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# Effects of Prior Exercise Habits and Adherence on Cognitive Function, Physical Fitness, and Vascular Health in Older Adults: An Exploratory Exercise-Based Intervention Trial

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## ABSTRACT

**Background and Purpose:** Given the irreversible nature of dementia, this study examined the effects of a 20-week exercise-based dementia prevention program in community-dwelling older adults, focusing on prior exercise experience and program adherence.
**Methods:** In this exploratory, non-randomized trial, 55 older adults (65–79 years) were allocated to an intervention (n=26) or control (n=29) group, and blinding was not feasible. The intervention comprised supervised rhythmic aerobic exercise with cognitive-motor components performed three times per week. Cognition was the primary outcome, and secondary outcomes included physical fitness, blood pressure, and blood biomarkers. Subgroup analyses classified participants by prior exercise experience and intervention exposure: G1 and G2 comprised control subgroups with no intervention exposure, whereas G3 and G4 comprised intervention-exposed subgroups stratified by adherence.
**Results:** No significant group-by-time interactions were observed for cognitive outcomes. Participants with prior exercise experience and low adherence (G2) showed significant improvement on the Korean Mini-Mental State Examination ($\beta$=1.66, $p$=0.024) despite declines in physical fitness, whereas higher adherence in G3–G4 was associated with stable or favorable physical performance, with G4 showing a positive trend in the 30-second sit-to-stand test. Systolic blood pressure decreased significantly in G2–G4.
**Conclusions:** Although overall cognitive gains were modest and not group specific, prior exercise experience and sustained adherence were associated with favorable changes in physical fitness and vascular outcomes, suggesting that tailored multicomponent exercise programs and long-term engagement may help promote cognitive health in older adults.

**Keywords:** Elderly; Physical Fitness; Cognitive Function; Physical Exercise; Dementia

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The authors have no financial conflicts of interest.

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## INTRODUCTION

With the global rise in the aging population, the prevalence of dementia is rapidly increasing, imposing substantial social and economic burdens. Since dementia is irreversible once it occurs, preventive strategies are essential, and non-pharmacological approaches have been increasingly emphasized.[1,2] Among these, physical exercise has received significant attention as a relatively safe and cost-effective intervention, providing advantages for both physical well-being and cognitive functioning.[3-5] Exercise involving aerobic and coordination elements may enhance cerebral blood flow, promote neuroplasticity, and increase levels of brain-derived neurotrophic factor, thereby improving cognitive outcomes.[6,7] In addition, exercise contributes to improved muscular strength, endurance, and flexibility, as well as favorable changes in body composition such as fat reduction and muscle mass maintenance.[8,9] These physiological mechanisms provide a strong basis for preventing cognitive decline and promoting cognitive recovery in older adults with mild cognitive impairment (MCI).

International guidelines, including those from the World Health Organization (WHO) and the American College of Sports Medicine (ACSM), recommend that adults aged 65 years and above engage in 150–300 minutes of moderate-intensity aerobic exercise each week, or alternatively 75–150 minutes of vigorous-intensity activity. These sessions should be distributed across at least three to five days each week, with a minimum duration of 30 minutes per session, in order to promote both cognitive and overall health.[10,11] Evidence indicates that adherence to these recommendations is linked to a lower likelihood of developing Alzheimer's disease and a decelerated rate of cognitive decline.[12]

However, the effects of exercise cannot be fully explained by program participation alone and may vary depending on prior exercise experience and adherence among those exposed to the intervention. Most previous studies have relied on simple intervention-versus-control comparisons, without adequately accounting for individual differences in baseline physical activity or long-term engagement.[13,14] Exercise-related benefits may differ depending on prior physical activity levels, and lack of sustained participation can diminish or abolish the observed effects.[15-17] Therefore, the objective of this research was to assess how a 20-week dementia-prevention exercise intervention influences cognitive outcomes in older adults, while also considering the roles of prior exercise experience and adherence to the intervention program.

