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Effects of Active vs Passive Cool-Down on Ultra-Early Post-Exercise Haemodynamics in Healthy Young Men: A Preliminary Study

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ABSTRACT

Objectives: This study investigated ultra-early effects of active cool-down (AC) and passive cool-down (PC) on post-exercise haemodynamics and cardiorespiratory responses in healthy young men, to inform strategies to reduce exercise-induced syncope.
Methods: Seven healthy, non-athletic male students performed an incremental cardiopulmonary exercise test (CPX) on a cycle ergometer, followed immediately by either AC (pedalling at 20 W) or PC (sitting without pedalling). Respiratory gas exchange was recorded breath-by-breath and averaged every 3 seconds. Heart rate (HR), oxygen uptake (VO_2), carbon dioxide output (VCO_2), and ventilation (VE) were measured at peak exercise and at 0, 3, and 6 seconds after the start of the cool-down. For descriptive purposes, responses up to 15 seconds were also plotted.
Results: HR decreased significantly more during the first 3 seconds of recovery in PC than in AC ($p = 0.03$, $d = 0.62$). No significant differences in HR changes were observed between 3 and 6 seconds after cool-down initiation ($p = 0.47$). VO_2, VCO_2, and VE did not differ significantly between AC and PC at any of the measured time points (all $p > 0.05$).
Conclusion: These findings suggest that PC immediately after exercise induces a more abrupt

early decline in HR than AC, potentially delaying ultra-early haemodynamic stabilisation during a time window in which exercise-induced syncope may occur. Incorporating light active recovery may help support safer haemodynamic recovery following exercise. Given the small sample size, these results should be confirmed in larger studies.
Keywords: Active cool-down, Passive cool-down, Haemodynamics, Cardiorespiratory responses, Exercise-induced syncope

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1. Introduction

Noncardiac syncope (NCS) in young individuals is often neurally mediated and frequently involves autonomic reflex mechanisms, which can lead to abrupt reductions in blood pressure and/or heart rate (Grubb & Karas, 1999; O'Connor et al., 1999). In athletes and physically active populations, most NCS events occur during orthostatic stress or shortly after exercise cessation rather than during exercise itself (O'Connor et al., 1999). Orthostatic intolerance and post-exercise hypotension are well-recognized triggers for NCS, making circulatory stability during recovery a critical factor in its prevention (Kenney & Seals, 1993; Halliwill, 2001; Murrell et al., 2009). As a preventive strategy, a cool-down (CD) period is widely recommended in both sports and rehabilitation settings (Van Hooren & Peake, 2018).

Cool-downs are typically categorized as active cool-down (AC), involving light exercise, and passive cool-down (PC), involving rest. PC eliminates the skeletal muscle pump, reduces venous return, and may reproduce physiological conditions associated with orthostatic intolerance and NCS during recovery (O'Connor et al., 1999; Romero et al., 2017). Conversely, AC is recommended to help maintain venous return and support a more stable cardiovascular transition during recovery (Van Hooren & Peake, 2018; Romero et al., 2017). However, whether AC and PC differentially influence the immediate post-exercise transition remains unclear.

Previous research on post-exercise recovery has largely focused on changes in HR, blood pressure, and autonomic function over minutes, not seconds, following exercise cessation (Halliwill, 2001; Romero et al., 2017; Peçanha et al., 2014). To our knowledge, no studies to date have specifically

examined cardiovascular and ventilatory/gas-exchange responses within the first 6 seconds after exercise cessation, when the decision to continue moving (AC) or stop abruptly (PC) is made in real-world settings. This ultra-early transition is characterised by an immediate loss of the skeletal muscle pump together with residual exercise-induced vasodilation, conditions that may rapidly alter venous return and arterial pressure (Kenney & Seals, 1993; Halliwill, 2001). Because practical instructions such as "keep pedalling/walking lightly for a few seconds" can be implemented immediately without special equipment, characterising responses in this 0–6 second window may provide actionable information for safer recovery strategies after CPX, rehabilitation sessions, and athletic training, particularly for individuals at risk of exercise-induced syncope (O'Connor et al., 1999; Murrell et al., 2009).

