

Research Article

A single daily 10-second sprint snack can improve left ventricular redox balance in male Wistar rats

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Abstract

Exercise snacks are time-efficient physical-activity strategies, but the cardiac redox effects of a single ultra-short daily sprint are unknown. This study examined whether one daily 10-second sprint snack modifies left ventricular oxidant-antioxidant balance in male Wistar rats. Twelve male Wistar rats were randomized to control (CO, n=6) and single daily sprint snack (DSS, n=6) groups. After treadmill familiarization and maximal treadmill running speed assessment, the DSS group performed one daily sprint, six days per week, for eight weeks. Forty-eight hours after the final session, left ventricular tissue was analyzed for malondialdehyde (MDA), superoxide dismutase (SOD), glutathione peroxidase (GPx), and total antioxidant capacity (TAC). Compared with CO, DSS reduced MDA ($p=0.004$) and increased SOD ($p=0.011$), GPx ($p=0.010$), and TAC ($p<0.001$). All four outcomes remained statistically significant in a post hoc Holm sensitivity analysis. A single daily 10-second sprint snack favorably modified left ventricular redox biomarkers in male Wistar rats. These findings support a minimal-volume model of redox adaptation, although effects on exercise performance, cardiac structure, and cardiac function remain to be established.

Key Words: Sprint snack, Lipid peroxidation, Antioxidant capacity, Left ventricle

Introduction

Physical inactivity contributes substantially to cardiometabolic disorders and cardiovascular disease (CVD) (Islam et al., 2022). Exercise snacks - brief bouts of exercise, often lasting 1 min or less and performed intermittently during the day - have emerged as a time-efficient strategy for improving cardiorespiratory fitness, glycemic control, and vascular function (Islam et al., 2022; Wang et al., 2025). This approach may help address barriers to regular physical activity such as limited time and access to equipment (Wang et al., 2025). Nevertheless, strenuous or unfamiliar exercise can transiently disturb redox homeostasis, and the magnitude of the response depends on exercise mode, duration, training status, and workload (Lee et al., 2023; Fisher-Wellman & Bloomer, 2009). Importantly, despite their time-efficient premise, existing exercise-snack models have not yet established long-term, let alone lifelong, adherence as a sustainable adjunct to predominantly chair-based modern lifestyles (Islam et al., 2022; Wang et al., 2025).

The heart is particularly sensitive to redox perturbation because of its continuous workload and high metabolic demand (Kim et al., 2016). Endogenous antioxidant defenses, including SOD, catalase, and GPx, limit oxidative injury, whereas sustained oxidative stress can contribute to maladaptive cardiac remodeling and dysfunction (Jomova et al., 2024; van der Pol et al., 2019). Exercise therefore has a dual redox role: transient RONS formation contributes to adaptive signaling, while excessive or poorly tolerated oxidant exposure may promote oxidative damage (Meng & Su, 2024; Morales-Alamo & Calbet, 2014; Costa et al., 2018; Akgul & Koz, 2019; Sutkowsky et al., 2022). Exercise dose, recovery, and conditioning status are consequently important determinants of the net response.

Despite growing interest in exercise snacks, heart-specific redox adaptations remain insufficiently characterized. Rodent models allow treadmill exposure and tissue sampling to be standardized under controlled conditions (Jia et al., 2020). Conventional exercise-snack paradigms generally use several

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brief bouts distributed across the day (Islam et al., 2022; Wang et al., 2025; Jenkins et al., 2023). In contrast, the present study deliberately tested one daily 10-second sprint as a reductionist, minimal-volume variant of the broader exercise-snack concept. We hypothesized that this ultra-short repeated sprint would reduce lipid peroxidation and enhance endogenous antioxidant defenses in left ventricular tissue.

