

Arterial stiffness acute changes following aerobic exercise in males with and without hypertension

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Abstract

While regular exercise exposure is considered the most effective therapy to reduce arterial stiffness, the effect of acute exercise training on arterial stiffness in adults with different blood pressure (BP) levels remains unclear. The authors aimed to investigate the effects of acute aerobic exercise on arterial stiffness in male with different BP levels. This cross-sectional study utilized data for 1200 males aged 20–49 years from the Kailuan study cohort who participated in the fifth National Fitness Monitoring project. A total of 940 participants (621 in the non-hypertensive group and 319 in the hypertensive group) aged 36.82 ± 7.76 who completed a twice-quantitative cycle ergometer exercise and measure of brachial-ankle pulse wave velocity (baPWV) at both the baseline and immediately after exercise were included in this study. The baPWV was decreased after acute aerobic exercise in the non-hypertension and hypertension groups (Δ 40.29 [95% confidence interval [CI], -47.72 to -32.86] vs. Δ 20.45 [95% CI, -31.32 to -9.58] cm/s). Participants without hypertension showed a greater decrease in baPWV (Δ 19.84 [95% CI, -33.83 to -5.84] cm/s) than participants with hypertension. Aerobic exercise had an acute positive effect on arterial stiffness. This study provides evidence of a greater reduction in arterial stiffness in individuals without hypertension than in those with hypertension.

KEYWORDS

arterial compliance, arterial stiffness, exercise/hypertension

1 | INTRODUCTION

Early vascular aging is a major contributing factor in the development of cardiovascular disease and is related to cardiovascular mortality.^{1,2} Arterial stiffness increases with aging, and is a marker of vascular damage that predicts future cardiovascular events.^{3,4} Epidemiological studies have consistently shown that unhealthy behavior (chronic cigarette smoking,⁵ excessive television time,⁶ and high dietary sodium intake⁷)

adversely affects vascular health in adulthood and promotes progression of vascular damage.

A recent meta-analysis validated the widely-accepted view that chronic exercise confers beneficial effects on maintaining healthy vascular function.⁸ However, the effects of acute aerobic exercise have received less attention and the reported results are inconsistent. Naka and colleagues⁹ reported a significant reduction in lower limb pulse wave velocity following maximal treadmill exercise in 50 healthy

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subjects. A study of 61 elderly patients with hypertension showed elevated carotid-femoral pulse wave velocity (cfPWV) immediately after acute maximal exercise compared with baseline levels.¹⁰ And the other investigations in nine hypertensive patients also showed no change in arterial stiffness indices immediately following maximum aerobic exercise.¹¹ These studies had small sample sizes and were mostly limited to Caucasian populations. To the best of our knowledge, no studies have investigated and compared the effect of acute aerobic exercise-induced changes on arterial stiffness in Asian men with different blood pressure (BP) levels.

Thus, we aimed to evaluate short-term changes in brachial-ankle pulse wave velocity (baPWV) following an acute aerobic exercise test in men with different BP levels in the Kailuan study population who participated in the fifth National Fitness Monitoring.

2 | METHODS

2.1 | Subjects

The Kailuan study (trial registration number: ChiCTR-TNC-11001489) is a community-based, longitudinal cohort study evaluating cardiovascular disease risk factors in a functional community population located in Tangshan City, China. In 2006, participants from the Kailuan community received their first survey at Kailuan General Hospital and 10 affiliated hospitals. Follow-up surveys were provided every 2 years in 2008–2009, 2010–2011, 2012–2013, 2014–2015, 2016–2017, and 2018–2020. To systematically understand the current status and changing laws of the Chinese national physique, the State Sports General Administration initiated the fifth National Physique Monitoring in 2020. Using the employee information database reported and established by the four affiliated companies of the Kailuan Group, 1200 male employees aged 20–49 years were randomly selected as participants of the fifth National Physique Monitoring. The National Physique Monitoring conducts annual checks within its defined area, including a cycling test, choice stepping reaction time, and handgrip strength. baPWV was measured before and immediately after each cycling exercise session. The study was carried out in accordance with the guidelines of the Declaration of Helsinki. All participants provided written informed consent to the study, which was approved by the Kailuan General Hospital Ethics Committee.