Therefore, this preliminary study aimed to investigate the immediate effects of AC and PC on ultra-early post-exercise responses (HR and ventilatory/gas-exchange variables) in healthy young men who do not engage in regular exercise. The findings may help inform practical CD strategies for safer recovery, especially in situations where rapid post-exercise circulatory transitions may contribute to symptoms.

2. Methods

- **Subjects**

This preliminary study was conducted at a single university from December 1 to 27, 2019. Seven healthy, non-athletic male students (mean age: 20.0 ± 1.1 years; height: 171.0 ± 8.8 cm; body mass: 61.2 ± 9.0 kg) participated. None of the participants had a history of cardiovascular or respiratory disease, and all reported engaging in structured exercise less than once per week.

Exclusion criteria included any history of neurological, metabolic, or orthopedic conditions that could affect exercise capacity. Participants were instructed to abstain from eating, smoking, and engaging in vigorous physical activity for at least 2 hours prior to the cardiopulmonary exercise test (CPX). Before the experiment, all participants received verbal and written explanations regarding the study purpose, procedures, potential risks, and data protection. Written informed consent was obtained from each participant. The study protocol was approved by the institutional research ethics committee (Approval No. R2-007) and conducted in accordance with the Declaration of Helsinki.

- ***Cardiopulmonary Exercise Test and Cool-Down Protocol***

Participants completed two separate laboratory visits in a randomized crossover design. In one visit, CPX was followed by active cool-down (AC), and in the other visit, CPX was followed by passive cool-down (PC). The order of conditions was randomized and counterbalanced, and visits were separated by at least 24 h. Visits were scheduled at a similar time of day. Participants were instructed to avoid strenuous exercise, caffeine, and alcohol for at least 3 h and to refrain from eating and smoking for at least 2 h before each visit.

The condition order (AC first vs PC first) was determined using a computer-generated random sequence prepared by an investigator not involved in outcome assessment. The allocation sequence was counterbalanced as evenly as possible across participants.

The CPX was performed using a ramp protocol on a cycle ergometer (AE-300S, Minato Medical Science, Japan). Respiratory gases were measured continuously using a metabolic cart providing breath-by-breath data, which were time-aligned by the system software (including compensation for sample-line/analyzer delay) and exported as 3-s averaged values. Accordingly, values labeled as "0 s", "3 s", and "6 s" represent the 0–3 s, 3–6 s, and 6–9 s time bins after exercise cessation (onset of cool-down), rather than instantaneous values at those exact time points. "End-exercise" was defined as the final 3-s averaged bin immediately before exercise cessation (−3–0 s).

Participants rested for 2 min and then completed a 2-min warm-up. During the ramp phase, the workload started at 20 W and increased by 20 $W \cdot min^{-1}$. Participants were instructed to maintain a cadence of 60 rpm, paced with a metronome to minimize cadence fluctuations. The test was terminated when participants reached an RPE of 17 ("very hard", Borg 6–20 scale) or when they were unable to maintain 60 rpm despite verbal encouragement. The mean time from the start of the ramp phase to reaching RPE 17 was 12.7 ± 2.1 min.

Immediately after exercise cessation, participants performed the cool-down condition assigned for that visit: AC (continued pedaling at 20 W) or PC (seated rest on the ergometer without pedaling). HR, VO_2, VCO_2, and $\dot{V}E$ were recorded at end-exercise (−3–0 s) and during the first 15 s after exercise cessation; values from the 0–3 s, 3–6 s, and 6–9 s bins ("0 s", "3 s", and "6 s") were used for statistical analyses. The cool-down period lasted 5 min for both conditions.

- ***Statistical Analysis***

Comparisons between AC and PC focused on the ultra-early recovery time bins immediately after exercise cessation. Specifically, we analyzed end-exercise (−3–0 s) and the subsequent 0–3 s, 3–6 s, and 6–9 s bins. Data are presented as mean ± SD. Normality of within-participant differences (AC–PC) at each time bin was assessed using the Shapiro–Wilk test. Between-condition comparisons were performed using paired t-tests (two-tailed). Effect sizes were calculated as Cohen's d_z (mean of within-participant differences divided by the SD of the differences) and interpreted as small (0.2), medium (0.5), and large (0.8). Statistical significance was set at $p < 0.05$. Because this was an exploratory preliminary study, we did not apply adjustments for multiple comparisons; findings should therefore be interpreted cautiously. All analyses were performed using R (version 3.0.2).