Materials and methods

Study design

Animals and experimental design

Twelve six-week-old male Wistar rats (185-240 g) were obtained from the Animal Laboratory of Baqiyatullah University of Medical Sciences. After a one-month acclimatization period, during which body mass increased to approximately 245-300 g, the rats were randomly assigned to control (CO, n=6) and single daily sprint snack (DSS, n=6) groups. Animals were maintained under a 12-h light/dark cycle and 45-50% humidity with ad libitum access to water and standard chow. All procedures were approved by the Ethics Committee of Shahed University (IR.Shahed.Rec.1400.005) and conducted in accordance with applicable Iranian guidelines for the care and use of laboratory animals.

Treadmill familiarization and maximal treadmill running speed assessment

All treadmill procedures were performed at 0° incline. General familiarization consisted of 10 min/day of continuous running at 10-15 m/min for five consecutive days. A maximal treadmill running speed (MTRS) test was then performed, beginning at 10 m/min and increasing by 3 m/min every 3 min until exhaustion (Asadi et al., 2022). The treadmill was fitted with a rear shock-grid assembly, but electrical stimulation was never activated or used. Exhaustion was operationally defined as inability to maintain belt speed despite non-noxious encouragement, followed by remaining in the rear grid area for approximately 5 s without resuming running. The group-level MTRS was approximately 40 m/min and was used only as a descriptive external-speed reference; it was not used to prescribe individual sprint speed. Published Wistar-rat data and animal exercise guidelines support the feasibility of treadmill speeds in this range (Brito Vieira et al., 2014; Poole et al., 2020). MTRS was used for baseline characterization and was not reassessed after the intervention.

Sprint-specific familiarization and exercise intervention

After MTRS assessment, the animals underwent six days of sprint-specific familiarization. Each session comprised three brief repetitions separated by 60 s of recovery. Belt speed was increa-

-sed over approximately 2 s until the highest speed the individual rat could execute without falling behind was reached. During early familiarization, the peak-speed phase lasted approximately 3-10 s and subsequently stabilized at approximately 8-10 s. Peak familiarization speeds varied according to individual capacity and were approximately 30-60 m/min. No overt exercise-related injury or adverse response was observed during familiarization.

During the eight-week intervention, the DSS group performed one sprint session per day, six days per week. Each session began with 3 min of running at 10-15 m/min. Belt speed was then increased over approximately 2 s to the highest speed the rat could execute while maintaining position on the treadmill. The target sprint duration was 10 s, and the peak-speed phase typically lasted approximately 8-10 s. Belt speed was reduced over approximately 3 s to 10 m/min, followed by 2 min of active cool-down at 10 m/min. Recorded peak sprint speeds ranged approximately from 50 to 70 m/min. Relative to the group-level reference MTRS (~40 m/min), these values corresponded descriptively to approximately 125-175% of the reference speed; these percentages were not training targets. The single-bout design was intentional and represented a minimal-volume experimental variant rather than a conventional multi-bout exercise-snack protocol. The protocol is summarized in Table 1.

Sampling and tissue preparation

Forty-eight hours after the final exercise session, the animals were deeply anesthetized, blood was collected from the heart, and the animals were then humanely euthanized. The body cavity was opened through a midline incision, and the heart was quickly

Table 1. Summary of treadmill familiarization, MTRS assessment, and the daily sprint protocol.

Component	Procedure
Treadmill incline	0° throughout familiarization, MTRS assessment, and the intervention
General treadmill familiarization	5 consecutive days; 10 min/day at 10-15 m/min
MTRS assessment	10 m/min start; +3 m/min every 3 min until exhaustion; electrical stimulation not used
Group-level reference MTRS	Approximately 40 m/min; descriptive external-speed reference only, not an individual training target
Sprint-specific familiarization	6 days; 3 repetitions/day; 60-s recovery; ~2-s acceleration; peak-speed phase initially ~3-10 s and subsequently ~8-10 s; peak speeds ~30-60 m/min
Training frequency	1 sprint/day; 6 days/week; 8 weeks
Warm-up	3 min at 10-15 m/min
Acceleration	~2 s to the highest executable belt speed for the individual rat
Peak-speed phase	Target 10 s; typically, ~8-10 s
Observed peak sprint speed	Approximately 50-70 m/min
Relative speed context	Approximately 125-175% of the ~40 m/min reference MTRS; descriptive only
Deceleration	~3 s to 10 m/min
Active cool-down	2 min at 10 m/min

