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Effects of 8-Week High-Dose Vitamin D Supplementation on Physical Performance in Female National League Handball Players During the Competitive Season

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Abstract

Background: Vitamin D insufficiency is common among indoor athletes, but supplementation effects on performance remain uncertain. This randomized, double-blind, placebo-controlled study examined eight-week vitamin D_3 supplementation in 20 female national level (second-division) handball players during the competitive season. **Methods:** Participants were randomized within pairs matched for baseline serum 25-hydroxyvitamin D [25(OH)D] to receive vitamin D_3 (5000 IU/day; $n = 10$) or placebo ($n = 10$). Serum 25(OH)D, parathyroid hormone, calcium, phosphorus, 10 m and 30 m sprint times, 20 m shuttle-run distance, Wingate peak power, and total work were assessed before and after supplementation. Exploratory between-group comparisons of change used Holm adjustment across nine outcomes. **Results:** Serum 25(OH)D increased from 26.70 ± 11.12 to 42.96 ± 16.20 ng/mL with supplementation and from 27.05 ± 8.01 to 29.65 ± 7.67 ng/mL with placebo. The between-group difference in change was 13.66 ng/mL (95% confidence interval: 7.32–20.00; Holm-adjusted $p = 0.002$). No other outcome showed a significant time-by-group interaction after adjustment. Confidence intervals for all between-group performance contrasts included zero. **Conclusions:** Vitamin D_3 supplementation improved vitamin D status, but a supplementation-specific performance benefit was not established. Given the small sample and imprecise estimates, these findings do not establish equivalence or exclude meaningful performance effects.

Keywords: vitamin D3; 25-hydroxyvitamin D; female athletes; handball; supplementation; sprint performance; anaerobic power

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1. Introduction

Vitamin D is increasingly recognized as a multifunctional secosteroid hormone with essential roles extending beyond classical skeletal metabolism into multiple physiological systems relevant to human performance and health [1–4]. Traditionally associated with calcium and phosphorus homeostasis, vitamin D contributes to bone mineralization and

endocrine regulation; however, growing evidence indicates that it also influences skeletal muscle function, immune regulation, inflammation and metabolic adaptation [5–8]. These broader physiological effects have positioned vitamin D as a central micronutrient within contemporary sports nutrition research, particularly in athletic populations exposed to high training loads and repetitive neuromuscular stress [4,9–11].

Vitamin D status is primarily assessed through circulating concentrations of 25-hydroxyvitamin D [25(OH)D], which reflect both dietary intake and endogenous synthesis via ultraviolet B exposure [2,5]. Despite its importance, vitamin D insufficiency remains highly prevalent worldwide, including among national level athletes [7,12,13]. Recent epidemiological investigations report that a substantial proportion of athletes exhibit suboptimal 25(OH)D concentrations during the competitive season, especially in indoor sports and regions characterized by limited sunlight exposure [9,14–17]. Female athletes may represent an especially vulnerable subgroup due to combined influences of training environment, lifestyle factors and nutritional variability [6,18–20].

From a mechanistic perspective, vitamin D may influence athletic performance through several biological pathways. Vitamin D receptors (VDRs) are expressed in skeletal muscle tissue, suggesting direct genomic effects on muscle protein synthesis, fiber composition and contractile function [1,5,21,22]. Experimental studies demonstrate that vitamin D signaling contributes to calcium transport regulation within muscle cells, mitochondrial function and neuromuscular coordination [15,23]. Additionally, vitamin D may modulate inflammatory responses and recovery processes following intense exercise, potentially affecting adaptation to training stimuli [4,24–26].

Within the sports nutrition literature, increasing attention has been directed toward endocrine interactions between vitamin D and calcium–phosphate metabolism. Adequate vitamin D levels regulate parathyroid hormone (PTH) secretion and maintain mineral balance essential for neuromuscular excitability and muscle contraction efficiency [1–3,5]. Alterations in the vitamin D–PTH axis have been associated with fatigue and impaired muscle function, highlighting the importance of examining biochemical markers alongside performance outcomes [7,9,16,25].

Despite strong mechanistic rationale, empirical findings regarding vitamin D supplementation and athletic performance remain inconsistent. Several randomized controlled trials and meta-analyses report improvements in muscle strength and sprint capacity following supplementation, particularly in individuals presenting baseline deficiency [10,12,16,23,27]. Conversely, other investigations demonstrate minimal performance benefits when baseline vitamin D concentrations are sufficient [7,9,28]. These conflicting findings likely reflect heterogeneity in supplementation protocols, dosage strategies, intervention duration, athlete characteristics and methodological approaches used to assess performance outcomes [4,6,11,29].

The recent literature emphasizes that performance improvements may depend primarily on correcting insufficiency rather than elevating already adequate vitamin D levels [7,12,16,28]. Furthermore, inter-individual variability in response to supplementation has emerged as an important research topic, with athletes exhibiting heterogeneous biochemical and functional responses despite similar dosing regimens [4,9,24,30].