## METHODS

### Participants

Community-dwelling older adults aged 65–79 years were recruited from the Songpa Center of the National Fitness Award 100, operated by the Korea Sports Promotion Foundation (KSPO), in Seoul, South Korea. Individuals were screened to exclude those with major chronic illnesses or musculoskeletal conditions that could interfere with moderate- to vigorous-intensity exercise. Eligibility criteria included independence in activities of daily living, defined as a Korean Instrumental Activities of Daily Living score <0.4, and the ability to safely participate in moderate- to vigorous-intensity exercise. Participants were excluded if they had severe visual, hearing, or communication impairment that could interfere with participation in the intervention or outcome assessments, or if they were concurrently enrolled in other intervention studies. Written informed consent was obtained from eligible

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**Table 3.** Baseline characteristics of participants stratified by exercise experience and intervention exposure (G1–G4)

| Variables | G1 (n=12) | G2 (n=17) | G3 (n=10) | G4 (n=16) | *p*-value |
|---|---|---|---|---|---|
| Demographics | | | | | |
| Age | 72.5 (68.0 to 75.0) | 71.0 (67.0 to 72.0) | 72.0 (69.5 to 74.8) | 73.0 (70.0 to 77.0) | 0.377 |
| Female | 11 (92) | 11 (65) | 8 (80) | 11 (69) | 0.394 |
| Education (yr) | 12.0 (8.2 to 12.5) | 14.0 (12.0 to 16.0) | 12.0 (12.0 to 16.0) | 12.0 (12.0 to 16.0) | 0.185 |
| Body composition | | | | | |
| BMI (kg/m$^2$) | 23.5 (21.3 to 25.5) | 23.8 (21.5 to 24.7) | 24.6 (24.0 to 25.8) | 22.9 (20.5 to 25.3) | 0.606 |
| Body fat (kg) | 33.3 (28.4 to 38.1) | 27.8 (24.3 to 35.5) | 34.5 (27.6 to 36.7) | 29.0 (27.6 to 35.0) | 0.373 |
| SMM (kg) | 19.6 (18.4 to 20.6) | 20.3 (19.2 to 26.6) | 21.1 (20.2 to 25.6) | 20.4 (17.1 to 22.8) | 0.156 |
| Blood pressure | | | | | |
| SBP (mmHg) | 130.0 (120.2 to 145.5) | 130.0 (116.0 to 144.0) | 142.0 (128.0 to 152.5) | 145.0 (135.0 to 156.2) | 0.178 |
| DBP (mmHg) | 71.5 (66.2 to 74.2) | 70.0 (67.0 to 75.0) | 78.5 (74.2 to 83.0) | 79.0 (74.5 to 88.0) | 0.012 |
| Physical fitness | | | | | |
| 30-sec sit-to-stand test (times) | 23.0 (19.5 to 24.5) | 24.0 (23.0 to 27.0) | 18.5 (17.2 to 21.5) | 25.0 (21.8 to 29.0) | 0.006 |
| 2-min stationary march (times) | 112.0 (107.8 to 118.2) | 117.0 (113.0 to 127.0) | 99.5 (91.5 to 109.0) | 112.0 (104.0 to 125.0) | 0.027 |
| Sit-and-reach test (cm) | 12.7 (5.3 to 16.9) | 8.6 (−1.5 to 15.2) | 10.8 (8.6 to 11.5) | 10.7 (4.6 to 19.4) | 0.760 |
| 3-m sit-walk-and-return test (sec) | 5.7 (5.5 to 5.9) | 5.4 (5.0 to 5.8) | 5.7 (5.4 to 6.2) | 5.5 (5.3 to 5.8) | 0.254 |
| Figure-8-walk (sec) | 22.8 (22.1 to 23.7) | 21.2 (19.1 to 24.5) | 23.0 (21.9 to 26.3) | 22.4 (21.6 to 23.6) | 0.390 |
| Hand grip (left) (kg) | 23.2 (20.9 to 25.9) | 25.8 (19.7 to 34.4) | 24.7 (23.7 to 25.3) | 23.5 (20.1 to 28.5) | 0.718 |
| Hand grip (right) (kg) | 24.5 (21.3 to 29.6) | 26.7 (22.2 to 37.1) | 26.9 (24.7 to 31.3) | 23.5 (20.1 to 28.5) | 0.427 |
| Blood test | | | | | |
| TG (mmol/L) | 89.5 (83.2 to 111.8) | 100.0 (82.0 to 108.0) | 125.0 (85.8 to 190.2) | 101.0 (73.8 to 109.8) | 0.528 |
| Total cholesterol (mmol/L) | 160.5 (155.8 to 188.0) | 186.0 (152.0 to 204.0) | 182.5 (169.0 to 207.0) | 183.5 (165.2 to 213.8) | 0.667 |
| HDL (mmol/L) | 58.0 (52.2 to 67.8) | 58.0 (49.0 to 70.0) | 51.0 (45.8 to 60.5) | 61.5 (57.0 to 73.5) | 0.202 |
| LDL (mmol/L) | 89.0 (79.8 to 101.2) | 100.0 (66.0 to 127.0) | 96.5 (83.0 to 125.2) | 99.0 (84.2 to 139.8) | 0.646 |
| Cortisol (μg/dL) | 8.8 (6.9 to 12.7) | 9.2 (7.0 to 11.9) | 9.9 (6.5 to 11.3) | 10.2 (7.6 to 12.8) | 0.863 |
| Serotonin (ng/dL) | 86.8 (67.9 to 113.3) | 83.2 (67.2 to 104.2) | 79.9 (73.0 to 96.3) | 103.3 (80.3 to 123.0) | 0.728 |
| Cognitive dysfunction | | | | | |
| K-MMSE | 29.0 (28.0 to 29.0) | 28.0 (26.0 to 29.0) | 29.0 (27.0 to 30.0) | 27.5 (26.0 to 28.2) | 0.126 |
| ADAS-Cog | 16.0 (13.5 to 17.8) | 15.0 (12.0 to 19.0) | 15.0 (13.2 to 21.5) | 17.5 (13.8 to 21.2) | 0.677 |

