

ORIGINAL RESEARCH

Difference in metabolic responses and hemorheological properties during submaximal aerobic exercise and recovery in nonobese and obese men

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Abstract

Background: Obesity is associated with metabolic dysregulation and impaired hemorheological function, which may affect oxygen delivery during exercise. However, how hemorheological properties change during submaximal exercise under controlled cardiovascular load remains unclear. **Objective:** This study compared hemorheological and metabolic responses during and after submaximal endurance exercise performed at a clamped heart rate (HR) in nonobese and obese Korean men. **Methods:** Twenty-four men participated: 12 in the obese group (OG; body mass index ≥ 25 kg/m² and body fat percentage $\geq 25\%$) and 12 nonobese (NG) according to Korean adult obesity criteria. Participants completed 30 min of cycling at 75% of maximal HR, followed by 30 min of recovery. Metabolic and cardiorespiratory variables (relative and absolute oxygen uptake (VO₂), relative and absolute carbon dioxide production (VCO₂), ventilation, respiratory exchange ratio, blood lactate), substrate utilization (carbohydrate oxidation, fat oxidation, energy expenditure), and hemorheological parameters (red blood cell (RBC) aggregation index, RBC deformability at 3 Pa) were measured at baseline, during exercise, and during recovery. **Results:** Despite comparable HR and workload, relative VO₂ and VCO₂ were significantly lower in the OG during exercise ($p < .001$), whereas absolute VO₂ and VCO₂ showed no group differences. Ventilation, respiratory exchange ratio, blood lactate, and substrate utilization showed no group differences. The OG exhibited consistently RBC aggregation index and lower RBC deformability at 3 Pa across all time points ($p < .01$). Time-dependent changes in hemorheological variables were small, with only transient increases in AI observed. **Conclusions:** Under clamped HR submaximal exercise, individuals with obesity show lower oxygen consumption despite similar ventilatory and substrate utilization responses. Persistent hemorheological impairments in obesity may restrict peripheral oxygen delivery, contributing to reduced metabolic responses under equivalent cardiovascular load.

Keywords: obesity, aerobic exercise, energy metabolism, energy substrate, hemorheology

Introduction

Obesity is defined as a chronic multifactorial disease characterized by excessive fat accumulation that impairs health and serves as a primary risk factor for cardiovascular disease and diabetes (World Health Organization, 2024). With global prevalence rising, projections estimate that approximately one billion adults will be living with obesity by 2030 (Barata Cavalcanti et al., 2022). Obesity is a major public health socioeconomic burden (Deng et al., 2025). Therefore, a comprehensive understanding of the physiological characteristics associated with obesity is essential for effective management.

Adipose tissue acts as an endocrine organ, secreting pro-inflammatory cytokines (Purdy & Shatzel, 2021). Obesity promotes adipose tissue expansion, increased immune cell infiltration into adipose tissue (Piché et al., 2020), and

elevated circulating leukocyte levels (Purdy & Shatzel, 2021), thereby contributing to a state of chronic low-grade inflammation (Gregor & Hotamisligil, 2011; Hotamisligil, 2006). This inflammatory milieu disrupts insulin signaling, leading to insulin resistance and impaired metabolic homeostasis (Saltiel & Kahn, 2001).

Furthermore, altered adipose function elevates free fatty acids liberation (Hotamisligil, 2006), inducing metabolic inflexibility – a reduced capacity to shift between carbohydrate and fat oxidation (Galgani & Ravussin, 2008; Kelley & Mandarino, 2000). Beyond substrate utilization, obesity-related inflammation and oxidative stress impair mitochondrial and endothelial function, contributing to microvascular dysfunction (Purdy & Shatzel, 2021).

Excessive visceral fat enhances lipolysis and free fatty acids release, which may induce lipotoxicity and reduced

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vascular responsiveness (Boden, 2008). Additionally, elevated coagulation factors and platelet activation in obesity influence blood flow and the microcirculatory environment. Collectively, these alterations drive a circulatory milieu characterized by combined changes in vascular function and the physical properties of blood. Under such circumstances, impaired oxygen and energy substrate delivery may not only reflect alterations in vascular and microvascular environments but also involve changes in the intrinsic physical properties of blood, potentially accompanied by hemorheological alterations such as reduced red blood cell (RBC) deformability and increased aggregation (Meiselman & Baskurt, 2003; Popel & Johnson, 2005).

Indeed, previous studies have reported that obese individuals exhibit hemorheological impairments at rest, including diminished RBC deformability and enhanced RBC aggregation (Tripolino et al., 2017), and that these changes are associated with increased flow resistance and microcirculatory disturbances (Wiewiora et al., 2007). Hemorheological function is determined by parameters closely related to blood flow properties, such as blood viscosity, RBC deformability, and RBC aggregation, and is considered an important determinant of flow resistance and oxygen transport efficiency within the vascular and microcirculatory systems (Nader et al., 2019). These obesity-related metabolic and circulatory abnormalities may become more pronounced not only at rest but also under conditions of increased physiological demand (Brun et al., 2007).

Exercise provides a representative physiological stimulus that simultaneously challenges energy metabolism and cardiovascular function and can thus serve as a useful model for evaluating obesity-related functional characteristics. Regular exercise training has been reported to improve hemorheological function by enhancing RBC deformability and reducing RBC aggregation, thereby inducing long-term improvements in vascular function (Brun, 2002; Romain et al., 2011). In contrast, acute bouts of aerobic exercise have been shown to transiently increase whole blood viscosity, augment RBC aggregation, and reduce RBC deformability, indicating that exercise-induced hemorheological responses may vary depending on the time course and specific exercise conditions (Connes et al., 2013). Furthermore, hemorheological characteristics associated with obesity have shown inconsistent findings across studies, and such discrepancies may be attributable to obesity heterogeneity as well as methodological differences, including assessment indices, exercise protocols, and measurement timing (Connes et al., 2013; Craveri et al., 1988; Romain et al., 2011).

