.2%, 95% CI 0.5 to 9.9; $p = 0.032$), but this difference was no longer significant after additional adjustment for BMI ($p = 0.319$) or with a 32% body fat threshold ($p = 0.322$). PP was lower in NWO than in NWL ($p = 0.012$). After Benjamini–Hochberg correction across the nine NWO-versus-NWL comparisons, the lower FMD ($q = 0.036$), higher baPWV (extreme value excluded; $q = 0.036$), and lower PP ($q = 0.036$) remained significant, whereas the higher aggregation index did not ($q = 0.072$), consistent with its attenuation after adjustment for BMI. At 20 women per group the hemorheological comparisons were adequately powered only for large effects: the observed between-group effect sizes were small for the elongation index (Cohen's $d = 0.22$; achieved power ≈ 10%) and critical shear stress ($d = 0.19$; ≈ 9%) and moderate for the aggregation index ($d = 0.63$; ≈ 49%), so these results should be read as inconclusive rather than as evidence of no difference, whereas the FMD difference was large (Hedges' $g = -1.21$) and adequately powered. The higher baPWV in NWO also persisted after additional adjustment for mean arterial pressure, with the extreme value excluded (127.8 cm/s, 95% CI 26.3 to 229.3; adjusted $p = 0.015$; $p = 0.154$ with the extreme value retained).

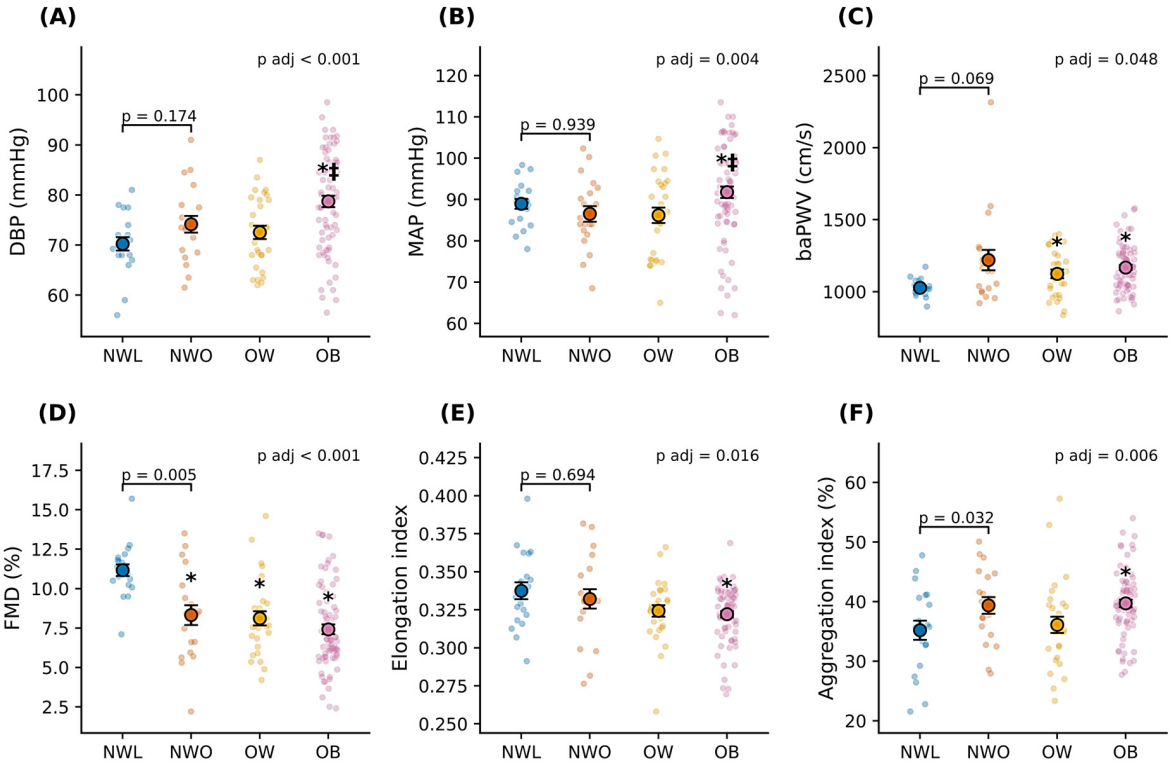


Figure 3. Vascular function and hemorheological properties according to adiposity phenotype. (**A**) DBP, (**B**) MAP, (**C**) baPWV, (**D**) FMD, (**E**) elongation index, and (**F**) aggregation index. Brackets show age-adjusted p values for NWO vs. NWL. Small symbols represent individual values; large symbols with error bars represent the mean ± standard error. * $p < 0.05$ vs. NWL; ‡ $p < 0.05$ vs. OW (post hoc test). NWL, normal-weight lean; NWO, normal-weight obesity; OW, overweight; OB, obese. Brackets denote age-adjusted NWO-versus-NWL comparisons; a high-resolution (600 dpi) version is provided for production.