Team sports represent a particularly relevant model for investigating these interactions due to their complex physiological requirements. Handball performance depends on repeated sprint ability, agility, jumping capacity and intermittent aerobic fitness [31–33]. These characteristics rely heavily on neuromuscular efficiency and metabolic regulation, systems potentially influenced by vitamin D-mediated pathways [4,7,15,16]. However, controlled experimental studies examining vitamin D supplementation in national level female handball athletes remain limited.

Female athletes remain underrepresented in sports nutrition research despite potential sex-specific physiological responses related to hormonal regulation, body composition and recovery dynamics [34,35]. Seasonal factors further complicate vitamin D dynamics in athletes, as indoor training and reduced sunlight exposure during competition periods may lead to declining 25(OH)D concentrations despite improvements in fitness [7,9,16].

Given inconsistencies in the literature and the limited number of controlled intervention studies involving national level female team-sport athletes, further research integrating biochemical and functional outcomes is warranted. Therefore, the aim of the present study was to investigate the effects of eight weeks of high-dose vitamin D_3 supplementation on vitamin D status, selected biochemical markers of calcium–phosphate–PTH homeostasis, and physical performance in national level female handball players during the competitive season.

2. Materials and Methods

Participants

All participants were members of the same team, competing in the Romanian National Handball League (2nd division) and were students at the University of Oradea. Most players (70%) were living in the university dormitory and shared a broadly similar living and meal environment. No vitamin D-containing supplements were provided by the research team before baseline or outside the assigned intervention.

No a priori sample size calculation was performed. The sample size was determined pragmatically by the number of eligible players available in a single national level women's handball team during the competitive season. Because recruiting players from multiple teams at the same competitive level under comparable training conditions was not feasible, the present study should be considered a pragmatic controlled intervention conducted in a real-world team setting.

The study was approved by the Ethics Committee of the University of Oradea (approval reference number FGTS_EFSK_5/16.02.2023) and was conducted in accordance with the principles of the Declaration of Helsinki. Before participation, all players received a full explanation of the study aims, procedures, potential risks and benefits and provided written informed consent.

Inclusion and Exclusion Criteria

The inclusion criteria were as follows: all players had to be members of the same club, follow the same training program, maintain a broadly similar diet and participate in more than 85% of training sessions. Of the 22 players on the team roster, two were excluded before baseline testing and allocation because prolonged injuries prevented them from completing the initial assessments. A total of 20 players met these criteria and were considered during the final analysis. This study design and participant allocation are illustrated in Figure 1.

Allocation procedure

Participants were allocated to the vitamin D_3 supplementation or placebo group using matched-pair randomization based on baseline serum 25-hydroxyvitamin D [25(OH)D] concentrations. Following baseline blood sampling, the 20 participants were ranked in descending order of serum 25(OH)D concentration. Adjacent participants were paired (ranks 1 and 2, 3 and 4, and so forth), forming 10 pairs with similar baseline vitamin D status. Within each pair, allocation was determined by drawing lots, with one participant assigned to the vitamin D_3 group and the other to the placebo group. This procedure resulted in two groups of 10 participants and was intended to balance baseline vitamin D status between groups.

Group codes were generated and held by an investigator not involved in testing or data analysis. Supplements were dispensed in identical bottles labeled with anonymous codes.

Participants, coaching staff and all assessors responsible for anthropometric measurements, performance testing and blood sampling were blinded to group assignment throughout the intervention and data processing. Blinding was maintained until completion of statistical analysis.

The supplementation phase was not prospectively registered.

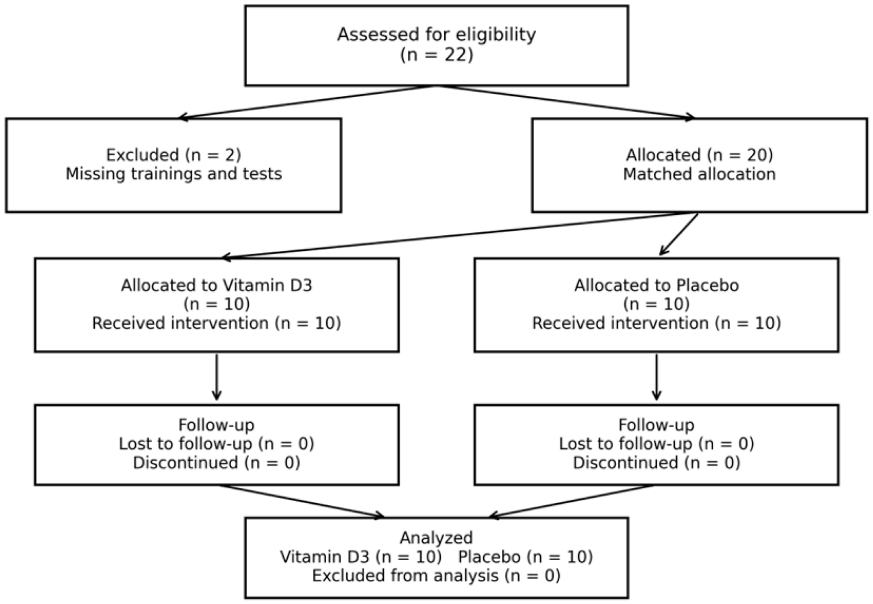


Figure 1. CONSORT-style participant flow for the supplementation phase (T1–T2).