Nevertheless, studies that have examined time-dependent hemorheological responses during submaximal endurance exercise in obese individuals remain scarce. In particular, although hemorheological variables can change progressively during exercise and may persist or be delayed during the post-exercise recovery period, many previous investigations have focused primarily on changes observed at a single time point immediately after exercise (O'Rourke, 2009). However, such a limited approach fails to capture the dynamic pattern of exercise-induced hemorheological responses and overlooks potential alterations during recovery.

The present study hypothesized that the time-course of hemorheological and metabolic responses during submaximal endurance exercise and the subsequent recovery period at an identical heart rate level would differ between obese and nonobese individuals. Therefore, we investigated and compared the time-course changes in hemorheological and metabolic responses during submaximal endurance exercise and the subsequent recovery period in obese and nonobese individuals at an identical heart rate level.

Methods

Participants

An a priori sample size calculation was conducted using G*Power to determine the minimum number of participants required for the repeated-measures design of the present study. Based on the primary outcome variable, erythrocyte aggregation, an effect size of $f = 0.503$ was estimated from a previous study (Bizjak et al., 2018) using Cohen's method (Shukla et al., 2005). The analysis was performed with an alpha level of .05, a desired statistical power of .90, two groups, and seven repeated measurements. The estimated minimum total sample size was 20 participants (10 per group). To account for potential dropouts, 24 participants were ultimately recruited, with 12 participants assigned to each group. A total of 24 adult men aged 20–30 years participated in this study. The physical characteristics of the participants are presented in Table 1. The inclusion criteria were as follows: (1) no regular participation in structured exercise within the previous six months, (2) absence of known physical or psychological disorders and no history of chronic disease, and (3) voluntary agreement to participate in the study with written informed consent provided prior to enrollment. The exclusion criteria were: (1) a history of orthopedic injury within the previous year; (2) a history of severe cerebrovascular disease, cardiovascular disease, endocrine disorders, or psychiatric disorders; (3) uncontrolled hypertension (blood pressure $\geq 160/100$ mmHg); and (4) any conflict of interest with the investigators.

Participants who met all inclusion and exclusion criteria were assigned to either a nonobese group (NG; $n = 12$) or an obese-classified group (OG; $n = 12$) according to Korean adult obesity criteria (Nam et al., 2021). Specifically, participants with both a body mass index (BMI) < 25 kg/m² and body fat percentage $< 25\%$ were assigned to the NG, whereas those with both a BMI ≥ 25 kg/m² and body fat percentage $\geq 25\%$ were assigned to the

Table 1 Participants' characteristics

Characteristics	Nonobese group ($n = 12$)	Obese group ($n = 12$)
Age (years)	21.25 $\pm$ 2.38	23.33 $\pm$ 2.15
Body height (cm)	175.42 $\pm$ 5.18	176.75 $\pm$ 5.03
Body mass (kg)	69.26 $\pm$ 6.36	91.80 $\pm$ 10.08*
Body mass index (kg/m ²)	22.48 $\pm$ 1.36	29.37 $\pm$ 2.83*
Percentage body fat (%)	15.33 $\pm$ 3.51	26.33 $\pm$ 4.62*
Lean body mass (kg)	55.38 $\pm$ 3.87	63.90 $\pm$ 6.35*

Note. Values are presented as means and standard deviations. * $p < .05$ nonobese vs. obese group.

OG. This classification was based on Korean population-specific obesity criteria, which adopt a lower BMI threshold ($\geq 25 \text{ kg/m}^2$) than the WHO international standard ($\geq 30 \text{ kg/m}^2$), reflecting the higher cardiometabolic risk observed at lower BMI values in Asian populations. Participants with discordant classifications (e.g., BMI $\geq 25 \text{ kg/m}^2$ but body fat percentage $< 25\%$, or vice versa) were excluded from the study. A total of 38 potential participants underwent pre-screening, of whom 14 met the criteria for the NG, and 13 met the criteria for the OG. The remaining 11 participants were excluded: 7 due to discordant BMI and body fat percentage classifications, and 4 due to uncontrolled hypertension (blood pressure $\geq 160/100 \text{ mmHg}$). From the eligible candidates, 12 participants from each group who were able to comply with the experimental schedule were finally recruited, resulting in a total of 24 participants.

Following a detailed explanation of the experimental procedures and potential risks, all participants provided written informed consent prior to the start of the study. The study protocol was approved by the Institutional Review Board of Konkuk University (approval number: 7001355-202405-HR-804) and was registered with the Clinical Research Information Service (CRIS; KCT0010783). All procedures were conducted in accordance with the principles outlined in the Declaration of Helsinki.

Study design

The overall study design is illustrated in Figure 1. During a 30-min bout of submaximal exercise and the subsequent 30-min recovery period, metabolic and cardiorespiratory responses, substrate oxidation, and hemorheological responses were assessed at 10-min intervals.

To ensure standardized testing conditions, participants were instructed to refrain from vigorous physical activity for 48 hours prior to testing, to fast for at least 8 hours, and to obtain a minimum of 7 hours of sleep before reporting to the laboratory. Upon arrival, all participants rested quietly for a standardized period, followed by light stretching to minimize the risk of musculoskeletal injury during exercise. Baseline measurements were obtained in a seated position

immediately before the start of exercise, after participants had been instrumented for physiological and gas exchange measurements. All testing sessions were conducted in controlled laboratory conditions at a consistent time of day, with ambient temperature and humidity maintained at approximately $20 \pm 2^\circ\text{C}$ and $60\% \pm 2\%$, respectively.