Data are presented as median (Q1–Q3) for continuous variables and number (%) for categorical variables. Group comparisons were performed using the Kruskal–Wallis test for continuous variables and Fisher's exact test for categorical variables. Statistical significance was set at $p<0.05$.

Group definitions: G1 (no prior exercise experience, no intervention exposure), G2 (prior exercise experience, no intervention exposure), G3 (no prior exercise experience, intervention exposure), and G4 (prior exercise experience, intervention exposure).

BMI: body mass index, SMM: skeletal muscle mass, SBP: systolic blood pressure, DBP: diastolic blood pressure, TG: triglyceride, HDL: high-density lipoprotein, LDL: low-density lipoprotein, K-MMSE: Korean version of Mini-Mental State Examination, ADAS-Cog: Alzheimer's Disease Assessment Scale–Cognitive Subscale.

fitness, or cognitive function, except for blood pressure. The intervention group showed higher baseline systolic blood pressure (SBP; 139.2±18.6 mmHg vs. 129.8±17.5 mmHg, $p=0.049$) and diastolic blood pressure (DBP; 79.8±10.2 mmHg vs. 71.5±7.8 mmHg, $p=0.001$) compared with the control group.

Baseline characteristics by subgroup revealed no significant differences in demographics or body composition (**Table 3**). However, significant differences were observed for DBP ($p=0.012$), 30-second sit-to-stand test ($p=0.006$), and 2-minute stationary march ($p=0.027$). Cognitive and biochemical markers did not differ significantly across the four subgroups.