3. Results

Participant characteristics and baseline cardiopulmonary values are presented in Table 1.

Table 1. Participant characteristics and pre-exercise baseline cardiopulmonary variables (n = 7)

Variable	All participants	Baseline (pre-exercise), AC visit	Baseline (pre-exercise), PC visit
Age (years)	20.0 ± 1.1	—	—
Height (cm)	171.0 ± 8.8	—	—
Body mass (kg)	61.2 ± 9.0	—	—
HR (beats·min⁻¹)	—	88.3 ± 10.1	90.1 ± 9.4
VO_2 (mL·min⁻¹)	—	601.7 ± 137.8	651.7 ± 125.3
VCO_2 (mL·min⁻¹)	—	569.3 ± 142.2	572.6 ± 148.0
$\dot{V}E$ (L·min⁻¹)	—	18.0 ± 3.3	18.3 ± 4.4
Time to test termination (RPE 17), min	12.7 ± 2.1	—	—
Estimated peak workload, W	274 ± 41	—	—

Values are mean ± SD. Abbreviations: AC, active cool-down; PC, passive cool-down; HR, heart rate; VO_2, oxygen uptake; VCO_2, carbon dioxide output; $\dot{V}E$, minute ventilation. Baseline indicates the value obtained immediately before the ramp phase (after warm-up).

Heart rate (HR) showed a greater immediate decrease during passive cool-down (PC) than during active cool-down (AC) in the first ultra-early recovery time bin after exercise cessation (end-exercise [−3–0 s] to 0–3 s; ΔHR).

Specifically, ΔHR was larger in PC than in AC (11.6 ± 10.1 vs 1.6 ± 1.7 beats·min⁻¹; p = 0.0394; Cohen's d_z = −0.99) (Table 2).

Table 2. Ultra-early post-exercise responses (end-exercise and first 6 s of recovery) in active vs passive cool-down (n=7).

Variable	Time point	Active cool-down (AC)	Passive cool-down (PC)	p-value	Cohen's d_z	Magnitude
HR (beats·min⁻¹)	End-exercise (−3–0 s)	151.9 ± 17.2	164.1 ± 15.4	0.0208	-1.18	large
	0 s (0–3 s)	150.3 ± 17.4	152.6 ± 15.4	0.7589	-0.12	trivial
	3 s (3–6 s)	150.0 ± 17.4	149.7 ± 15.6	0.9571	0.02	trivial
	6 s (6–9 s)	147.3 ± 18.4	154.6 ± 15.1	0.3341	-0.4	small
	Δ (End-exercise → 0 s)	1.6 ± 1.7	11.6 ± 10.1	0.0394	-0.99	large
VO_2 (mL·min⁻¹)	End-exercise (−3–0 s)	1699 ± 404	1987 ± 318	0.1125	-0.7	medium
	0 s (0–3 s)	1684 ± 272	1694 ± 395	0.955	-0.02	trivial
	3 s (3–6 s)	1726 ± 363	1764 ± 323	0.8007	-0.1	trivial
	6 s (6–9 s)	1640 ± 208	1753 ± 465	0.4813	-0.28	small
VCO_2 (mL·min⁻¹)	End-exercise (−3–0 s)	2024 ± 507	2298 ± 359	0.1727	-0.58	medium
	0 s (0–3 s)	2011 ± 369	1985 ± 507	0.9069	0.05	trivial
	3 s (3–6 s)	2048 ± 479	2101 ± 397	0.7566	-0.12	trivial
	6 s (6–9 s)	1960 ± 291	2148 ± 619	0.3385	-0.39	small
$\dot{V}E$ (L·min⁻¹)	End-exercise (−3–0 s)	54.9 ± 14.8	61.7 ± 8.7	0.1483	-0.63	medium
	0 s (0–3 s)	54.7 ± 10.1	53.7 ± 12.8	0.8598	0.07	trivial
	3 s (3–6 s)	54.3 ± 12.7	57.1 ± 7.6	0.5127	-0.26	small
	6 s (6–9 s)	53.0 ± 10.3	58.7 ± 15.9	0.2422	-0.49	small

Values are mean ± SD. HR, heart rate (beats·min⁻¹); VO_2, oxygen uptake (mL·min⁻¹); VCO_2, carbon dioxide output (mL·min⁻¹); $\dot{V}E$, minute ventilation (L·min⁻¹).