In this study, we included the following participants: (1) those who had participated in the survey in 2018–2020 and had participated in the fifth National Physique Monitoring; and (2) those who had undergone cycling exercise and had baPWV measured at both the baseline and follow-up exercise.

2.2 | Exercise protocol

The procedure of performing cycling exercises complies with the Exercise Standards for Testing and Training released by the American Heart Association (AHA) and is briefly described below.¹² All interventions

and measurements were performed in a quiet, temperature-controlled room (22°C–25°C). Before cycling exercise, the subjects had avoided alcohol, tobacco, and caffeinated beverages for 12 h. After an initial period of rest, baseline measurements of baPWV were made in the supine position. Cycling exercise was then performed with individually tailored ramp protocols via upright cycle ergometry (GMCS-GLC3, Beijing, China). Following a brief warm-up by cycling against unloaded exercise (0 watts) for 30 s, participants started to cycle at a moderate intensity (50 watts~80 watts) for 3 min according to the individual's conditions so that the heart rate reached 60%~80% of their estimated maximum (calculated as 220 minus age in years), and the resistance was increased by 25 watts in the next 3 min. Throughout the exercise, participants were instructed to maintain a pedaling cadence of 60 revolutions per minute (rpm). The cycling exercise finished with 30 seconds of unloaded (0 watts). HR was monitored and recorded continuously. Maximum oxygen consumption ($\text{VO}_2 \text{ max}$) was indirectly estimated based on the cycling exercise and calculated by the ergometry according to a previously described equation.¹³

2.3 | baPWV, BP, and HR measurement

baPWV, BP, and HR were recorded by a BP-203 RPE III networked arterial stiffness detection device (Omron Health Medical Co., Ltd., China), which simultaneously measures arterial pulse waves and systolic/diastolic arterial pressures in both the brachial and posterior tibial arteries.¹⁴ Previous studies have demonstrated the strong association between baPWV and cfPWV (correlation coefficient .73), and similarly has a strong ability to predict future cardiovascular events, and thus, is widely used especially in East Asian countries.^{15,16} A minimum of 5 min of resting time was required before the baseline baPWV measurements. Participants in thin clothes were asked to lie down on an examination couch in the supine position and remain quiet during measurements. Cuffs were wrapped on both arms and ankles. The lower edge of the arm cuffs was positioned 2–3 cm above the transverse striation of the cubital fossa, while the lower edge of the ankle cuffs was positioned 1–2 cm above the superior aspect of the medial malleolus. Measurements were repeated twice for each participant, with the second data considered the final value. For analysis, the higher values of left and right sides of baPWV and brachial blood pressures (systolic, diastolic, and mean arterial pressure) were used. Based on the systolic BP and diastolic BP, we calculated MAP as follows: $\text{MAP} = 1/3 \text{ systolic BP} + 2/3 \text{ diastolic BP}$. The measurement was performed under controlled conditions in a quiet room and using the same protocol at both the pre- and post-exercise.

2.4 | Data collection

Demographic data, lifestyle factors (e.g., age, smoking status, alcohol use, and physical activity), and medical history (e.g., anti-hypertensive and hypoglycemic agents) were collected using a self-reported questionnaire, as detailed previously.¹⁷ Height, weight, and waist

circumference were measured by trained nurses, and body mass index (BMI) was calculated as weight (kg)/height² (m²). Hypertension was defined as systolic BP \geq 140 mmHg, diastolic BP \geq 90 mmHg, or use of antihypertensive medications.¹⁸ The definition of diabetes mellitus was laboratory based, with fasting blood glucose levels \geq 7.0 mmol/L combined with medication use.^{19,20} The smoking status was defined as smoking at least one cigarette/day on average in the past 6 months. The drinking status was defined as 100 ml/day on average for more than 1 year. Physical activity was defined as aerobic exercise three times/week for \geq 30 min periodically.