*Relative percentages are provided only to contextualize external running speed and were not used as fixed training targets.

removed for tissue sampling. The 48-h interval was selected to minimize the influence of transient acute exercise-induced changes in redox markers and to better reflect adaptations associated with the eight-week intervention. Hearts were rinsed in physiological saline, weighed to the nearest 0.001g, and dissected to isolate the left ventricle. Left ventricular tissue was placed in cryovials and stored at -80°C until biochemical analysis.

Measurement of oxidative stress markers

Left ventricular tissue was homogenized in phosphate-buffered saline (PBS) and centrifuged, and the clear supernatant was used for biochemical analyses. All assays were performed in duplicate using commercial kits (ZellBio GmbH, Germany) according to the manufacturer's instructions with appropriate blank controls. MDA, an index of lipid peroxidation, was measured using the thiobarbituric acid reactive substances method (ZB-MDA-48A) at 532 nm (sensitivity: 0.1 µM; intra-assay coefficient of variation [CV]: 5.8%). SOD activity was measured using the ZB-SOD-48A kit by inhibition of the autoxidation reaction at 560 nm (sensitivity: 1 U/mL; CV: 5.8%). GPx activity was assessed using the ZB-GPx-48A kit by monitoring NADPH oxidation at 340 nm (sensitivity: 1 U/mL; CV: 3.5%). TAC was quantified using the ZB-TAC-48A kit with an oxidation-reduction colorimetric assay read at 490 nm (sensitivity: 0.1 mM; intra-assay CV: <3.4%).

Statistical analysis

Data are presented as mean ±SD. Normality was assessed using the Shapiro-Wilk test and homogeneity of variances using Levene's test. Between-group differences were analyzed using two-sided independent-samples t tests. Mean differences were defined as DSS minus CO. Effect sizes were expressed as eta-squared (η^2), calculated as $t^2/(t^2 + df)$, and 95% confidence intervals (CIs) were reported for mean differences. To assess robustness to multiplicity across MDA, SOD, GPx, and TAC, an additional post hoc sensitivity analysis using the Holm step-down procedure was performed. This analysis was not prespecified and was not treated as a planned confirmatory analysis. Statistical significance was set at $p < 0.05$. Analyses were performed using SPSS version 22.0.

Results

Table 2. Body and heart measurements.

Measure	CO (n = 6)	DSS (n = 6)	p value
Body weight, pre-intervention (g)	286.33 ± 10.33	282.67 ± 17.04	0.662
Body weight, post-intervention (g)	370.17 ± 14.27	351.67 ± 21.12	0.106
Heart weight (g)	1.27 ± 0.09	1.19 ± 0.13	0.243
Heart-to-body weight ratio (%)	0.343 ± 0.025	0.338 ± 0.035	0.778

Data are presented as mean ± SD. CO, control group; DSS, single daily sprint snack group.

Body and heart measurements are summarized in Table 2. There were no significant between-group differences in pre-intervention body weight ($p=0.662$), post-intervention body weight ($p=0.106$), heart weight ($p=0.243$), or the heart-to-body weight ratio ($p=0.778$).