End-exercise indicates the last 3-s averaged value immediately before exercise cessation (−3–0 s). “0 s”, “3 s”, and “6 s” correspond to the 0–3 s, 3–6 s, and 6–9 s bins after cessation, respectively, based on 3-s averaged breath-by-breath outputs. Δ represents the change from End-exercise to 0 s.

Between-condition comparisons (AC vs PC) were performed using paired t-tests (two-tailed). Effect size was calculated as Cohen’s d_z (mean of within-participant differences divided by the SD of the differences) and interpreted as trivial (<0.2), small (0.2–0.49), medium (0.5–0.79), and large (≥0.8).

In contrast, HR values during the subsequent ultra-early recovery bins (3–6 s and 6–9 s) did not differ significantly between conditions (all $p > 0.05$) (Table 2).

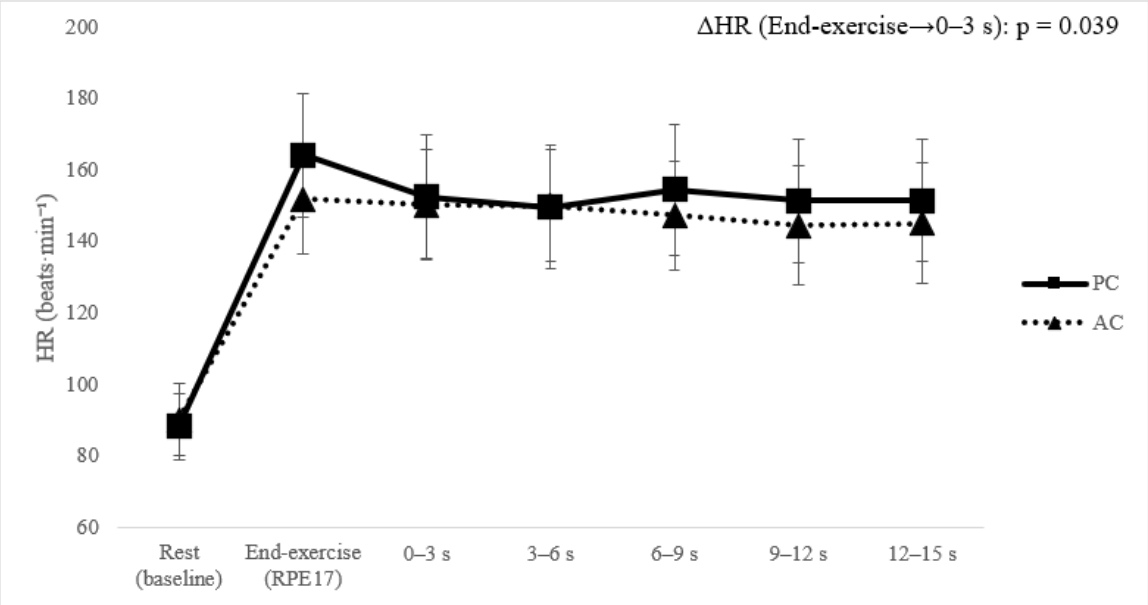

Figure 1: Heart rate response from baseline to the ultra-early recovery period after CPX.

Values are mean ± SD (n = 7). Rest indicates baseline prior to warm-up. End-exercise indicates exercise termination at RPE 17 and represents the final 3-s bin before cessation (−3–0 s). Ultra-early recovery points represent 3-s averaged bins after exercise cessation (0–3, 3–6, 6–9 s, etc.). A significant between-condition difference was observed for ΔHR (end-exercise minus 0–3 s; $p = 0.039$). AC, active cool-down; PC, passive cool-down.

No significant between-condition differences were detected in oxygen uptake (VO_2), carbon dioxide output (VCO_2), or minute ventilation ($\dot{V}E$) at any measured time bin (all $p > 0.05$) (Table 2).