2.5 | Statistical analysis

Normally distributed variables are shown as the mean and standard deviation, and variables with a skewed distribution are shown as the median with interquartile range (25%, 75%). Categorical measures are shown as the frequency and percentage (%). We examined baseline characteristics in the hypertension versus non-hypertension groups using the Student's *t*-test or the Wilcoxon–Mann–Whitney test, as appropriate. Categorical variables were compared using the chi-square test. The changes in all hemodynamic parameters from rest to post-exercise were compared using the paired Student's *t*-test. Statistical significance was set at $p < .05$.

Linear regression models were used to evaluate group changes in hemodynamic parameters post-exercise. These models were adjusted for the pre-exercise value, age (years), BMI (continuous), smoking status (never or ever-smoker), drinking status (never or ever-drinker), physical activity (inactive or active), and duration of measurement, with the hypertension group as the reference group. The changes in HR (post-exercise minus rest), brachial MAP (post-exercise minus baseline), and ankle MAP (post-exercise minus baseline) were dependent variables. Further, VO₂max and Max HR are adjusted for age (year) and BMI (continuous).

In the multivariate models, we adjusted for baseline baPWV in Model 1. In Model 2, we further adjusted for age (years), BMI (continuous), smoking status (never or ever-smoker), drinking status (never or ever-drinker), and physical activity (never or active). In Model 3, we further adjusted for the changes in HR (exercise minus baseline), changes in brachial MAP (post-exercise minus baseline), VO₂max and duration of the interval between measurements.

We performed several sensitivity analyses to assess the robustness of our findings. First, we excluded participants with diabetes mellitus, which is associated with a high baPWV and is a potential risk factor for arterial stiffness. To reduce the effect of the interval duration, we also conducted another sensitivity analysis by excluding participants with an interval duration between measurements greater than the 95% quantile (26.7 min). Since the proportion of physical activity was lower in the hypertension group, which is the first line of therapy for hypertension and positive effects of baPWV, we performed an analysis comparing the arterial stiffness response to cycling exercise among physical activity groups (inactive and active groups). Lastly, we performed a sensitivity analysis to adjust for the time of day of assessment which

was may influence model results. SAS (version 9.4; SAS Institute, Inc., Cary, NC, USA) was used for all statistical analyses.

3 | RESULTS

3.1 | Baseline characteristics

A total of 1200 male employees of the Kailuan Group were recruited to the fifth National Physique Monitoring of whom 1138 participated in the 2018–2020 survey in the Kailuan Study. After excluding participants who did not complete baPWV measurements before and after the cycling test ($n = 185$) and those who were missing cycling exercise details ($n = 13$), 940 eligible participants were assigned to the hypertension ($n = 319$) and non-hypertension ($n = 621$) groups, and were included in the data analysis. All participants had a mean age of 36.82 ± 7.76 years. Generally, participants with hypertension were more likely to have a higher BMI, waist circumference, hip circumference, concentrations of fasting blood glucose, total cholesterol, triglyceride, high-sensitivity C-reactive protein and low-density lipoprotein, and baPWV (Table 1).

baPWV was significantly decreased post ($p < .001$) in comparison to values at pre-exercise (Table S1). Unadjusted values of hemodynamics and parameters post-exercise are presented in the Data Supplement (Table S2). In adjusted analyses, the HR increased in the non-hypertension group by 4.82 bpm (95% CI, 4.16–5.49) and in the hypertension group by 6.79 bpm (95% CI, 5.82–7.75). The brachial MAP decreased in the non-hypertension group by Δ .77 mmHg (95% CI, -1.37 to $-.19$) whereas increased in the hypertension group by 2.49 mmHg (95% CI, 1.62–3.35). The interval of baPWV measurement after the cycling test in the non-hypertension group was 9.56 min (95% CI, 8.93–10.19) and that in the hypertension group was 10.25 min (95% CI, 9.37–11.13). VO₂max predicted by the exercise protocol were 43.33 ml/kg per min (95% CI, 42.70–43.97) and 41.15 ml/kg per minute (95% CI, 40.24–42.07), respectively (Table 2).