Study Design

The study followed a parallel-group, double-blind, placebo-controlled intervention design conducted during the in-season period, from April to May 2024, in a single national level women's handball club. Players were allocated 1:1 to a vitamin D_3 group (VitD) or a placebo group (PL), with two measurement time points: baseline (T1) and post-intervention (T2). Blood sampling was performed on 04 April and 03 June, field tests on 05 April and 04 June and Wingate tests on 06 April and 05 June. The intervention lasted eight weeks.

Intervention

The VitD group received a high-dose oral vitamin D_3 supplement of 5000 IU/day (cholecalciferol, oil-based liquid preparation), administered as drops once daily in the morning with breakfast. The placebo group received an identical oil-based preparation without vitamin D_3, matched for appearance, viscosity and flavor. Supplements were provided in identical bottles and dosed with the same dropper system.

The intervention period lasted eight weeks, from the first blood sampling in early April to the final sampling and performance testing at the beginning of June. Players were instructed to maintain their usual diet, not to initiate any additional vitamin D or calcium supplements and to keep their habitual caffeine and alcohol intake stable.

Compliance monitoring. Supplementation was administered in liquid form and compliance was supported by a daily reminder message posted by the coach in the team group chat. In addition, the coach performed periodic bottle checks during the intervention and all bottles were returned at the end of the study. Residual bottle volume was not objectively quantified; therefore, an exact adherence percentage could not be calculated.

Calculation of Average Vitamin D Intake

Dietary vitamin D and calcium intakes were estimated exclusively from food sources using Nutrilog software (v2.10d) and the CIQUAL food composition database [36]. These

estimates excluded vitamin D administered as part of the study intervention and therefore represent dietary intake rather than total vitamin D exposure. Each athlete completed a nutritional questionnaire incorporating a seven-day dietary record (7-DR) during the week preceding the baseline and post-intervention assessments. For seven consecutive days, participants recorded the types and weights of foods consumed at breakfast, lunch, and dinner, as well as foods consumed between meals.

In the VitD group, mean daily dietary vitamin D intake was 11.85 ± 1.49 µg/day at baseline and 11.96 ± 1.57 µg/day after the intervention ($p = 0.051$). In the placebo group, the corresponding values were 11.69 ± 1.71 and 12.04 ± 1.48 µg/day ($p = 0.057$). Neither group showed a statistically significant within-group change in dietary vitamin D intake. Mean daily dietary calcium intake increased from 870 ± 120 to 900 ± 145 mg/day in the VitD group ($p = 0.049$) and from 880 ± 130 to 890 ± 140 mg/day in the placebo group ($p = 0.042$). All values are presented as mean ± standard deviation. The dietary reference intake for vitamin D in this age group is 15 µg/day (1 µg = 40 IU). Dietary vitamin D intake was assessed as a control variable to determine whether changes in habitual food intake could have contributed to the observed changes in serum 25(OH)D. Dietary intake was not considered an intervention outcome because the study did not include a dietary modification. Historical monthly average UV index values for April and May, corresponding to the intervention months, were obtained from World Weather Online. These data were used to characterize the general ambient UV radiation conditions during the study period (Figure 2). However, the reported values represent historical climatic averages rather than direct measurements from April–May 2024 and do not reflect individual sunlight exposure.

UV Index

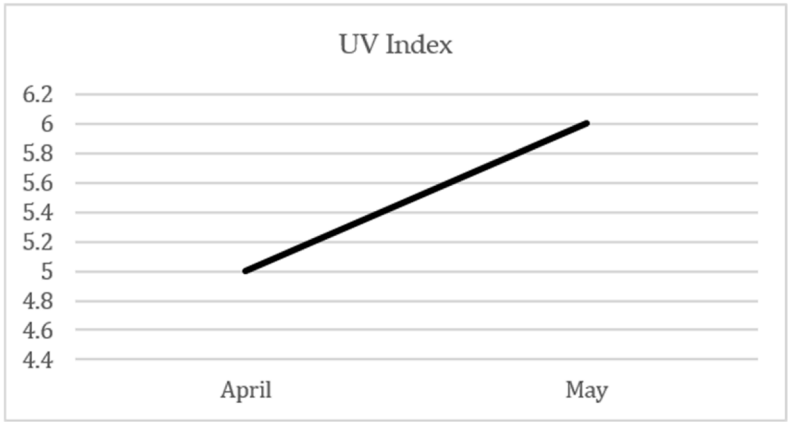


Figure 2. UV index values in Oradea (Romania) during the study period (source: https://www.worldweatheronline.com/oradea-weather-averages/bihor/ro.aspx, accessed on 10 June 2026).

Training load

The intervention covered an 8-week period from early April to late May. During the competitive season, players completed two daily training sessions on two days per week. The standard weekly schedule comprised seven training sessions and one competitive match. Session duration varied, resulting in approximately 15–17 h of combined training and match exposure per week, with an average of approximately 950 min per week over the intervention period, including the competitive match. Attendance and session duration were recorded in the coach's training diary throughout the intervention. These records were used to monitor training attendance and weekly exposure and to assess compliance with the minimum attendance criterion of 85% of training sessions.