MDA was lower in the DSS group than in the CO group ($t(10) = -3.65$, $p=0.004$, $\eta^2=0.57$), with a DSS-minus-CO mean difference of $-1.58 \mu\text{M}$ (95% CI: -2.55 to -0.62) (Figure 1A). SOD activity was higher in DSS ($t(10)=3.10$, $p=0.011$, $\eta^2=0.49$), with a mean difference of 6.16 U/mL (95% CI: 1.73 to 10.60) (Figure 1B). GPx activity was also higher in DSS ($t(10)=3.19$, $p=0.010$, $\eta^2=0.50$), with a mean difference of 1.61 U/mL (95% CI: 0.49 to 2.74) (Figure 1C). TAC was higher in DSS ($t(10)=8.52$, $p < 0.001$, $\eta^2 = 0.88$), with a mean difference of 3.97 mM (95% CI: 2.93 to 5.00) (Figure 1D). In the post hoc Holm sensitivity analysis, all four between-group differences remained statistically significant (adjusted $p=0.013$ for MDA, 0.019 for SOD, 0.019 for GPx, and <0.001 for TAC).

Discussion

Oxidative stress contributes to cardiovascular disease, whereas transient redox signaling is also integral to physiological adaptation (Sies, 2017). The principal finding of this study was that one daily 10-second sprint snack for eight weeks reduced left ventricular MDA and increased SOD, GPx, and TAC. The concurrent reduction in lipid peroxidation and enhancement of enzymatic and total antioxidant measures indicates a favorable chronic shift in left ventricular redox status after an extremely brief repeated sprint stimulus. This pattern is consistent with established concepts of exercise-induced redox signaling and adaptation (Merry & Ristow, 2016; Powers & Schrager, 2022).

The protocol should be distinguished from conventional exercise-snack paradigms, which generally use several brief bouts distributed across the day (Islam et al., 2022; Wang et al., 2025; Jenkins et al., 2023). Here, one daily sprint was deliberately selected as a reductionist minimal-volume model. The findings therefore should not be interpreted as directly equivalent to accumulated multi-bout exercise-snack interventions; rather, they provide evidence from a single-bout variant of the broader concept.

The present results are broadly consistent with studies reporting improved antioxidant status after repeated sprint or interval exercise. Eight weeks of sprint interval training reduced lipid peroxidation and increased antioxidant capacity in cardiac tissue of Wistar rats (Radaei et al., 2022), while another rat study reported improved cardiac oxidant-antioxidant balance after sprint interval training (Bieranvand & Rahmani, 2022). In young adults, sprint interval training has also been associated with enh-