After adjusted for baseline baPWV, there was a greater net reduction in baPWV in the non-hypertension group compared with the hypertension group (non-hypertension group, -44.90 cm/s [95% CI, -52.39 to -37.42]; hypertension group, -11.46 cm/s [95% CI, -22.19 to $-.37$]; difference of -33.44 cm/s [95% CI, -47.00 to -19.89]). After adjusting for the variables of baseline baPWV, age, BMI, smoking status, drinking status, and physical activity, there were notable changes in baPWV from baseline to post-exercise. Both groups showed a favorable change in baPWV, with a significant difference between the groups (non-hypertension group, -40.55 cm/s [95% CI, -48.08 to -33.01]; hypertension group, -19.94 cm/s [95% CI, -30.94 to -8.94]; difference of -20.61 cm/s [95% CI, -34.74 to -6.47]). After further adjustments for the change in HR (post-exercise minus baseline), change in brachial MAP (post-exercise minus baseline), VO₂max and interval duration between measurements, there was still a significant between-group difference in the change in baPWV (non-hypertension group, -40.29 cm/s [95% CI, -47.72 to -32.86]; hypertension group,

TABLE 1 Baseline characteristics

Variable	Total N = 940	Non-hypertension N = 621	Hypertension N = 319
Demographic factors			
°Age, year	36.82 ± 7.76	34.73 ± 7.52	40.88 ± 6.52 ^a
°BMI, kg/m ²	26.25 ± 3.89	25.59 ± 3.86	27.54 ± 3.62 ^a
°Waist, cm	89.68 ± 9.74	88.08 ± 9.66	92.79 ± 9.13 ^a
°Hip, cm	98.46 ± 7.18	97.80 ± 7.17	99.73 ± 7.05 ^a
°Waist: hip ratio	.91 ± .06	.90 ± .06	.93 ± .06 ^a
Laboratory parameters			
°FBG, mmol/L	5.50 ± .94	5.39 ± .87	5.73 ± 1.04 ^a
°TC, mmol/L	4.79 ± 1.09	4.69 ± 1.11	5.00 ± 1.02 ^a
°TG, mmol/L	1.19 (.83, 1.91)	1.10 (.80, 1.72)	1.39 (.97, 2.35) ^a
°HDL, mmol/L	1.37 ± .36	1.38 ± .38	1.35 ± .32
°LDL, mmol/L	2.81 ± .81	2.75 ± .85	2.93 ± .73 ^a
°Hs-Crp	−1.26 (−4.61, .41)	−1.11 (−4.61, .53)	−1.56 (−4.61, .15) ^a
°Smoke, n (%)	607 (64.57)	412 (66.34)	195 (61.13)
°Drink, n (%)	652 (69.36)	421 (67.79)	231 (72.41)
°Physical activity, n (%)	350 (37.23)	245 (39.45)	105 (32.92) ^a
°Diabetes, n (%)	65 (7.27)	25 (4.33)	40 (12.66)
Medications, n (%).			
°Antihypertensive agents, n (%)	93 (9.89)	—	93 (32.75)
BP (measured seated)			
°SBP, mmHg	131.35 ± 12.21	136.43 ± 8.77	140.92 ± 12.30 ^a
°DBP, mmHg	81.50 ± 9.09	77.94 ± 6.86	88.42 ± 8.90 ^a
°MAP, mmHg	98.11 ± 9.38	94.10 ± 6.53	105.92 ± 9.16 ^a
Pre-exercise: Arterial stiffness and hemodynamics (measured supine)			
°baPWV, cm/s	1375.08 ± 209.09	1316.65 ± 172.76	1488.84 ± 226.40 ^a
°HR, bpm	80.24 ± 12.54	79.42 ± 12.35	81.85 ± 12.76 ^a
°Brachial SBP, mmHg	131.84 ± 15.97	127.33 ± 13.73	140.63 ± 16.40 ^a
°Brachial DBP, mmHg	78.99 ± 11.27	75.31 ± 9.69	86.15 ± 10.69 ^a
°Brachial MAP, mmHg	99.09 ± 12.91	95.01 ± 10.75	107.03 ± 13.09 ^a
°Ankle SBP, mmHg	141.82 ± 19.80	135.77 ± 16.02	153.59 ± 21.13 ^a
°Ankle DBP, mmHg	74.39 ± 11.35	70.76 ± 9.97	81.48 ± 10.52 ^a
°Ankle MAP, mmHg	96.60 ± 12.77	92.40 ± 10.60	104.75 ± 12.69 ^a

Note: Values expressed as mean ± SD, median (interquartile range), or number (%) as appropriate.