### Primary cognitive outcomes (2-group analysis)

As shown in **Table 4** and **Supplementary Fig. 1**, linear mixed-effects models indicated no significant group-by-time interactions for K-MMSE ($p=0.581$) or ADAS-Cog ($p=0.964$). No significant main effects of group were found (K-MMSE: $p=0.199$; ADAS-Cog: $p=0.339$). However, a significant main effect of time was observed for ADAS-Cog ($p<0.001$), reflecting overall improvements in cognitive performance over the 20-week intervention period. *Post hoc* analyses showed significance within-group improvements in ADAS-Cog scores in both the intervention group (from 17.0±5.3 to 14.9±7.1, $p=0.007$) and the control group (from 15.8±4.1 to 13.5±4.8, $p=0.004$), without between-group differences.

**Table 4.** Linear mixed-effects model results for primary cognitive outcomes

| Outcome | Group effect (*p*) | Time effect (*p*) | Group × time interaction (*p*) |
|---|---|---|---|
| K-MMSE | 0.199 | 0.234 | 0.581 |
| ADAS-Cog | 0.319 | <0.001 | 0.964 |

The *p*-values represent fixed effects of group, time, and group × time interaction based on linear mixed-effects models. Group classification was based on intervention assignment: intervention group vs. control group.
K-MMSE: Korean version of the Mini-Mental State Examination, ADAS-Cog: Alzheimer's Disease Assessment Scale–Cognitive Subscale.

### Subgroup analysis based on exercise experience and intervention exposure (G1–G4)

Regression results for subgroup comparisons are presented in **Table 5** and **Fig. 2**, with G1 as the reference group. To ensure transparency and alignment with reporting guidelines, all pre-specified primary and secondary outcomes were reported regardless of statistical significance. SBP decreased significantly in G2 (β=–14.30; 95% CI, –26.45 to –2.15; *p*=0.022), G3 (β=–14.35; 95% CI, –27.65 to –1.04; *p*=0.035), and G4 (β=–13.13; 95% CI, –25.72 to –0.53; *p*=0.041) compared with G1, whereas DBP did not differ significantly across subgroups. For physical fitness, G2 showed significantly poorer performance in the 30-second sit-to-stand test (β=–4.60; 95% CI, –8.26 to –0.95; *p*=0.015) and the 2-minute stationary march (β=–13.30; 95% CI,

**Table 5.** Adjusted group effects on outcomes from multiple linear regression models

| Dependent (Δ) | Coefficient (95% CI) | *p*-value |
|---|---|---|
| Blood pressure | | |
| SBP | | |
| G1 | - | - |
| G2 | −14.30 (−26.45 to −2.15) | 0.022 |
| G3 | −14.35 (−27.65 to −1.04) | 0.035 |
| G4 | −13.13 (−25.72 to −0.53) | 0.041 |
| DBP | | |
| G1 | - | - |
| G2 | 0.61 (−8.11 to 9.33) | 0.888 |
| G3 | 0.53 (−9.12 to 10.17) | 0.913 |
| G4 | 1.40 (−7.73 to 10 .54) | 0.759 |
| Physical fitness | | |
| 30-sec sit-to-stand test | | |
| G1 | - | - |
| G2 | −4.60 (−8.26 to −0.95) | 0.015 |
| G3 | −1.06 (−5.32 to 3.21) | 0.620 |
| G4 | 0.86 (−2.91 to 4.63) | 0.647 |
| 2-min stationary march | | |
| G1 | - | - |
| G2 | −13.30 (−23.76 to −2.84) | 0.014 |
| G3 | 0.19 (−11.39 to 11.77) | 0.974 |
| G4 | −3.99 (−14.77 to 6.78) | 0.459 |
| Sit-and-reach test | | |
| G1 | - | - |
| G2 | −0.92 (−4.47 to 2.62) | 0.603 |
| G3 | 3.25 (−0.60 to 7.11) | 0.096 |
| G4 | 3.17 (−0.46 to 6.79) | 0.085 |
| 3-m sit-walk-and-return test | | |
| G1 | - | - |
| G2 | 0.21 (−0.21 to 0.63) | 0.318 |
| G3 | −0.10 (−0.56 to 0.36) | 0.661 |
| G4 | −0.35 (−0.79 to 0.08) | 0.108 |
| Figure-8-walk | | |
| G1 | - | - |
| G2 | 1.70 (−0.05 to 3.44) | 0.056 |
| G3 | −0.44 (−2.34 to 1.47) | 0.646 |
| G4 | −1.13 (−2.93 to 0.67) | 0.211 |