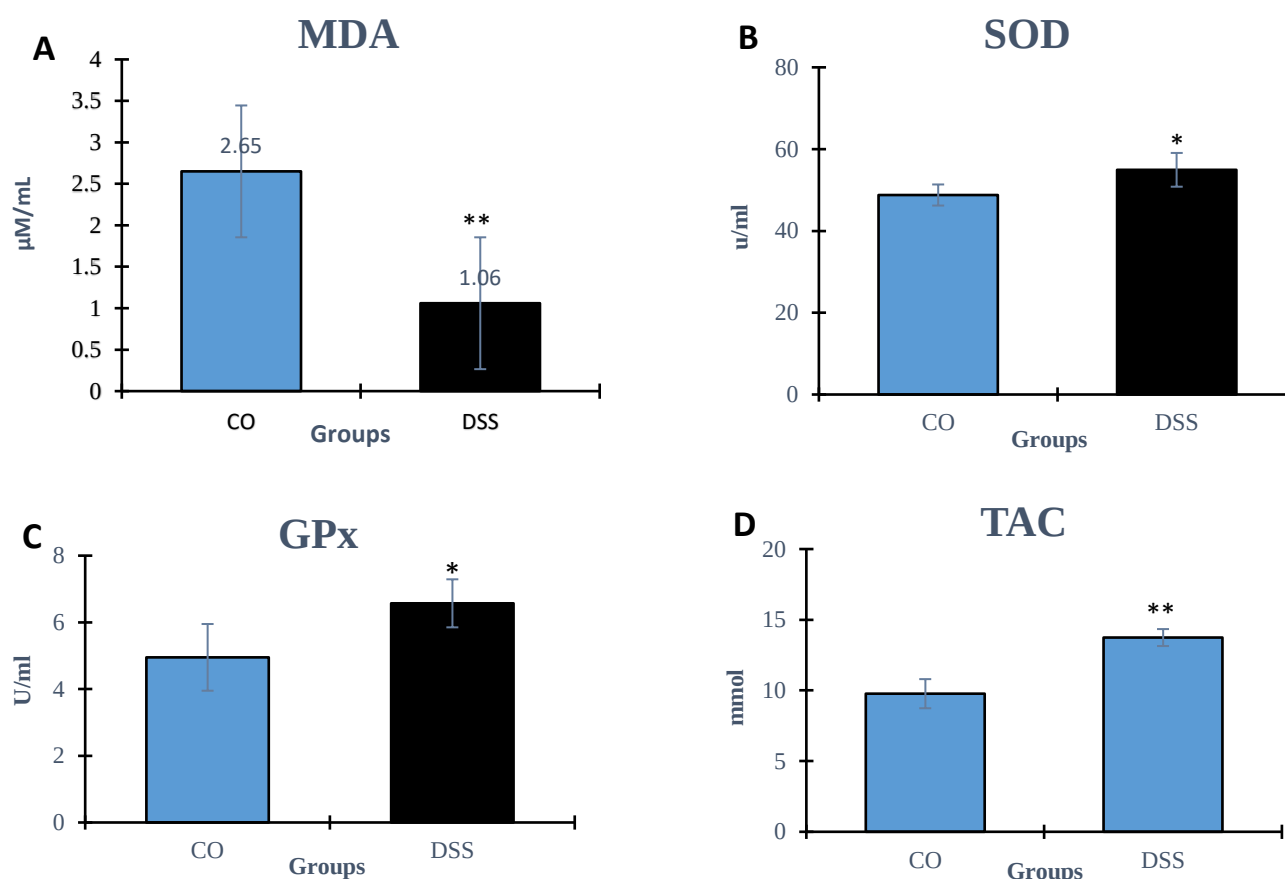


Figure 1. Left ventricular redox markers in the control and single daily sprint snack groups. (A) Malondialdehyde (MDA), (B) superoxide dismutase (SOD), (C) glutathione peroxidase (GPx), and (D) total antioxidant capacity (TAC). CO: control group (n=6); DSS: single daily sprint snack group (n=6). *p<0.05, **p<0.01 vs. CO group.

-anced antioxidant potential and lower oxidative stress markers (Rudarli Nalcakan et al., 2021). Differences among studies likely reflect bout duration, accumulated work, recovery, tissue specificity, and training status. Brief exercise exposures may remain within an adaptive redox range more readily than longer or more metabolically demanding efforts (Radak et al., 2013; Gomez-Cabrera et al., 2008). Tissue-specific findings from other models support the broader possibility of antioxidant adaptation after brief or interval exercise. Interval training has increased antioxidant capacity in rat cerebellar tissue (Freitas et al., 2019), a single interval-exercise session enhanced antioxidant defenses in the rat hippocampus without significant lipid peroxidation (Melo et al., 2019), and short-term interval training in humans reduced oxidative stress markers while increasing antioxidant enzyme activity (Bogdanis et al., 2013). Conversely, some protocols have produced less favorable redox responses. Gorzi et al. reported increased MDA and reduced cardiac GPx after sprint interval training in Wistar rats (Gorzi et al., 2018), 30 min of swimming increased cardiac MDA in rats (Turgut et al., 2003), and acute oxidative stress after demanding exercise in humans varies with exercise characteristics and participant status (Lu et al., 2021; Cipryan, 2017). These contrasting findings reinforce the importance of bout duration and cumulative exercise dose.