3.5. Independent Associations of BMI and Body Fat

BMI and body fat percentage were strongly correlated (r = 0.81), but collinearity was acceptable (maximum variance inflation factor 3.03). When both were entered together with age (Table 5), body fat percentage, but not BMI, was independently associated with FMD (β = −0.30, 95% CI −0.56 to −0.05; $p = 0.019$), and showed borderline associations with baPWV (β = 0.20; $p = 0.082$) and AI (β = 0.24; $p = 0.085$). Conversely, BMI, but not body fat, was independently associated with DBP (β = 0.30; $p = 0.021$) and MAP (β = 0.30; $p = 0.023$). Neither BMI nor body fat was independently associated with EI or CSS, although EI and AI were correlated with both BMI and body fat in single-variable analyses (Table S2, Figure S1).

Table 5. Independent associations of BMI and body fat percentage with vascular function and hemorheological properties (N = 150).

Outcome	BMI		Body Fat (%)		R^2
	β (95% CI)	*p*	β (95% CI)	*p*	
SBP	0.25 (−0.02, 0.52)	0.071	−0.04 (−0.32, 0.24)	0.783	0.050
DBP	0.30 (0.05, 0.55)	0.021	0.14 (−0.12, 0.40)	0.291	0.172
MAP	0.30 (0.04, 0.56)	0.023	0.07 (−0.19, 0.34)	0.587	0.131
PP	0.04 (−0.24, 0.32)	0.775	−0.20 (−0.48, 0.09)	0.170	0.025
baPWV †	0.00 (−0.22, 0.23)	0.965	0.20 (−0.03, 0.43)	0.082	0.138
FMD	−0.02 (−0.27, 0.23)	0.874	−0.30 (−0.56, −0.05)	0.019	0.203
Elongation index	−0.08 (−0.35, 0.19)	0.543	−0.18 (−0.46, 0.10)	0.200	0.059
CSS	0.16 (−0.11, 0.44)	0.249	−0.01 (−0.29, 0.27)	0.930	0.026
Aggregation index	0.13 (−0.13, 0.39)	0.335	0.24 (−0.03, 0.50)	0.085	0.111

β, standardized regression coefficient from linear regression including BMI, body fat percentage, and age; R^2, coefficient of determination of the full model. The maximum variance inflation factor was 3.03. † One extreme value excluded (n = 149). BMI, body mass index; SBP, systolic blood pressure; DBP, diastolic blood pressure; MAP, mean arterial pressure; PP, pulse pressure; baPWV, brachial–ankle pulse wave velocity; FMD, flow-mediated dilation; CSS, critical shear stress.

4. Discussion

This study showed that the cardiovascular correlates of adiposity in women follow two distinct patterns. Endothelial function and arterial stiffness were related to body fat: they were already impaired in women with NWO, whose BMI was normal, and body fat percentage was associated with FMD independently of BMI; the lower FMD in NWO also persisted after adjustment for BMI. In contrast, blood pressure was related to BMI: it rose only in women with obesity and was independently associated with BMI rather than body fat. RBC deformability and aggregation worsened with increasing BMI; in NWO, deformability was preserved, and the higher aggregation index was not robust to adjustment for BMI.

The lower FMD observed in NWO extends earlier reports that NWO is accompanied by an adverse cardiometabolic profile [6,8,9]. The magnitude of the difference (−2.4 percentage points) is clinically relevant, because each 1% increase in FMD is associated with an approximately 13% lower risk of future cardiovascular events [13]. Adipose tissue expansion, even without weight gain, is associated with adipocyte hypertrophy, macrophage infiltration, and secretion of pro-inflammatory cytokines and adipokines that impair endothelial nitric oxide synthase activity and increase oxidative stress [15,16]. The lower fat-free mass in NWO women may also contribute, since skeletal muscle is a major site of glucose disposal and a source of vasoprotective myokines. Similarly, the higher baPWV in NWO suggests that structural or functional stiffening of the arterial wall may accompany excess adiposity before BMI increases [14]. This is consistent with a study in young women

in which adiposity, but not BMI-defined obesity, was associated with arterial stiffness [24], although that study found no association with FMD.

These findings can be situated within the small but growing literature on vascular function in NWO. A systematic review and meta-analysis linked NWO to higher blood pressure and an adverse lipid and glycemic profile relative to normal-weight lean status but did not examine endothelial function or arterial stiffness [8]; the present data extend that cardiometabolic picture to directly measured macrovascular function. Phillips et al. found that adiposity, but not BMI-defined obesity, was associated with arterial stiffness in young nulliparous women yet reported no association with FMD [24]; our larger sample and wider age range may explain why we additionally detected a body-fat-related impairment in FMD. That the FMD difference was consistent across age- and BMI-adjusted models and survived correction for multiple comparisons strengthens the inference that endothelial dysfunction in NWO reflects adiposity itself rather than confounding by body size or age.