The exercise protocol was performed on a cycle ergometer (Aero bike 75XLII; Konami Corporation) under a clamped heart rate exercise condition, in which external workload was continuously adjusted to maintain a target heart rate corresponding to 75% of age-predicted maximal heart rate. Heart rate was continuously monitored during exercise using an earlobe heart rate sensor integrated into the cycle ergometer, which was attached to the participant's earlobe. Maximal heart rate (HR_{max}) was estimated using the Miyashita formula for men (HR_{max} = $209 - 0.69 \times \text{age}$; Miyashita et al., 1985). Participants exercised at the target heart rate for 30 min, after which they remained seated for an additional 30 min while physiological responses were monitored during recovery. Venous blood samples (3 mL) were collected by an experienced nurse at the designated time points throughout exercise and recovery.

Measurements

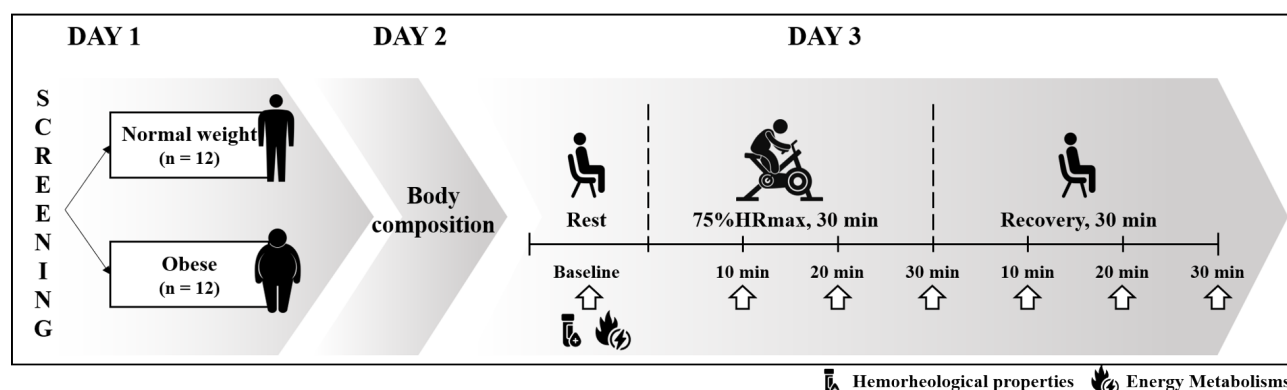
Body composition

Anthropometric and body composition measurements were obtained using a stadiometer (BSM 330, InBody) and a bioelectrical impedance analyzer (InBody 770, InBody). Height (cm), body mass (kg), fat mass (kg), body fat percentage (%), and lean body mass (kg) were measured, and BMI (kg/m^2) was calculated from height and body mass. All measurements were performed with participants wearing light clothing, barefoot, and with all metallic accessories removed.

Mean exercise load during submaximal exercise

During the 30-min bout of submaximal exercise performed at 75% of maximal heart rate, mean exercise load (in Watts) was automatically calculated by the cycle ergometer (Aero bike 75XLII, Konami). This variable was

Figure 1 Study design. The schematic illustrates the overall study design and experimental timeline. After baseline measurements, participants performed 30 min of submaximal cycling exercise at 75% of maximal heart rate (HR_{max}), followed by 30 min of recovery. Energy metabolism variables and hemorheological parameters were assessed at baseline and at 10-min intervals during exercise and recovery in the normal-weight group and obese group.



used to quantify the average external workload required to maintain the target heart rate during the clamped heart rate exercise condition.

Metabolic and cardiorespiratory responses

During the 30-min submaximal exercise and the subsequent 30-min recovery period, oxygen uptake (VO_2 , relative in mL/min/kg and absolute in L/min), carbon dioxide production (VCO_2 , relative in mL/min/kg and absolute in L/min), minute ventilation (VE, L/min), and respiratory exchange ratio (RER) were measured using a breath-by-breath automated gas analyzer (K5, Cosmed). Blood lactate concentration was determined from fingertip capillary blood (0.3 μL) using a portable lactate analyzer (Lactate Pro2, Arkra). All metabolic and cardiorespiratory variables were analyzed at baseline, during exercise at 10, 20, and 30 min, and during recovery at 10, 20, and 30 min.

Substrate oxidation

Rates of substrate oxidation during exercise and recovery were calculated from absolute VO_2 and absolute VCO_2 values obtained with the automated gas analyzer (K5, Cosmed). Carbohydrate oxidation (CHO), fatty acid oxidation (FAO), and energy expenditure (EE) were estimated using the equations proposed by Kelly and Basset (2017):
 $\text{CHO (g/min)} = 4.210 \times \text{VCO}_2 \text{ (L/min)} - 2.962 \times \text{VO}_2 \text{ (L/min)}$
 $\text{FAO (g/min)} = 1.695 \times \text{VO}_2 \text{ (L/min)} - 1.701 \times \text{VCO}_2 \text{ (L/min)}$
 $\text{EE (kcal/min)} = 4.07 \times \text{CHO (g/min)} + 9.75 \times \text{FAO (g/min)}$

All variables were calculated at baseline, during exercise at 10, 20, and 30 min, and during recovery at 10, 20, and 30 min.

Hemorheological variables

RBC deformability and aggregation during exercise and recovery were assessed using a hemorheometer (RheoSCAN-AnD300, Rheo Meditech Inc.). Blood samples were collected at baseline, during exercise at 10, 20, and 30 min, and during recovery at 10, 20, and 30 min, and were stored in tubes containing K_3EDTA as an anticoagulant. All samples were analyzed within 2 hours of collection. Measurements were performed at a temperature of 25 °C under a standardized shear stress of 3 Pa, in accordance with the manufacturer's recommendations.