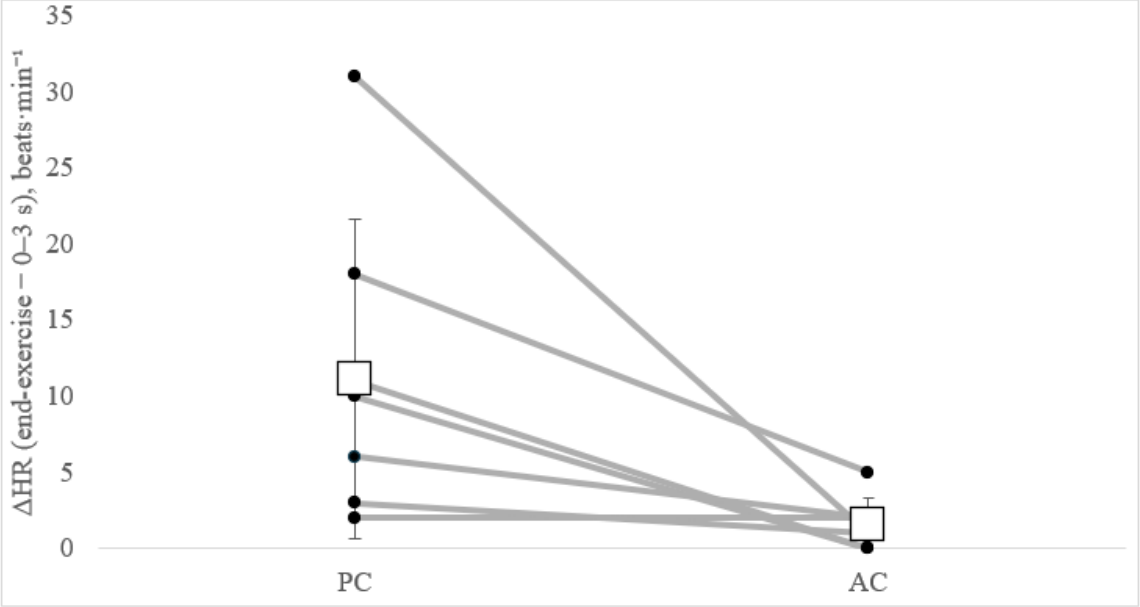

Figure 2. Individual and mean changes in heart rate (ΔHR) during the first ultra-early recovery bin (end-exercise to 0–3 s) under passive and active cool-down.

ΔHR was calculated as HR at end-exercise (−3–0 s) minus HR averaged over the 0–3 s bin after exercise cessation. Thin lines represent individual participants (n = 7). Open squares represent group means and error bars indicate SD. ΔHR was greater in PC than in AC ($p = 0.039$, paired two-tailed t-test; Cohen's $d_z = -0.99$). HR, heart rate; PC, passive cool-down; AC, active cool-down.

4. Discussion

The present preliminary study showed that the immediate decrease in HR from end-exercise to the first ultra-early recovery bin (0–3 s) was greater in passive cool-down (PC) than in active cool-down (AC) following incremental cycling exercise in healthy young men. In contrast, HR values at subsequent ultra-early bins (3–6 s and 6–9 s) did not differ between conditions, indicating that the between-condition divergence was brief and confined to the earliest seconds after exercise cessation. Given the absence of direct haemodynamic and autonomic measurements in this study, the mechanisms underlying this transient difference cannot be determined; however, the finding suggests that even

minimal behavioural differences immediately at exercise stop (continued light pedalling vs complete rest) may influence the earliest recovery dynamics.

Importantly, the 0–6 s window should not be interpreted as the period during which syncope or frank haemodynamic collapse necessarily occurs. In healthy individuals, a true circulatory collapse leading to loss of consciousness is unlikely to develop within only a few seconds. Rather, this window represents the earliest transition immediately after exercise cessation, when the skeletal muscle pump is abruptly withdrawn while exercise-induced vasodilation may persist and reflex control begins to re-adjust. This period is practically relevant because recovery behaviour is decided at the moment exercise stops, and any preventive instruction (e.g., "keep moving lightly for a few seconds") must be implemented without delay. Therefore, characterising responses in the first seconds after cessation is clinically meaningful as an "actionable" window, even if it is not the time frame in which syncope itself typically manifests.