In contrast, blood pressure was not elevated in NWO and was associated with BMI rather than body fat. Obesity-related hypertension is driven in part by expansion of blood volume and cardiac output, sympathetic activation, and renal sodium retention, all of which scale with total body mass [25], and in a large Asian cohort, blood pressure was more strongly related to general adiposity than to central adiposity [26]. Our findings therefore suggest that normal blood pressure should not be taken as evidence of low vascular risk in women with a normal BMI but high body fat, because endothelial dysfunction may precede blood pressure elevation. The lower PP in NWO likely reflects the relatively high SBP of the young NWL group and should be interpreted cautiously.

RBC deformability was lower and RBC aggregation higher with increasing BMI, consistent with previous studies in individuals with obesity [18,20]. Proposed mechanisms include increased plasma fibrinogen and acute-phase proteins, which promote aggregation, and oxidative damage and altered cholesterol content of the RBC membrane, which reduce deformability [17,21]. In NWO, RBC deformability was preserved, and the higher aggregation index was attenuated by adjustment for BMI and was sensitive to the body fat threshold. Together with the absence of an independent association with either BMI or body fat in mutually adjusted models, this suggests that hemorheological impairment reflects the overall burden of adiposity and may appear at a later stage than endothelial dysfunction, although a modest early increase in aggregation in NWO cannot be excluded. Because reduced deformability and enhanced aggregation increase microvascular flow resistance [17], they may compound the effects of endothelial dysfunction in women with obesity.

This study has several limitations. First, the cross-sectional design precludes causal inference. Second, the NWO and NWL groups were small (n = 20 each); NWO women were older than NWL women; and the 30% body fat threshold, although commonly used, is not universally standardized. Nevertheless, the FMD difference remained significant after adjustment for age and BMI and with a 32% threshold ($p = 0.010$). Third, the classification of NWO depends on the body fat threshold and on the precision of body fat estimation, and a single assessment may not capture day-to-day variability. Fourth, metabolic and inflammatory markers (e.g., lipids, glucose, insulin, and fibrinogen), menopausal status, physical activity, and medication use were not available and could not be adjusted for. Finally, all participants were Korean women, which limits generalizability to men and other populations. In addition, with only 20 women per group, the phenotype comparisons were underpowered for small between-group differences in hemorheological indices (minimum detectable standardized effect ≈ 0.91 at 80% power), so the preserved red blood cell deformability and the borderline aggregation findings in NWO require confirmation in larger

samples; the arterial-stiffness comparisons were, however, additionally adjusted for mean arterial pressure to limit hemodynamic confounding; and heart rate was not measured in the original protocol, so adjustment was limited to mean arterial pressure.

Nevertheless, this study is among the first to examine vascular function and hemorheological properties together in women with NWO and to separate the contributions of BMI and body fat. From a practical perspective, measuring body composition in addition to BMI may help identify women with normal BMI who already have impaired vascular function and who could benefit from interventions targeting fat loss and preservation of lean mass, such as combined exercise and dietary education. These observations should also be read within the broader lifestyle context of NWO, which commonly arises from a combination of chronic physical inactivity, sedentary behavior, and energy-dense, nutrient-poor dietary patterns that promote fat accumulation while sparing body weight [27]. This background reinforces the clinical value of assessing body composition and of targeting diet quality and physical activity—rather than body weight alone—in women with a normal BMI.

5. Conclusions

In this cross-sectional study of Korean women, higher body fat—rather than higher BMI—was associated with lower endothelial function and greater arterial stiffness, and these associations were already apparent in women with normal-weight obesity, whereas blood pressure tracked BMI. Because the design is observational, these associations require longitudinal confirmation, but they suggest that assessing body fat in addition to BMI may help identify women with a normal BMI who already have early vascular impairment.

Supplementary Materials: The following supporting information can be downloaded at https://www.mdpi.com/article/10.3390/metabo16120935/s1. Table S1: Age-adjusted vascular function and hemorheological properties according to BMI group; Table S2: Correlations of BMI and body fat percentage with vascular function and hemorheological properties (N = 150); Figure S1: Relationships of BMI with (A) DBP, (B) FMD, (C) elongation index, and (D) aggregation index (N = 150). Symbols indicate BMI groups; solid lines represent least-squares regression lines.

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Informed Consent Statement: Patient consent was waived because the study used anonymized existing data and no identifiable information was collected.

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Abbreviations

The following abbreviations are used in this manuscript:

BMI	body mass index
baPWV	brachial–ankle pulse wave velocity
CI	confidence interval
CSS	critical shear stress
DBP	diastolic blood pressure
EI	elongation index
FMD	flow-mediated dilation
MAP	mean arterial pressure
NWL	normal-weight lean
NWO	normal-weight obesity
OB	obese
OW	overweight
PP	pulse pressure
RBC	red blood cell
SBP	systolic blood pressure

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