For the assessment of RBC deformability, 6 μL of whole blood was mixed with 600 μL of polyvinylpyrrolidone solution and introduced into a disposable test kit (RSD-K02, Rheo Meditech Inc.). The device generates a pressure difference across a microchannel, forcing the blood sample to flow through a narrow capillary. As the differential pressure gradually decreases during flow, RBCs undergo progressive deformation from a prolate ellipsoid shape toward a biconcave discoid shape. A laser beam is directed through the flowing blood, and the diffraction pattern produced by the deformed RBCs is recorded by a video camera and analyzed to quantify cell deformability, as previously described. The elongation index (EI) is calculated from the major (L) and minor (W) axes of the cell according to the formula $\text{EI} = (\text{L} - \text{W})/(\text{L} + \text{W})$, and values are expressed as a percentage (RBC deformability at 3 Pa, EI_3Pa).

For the assessment of RBC aggregation, 8 μL of whole blood was applied to a specific aggregation test chip (RSA-C01, Rheo Meditech Inc.). The device first subjects the blood sample to high shear using an internal stirrer for 10 seconds to completely disperse RBC aggregates, after which stirring is abruptly stopped. Under these low-shear conditions, RBCs exhibit an immediate tendency to re-aggregate. The intensity of light transmitted through the blood sample is then recorded over time, and the time-dependent changes in light intensity are analyzed to quantify the degree of RBC aggregation (AI; %).

Statistical analysis

All data collected in this study were analyzed using IBM SPSS Statistics (Version 28; IBM Corp.). Descriptive statistics for all dependent variables are presented as means and standard deviations. Prior to analysis, the normality of the data distribution was verified using the Shapiro–Wilk test. To examine differences in dependent variables between groups and across time points, a two-way analysis of variance with repeated measures (group $\times$ time) was performed. When a significant main effect or interaction effect was observed, independent-samples t-tests were conducted to compare groups at each time point, and one-way repeated-measures analyses of variance were applied to examine within-group changes over time, followed by Bonferroni post hoc tests to identify specific time points at which significant differences occurred. The level of statistical significance was set at $p < .05$ for all analyses. Effect size was calculated as partial eta-squared (η_p^2), with values of $\geq .01$, $\geq .06$, and $\geq .14$ interpreted as small, medium, and large effects, respectively.

Results

Heart rate and exercise intensity during submaximal exercise

Heart rate and exercise intensity during submaximal exercise in both groups are presented in Figure 2. As the two groups were of similar age and exercised at the same relative intensity calculated from the same prediction equation, no statistically significant differences were observed in heart rate or exercise intensity between groups.

Metabolic and cardiorespiratory response

Metabolic and cardiorespiratory responses at baseline, during exercise, and during recovery are presented in Figure 3. Relative VO_2 and relative VCO_2 showed significant time $\times$ group interaction effects ($p < .001$) as well as significant main effects of time ($p < .001$) and group ($p < .001$). In contrast, absolute VO_2 , absolute VCO_2 , VE, RER, and blood lactate concentration showed a significant main effect of time only ($p < .001$), with no significant main effect of group or time $\times$ group interaction effect.

Post hoc analyses revealed that, in the NG, relative VO_2 , relative VCO_2 , and VE were significantly higher at 10, 20, and 30 min of exercise compared with baseline ($p < .007$). RER was significantly higher at 10 min of exercise compared with baseline ($p < .007$), and lactate concentration

was significantly elevated at 10, 20, and 30 min of exercise and at 10 min of recovery relative to baseline ($p < 0.007$). In the OG, relative $\dot{V}O_2$, relative $\dot{V}CO_2$, and VE were significantly increased at 10, 20, and 30 min of exercise compared with baseline ($p < .007$), and significantly decreased at 20 and 30 min of recovery compared with the end of exercise ($p < .007$). RER was significantly higher at 10, 20, and 30 min of exercise compared with baseline ($p < 0.007$), and significantly lower at 30 min of recovery than at baseline ($p < .007$). Lactate concentration was significantly higher at all exercise and recovery time points compared with baseline ($p < .007$).

For absolute $\dot{V}O_2$ and absolute $\dot{V}CO_2$, post hoc analyses showed that both groups exhibited significantly higher values at 10, 20, and 30 min of exercise compared with baseline ($p < .001$). In the OG, absolute $\dot{V}O_2$ and absolute $\dot{V}CO_2$ remained significantly elevated at 20 and 30 min of recovery compared with baseline ($p < .001$), whereas no significant differences were observed during recovery in the NG.

Between-group comparisons showed that relative $\dot{V}O_2$ was significantly lower in the OG than in the NG at 10 min ($p < .01$), 20 min ($p < .001$), and 30 min ($p < .001$) of exercise and at 20 min of recovery ($p < .05$). Similarly, relative $\dot{V}CO_2$ was significantly lower in the OG than in the NG at 10 min ($p < .01$), 20 min ($p < .001$), and 30 min ($p < .001$) of exercise. No significant between-group differences were observed for absolute $\dot{V}O_2$ or absolute $\dot{V}CO_2$ at any time point.

Substrate oxidation responses

Substrate oxidation responses at baseline, during exercise, and during recovery are presented in Figure 4. All substrate-related variables showed significant main effects of time ($p < .001$), whereas time $\times$ group interaction effects were not significant.