A larger immediate HR decrease in PC may reflect a more abrupt cardiovascular transition immediately after cessation. One plausible interpretation is that the withdrawal of rhythmic leg contractions during PC reduces venous return via loss of the muscle pump, while vasodilation remains, potentially challenging central haemodynamics. However, because beat-to-beat blood pressure and stroke volume were not available, we cannot infer whether the observed HR pattern corresponded to meaningful differences in arterial pressure or cardiac output. Accordingly, we interpret the present HR finding as a sensitive marker of the earliest recovery transition rather than direct evidence of improved or worsened haemodynamic stability.

No significant between-condition differences were detected in VO_2, VCO_2, or $\dot{V}E$ at any measured time point. This lack of differences is physiologically plausible for several reasons. First, pulmonary gas-exchange and ventilatory kinetics do not change instantaneously at exercise cessation; VO_2 and VCO_2 typically decline over tens of seconds as metabolic demand and CO_2 stores equilibrate, whereas the most immediate mechanical change at cessation is the withdrawal of the muscle pump. Second, breath-by-breath measures averaged over very short bins are inherently variable immediately after cessation because the timing of individual breaths relative to the cessation point can influence 3-s averages. Together, these factors may render VO_2, VCO_2, and $\dot{V}E$ less sensitive than HR to small between-condition differences within the first seconds of recovery, particularly in a small sample. Thus, the present findings suggest that AC and PC may differ in the earliest HR transition without

producing detectable between-condition differences in gas-exchange or ventilatory variables within the same ultra-early window.

Although the ramp protocol used here is common in clinical exercise testing, the present results were obtained in a small sample of healthy young men and should be interpreted as exploratory. The absence of differences in VO_2, VCO_2, and $\dot{V}E$ does not exclude small effects, and the HR finding should not be generalised to clinical outcomes such as syncope risk. It remains possible that individuals with impaired cardiovascular compensation (e.g., older adults or patients in rehabilitation settings) may exhibit larger or more clinically meaningful responses during the ultra-early transition; however, this hypothesis requires direct testing with appropriate haemodynamic monitoring.

This study has several limitations. First, the sample size was small (n = 7), limiting statistical power and generalisability. Second, only HR and respiratory variables were available; blood pressure data were unavailable, and thus we could not evaluate post-exercise hypotension or haemodynamic instability directly. Future studies should incorporate continuous beat-to-beat blood pressure and additional indices (e.g., stroke volume, cardiac output, and O_2 pulse) to determine whether brief active movement immediately after cessation meaningfully attenuates early haemodynamic perturbations. Third, the ultra-early time points were derived from 3-s averaged breath-by-breath outputs; while this approach enables practical assessment of the earliest recovery phase, it is sensitive to breath timing and system delays. Finally, participants were young men without regular exercise habits; sex differences, training status, and clinical conditions were not addressed.

From a practical standpoint, these preliminary results support the notion that recovery instructions implemented immediately at exercise cessation may influence the earliest HR transition, even if differences in ventilatory and gas-exchange variables are not detectable within the same seconds. Future research should examine whether ultra-early active recovery strategies translate into measurable differences in haemodynamics and symptom risk across different populations and exercise contexts.

5. Conclusion

This preliminary study examined ultra-early changes in cardiorespiratory function when active cool-down (AC) and passive cool-down (PC) were performed immediately after incremental exercise in healthy young men without regular exercise habits. HR decreased significantly more during the first 3

seconds of recovery in PC than in AC, whereas no differences were observed from 3 to 6 seconds after cool-down initiation. These findings suggest that PC may induce a more abrupt early decline in HR and potentially delay haemodynamic recovery compared with AC during the initial seconds following exercise cessation.

However, the results should be interpreted cautiously given the small sample size, the exclusion of older adults and clinical populations, and the use of a gradual-load exercise protocol. Additionally, only HR, VO_2, VCO_2, and VE were measured due to equipment limitations. Future studies should include larger and more diverse populations, incorporate continuous blood pressure and additional haemodynamic indices such as O_2 pulse, and compare cool-down methods across different exercise modalities to further clarify their clinical and practical relevance.

- ***Preprint Declaration***

Not applicable.

- ***Data Availability Statement***

The datasets generated during the current study are available from the corresponding author upon reasonable request.

- ***AI-assisted technologies***

We utilized AI-based language tools, such as ChatGPT, to review and refine the grammar and vocabulary of the manuscript. No content generation or interpretation of results was performed using these tools

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