(continued to the next page)

**Table 5.** (Continued) Adjusted group effects on outcomes from multiple linear regression models

| Dependent (Δ) | Coefficient (95% CI) | *p*-value |
|---|---|---|
| Hand grip (left) | | |
| G1 | - | - |
| G2 | −0.96 (−3.87 to 1.95) | 0.510 |
| G3 | 0.47 (−2.72 to 3.66) | 0.769 |
| G4 | −2.41 (−5.41 to 0.59) | 0.113 |
| Hand grip (right) | | |
| G1 | - | - |
| G2 | −1.13 (−4.24 to 1.99) | 0.470 |
| G3 | 1.63 (−1.79 to 5.04) | 0.343 |
| G4 | −1.55 (−4.78 to 1.69) | 0.341 |
| Cognitive dysfunction | | |
| K-MMSE | | |
| G1 | - | - |
| G2 | 1.66 (0.23 to 3.09) | 0.024 |
| G3 | 0.43 (−1.07 to 1.94) | 0.565 |
| G4 | 1.04 (−0.43 to 2.52) | 0.161 |
| ADAS-Cog | | |
| G1 | - | - |
| G2 | 0.86 (−3.85 to 5.58) | 0.715 |
| G3 | 0.48 (−4.61 to 5.58) | 0.849 |
| G4 | 1.47 (−3.35 to 6.29) | 0.542 |
| Blood biomarkers | | |
| TG | | |
| G1 | - | - |
| G2 | 33.86 (−6.60 to 74.32) | 0.099 |
| G3 | 33.92 (−10.84 to 78.68) | 0.134 |
| G4 | 15.67 (−26.13 to 57.47) | 0.454 |
| Total cholesterol | | |
| G1 | - | - |
| G2 | 0.57 (−30.14 to 29.00) | 0.969 |
| G3 | 5.79 (−26.65 to 38.23) | 0.721 |
| G4 | −0.12 (−30.86 to 30.61) | 0.994 |
| HDL | | |
| G1 | - | - |
| G2 | −5.64 (−13.64 to 2.36) | 0.162 |
| G3 | −2.31 (−11.01 to 6.39) | 0.596 |
| G4 | −1.88 (−10.19 to 6.43) | 0.654 |
| LDL | | |
| G1 | - | - |
| G2 | −0.40 (−26.77 to 25.97) | 0.976 |
| G3 | 9.56 (−19.32 to 38.43) | 0.509 |
| G4 | −1.10 (−28.45 to 26.24) | 0.936 |
| Cortisol | | |
| G1 | - | - |
| G2 | −0.99 (−3.37 to 1.38) | 0.405 |
| G3 | 0.35 (−2.29 to 2.99) | 0.789 |
| G4 | 1.07 (−1.38 to 3.57) | 0.385 |
| Serotonin | | |
| G1 | - | - |
| G2 | −9.02 (−32.49 to 14.44) | 0.443 |
| G3 | 0.88 (−24.75 to 26.50) | 0.945 |
| G4 | −3.26 (−27.79 to 21.27) | 0.791 |

Regression coefficients (β), 95% CIs, and *p*-values were obtained from multiple linear regression models adjusted for baseline values, age, sex, education, and exercise adherence. G1 was used as the reference group.

Group definitions: G1 (no prior exercise experience, no intervention exposure), G2 (prior exercise experience, no intervention exposure), G3 (no prior exercise experience, intervention exposure), and G4 (prior exercise experience, intervention exposure).