Post hoc analyses indicated that, in the NG, CHO and EE were significantly higher at 10, 20, and 30 min of exercise compared with baseline ($p < .007$), and FAO was significantly higher at 20 and 30 min of exercise than at baseline ($p < .007$). In the OG, CHO was significantly increased at 10, 20, and 30 min of exercise compared with baseline ($p < .007$), and significantly decreased at 30 min of recovery ($p < .007$). FAO was significantly higher at 20

and 30 min of exercise compared with baseline ($p < .007$). EE was significantly increased at 10, 20, and 30 min of exercise compared with baseline ($p < .007$), and significantly decreased at 20 and 30 min of recovery ($p < .007$).

At no time point were there statistically significant differences between groups in CHO, FAO, or EE.

Hemorheological responses

Hemorheological responses at baseline, during exercise, and during recovery are presented in Figure 5. The AI showed a significant time $\times$ group interaction ($p = .011$), as well as significant main effects of time ($p < .001$) and group ($p = .002$). In contrast, EI_3Pa showed a significant main effect of group only ($p < .001$), with no significant main effect of time or time $\times$ group interaction.

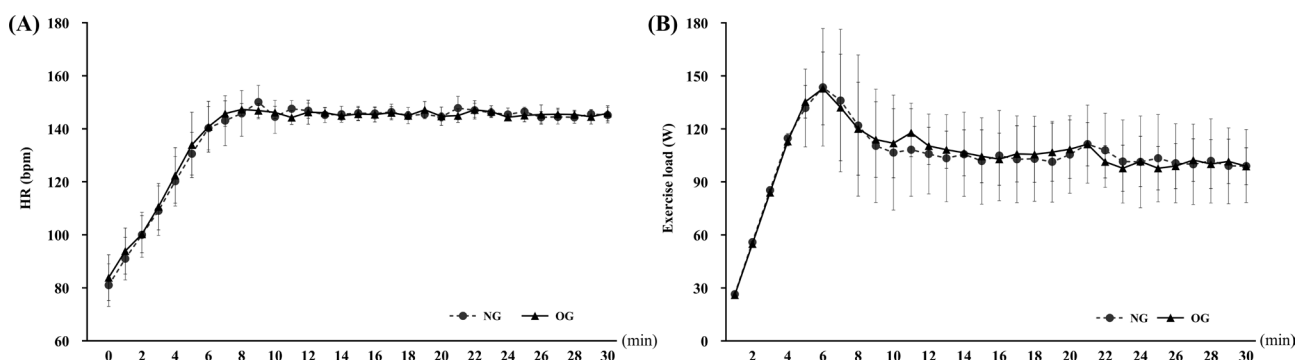
Post hoc analyses showed that, in the NG, AI was significantly higher at 10 and 20 min of exercise and at 10 min of recovery compared with baseline ($p < .007$). In the OG, AI was significantly higher than baseline only at 10 min of exercise ($p < .007$).

Between-group comparisons revealed that AI was significantly higher in the OG than in the NG at baseline ($p < .001$), at 10, 20, and 30 min of exercise ($p < .05$), and at 10, 20, and 30 min of recovery ($ps < .05$, with $p < .001$ at 20 min of recovery). In addition, EI_3Pa was significantly lower in the OG than in the NG at all time points ($p < .001$).

Discussion

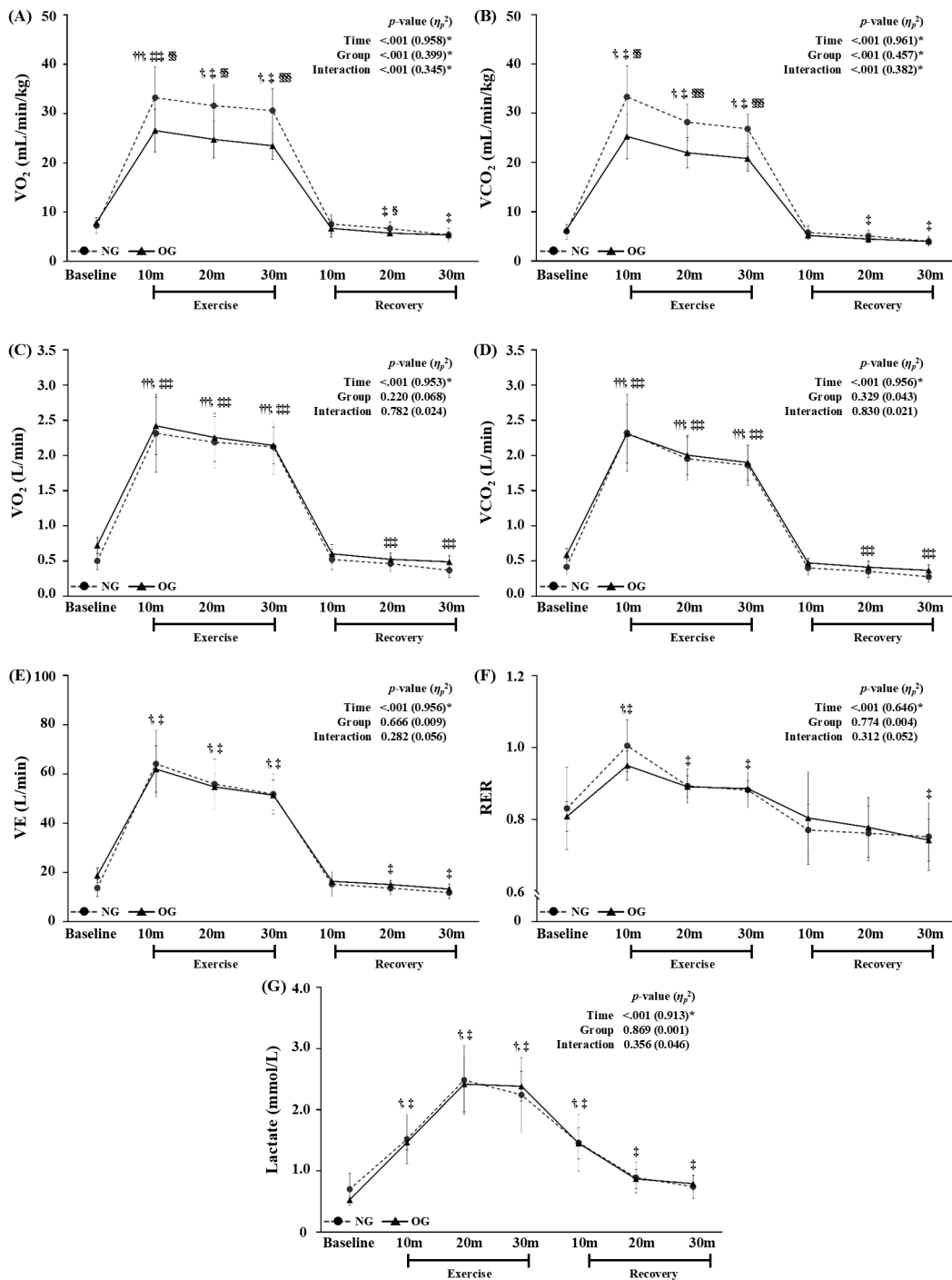
The present study compared temporal changes in hemorheological properties and energy metabolic responses during and after submaximal endurance exercise performed under a clamped heart rate exercise (75% HRmax). The heart rate-clamped exercise protocol was successfully implemented, and heart rate and external work rate were maintained at comparable levels between the two groups during submaximal exercise, confirming that cardiovascular load was effectively controlled across groups (Burger, 1969; Li et al., 2023). Within this controlled physiological context, hemorheological variables exhibited the most consistent between-group differences across baseline, exercise, and recovery, indicating that these variables may represent a