A hormetic and mitohormetic interpretation is plausible. In the present study, the intervention was defined by very short duration, high executable belt speed, and extremely low cumulative exercise volume rather than by a directly measured physiological intensity. Repeated brief redox perturbations may activate adaptive signaling and strengthen endogenous antioxidant defenses. This interpretation is consistent with mitohormesis, in which transient mitochondrial redox signals promote stress resistance while prolonged or excessive oxidant exposure may become detrimental (Radak et al., 2008; Ristow & Schmeisser, 2014). Exercise-related RONS can engage Nrf2-dependent antioxidant responses, interact with PGC-1 α -related mitochondrial remodeling, and modulate NF- κ B as a context-dependent redox-sensitive transcription factor (Done & Traustadottir, 2016; Davies, 2018). Because acute RONS production, Nrf2, PGC-1 α , NF- κ B, and mitochondrial adaptations were not measured, these mechanisms remain hypotheses rather than demonstrated mediators.

Heart weight and the heart-to-body weight ratio did not differ between groups. Given the very low cumulative exercise volume, favorable redox adaptation may therefore occur without a measurable increase in gross cardiac mass. However, unchang-

-ed heart weight does not exclude subtle structural remodeling because histological and morphometric analyses were not performed.

Several limitations should be considered. The small sample size (n=6 per group) limits precision and generalizability and increases the influence of sampling variability. Only male rats were studied, so sex-specific responses cannot be inferred. Although effect sizes and 95% CIs were reported and all four redox outcomes remained significant in a post hoc Holm sensitivity analysis, the multiplicity adjustment was not prespecified and should be interpreted as a robustness analysis. The study did not compare the single daily sprint with conventional multi-bout exercise-snack patterns or alternative distributions of equivalent weekly exercise dose. A standardized post-intervention performance test was not performed, and cardiac function was not assessed by echocardiography or hemodynamics. Consequently, the observed biochemical adaptations cannot be assumed to represent improved exercise capacity, contractile function, or protection against cardiac injury. Larger, prospectively powered studies should include both sexes and combine redox biomarkers with standardized performance, structural, and functional cardiac outcomes.

Conclusion

A single daily 10-second sprint snack for eight weeks reduced lipid peroxidation and enhanced antioxidant defenses in left ventricular tissue of male Wistar rats. The findings support the possibility that an ultra-short sprint with very low cumulative exercise volume can favorably modify cardiac redox biomarkers. The physiological and cardioprotective significance of these adaptations should be established in larger studies that include direct functional and structural assessments.

What is already known on this subject?

Exercise snacks can improve cardiometabolic and fitness outcomes, and repeated exercise can strengthen endogenous antioxidant defenses through redox-sensitive adaptation. Heart-specific redox effects of a single ultra-short daily sprint, however, remain poorly characterized.

What this study adds?

In male Wistar rats, one approximately 8-10-s sprint per day, six days per week for eight weeks, reduced left ventricular MDA and increased SOD, GPx, and TAC. The findings remained significant in a post hoc Holm sensitivity analysis and support a minimal-volume redox adaptation without establishing functional cardioprotection.

Organ Cross-Talk Tips:

- Heart, cerebellum, and hippocampus showed antioxidant gains after interval exercise, while some cardiac protocols increased oxidative damage, indicating tissue-specific crosstalk shaped by exercise dose.

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Data availability

The data that support the findings of this study are available from the corresponding author upon reasonable request.

Compliance with ethical standards

Conflict of interest: The authors declare that there is no conflict of interest in the present research.

Ethical approval: All procedures were approved by the Ethics Committee of Shahed University (IR.Shahed.Rec.1400.005) and conducted in accordance with applicable Iranian guidelines for the care and use of laboratory animals.

Informed consent: Not applicable (animal study).

Author contributions

Conceptualization: F.P.; Methodology: M.R.; Software: A.S.; Validation: F.P.; Formal analysis: M.R.; Investigation: A.S.; Resources: F.P.; Data curation: M.R.; Writing—original draft: A.S.; Writing—review & editing: F.P.; Visualization: A.S.; Supervision: M.R.; Project administration: F.P.; Funding acquisition: M.R.

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