Training load (min) considering the type (overall and special drills) and intensity (aerobic, anaerobic) of exercises of typical weekly training during competitive season, implemented during the research project is shown in Table 1.

Table 1. Overview of the typical weekly training load during competitive season.

Preparation Period	**Training Drills**	
Day of the Week	Morning	Afternoon
Monday	strength and speed, technical individual training (formation)	technical, tactical, aerobic power
Tuesday	-	speed endurance, technical, tactical
Wednesday	-	technical, tactical, mixt aerobic-anaerobic
Thursday	plyometric and change of direction speed, technical individual training (formation)	technical, tactical, aerobic capacity
Friday	-	agility, technical, tactical
Saturday	competition game	-
Sunday	Day off (recovery)	

Anthropometry and body composition

Body height (cm) was measured using a stadiometer with players barefoot. Body mass (kg), body fat mass (BFM, kg), skeletal muscle mass (SMM, kg), body mass index (BMI, kg/m^2) and percent body fat (PBF, %) were assessed by InBody 770 bioelectrical impedance analysis using the same device and protocol for all participants. Measurements were conducted in the morning, in a fasted state when feasible, after voiding and under standardized hydration conditions, with no intense exercise within the previous 24 h. Body fat mass, skeletal muscle mass and percent body fat were assessed as descriptive variables to characterize the participants and to identify potential changes in body composition that could influence the interpretation of the performance outcomes. These variables were not considered primary or secondary outcomes of the supplementation intervention.

Blood markers

Venous blood samples were collected from an antecubital vein in the morning under fasting conditions at baseline (T1) and post-intervention (T2). Analyses were performed by an accredited analytical laboratory (Synevo Romania SRL, Oradea, Romania). All four analytes were measured in serum. Serum 25-hydroxyvitamin D [25(OH)D, ng/mL] and intact parathyroid hormone (PTH, pg/mL) were measured using electrochemiluminescence immunoassays, while total calcium and phosphorus (mg/dL) were measured using colorimetric spectrophotometric methods. The laboratory reference intervals were 15–65 pg/mL for intact PTH, 8.6–10.0 mg/dL for total calcium and 2.5–4.5 mg/dL for phosphorus. For descriptive reporting, serum 25(OH)D concentrations were grouped into the following intervals: <20, 20 to <30, 30 to <56, 56 to 100, and >100 ng/mL.

Field performance tests—sprint performance and Beep Test

Field tests were conducted on an indoor handball court under consistent environmental conditions, using a standardized warm-up before each test battery. All participants were familiar with the full performance test battery. They had completed all these tests on four occasions before the present baseline assessment as part of earlier research assessments.

The baseline and post-intervention assessments reported here therefore represented their fifth and sixth administrations of the performance test battery, respectively.

Sprint Performance

10 m and 30 m sprint tests

Before testing, players completed a 20 min standardized warm-up including two 10 m sprints and one 30 m sprint. Sprint times were recorded using double photocell timing gates (Witty Gate system, Microgate, Bolzano, Italy) positioned at the start and finish lines, with the start line placed 0.3 m behind the first timing gate. Timing gates were positioned at a height of 0.7 m. Each player performed two maximal attempts over 10 m and 30 m and the best time for each distance was retained for analysis. Recovery intervals were 120 s after the 10 m sprint and 240 s after the 30 m sprint.

Beep Test

Intermittent aerobic capacity was evaluated using the standard 20 m progressive shuttle run test described by Léger et al. (1988) [37]. The test started at 8.5 km·h^{-1}, with speed increasing by 0.5 km·h^{-1} each minute. The test was performed on an indoor handball court using two marked lines placed 20 m apart. Running pace was controlled by standardized audio signals and the test was terminated when the player failed twice consecutively to reach the required line in time or stopped voluntarily. Cumulative distance covered was recorded upon the athlete's volitional exhaustion or failure to maintain synchronization.

Wingate Anaerobic Test

Anaerobic power production and fatigue characteristics were assessed using the 30 s Wingate Anaerobic Test, following established methodological recommendations [38]. The test was performed on a mechanically braked cycle ergometer (Monark LC7TT, Sweden). After a standardized 5 min warm-up at approximately 50 W, including two 3 s run-up practices against the actual test load, athletes performed one all-out 30 s bout against a braking load equivalent to 7.5% of individual body mass. Peak power output (W) and total work (J) were derived from the ergometer software for subsequent analysis.

At baseline and follow-up, the field tests were performed in a fixed sequence: the 10 m sprint, the 30 m sprint, and the 20 m shuttle-run test (Beep test). A five-minute recovery interval was provided between the different field tests, in addition to the recovery intervals between individual sprint attempts specified above. The Wingate anaerobic test was conducted 24 h after the field-testing session. Assessments were performed at the same time of day, using the same test-specific warm-up procedures, testing equipment, and assessors at both time points. The type of training undertaken on the preceding day was also kept consistent between the two assessment periods.

Statistical analysis

Continuous variables are presented as mean ± standard deviation (SD). All 20 participants were included in the analyses (10 per group). No data were missing for the reported outcomes; therefore, no imputation was required. As no single primary outcome was prospectively designated, the analyses were considered exploratory.