Abbreviations: baPWV, brachial-ankle pulse wave velocity; BMI, body mass index; DBP, diastolic blood pressure; FBG, fasting blood glucose; HDL-C, high-density lipoprotein cholesterol; HR, heart rate.; hsCrp, high-sensitivity C-reactive protein; LDL-C, low-density lipoprotein cholesterol; MAP, mean arterial pressure; SBP, systolic blood pressure; TC, total cholesterol; TG, triglyceride.

^aIndicates a significant between-group difference.

−20.45 cm/s [95% CI, −31.32 to −9.58; difference of −19.84 cm/s [95% CI, −33.83 to −5.84]] (Table 3).

In sensitivity analyses, the change in baPWV from baseline to post-exercise in participants in the non-hypertension and hypertension groups, and differences between the groups, remained consistent after excluding participants with diabetes mellitus (Supplementary Table S2). Additionally, after excluding participants with an interval

duration between measurements greater than the 95% quantile (26.7 min), the results were similar to the main results (Supplementary Table S3). There were no significant differences in ΔbaPWV between inactive and active adults (Supplementary Table S5). In addition, the difference between-group remained stable after further adjustment for the time of day of assessment (morning or afternoon) (Supplementary Table S6).

TABLE 2 Adjusted values of changes in hemodynamics

Variable	Non-hypertension N = 621	With hypertension N = 319
△Brachial MAP (mmHg)	-.77 (-1.37 to -.19)	2.49 (1.62 to 3.35)
△ankle MAP (mmHg)	-1.83 (-2.51 to -1.15)	.13 (-.88 to 1.13)
△HR (bpm)	4.82 (4.16 to 5.49)	6.79 (5.82 to 7.75)
VO ₂ max (ml/kg per min)	43.33 (42.70 to 43.97)	41.15 (40.24 to 42.07)
Max HR (bpm)	138.15 (137.09 to 139.22)	141.84 (140.31 to 143.38)
Interval measurement duration (min)	9.56 (8.93 to 10.19)	10.25 (9.37 to 11.13)

Note: Adjusted means (95% CI) are presented. △(changes) from baseline comparisons.

△HR, △Brachial MAP, △ankle MAP are adjusted for the pre-exercise value, age(year), BMI, smoking status (never or ever-smoker), drinking status (never or ever-drinker), physical activity (inactive or active) as well as interval measurement duration.

VO₂max and Max HR are adjusted for age (year), BMI.

Abbreviations: BMI, body mass index; DBP, diastolic blood pressure; HR, heart rate.; MAP, mean arterial pressure; SBP, systolic blood pressure.

VO₂max: maximum oxygen consumption.

TABLE 3 Mean difference in △baPWV between adults without non-hypertension and with hypertension in univariate, partially adjusted, and fully adjusted models

	Non-hypertension N = 621	With hypertension N = 319	Adjusted mean difference (non-hypertension minus with hypertension) (95% CI)
Model 1	-44.90 (-52.39 to -37.42)	-11.46 (-22.19 to -3.7)	-33.44 (-47.00 to -19.89)
Model 2	-40.55 (-48.08 to -33.01)	-19.94 (-30.94 to -8.94)	-20.61 (-34.74 to -6.47)
Model 3	-40.29 (-47.72 to -32.86)	-20.45 (-31.32 to -9.58)	-19.84 (-33.83 to -5.84)

Note: △(changes) from baseline comparisons; △HR, △Brachial MAP, postexercise minus baseline;

Model 1 is adjusted for baseline baPWV.