CI: confidence interval, SBP: systolic blood pressure, DBP: diastolic blood pressure, K-MMSE: Korean version of Mini-Mental State Examination, ADAS-Cog: Alzheimer's Disease Assessment Scale–Cognitive Subscale, TG: triglyceride, HDL: high-density lipoprotein, LDL: low-density lipoprotein.

–23.76 to –2.84; *p*=0.014) relative to G1, indicating detraining effects among participants with prior exercise experience who were not exposed to the intervention. No significant subgroup differences were detected for flexibility, balance, agility, or hand grip strength, although several outcomes demonstrated non-significant trends favoring the higher-adherence groups (G3 and G4). For cognitive outcomes, K-MMSE scores improved significantly in G2 (β=1.66; 95% CI, 0.23 to 3.09; *p*=0.024), whereas no significant changes were observed in G3 or G4, and ADAS-Cog scores did not differ significantly across subgroups. Finally, no statistically significant subgroup differences were detected for lipid profiles, cortisol, or serotonin levels, although effect estimates for cortisol and serotonin varied across subgroups without a consistent pattern, suggesting heterogeneity in neuroendocrine responses.

As illustrated in **Fig. 2**, subgroup G2 demonstrated multiple significant changes, including higher K-MMSE scores but lower physical fitness and reduced blood pressure compared