Between-group differences in response to supplementation were evaluated by comparing individual changes from baseline to post-intervention. For each outcome, the treatment contrast was calculated as the mean change in the vitamin D group minus the mean change in the placebo group (ΔVitD − ΔPlacebo). Differences in change are presented with two-sided 95% confidence intervals (CIs), calculated using the pooled within-group variance of change scores. The corresponding time × group interaction is reported as an F statistic with 1 and 18 degrees of freedom, together with its p-value and partial eta squared (η^2p).

For descriptive interpretation, partial η^2 values of 0.01, 0.06, and 0.14 were used as conventional benchmarks for small, moderate, and large effects, respectively. These benchmarks were interpreted cautiously because effect-size interpretation depends on study

design and context; the magnitude labels do not imply statistical significance or clinical importance [39].

Holm adjustment was applied across the nine time × group comparisons, comprising four biochemical and five performance outcomes, to account for multiple testing. Both unadjusted and adjusted *p*-values are reported. Statistical significance was assessed using Holm-adjusted *p*-values at a two-sided threshold of 0.05. The 95% CIs were not adjusted for multiple comparisons.

Within-group standardized changes in performance were calculated as Cohen's dz, defined as the mean individual change divided by the SD of individual changes, and are presented in Table S1. These effect sizes describe within-group changes and do not establish differences between the interventions. Body-composition measurements are summarized descriptively in Table S2. Vitamin D status categories are presented as counts and percentages at each assessment.

No a priori sample size calculation was performed; the sample size was determined by the number of eligible players available from a single team. A sensitivity power analysis was performed for the two-sided pooled-variance comparison of between-group change scores, corresponding to the reported time × group contrast with one numerator degree of freedom. With $n = 10$ per group, 18 degrees of freedom, and 80% target power, the detectable standardized difference in mean changes was $|d_change| = 1.325$ at $\alpha = 0.05$. Here, d_change denotes the difference in population mean changes divided by the common population standard deviation of change scores. Using $\alpha = 0.05/9$ as a conservative Bonferroni benchmark yielded $|d_change| = 1.813$; this does not represent an exact power calculation for the full Holm procedure. The analysis did not use the observed effect sizes as inputs. These findings characterize the limited sensitivity of the sample but do not determine whether any particular nonsignificant result reflects an absent effect or an undetected effect.

All statistical analyses were performed using JASP Team (2026). JASP (Version 0.99.0) [Computer software].

3. Results

3.1. Participants and Baseline Characteristics

All 20 participants completed baseline and post-intervention assessments and were included in the analyses (10 in the vitamin D group and 10 in the placebo group). No participants were excluded after allocation. Baseline demographic, anthropometric and vitamin D characteristics are summarized in Table 2. Mean baseline 25(OH)D concentrations were 26.70 ± 11.12 ng/mL in the vitamin D group and 27.05 ± 8.01 ng/mL in the placebo group.

Table 2. Baseline characteristics of the participants.

Characteristic	Vitamin D ($n = 10$)	Placebo ($n = 10$)
Age (years)	22.20 ± 3.39	23.70 ± 2.58
Height (cm)	169.10 ± 8.32	168.60 ± 6.29
Body mass (kg)	63.50 ± 10.16	65.35 ± 11.84
BMI (kg/m^2)	22.18 ± 3.01	22.93 ± 3.59
25(OH)D (ng/mL)	26.70 ± 11.12	27.05 ± 8.01

Values are mean ± SD. BMI, body mass index; 25(OH)D, 25-hydroxyvitamin D. Baseline significance tests are omitted; the table describes the observed sample and does not establish equivalence between groups.

3.2. Biochemical Outcomes

Pre- and post-intervention values are reported in Table 3 and between-group differences in change are reported in Table 4. Serum 25(OH)D increased by 16.26 ng/mL in the vitamin

D group and by 2.60 ng/mL in the placebo group. The between-group difference in change was 13.66 ng/mL (95% CI 7.32 to 20.00; F(1,18) = 20.49; unadjusted $p < 0.001$; Holm-adjusted $p = 0.002$; $\eta^2p = 0.532$). No other biochemical outcome showed a significant interaction after adjustment. For serum calcium, the mean change was +0.014 mg/dL in the vitamin D group and −0.225 mg/dL in the placebo group. The between-group difference in change was 0.239 mg/dL (95% CI −0.021 to 0.499), with a conventionally large interaction effect size (partial $\eta^2 = 0.172$), but the interaction was not statistically significant (unadjusted $p = 0.069$; Holm-adjusted $p = 0.554$).

Table 3. Biochemical and performance outcomes before and after the intervention.