Model 2 is adjusted for baseline baPWV, age(year), BMI, smoking status (never or ever-smoker), drinking status (never or ever drinker), and physical activity (inactive or active).

Model 3 is adjusted for baseline baPWV, age(year), BMI, smoking status (never or ever-smoker), drinking status (never or ever drinker), physical activity (inactive or active), △HR, △Brachial MAP, VO₂max, and interval measurement duration.

Abbreviations: BMI, body mass index; HR, heart rate; MAP, mean arterial pressure; VO₂max: maximum oxygen consumption.

4 | DISCUSSION

In the present study, we found that 7 min of cycling exercise was effective in reducing arterial stiffness in male with and without hypertension. Remarkably, the beneficial effects of exercise on arterial stiffness appeared to be more evident in male without hypertension than in those with hypertension.

Age-related differences in arterial stiffness are diminished in healthy individuals who undertake regular exercise.²¹ Following a 7-min bout of exercise, we also observed a significant reduction in baPWV of 44.90 cm/s from resting values in young and middle-aged (36.82 ± 7.76 years) participants without hypertension. This reduction remained consistent after adjusting for potential confounders. In line with our results, a previous study conducted by Kingwell and colleagues²² reported a significant decrease in femoral pulse wave velocity 30 min after a single bout of cycling at 65% of the maximal oxygen uptake in 12 young (24 ± 6 years) healthy men.

Importantly, in our study, baPWV of participants with hypertension was immediately decreased by 11.46 cm/s after exercise compared to that pre-exercise. However, Cooke and colleagues¹⁰ showed

elevated cfPWV 3 min after a maximal treadmill exercise test compared with baseline levels in elderly adults with hypertension. A similar finding was also reported by Lefferts and colleagues²³ The greater reflected wave amplitude during exercise in older subjects (>50 years) is due to increased large artery stiffness and impaired vasodilatory capacity in association with the aging process.²⁴ Discrepancies in findings between this study and the above-mentioned studies could partly be explained by the age difference. Notably, different from the above-mentioned studies, our study used baPWV as the measurement methodology for arterial stiffness. It has been found that immediately post-exercise (0–5 min), the central and upper body arterial segments show an overall increase in arterial stiffness whereas, measurements taken in lower limb segments (proximal to the primary working muscles in most cases) show an immediate decrease in arterial stiffness.²⁵

In our study, we further compared the arterial stiffness response to cycling exercise between individuals with and without hypertension. Both groups showed a significant reduction in baPWV following cycling exercise compared with baseline measurements. Participants without hypertension had a greater reduction (19.84 cm/s) in baPWV than those with hypertension. In contrast to our study, Lefferts

and colleagues²³ reported that no obvious differences between middle-aged adults with hypertension and without hypertension were observed in hemodynamic response following exercise. The difference of the participant's health status may be the major contributor to the discrepancies between the two studies. In the study conducted by Lefferts and colleagues,²³ there was no significant difference in health status between participants with and without hypertension, including physical activity, traditional cardiovascular disease profiles, and body composition. Moreover, in this study, BP in their participants with hypertension was well controlled according to at-home BP values. However, in our study, age and RHR in subjects with hypertension were higher than subjects without hypertension. Accumulative evidences have indicated that an older age³ and increased RHR²⁶ are independently associated with vascular aging. Therefore, we speculate that the between-group difference in baPWV after cycling exercise may be related to the above-mentioned factors (age and RHR) attenuating the benefit of exercise on arterial stiffness in participants with hypertension.

Each additional year of age is associated with a 10.7 cm/s increase in baPWV in a Chinese male population.²⁷ This previous finding has theoretical and clinical importance. Yang and colleagues²⁸ showed that short-term (≤ 3 months) regular aerobic exercise enhanced circulating endothelial progenitor cells and attenuated the age-related decline in arterial elasticity in 20 healthy men. We found that baPWV was decreased immediately after exercise in men without hypertension and in those with hypertension by 40.29 and 20.45 cm/s, respectively. As functional and structural changes are interrelated and inseparable aspects of an integrated process of vessel adaptation, the acute benefits of regularly applied exercise may result in a structural improvement.²⁹ Repeated exposures of exercise may contribute to maintain the reduction in baPWV and reduce the vascular age of non-hypertensive people and hypertensive people by 3.8 and 2 years, respectively. Therefore, the findings of our study highlight the need to prescribe exercise by defining the number of weekly sessions, the intensity, and duration in clinical practice.