Figure 2 Heart rate and exercise intensity during submaximal exercise. (A) Heart rate (responses during submaximal exercise. (B) Exercise load during submaximal exercise.



Note. HR = heart rate; bpm = beats/minute; NG = nonobese group; OG = obese group. Data are presented as means and standard deviations.

Figure 3 Metabolic and cardiorespiratory responses. (A) Oxygen uptake normalized to body mass. (B) Carbon dioxide production normalized to body mass. (C) Absolute oxygen uptake; (D) Absolute carbon dioxide production. (E) Minute ventilation. (F) Respiratory exchange ratio. (G) Blood lactate concentration.



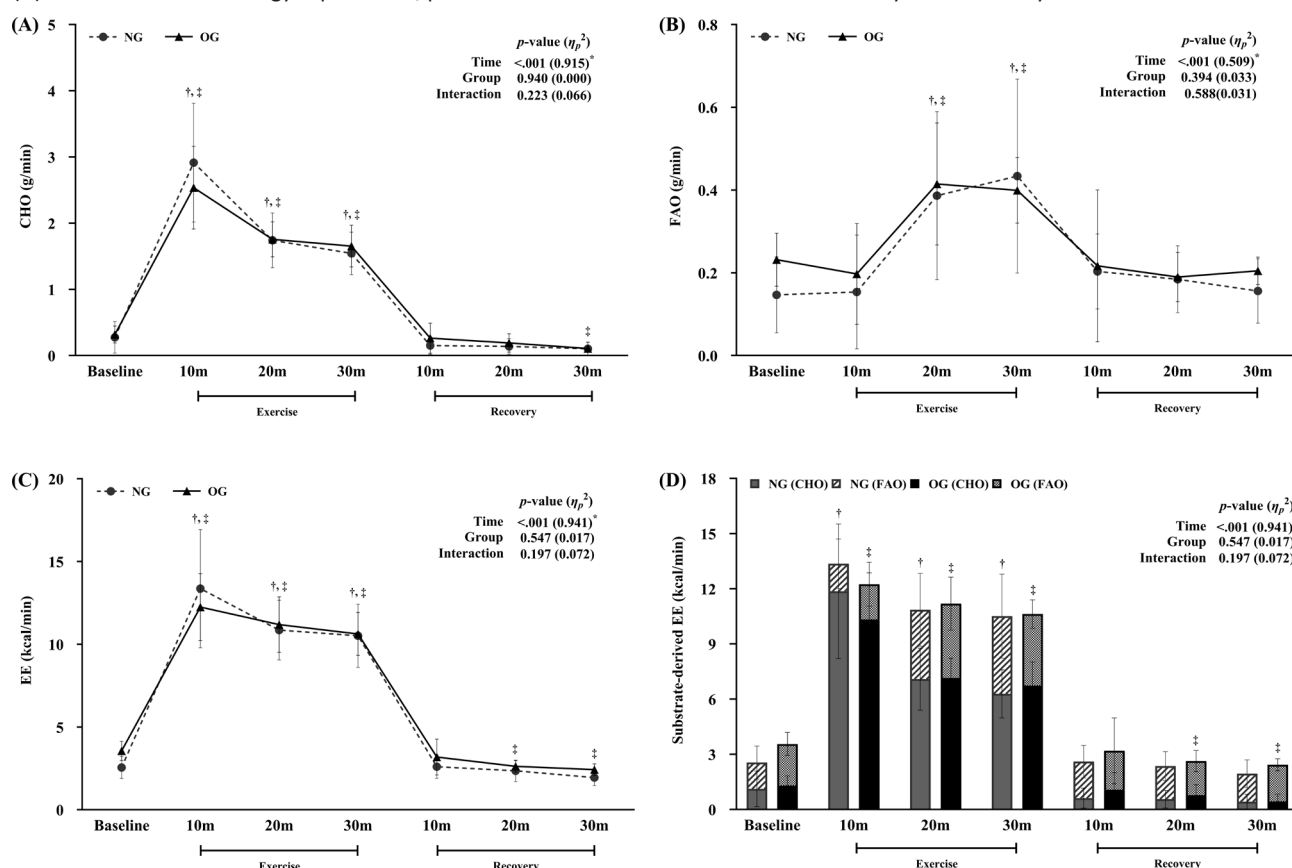
Note. NG = nonobese group; OG = obese group; VO_2 = oxygen consumption; VCO_2 = carbon dioxide production; VE = minute ventilation; RER = respiratory exchange ratio. Data are presented as means and standard deviations. * $p < .05$ significant interaction or main effect. $^{\dagger}p < .007$ significant differences compared between baseline and terminal time within normal group by Bonferroni. $^{\ddagger}p < .007$ significant differences compared between baseline and terminal time within OG (Bonferroni). $^{\S}p < .05$, $^{\S\S}p < .01$, $^{\S\S\S}p < .001$ for NG vs. OG.