Outcome	Vitamin D Baseline	Vitamin D Post	Placebo Baseline	Placebo Post
25(OH)D (ng/mL) *	26.70 ± 11.12	42.96 ± 16.20	27.05 ± 8.01	29.65 ± 7.67
PTH (pg/mL)	28.91 ± 13.09	24.59 ± 7.84	25.66 ± 8.36	26.05 ± 9.79
Calcium (mg/dL)	9.562 ± 0.204	9.576 ± 0.347	9.610 ± 0.209	9.385 ± 0.279
Phosphorus (mg/dL)	4.307 ± 0.409	4.452 ± 0.398	4.286 ± 0.498	4.341 ± 0.427
10 m sprint (s)	1.921 ± 0.063	1.961 ± 0.074	1.924 ± 0.132	1.957 ± 0.145
30 m sprint (s)	4.730 ± 0.153	4.777 ± 0.149	4.759 ± 0.298	4.816 ± 0.344
20 m shuttle run (m)	1406.00 ± 147.59	1438.00 ± 133.48	1490.00 ± 268.20	1502.00 ± 276.08
Peak power (W)	603.00 ± 76.42	612.00 ± 79.86	585.80 ± 60.20	590.30 ± 66.08
Total work (J)	12456.90 ± 1603.25	12662.13 ± 1732.43	12127.47 ± 1435.57	12225.39 ± 1540.70

Values are mean ± SD; $n = 10$ per group at both assessments. PTH, parathyroid hormone; 25(OH)D, 25-hydroxyvitamin D. Post, post-intervention. Inferential comparisons are reported in Table 4. Longer sprint times indicate poorer performance. * Significant time × group interaction after Holm adjustment across the nine outcomes (adjusted $p = 0.002$; see Table 4). The asterisk indicates a between-group difference in change, not a within-group pre–post comparison.

Table 4. Between-group differences in change and time × group interaction results.

Outcome	Δ VitD	Δ Placebo	ΔVitD − ΔPlacebo [95% CI]	F(1,18)	*p*	*p* Holm	η^2p
25(OH)D (ng/mL)	16.26	2.60	13.66 [7.32, 20.00]	20.49	<0.001	0.002	0.532
PTH (pg/mL)	−4.32	0.39	−4.71 [−15.02, 5.60]	0.92	0.350	1.000	0.049
Calcium (mg/dL)	0.014	−0.225	0.239 [−0.021, 0.499]	3.73	0.069	0.554	0.172
Phosphorus (mg/dL)	0.145	0.055	0.090 [−0.350, 0.530]	0.18	0.673	1.000	0.010
10 m sprint (s)	0.040	0.033	0.007 [−0.040, 0.054]	0.10	0.759	1.000	0.005
30 m sprint (s)	0.047	0.057	−0.010 [−0.141, 0.121]	0.03	0.874	1.000	0.001
20 m shuttle run (m)	32.00	12.00	20.00 [−26.20, 66.20]	0.83	0.375	1.000	0.044
Peak power (W)	9.00	4.50	4.50 [−3.15, 12.15]	1.53	0.232	1.000	0.078
Total work (J)	205.23	97.92	107.31 [−134.14, 348.76]	0.87	0.363	1.000	0.046

Δ = post minus baseline. The contrast and its CI have the same units as the outcome. F tests the time × group interaction; η^2p is partial eta squared. CIs are pointwise, unadjusted intervals. Holm adjustment covers all nine comparisons in this table. Values of $p < 0.001$ are displayed as <0.001; the unrounded p for 25(OH)D was approximately 0.000261, giving a Holm-adjusted p of approximately 0.00235. VitD, vitamin D group. Conventional benchmarks for partial η^2 are 0.01, 0.06, and 0.14 for small, moderate, and large effects, respectively. The calcium interaction effect was conventionally large (partial $\eta^2 = 0.172$), whereas the Wingate peak-power interaction effect was moderate (partial $\eta^2 = 0.078$). These magnitude classifications do not indicate statistical significance.

Individual vitamin D status distributions are provided in Table 5. At baseline, 4/10 participants in the vitamin D group and 2/10 in the placebo group had concentrations below

20 ng/mL. At follow-up, no participant had a concentration below 20 ng/mL; 8/10 and 4/10, respectively, had concentrations of at least 30 ng/mL. These categories describe the measured distribution and should not be inferred from group means alone. Individual changes in serum 25(OH)D concentrations from baseline to post-intervention are illustrated in Figure 3.

Table 5. Distribution of 25(OH)D concentrations at baseline and follow-up.

25(OH)D (ng/mL)	Vitamin D Baseline	Vitamin D Post	Placebo Baseline	Placebo Post
<20	4 (40%)	0 (0%)	2 (20%)	0 (0%)
20 to <30	1 (10%)	2 (20%)	5 (50%)	6 (60%)
30 to <56	5 (50%)	6 (60%)	3 (30%)	4 (40%)
56 to 100	0 (0%)	2 (20%)	0 (0%)	0 (0%)
>100	0 (0%)	0 (0%)	0 (0%)	0 (0%)

Values are *n* (%), with $n = 10$ per group. The intervals are operational categories used to describe this sample, not a universal clinical classification. The 30 to <56 ng/mL interval avoids gaps in the original manuscript's category boundaries. None of the observed values fell between 55.5 and 56 ng/mL. Category definitions should be harmonized with Methods and the cited laboratory reference.