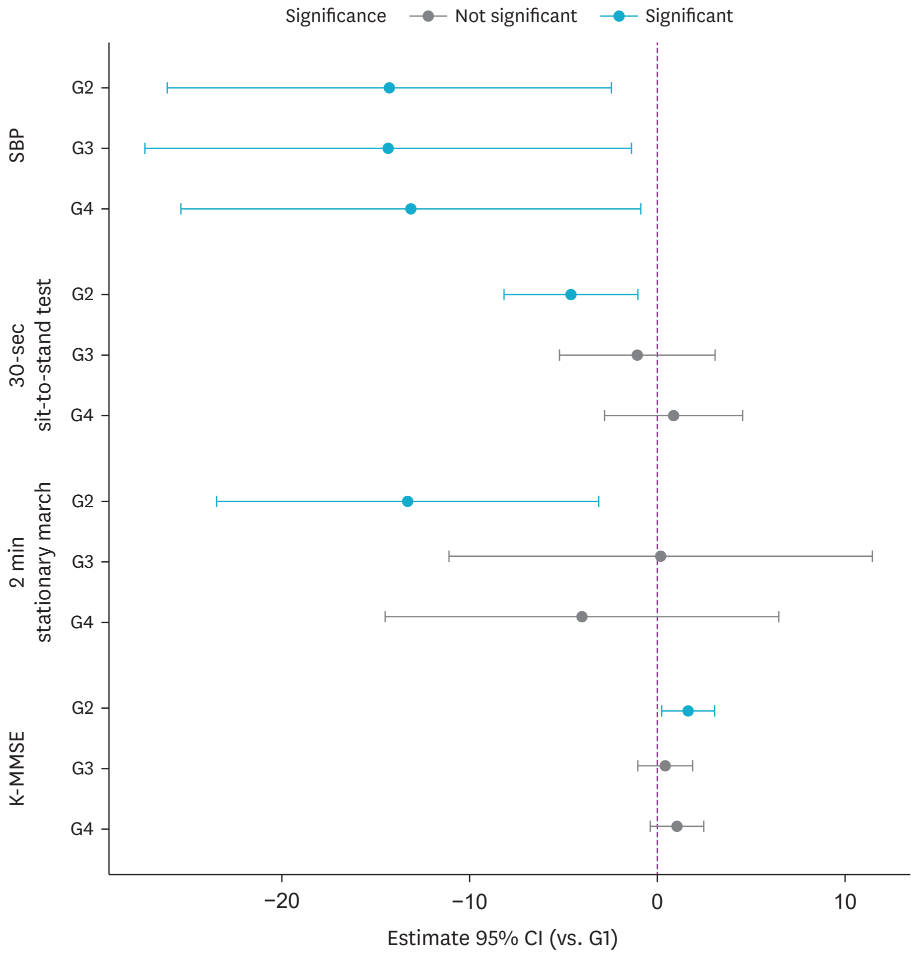

**Fig. 2.** Forest plot of group effects on outcome measures adjusted for covariates (vs. G1). Forest plot showing adjusted regression coefficients (β) and 95% CIs for G2, G3, and G4 relative to the reference group (G1). Estimates were adjusted for baseline values, age, sex, education, and post-intervention physical activity. Blue markers indicate statistically significant effects (*p*<0.05); gray markers are non-significant.
Group definitions: G1 (no prior exercise experience, no intervention exposure), G2 (prior exercise experience, no intervention exposure), G3 (no prior exercise experience, intervention exposure), and G4 (prior exercise experience, intervention exposure). ADAS-Cog was excluded due to non-significant findings across all groups.
SBP: systolic blood pressure, K-MMSE: Korean version of the Mini-Mental State Examination, CI: confidence interval, ADAS-Cog: Alzheimer's Disease Assessment Scale–Cognitive Subscale.

with G1. In contrast, G3 and G4 maintained relatively stable outcomes without marked deterioration or further improvement. These results suggest that lack of intervention exposure among previously physically active individuals (G2) may negatively influence outcomes, whereas continued intervention participation (G3, G4) supports the preservation of baseline function.

# DISCUSSION

This study examined the effects of a 20-week structured dementia prevention exercise program on cognitive function, physical fitness, vascular health, and blood biomarkers in community-dwelling older adults, with particular attention to baseline exercise experience and program adherence. The primary analyses revealed no significant group-by-time interactions for global cognition; however, a significant time effect on ADAS-Cog indicated general improvements across both groups, and subgroup analyses highlighted the potential importance of prior exercise experience and sustained participation in shaping intervention outcomes.

Participants with prior exercise experience who were not exposed to the intervention (G2) demonstrated significant improvements in K-MMSE scores despite concomitant declines in 30-second sit-to-stand and 2-minute stationary march performance compared with inactive reference group (G1). In contrast, those who participated in the intervention (G3 and G4) generally maintained more stable physical fitness, with G4 showing a slight but non-significant positive trend in 30-second sit-to-stand performance. This pattern is unlikely to reflect superior intervention efficacy in G2 and is more plausibly attributable to residual cognitive reserve accumulated through long-term exercise habits, in combination with the limited sensitivity and potential ceiling effects of the K-MMSE in cognitively intact older adults; the absence of corresponding benefits in ADAS-Cog further supports a cautious interpretation of the cognitive changes observed in these subgroups.

The absence of significant group-by-time interactions for ADAS-Cog, despite overall improvement over time, may partly reflect practice effects or natural variability rather than clear intervention-specific benefits. Previous work has indicated that repeated exposure to cognitive tests can yield modest score gains, particularly among non-demented older adults,[23,24] suggesting that learning effects may have contributed to the changes observed in this study, and underscoring the need for cognitive outcome measures that are less susceptible to practice effects in future exercise-based dementia prevention trials.[25] Considering the limited sensitivity and potential ceiling effects of K-MMSE, relative differences across subgroups may therefore reflect baseline cognitive reserve rather than short-term intervention effects; importantly, parallel improvements across both groups indicate that learning or practice effects may also have contributed to the observed cognitive changes. The cognitive gains observed in G2 may represent residual benefits of long-term exercise habits on cognitive reserve, aligning with evidence that physically active older adults demonstrate less decline and better executive functioning than their sedentary peers, and with studies linking higher levels of moderate-to-vigorous activity to larger hippocampal volumes in older adults,[26-29] suggesting that accumulated exercise history can bolster resilience against cognitive deterioration even after structured training ends.

