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Arterial stiffness results from eccentrically biased downhill running exercise

J.F. Burr*, M. Boulter, K. Beck

Applied Human Science, Human Performance and Health Research Laboratory, University of PEI, Canada

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ABSTRACT

Objectives: There is increasing evidence that select forms of exercise are associated with vascular changes that are in opposition to the well-accepted beneficial effects of moderate intensity aerobic exercise. To determine if alterations in arterial stiffness occur following eccentrically accentuated aerobic exercise, and if changes are associated with measures of muscle soreness.
Design: Repeated measures experimental cohort.
Methods: Twelve ($m = 8/f = 4$) moderately trained ($VO_2max = 52.2 \pm 7.4\ ml\ kg^{-1}\ min^{-1}$) participants performed a downhill run at -12° grade using a speed that elicited 60% VO_2max for 40 min. Cardiovascular and muscle soreness measures were collected at baseline and up to 72 h post-running.
Results: Muscle soreness peaked at 48 h ($p = <0.001$). Arterial stiffness similarly peaked at 48 h ($p = 0.04$) and remained significantly elevated above baseline through 72 h.
Conclusions: Eccentrically accentuated downhill running is associated with arterial stiffening in the absence of an extremely prolonged duration or fast pace. The timing of alterations coincides with the well-documented inflammatory response that occurs from the muscular insult of downhill running, but whether the observed changes are a result of either systemic or local inflammation is yet unclear. These findings may help to explain evidence of arterial stiffening in long-term runners and following prolonged duration races wherein cumulative eccentric loading is high.

1. Introduction

The health benefits of participation in regular aerobic exercise are well established.[1] Amongst the cardiovascular benefits of regular moderate intensity exercise is the reduction of arterial stiffness, which is associated with a reduced risk for cardiovascular disease and ischaemic events owing to decreased atherosclerosis, an improvement of coronary artery perfusion,[2] and a lowering of both pulse pressure and wall stress.[2,3] Given the clear relationship to future morbidity and mortality, arterial stiffness is now recognized as an important independent predictor of cardiovascular risk.[4] Despite the known beneficial cardiovascular effects of habitual moderate intensity aerobic exercise, high-volume resistance training has been shown to elicit transient arterial stiffening[5] and long-term participants manifest these changes persistently such that their resting levels substantially differ from controls who are only recreationally active;[6] however, this area remains a controversial area and results are mixed.[7,8] Counter intuitively, recent evidence examining the arterial properties and cardiovascular health of long distance runners has similarly demonstrated higher than expected baseline arterial stiffness compared to recreationally active controls[9,10] and there is conflicting evidence as to whether or not participation in a long-distance race itself causes arterial stiffness.[9,11,12] From our own published and unpublished observations, our laboratory has noted that substantial post-race arterial stiffening occurred only in races that were of prolonged duration (>24 h baseline to follow-up) and which involved heightened physical stresses including a higher exercise intensity or substantial terrain challenges, such as the traversing of mountains.[11,12] As such, we posit that the observed effect on arterial stiffening may be related to exercise induced muscle damage and inflammation, which typically requires 24–48 h to manifest[13] and is enhanced by eccentric muscle contractions, which occur repeatedly during running – particularly while descending hills. This theory fits with existing evidence showing that long distance running causes high levels of oxidative stress and inflammation.[13] In fact, recent strength training literature shows that eccentric training results in notable arterial stiffening from baseline and is associated with measures of muscle damage and inflammation whether using small or large muscle mass.[14] Using experimentally induced

* Corresponding author.
E-mail address: jburr@upei.ca (J.F. Burr).

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inflammation Vlachopoulos et al.[15] have convincingly demonstrated a cause-and-effect relationship of systemic inflammation with arterial stiffening, however, some difference in the progress of the inflammatory cascade may occur depending on whether inflammation is systemically induced or stimulated by exercise.[16]

Treadmill-based downhill running has long been established as a valid and reliable method to induce delayed onset muscle soreness and inflammation,[17,18] particularly when the exposure exceeds an amount normally encountered by the participants. A downhill running exercise model allows investigation of the effects of the eccentric muscle contractions associated with running in the absence of extreme aerobic exercise intensity or duration. The purpose of the present investigation was thus to determine the role of eccentric muscle contractions during aerobically based exercise for affecting changes in arterial stiffness. Furthermore, we sought to investigate the temporal course of alterations from baseline to determine the time of onset, duration of the effect, and the association of changes in arterial stiffness with measures of muscle soreness. We hypothesized that even moderate intensity eccentrically biased running would result in significant muscle soreness, and associated arterial stiffening within twenty-four to forty-eight hours.

2. Methods

A mixed sex population of recreationally active subjects ($n = 13$, $m = 9$, $f = 4$) were recruited from the university community. Inclusion criteria indicated good general health, free from injury or pregnancy, not currently or previously engaging in eccentric exercise training, and no history of smoking, alcohol dependence, diagnosed heart disease, peripheral vascular disease, diabetes, cancer, pulmonary disease, orthopaedic conditions or use of medication. Participants were 25 ± 6 years of age, had a mean systolic pressure of 115 ± 9, diastolic pressure of 75 ± 5, were 175 ± 8 cm tall, and weighed 74.6 ± 16 kg. VO_2max was 52.2 ± 7.4 ml kg min^{-1} (male 55.6, female 45.4). All participants provided written informed consent and this study was approved by the institutional Research Ethics Board for investigations involving human participants.