robust physiological distinction between obese and non-obese individuals.

During exercise, relative VO_2 and relative VCO_2 were significantly lower in the OG than in the NG. However, as absolute VO_2 and absolute VCO_2 did not differ significantly between groups, these differences should be interpreted primarily in the context of body mass normalization

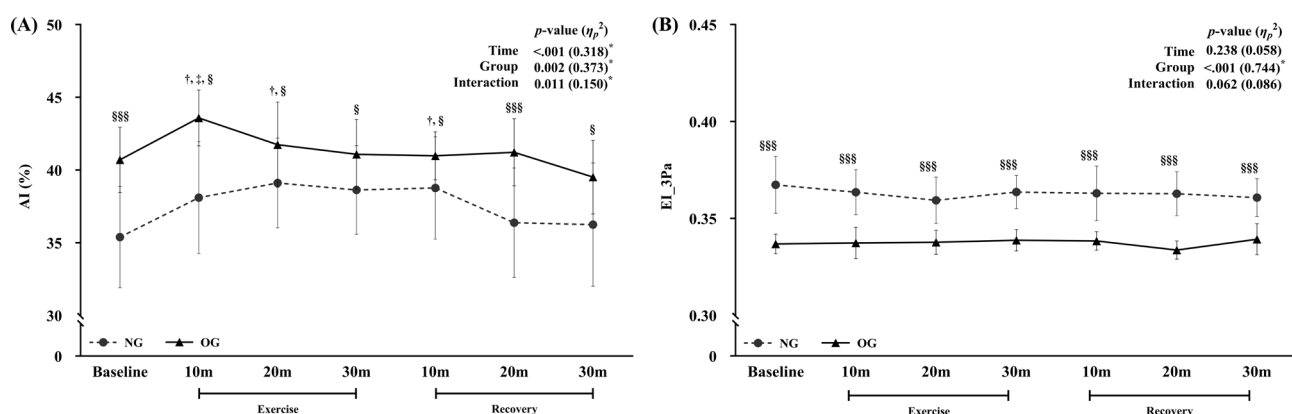
rather than as reflecting impaired metabolic capacity or oxygen utilization. In contrast, during the recovery period, relative VO_2 in the OG was significantly lower only at 20 min of recovery, with no group differences observed at any other time point. Considering that maximal oxygen uptake in obesity can be comparable to that of normal-weight individuals (Salvadori et al., 1992, 2004), the VO_2 differences

Figure 4 Energy substrate utilization. (A) Carbohydrate oxidation (CHO). (B) Fatty acid oxidation (FAO). (C) Total energy expenditure. (D) Substrate-derived energy expenditure, presented as stacked contributions from carbohydrate and fatty acid oxidation.



Note. NG = nonobese group; OG = obese group. Data are presented as means and standard deviations. * $p < .05$ significant interaction or main effect. † $p < .007$ significant differences compared between baseline and terminal time within NG (Bonferroni). ‡ $p < .007$ significant differences compared between baseline and terminal time within OG (Bonferroni).

Figure 5 Hemorheological properties. (A) Red blood cell aggregation index. (B) Red blood cell elongation index measured at a shear stress of 3 Pa.



Note. NG = nonobese group; OG = obese group. Data are presented as means and standard deviations. * $p < .05$ significant interaction or main effect. † $p < .007$ significant differences compared between baseline and terminal time within NG (Bonferroni); ‡ $p < .007$ significant differences compared between baseline and terminal time within OG (Bonferroni). § $p < .05$, §§ $p < .01$, §§§ $p < .001$ for NG vs. OG.

observed reflect differences in relative metabolic load under the same heart rate response.

In other words, an identical heart rate response does not necessarily imply an identical metabolic response between groups, suggesting that heart rate alone may not fully reflect the physiological demands of exercise in individuals with different body composition characteristics. Notably,

when VO_2 and VCO_2 were expressed in absolute terms, no significant between-group differences were observed, and only time-dependent changes were evident. This suggests that the observed group differences were primarily apparent when these variables were normalized to body mass. Accordingly, the lower relative VO_2 and VCO_2 observed in the OG should be interpreted with consideration of body

mass-related normalization rather than as an isolated indication of impaired whole-body metabolic capacity.

Previous studies have reported that, when exercising at the same absolute intensity, obesity exhibits higher oxygen consumption and lower heart rate compared with normal-weight individuals (Cavuoto & Maikala, 2016), which has been attributed to the additional energy cost associated with moving a greater body mass (Ehlen et al., 2011; Salvadori et al., 2004). In the present study, however, the application of a clamped heart rate protocol effectively controlled cardiovascular stress, enabling a precise comparison of metabolic responses.