Physical performance outcomes revealed marked detraining effects in G2, highlighting the necessity of continued participation for maintaining muscular and aerobic capacity.

Previous research shows that detraining leads to rapid declines in muscle strength and aerobic fitness, particularly among older adults, whereas long-term exercisers tend to achieve superior functional capacity and balance compared with less active counterparts.[15,16,30,31] In the present study, G3 and G4 maintained relatively stable performance, reinforcing the protective role of continued intervention participation and suggesting that prior exercise experience may preferentially contribute to cognitive reserve, whereas sustained adherence appears essential for the preservation of physical function. This pattern appears to align with previous research on physical activity trajectories in individuals with MCI, which categorizes participants into "withdrawal," "initiation," and "maintenance" activity patterns and reported differential risks of functional decline and dementia progression across these groups. Within this framework, G2 may approximate a withdrawal trajectory, whereas G3 and G4 more closely resemble initiation or maintenance trajectories, tentatively highlighting the importance of sustained engagement in physical activity for preserving function and potentially mitigating dementia risk.[32]

Vascular adaptations provided additional evidence of the benefits of sustained engagement, as SBP was significantly reduced in all subgroups with prior exercise experience or intervention exposure (G2–G4), consistent with previous evidence that multicomponent exercise programs improve vascular health and reduce dementia risk.[33-35] Notably, these effects were observed regardless of baseline physical fitness, demonstrating that structured, supervised exercise can yield cardiovascular benefits even in previously inactive individuals, which is particularly relevant given the established links between midlife hypertension and dementia and supports the integration of structured exercise into community-based dementia prevention strategies.[36,37]

With respect to neuroendocrine biomarkers, no statistically significant subgroup differences were detected for cortisol or serotonin levels. Although these markers were assessed as exploratory outcomes reflecting stress and neurobiological regulation, the heterogeneous directions of effect across subgroups suggest substantial inter-individual variability rather than consistent intervention-related changes. These findings imply that longer intervention durations, repeated measurements, or more tightly controlled study designs may be necessary to elucidate the responsiveness of cortisol and serotonin to structured exercise interventions in older adults.

Taken together, the findings tentatively highlight the multidimensional impact that structured exercise may have on cognitive, physical, and vascular health in older adults. Prior exercise experience appeared to provide some residual protection related to cognitive reserve, whereas sustained adherence seemed essential for preserving physical capacity and realizing cardiovascular benefits, supporting the notion that future dementia prevention programs should consider participants' baseline activity levels and incorporate behavioral strategies—such as personalized goal setting, motivational support, and social engagement—to encourage long-term adherence to multicomponent exercise interventions.[38,39]

Strengths of this study include its supervised, multicomponent design, objective monitoring of exercise intensity, and novel subgroup analyses considering prior exercise experience and adherence. Nonetheless, several limitations should be acknowledged, including non-randomized allocation, modest sample size, and the absence of controls for dietary, psychosocial, and sleep factors, as well as the potential influence of practice effects or spontaneous lifestyle changes on cognitive outcomes. Moreover, given the exploratory nature of subgroup comparisons and the number of outcomes assessed, the findings should be regarded as hypothesis-generating rather than confirmatory; future research with randomized controlled designs, larger and more diverse samples, and extended follow-up is needed to confirm these results and establish the long-term efficacy of exercise-based dementia prevention, and integrating lifestyle factors together with adherence-enhancing strategies will also be critical for optimizing scalability and effectiveness at the community level.

## SUPPLEMENTARY MATERIAL

### Supplementary Fig. 1

Changes in cognitive function from baseline (Time 1) to post-intervention (Time 2) in the Tr and Ct groups. (A) Changes in K-MMSE scores. Higher scores indicate better global cognitive function. (B) Changes in ADAS-Cog scores. Lower scores indicate better cognitive performance. Both graphs present mean values at each time point. Blue lines indicate the Tr group, and red lines indicate the Ct group.

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