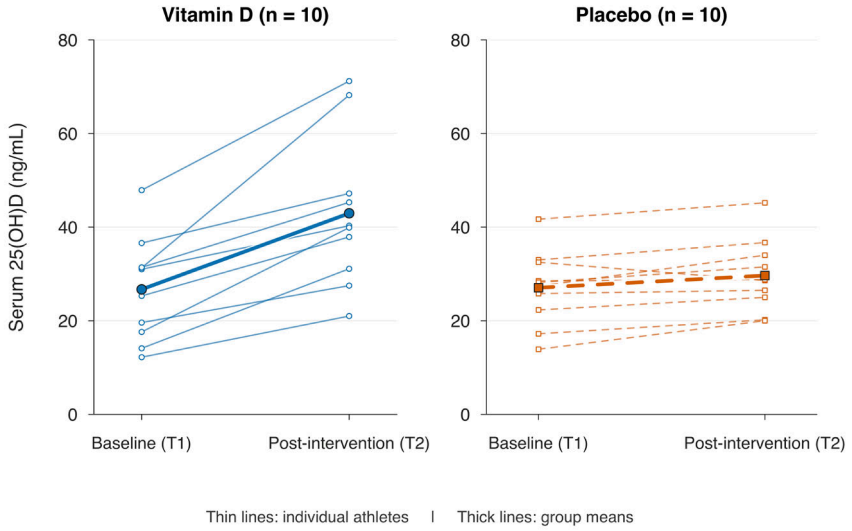


Figure 3. Individual changes in serum 25(OH)D concentrations from baseline (T1) to post-intervention (T2) in the vitamin D and placebo groups ($n = 10$ per group). Thin lines connect measurements from individual athletes; thick lines represent group means. The vitamin D group is shown with blue solid lines and circles, and the placebo group with orange dashed lines and squares.

3.3. Physical Performance

No performance outcome showed a significant time × group interaction, either before or after Holm adjustment (Table 4). The between-group difference in change was 0.007 s for the 10 m sprint (95% CI −0.040 to 0.054) and −0.010 s for the 30 m sprint (95% CI −0.141 to 0.121). Shuttle-run distance increased by 32 m in the vitamin D group and 12 m in the placebo group, yielding a difference of 20 m (95% CI −26.20 to 66.20).

Wingate peak power increased by 9.00 W in the vitamin D group and 4.50 W in the placebo group. The between-group difference in change was 4.50 W (95% CI −3.15 to 12.15), with a conventionally moderate interaction effect size (partial $\eta^2 = 0.078$), but the interaction was not statistically significant (unadjusted $p = 0.232$; Holm-adjusted $p = 1.000$). Total work increased by 205.23 J and 97.92 J, respectively; the difference was 107.31 J (95% CI −134.14 to

348.76). Within-group standardized changes are reported in Table S1. These changes do not establish an intervention effect because the direct between-group comparisons remained inconclusive. Descriptive body-composition data are provided in Table S2.

4. Discussion

The intervention produced a greater increase in serum 25(OH)D than placebo, and this finding remained significant after Holm adjustment across the nine reported outcomes. In contrast, the between-group CIs for all performance outcomes included zero. The data therefore did not establish a supplementation-specific performance benefit, but neither do they establish equivalence or exclude potentially relevant effects. Favorable within-group changes must be distinguished from evidence of a difference between interventions.

The marked increase in serum 25(OH)D in the supplemented group confirms the biological efficacy of the intervention. This finding is consistent with previous evidence showing that vitamin D supplementation reliably increases circulating 25(OH)D concentrations in athletes, especially when baseline values are suboptimal [12,40]. Farrokhyar et al. reported that vitamin D supplementation increased serum 25(OH)D concentrations in athletes, although improvements in physical performance were not consistently observed. Similarly, the systematic review by Wyatt et al. emphasized that vitamin D supplementation may be beneficial in national level athletes but that the magnitude and type of benefit depend on baseline vitamin D status, sport-specific demands and the performance outcome assessed. Baseline mean 25(OH)D concentrations concealed substantial individual variation: four participants in the vitamin D group and two in the placebo group had concentrations below 20 ng/mL, whereas five and three, respectively, had concentrations of at least 30 ng/mL. Baseline status may therefore have contributed to response heterogeneity. However, a performance ceiling effect was not directly tested and should not be presented as an established explanation.

This biochemical response is particularly relevant in handball, where athletes train predominantly indoors and are exposed to high neuromuscular and metabolic demands. Limited ultraviolet B exposure, combined with the competitive calendar and high training volume, may increase the risk of suboptimal vitamin D status, especially in indoor athletes during low-sunlight periods [41]. Therefore, the present findings support the practical value of monitoring and correcting vitamin D insufficiency in female indoor team-sport athletes, while also showing that improved vitamin D status should not be interpreted automatically as evidence of enhanced performance [14].

The performance estimates did not establish differential improvements attributable to supplementation. The between-group differences in change were 20 m for shuttle-run distance, 4.50 W for Wingate peak power and 107.31 J for total work, with all corresponding 95% CIs including zero. Similarly, neither sprint outcome showed a significant interaction. Within-group improvements and their standardized effect sizes describe changes over time but cannot establish a supplementation-specific benefit. The intervals should be considered when judging the range of effects compatible with this small sample.

The absence of clear supplementation-specific effects on performance is not unexpected. Several reviews and meta-analyses have shown that vitamin D supplementation may produce inconsistent effects on strength, power and sport performance, particularly when baseline deficiency is mild or intervention duration is relatively short [12]. Han et al. reported potential benefits of vitamin D_3 supplementation for muscle strength in athletes but also noted that evidence remains limited and that clear overall improvements are not guaranteed once adequate serum 25(OH)D concentrations are achieved. This aligns with the present findings, where the biochemical response was clear, but functional

improvements were not sufficiently distinct from the placebo group to support a strong ergogenic conclusion.