Despite between-group differences in relative $\dot{V}O_2$ and $\dot{V}CO_2$, no significant differences were observed in VE, RER, or blood lactate concentration. This pattern is consistent with the absence of group differences in CHO, FAO, and EE, and suggests that metabolic regulation and substrate utilization were similar between groups. According to previous studies, obese individuals are able to maintain ventilatory output during exercise at a level comparable to that of normal-weight individuals, but may require a greater neural respiratory drive to do so (Babb, 2013; DeLorey et al., 2005). In the present study, the absence of differences in VE implies that while ventilatory response was similar, the mechanisms in ventilatory control may differ. Blood lactate is influenced by external exercise load and the degree of glycolytic pathway activation (Gladden, 2004). Since external workload was similar, it is plausible that substrate utilization and glycolytic engagement followed a similar pattern. Accordingly, the absence of group differences in blood lactate concentration and RER can be interpreted as reflecting comparable reliance on glycolytic metabolism and substrate use and suggests that the exercise intensity employed in this study was a primary determinant of the metabolic response.

In terms of substrate utilization, obesity exhibits reduced metabolic flexibility and a greater reliance on lipid oxidation during exercise under low-to-moderate intensity exercise conditions (Brooks & Jacques Mercier, 1994; Kelley & Mandarino, 2000). In the present study, carbohydrate oxidation increased, whereas fat oxidation increased only during later exercise, irrespective of obesity status. The predominance of carbohydrate is consistent with the intensity applied (75% HRmax; Brooks & Jacques Mercier, 1994), and this relatively high intensity may have attenuated group differences in substrate utilization. This suggests that metabolic responses in obesity are strongly influenced by intensity and, more broadly, by the mode of exercise prescription.

Although the overall metabolic responses were largely similar between groups, hemorheological indices showed the clearest and most consistent between-group differences across the experimental period. The OG consistently exhibited higher AI and lower EI_{3Pa} at all time points. These findings indicate that unfavorable hemorheological properties persist regardless of acute exercise or recovery status. Temporal changes in hemorheological parameters were limited. EI_{3Pa} did not differ significantly from baseline at any time point in either group during exercise

or recovery, suggesting that the exercise was not sufficient to induce acute structural alterations in RBC deformability (Brun et al., 1998; Simmonds et al., 2013; Triplette et al., 2011). In contrast, AI transiently increased during the early phase of exercise, and an additional transient increase was observed at 10 min of recovery in the NG. This is more indicative of acute hemodynamic transitions than of a time-dependent hemorheological adaptation throughout the exercise bout (El-Sayed et al., 2011; Meiselman & Baskurt, 2003). The more limited temporal variation observed in the OG may be related to the already elevated baseline level of AI, which could limit the magnitude of further exercise-induced changes.

Hemorheological properties are closely related to the microcirculatory environment for blood flow and oxygen delivery (Meiselman & Baskurt, 2003). Increases in AI can elevate flow resistance under low-shear conditions and impair capillary perfusion, whereas reductions in EI_{3Pa} can diminish the ability of erythrocytes to traverse narrow capillaries efficiently, thereby acting unfavorably on tissue oxygenation (Popel & Johnson, 2005). The persistently unfavorable hemorheological profile observed in the OG in the present study may therefore have imposed constraints on oxygen delivery at the level of the peripheral circulation. Under such constrained peripheral conditions, it is conceivable that the increase in oxygen consumption required to maintain a given heart rate response was relatively attenuated in obesity, resulting in the lower relative $\dot{V}O_2$ observed despite comparable heart rate responses.

In this context, the lower relative $\dot{V}O_2$ observed in the OG may reflect differences in oxygen demand relative to body mass under matched cardiovascular load, rather than a difference in absolute oxygen consumption per se. Thus, the observed differences in metabolic demand are more appropriately interpreted not as evidence of reduced aerobic capacity, but as reflecting distinct peripheral oxygen delivery environments under identical heart rate responses between groups. Although muscle oxygenation and oxygen extraction were not directly assessed in this study, hemorheological properties can act as important physical determinants of peripheral oxygen transport.

Limitations

Despite the use of an a priori power analysis and a repeated-measures design, the present study included a relatively modest sample size, which may limit the generalizability of the findings. In addition, exercise intensity was prescribed using an age-predicted maximal heart rate equation rather than directly measured maximal heart rate or heart rate reserve, which may have introduced some variability in the relative exercise intensity across participants. Furthermore, central cardiovascular variables such as stroke volume and cardiac output were not assessed, limiting the ability to fully interpret the cardiovascular mechanisms underlying the observed responses.

The present study also did not adjust oxygen consumption for body composition variables such as fat-free mass or lean body mass, which may influence the interpretation of group differences in metabolic responses. Finally, given

the cross-sectional and acute experimental design, causal relationships between hemorheological properties and metabolic responses cannot be established. Future studies with larger and more diverse samples, as well as more comprehensive physiological measurements, are warranted to confirm and extend these findings.

Conclusions

In summary, the most consistent finding of the present study was that the obese group exhibited persistently altered hemorheological properties, characterized by greater red blood cell aggregation and lower deformability, across baseline, exercise, and recovery. Even when heart rate was kept the same during exercise, the obese group exhibited lower oxygen consumption relative to body weight than the normal-weight group, whereas absolute oxygen consumption, breathing responses, and substrate use remained largely similar.

These features may reflect a less favorable peripheral environment for oxygen delivery to working muscles and may be associated with the lower relative oxygen consumption observed under heart-rate-matched conditions. From a practical standpoint, this means that heart rate alone may not fully represent the actual metabolic and circulatory demands experienced by obesity during aerobic exercise. Considering additional physiological indicators may therefore help make exercise prescriptions more accurate and appropriate for this population.

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Conflict of interest

The authors report no conflict of interest.

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