Participants visited the laboratory for baseline testing either 3 or 4 days prior to the downhill running test. All baseline testing commenced on either Thursday or Friday morning, with experimental procedures taking place the following week. During the study period, participants were requested to abstain from exercise, and NSAID use was prohibited. Baseline measures including blood pressure, carotid-femoral pulse wave velocity (PWV), radial artery augmentation index (AIx), and both subjective and objective muscle pain were recorded at each time point. Participants were given a period of no less than 2 full days recovery following the VO_2max test, prior to the commencement of the downhill running intervention. Fig. 1 graphically illustrates the procedural timing of all measurements. With the exception of the 6 h follow-up, all measures were taken at the same time each morning to control for diurnal variation. Participants were instructed to eat a light breakfast 2 h prior to arrival, and to standardize their morning meal from baseline through the last day of follow-up.

Following the assessment of baseline muscle pain and the cardiovascular measures described to follow, aerobic fitness testing was performed using a mechanically driven treadmill with analysis of expired gases. The fitness test commenced at a fast walking speed (4 mph) and progressed 0.45 ms^{-1} (1 mph) and 2% grade until the participant reached his or her maximal safe running speed, after which only the incline was progressed. Breath-by-breath metabolic measurements were performed using a Cosmed Quark CPET system (Cosmed, Rome, Italy) and were averaged over 30 s for analysis. Metabolic testing required an average of 11.7 ± 1.2 min. The test was terminated when participants reached volitional fatigue (RPE = 19 or 20) and had an RER greater than 1.15. Attainment of a true VO_2max was verified by a plateau in oxygen consumption (increase < 150 ml min^{-1}) with an increase in workload. All participants reached a true max according to these criteria.

Blood pressure was measured using a standard sphygmomanometer at the brachial artery of the left arm while the participant was seated with his or her arm supported on a table at the level of the heart. Prior to the measurement of blood pressure, participants sat quietly for a period of 10 min to ensure accurate resting values. All measurements were taken in a quiet temperature and humidity controlled laboratory.

Subjective muscle pain was recorded using a 10 cm visual analogue scale wherein the participant marked the line corresponding to their rating of leg pain in daily life since the last assessment. Participants were instructed to make a mark on the line with the far left indicating "no discomfort at all" and the far right being "the most extreme muscular pain you have experienced". This scale was quantified by measuring the distance to the nearest 0.1 cm from the left edge of the scale to the participant's mark. These methods have been previously used and validated.[19] Objective leg pain was quantified using a push-pressure strain gauge dynamometer (Fabrication Enterprises, White Plains, NY) with a cylindrical 1 cm^2 flat contact head applied to the mid muscle belly (measured from the superior patella to the anterior inferior iliac crest) of the vastus lateralis, immediately anterior to the iliotibial band. Participants were instructed to look away from the dynamometer and verbally indicate when the progressively applied pressure first became uncomfortable. Push-pressure muscle pain measures have been shown to be highly reproducible, and have been recommended for experimentally induced alterations in muscle pain sensitivity.[20]

Carotid to femoral PWV was measured with participants in the supine position. Using an electrocardiogram, pressure waves were gated to systolic contraction and were collected consecutively from the carotid then femoral artery. Signal capture at each site was performed manually once a sufficient quality signal was obtained. Pulse wave arrival was determined using the intersecting tangent method to detect the foot of each wave. For velocity calculations, distance was measured with a standard anthropometric tape using a straight line above the body from the carotid to femoral sites, with an adjustment for the distance from the carotid site to the suprasternal notch. Pulse wave velocity of the descending aorta was our primary outcome variable to estimate arterial stiffness as PWV is recognized as the gold standard measure and is less affected by the physiological alterations known to occur in conjunction with a bout of exercise compared to other common estimation methods.[25]

Resting AIx was calculated from pulse waves collected at the right radial artery using a high fidelity Millar strain gauge transducer (Millar instruments, Houston, TX). Participants sat quietly with the right forearm resting on a table and the wrist supported in slight hyperextension. Signal quality was controlled using the autocapture feature of the Sphygmocor CPVH software (Atcor Medical, Sydney, Australia) and the operator index was recorded at $93 \pm 5\%$ for all captures. AIx measures standardized to a heart rate of 75 bpm were employed for analysis to reduce the tachycardic effect of the downhill run on post-run measures. Augmentation index is a measure of the reflected waves within the arterial tree extending down to the radial artery level, and although this can offer some insight into stiffness properties and the work of the myocardium, reflected waves can be affected by changes in other exercise related cardiovascular factors, such as cardiac time intervals. As such, AIx was included only as a secondary measure.

The downhill run was performed using the same treadmill as the VO_2max test set to a decline of -12° with a specifically constructed elevation device placed under the rear of the belt platform. Participants ran at a speed eliciting 60% VO_2max (pace range 1.9–3.2 m/s)