Because structural changes in a short time frame (minutes) are unlikely, we mainly attribute the decrease in baPWV after the cycling exercise test in our study to functional changes. Although the mechanisms underlying this reduction by acute aerobic exercise remain to be investigated, there are several potential mechanisms. First, some previous studies have suggested that, under acute conditions, physical exercise increases blood flow and shear stress,³⁰ leading to an increased release of endothelial vasodilatory signals (e.g., nitric oxide and prostaglandins), resulting in relaxation of vascular tone, and it explains the sharp rise in arterial compliance and deceleration of pulse wave velocity after exercise. Second, other mechanisms involved could be vasodilation mediators and decreased release of vasoconstriction factors (e.g., endothelin-1 or angiotensin-2), which attenuate the vasoconstrictor effect of endothelin-1 and angiotensin 2 in the vasculature.³¹ Furthermore, the acute effect of exercise on arterial stiffness may be mediated through changes in blood metabolite levels. The Framingham Heart Study showed a large shift in $>80\%$ of annotated metabolites in the circulating metabolome in response

to approximately 12 min of incremental exercise.³² This shift led to beneficial alterations in levels of metabolites representing key metabolic pathways that are central to obesity, insulin resistance, oxidative stress, inflammation, and vascular reactivity and longevity, including a variety of novel metabolic mediators.

There are some limitations to our study that should be acknowledged. (1) This was a cross-sectional study, and we did not examine the causal relationship between various factors and the change in baPWV before and post-exercise. (2) It should be noted that our study was based on a population from an industrial district who were selected to participate in the fifth National Fitness Monitoring were male, which may not be generalized to other populations. (3) Although cfPWV is presently considered as the gold standard method for measuring arterial stiffness,³³ whereas baPWV is similarly validated as an independent predictor of the risk of development of CVD and is also closely correlated with cfPWV,³⁴ reproducibly, and noninvasively. (4) Owing to the limitations of the site conditions, the time for baPWV measurement after the cycling exercise test could not be unified. However, when evaluating the short-term effect of acute aerobic exercise on arterial stiffness, we adjusted for the duration of baPWV measurements. (5) The duration of the cycling exercise test in this study was only 7 min, which is slightly shorter than that in previous reports, but the maximum peak HR of the subjects was close to the sub-maximal HR.

5 | CONCLUSIONS

This study suggests that acute aerobic exercise favorably affects endothelial function. We also provide evidence of a greater decrease in arterial stiffness in male individuals without hypertension than in those with hypertension.

5.1 | Perspectives

In this study, we showed the beneficial effects of acute aerobic exercise by reducing arterial stiffness in young and middle-aged males with and without hypertension. Future studies are warranted to address whether acute aerobic exercise can prevent or delay the onset of early vascular aging, benefit BP control, and how acute exercise influences the human metabolome.

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DISCLOSURES

None.

CONFLICTS OF INTEREST

There are no conflicts of interest.

AUTHOR CONTRIBUTIONS

We thank all the survey teams of the Kailuan Study Group for their contribution and the study participants who contributed their information. Lixia Sun and Shouling Wu conceived designed the study and revised it critically for important intellectual content; Yiran Zang, Xiong Ding, MaoXiang Zhao, Xiaoling Zhang, and Li zhang acquired the data; Yiran Zang, Xiong Ding, and MaoXiang Zhao supervised the analysis and contributed to the discussion; Yiran Zang drafted the manuscript. All authors approved the version to be published and agreed to be accountable for all aspects of the work.

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SUPPORTING INFORMATION

Additional supporting information may be found in the online version of the article at the publisher's website.

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