For serum calcium, the time × group interaction had a partial η^2 of 0.172, conventionally classified as large. The contrast primarily reflected a decrease in the placebo group (−0.225 mg/dL), whereas the vitamin D group showed little change (+0.014 mg/dL). However, the between-group difference in change was imprecisely estimated (0.239 mg/dL; 95% CI −0.021 to 0.499) and was not statistically significant (unadjusted $p = 0.069$; Holm-adjusted $p = 0.554$). These findings do not establish a calcium-stabilizing effect of supplementation or demonstrate that a true effect was missed because of the small sample size. The biochemical measurements do not provide a comprehensive safety assessment.

Taken together, these findings suggest that vitamin D_3 supplementation in national level female handball players may be more justified as a strategy for correcting or preventing insufficiency than as a short-term method for improving physical performance. This distinction is practically important. In applied sport settings, restoring adequate vitamin D status may support general health, skeletal integrity and physiological readiness, but expectations regarding immediate improvements in sprint, aerobic or anaerobic performance should remain cautious unless supported by clear group-specific effects.

Several limitations should be acknowledged. The small sample from a single team limits precision and generalizability. No a priori sample size calculation was performed, no single primary outcome was prospectively documented, and the supplementation phase was not prospectively registered. The analyses are therefore exploratory, and Holm adjustment addressed multiplicity across the nine reported outcomes, not across every variable collected in the broader dataset. The confidence intervals remained compatible with both no differential performance change and effects in either direction. Participants were randomized within matched pairs based on baseline serum 25(OH)D concentrations, with allocation determined by drawing lots within each pair. Although this procedure was intended to balance baseline vitamin D status between groups, the small sample size means that imbalances in other measured or unmeasured characteristics cannot be excluded. Complete follow-up avoids attrition in the analyzed cohort but does not remove these design limitations. Supplement adherence was monitored through daily reminders, periodic bottle checks and the return of all bottles at the end of the study, but residual volume was not objectively quantified. Consequently, the proportion of prescribed doses consumed could not be calculated. The greater increase in serum 25(OH)D in the supplemented group than in the placebo group supports a biological response to supplementation at the group level but does not quantify individual adherence or confirm that every prescribed dose was taken. Variation in supplement intake therefore remains a potential source of variability. Individual sunlight exposure, recovery status, menstrual-cycle phase, sleep and detailed training-load responses were not quantified in sufficient detail, although they may have influenced both biochemical and performance outcomes. Dietary intake was monitored, but residual variability in vitamin D and calcium intake cannot be excluded. The 8-week intervention period may have been sufficient to improve serum 25(OH)D concentrations but may not have been long enough to produce detectable neuromuscular or performance-related adaptations.

Future studies should include larger samples, preferably involving multiple teams and competitive levels and should stratify participants according to baseline vitamin D status. Longer intervention periods may help clarify whether sustained correction of insufficiency produces more meaningful functional benefits. Future research should also include more precise monitoring of sunlight exposure, dietary intake, menstrual-cycle status, training load, recovery and individual adherence. In addition, responder–non-responder analyses may be useful, as athletes may show heterogeneous biochemical and functional responses even when receiving the same supplementation dose. Such approaches would allow a

more individualized interpretation of vitamin D supplementation in national level female team-sport athletes.

5. Conclusions

Vitamin D_3 supplementation increased serum 25(OH)D more than placebo in this cohort of female national level handball players. No significant supplementation-specific changes were identified in sprint, shuttle-run or Wingate performance. The estimates were imprecise, and the findings should not be interpreted as evidence of equivalence or proof that supplementation has no meaningful performance effect.

Supplementary Materials: The following supporting information can be downloaded at https://www.mdpi.com/article/10.3390/nu18223743/s1. Table S1: Standardized within-group changes in performance; Table S2: Descriptive body-composition measurements before and after the intervention.

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Abbreviations

1W/2W	One week before the first/second testing session
25(OH)D	25-hydroxyvitamin D
7-DR	7-day diet record
ANOVA	Analysis of variance
BFM	Body fat mass
BMI	Body mass index
Ca	Calcium
CIQUAL	French food composition database (Centre d'Information sur la Qualité des Aliments)
CONSORT	Consolidated Standards of Reporting Trials
ECLIA	Electrochemiluminescence immunoassay
EDTA	Ethylenediaminetetraacetic acid
IU	International unit(s)
JASP	Jeffreys's Amazing Statistics Program (statistical software)
P	Phosphorus

PBF	Percent body fat
PL	Placebo (group)
PTH	Parathyroid hormone
SD	Standard deviation
SMM	Skeletal muscle mass
T1/T2	Time point 1 (baseline)/Time point 2 (post-intervention)
UV	Ultraviolet
VDR(s)	Vitamin D receptor(s)
VitD	Vitamin D (supplementation group)
η^2p	Partial eta squared
Wingate	30 s Wingate Anaerobic Test

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