A Mechanistic Perspective on Post-Activation Performance Enhancement Responsiveness: A Randomized Controlled Study of Acute Changes in Muscle Architecture, Contractile Property Kinetics, and Muscle Excitability

NCT06982937

18.05.2025

Objectives and endpoints.

The primary objective is to test whether a heavy conditioning activity (CA; one set of 2–3 repetitions at ~90% 1RM) produces post-activation performance enhancement (PAPE) in countermovement jump (CMJ) height compared with a control treadmill warm-up within the 4–12 min window after the intervention. The analysis does not assume a priori when the maximal effect occurs; instead, the minute of maximal between-group difference (denoted T*) will be identified empirically. The primary endpoint is the change in CMJ height from baseline (ΔCMJ) at post-intervention time points (4, 6, 8, 10, 12 min). Secondary endpoints include the time-course of ΔCMJ within the experimental group (EXP), individual responder status at each time point, and the associations between neuromuscular phenotype—estimated as %MHC-I from tensiomyography (TMG)—and the magnitude, timing, and likelihood of response. Ultrasound (USG) morphology (muscle thickness, pennation angle, derived fascicle length) will be analysed in parallel to performance endpoints. Electromyography (EMG), where available, will be treated analogously to USG/TMG (see below).

Data handling and derived variables.

At each time point, CMJ height is the mean of three trials; ΔCMJ and percentage change (%ΔCMJ) will be computed relative to baseline. Individual response is classified using MDC. USG outcomes will be averaged across three images; fascicle length is calculated as thickness divided by the sine of pennation angle (degrees converted to radians). TMG-based %MHC-I is estimated from delay, contraction, and half-relaxation times using the published multivariable model; TMG amplitudes and time constants may also be expressed as Δ or %Δ. If EMG is collected, signals will be band-pass filtered, rectified, quantified with 50-ms RMS, normalized (to MVC or M-wave), and summarized over task-relevant phases; %Δ at 4–12 min is computed analogously. Values >3.5 SD from group-time means will be flagged; primary analyses include all data, with winsorized sensitivity checks reported only if inferences change. Missing data will be not imputed for the primary analysis; mixed-effects models accommodate missingness assuming MAR.

Primary analysis and identification of the peak time (T*).

The confirmatory analysis uses a RM-ANOVA with ΔCMJ as the outcome, Group (EXP vs CON), Time (time effect: 4, 6, 8, 10, 12 min), and Group×Time. If the interaction is significant, we estimate adjusted between-group contrasts at each minute with bonferoni correction across the five time points. T* is defined as the minute showing the largest positive EXP–CON difference in estimated marginal means (EMMs) with the smallest p-value. We report the EMM difference at T* (95% CI) and effect size (d'Cohen). Greenhouse–Geisser correction if needed.

Within-group time-course and kinetics.

To describe the temporal profile without prespecifying a target minute, we analyse ΔCMJ across time within EXP using a one-way repeated-measures ANOVA (or LMM if assumptions are violated), followed by Bonferoni-adjusted pairwise EMMs to locate the within-group peak. To characterise response timing at the individual level, we model time to first and time to peak response (4–12 min) using Kaplan–Meier curves with censoring at 12 min, compare strata by log-rank tests, and fit Cox proportional-hazards models to estimate hazard ratios (HR, 95% CI) and concordance (C-index). This kinetics framework provides an assumption-light estimate of when athletes typically express PAPE.

Responder analyses and discrimination.

Responder distributions (Positive/neutral/adverse) will be compared between EXP and CON at each minute using likelihood χ^2 test.. For mechanistic discrimination at specific minutes (including T* once identified), we fit logistic models with %MHC-I as a continuous predictor and report ORs (95% CI),

Nagelkerke R^2, AIC, Hosmer–Lemeshow calibration, and ROC-AUC with sensitivity, specificity, and Youden's J cut-offs. Moderation nalysis also will be used with simple slope analysis.

Phenotype and morphology linkages (USG/TMG/EMG).

Associations between %MHC-I and ΔCMJ will be quantified at each minute using Pearson's r.USG variables (thickness, pennation angle, fascicle length) will be analysed with the same three lenses applied to performance: (i) within-EXP time-course (rmANOVA/LMM with Tukey), (ii) responder linkage at each minute (one-way ANOVA or robust alternative; Hedges' g for P vs X), and (iii) phenotype linkage (correlations and moderation models). TMG (e.g., Dm, Tc, Tr) and EMG (e.g., normalized RMS, median frequency) follow this identical framework to ensure coherence across modalities.

Moderation and mediation (pre-specified).

All moderators and predictors will be mean-centered; multicollinearity is checked (VIF < 5). Primary moderation models test whether (a) baseline CMJ modifies the effect of %MHC-I on ΔCMJ and (b) baseline morphology (thickness, pennation angle, fascicle length) modifies the effect of baseline CMJ on ΔCMJ. Significant interactions will be decomposed with simple slopes at −1 SD, mean, and +1 SD, with 95% CIs via nonparametric bootstrap (1,000 resamples) and publication-ready interaction plots. Exploratory mediation tests whether acute morphological change (e.g., %Δthickness at a given minute) transmits baseline morphology effects to %ΔCMJ; indirect, direct, and total effects will be estimated by bootstrap (5,000 resamples; bias-corrected CIs) and interpreted cautiously.

Assumptions, multiplicity, and robustness.

Model diagnostics include residual normality (Shapiro–Wilk), homoscedasticity (residual–fitted patterns, Breusch–Pagan), linearity, and independence by design. Sphericity is tested for rmANOVA (Mauchly) with Greenhouse–Geisser corrections as needed. If mechanistic variables are skewed, appropriate transformations (e.g., log for EMG amplitude) are considered; otherwise we rely on LMM robustness. Multiplicity is controlled via Holm across the five between-group contrasts, FDR for correlation families, and restraint in post-hoc testing; moderation/mediation will be estimation-focused with full interval reporting and are not used to redefine the primary inference.

Reporting, visualization, and software.

We report means ± SD with 95% CIs; between-group effects as Hedges' g; within-subject effects as d_nz; ANOVA-style effects as ηp^2; logistic results as ORs with AUC; and survival results as HRs with concordance. Figures include EMM trajectories with 95% CIs, individual waterfall plots with MDC bands, ROC curves for %MHC-I, Kaplan–Meier curves for first/peak response, interaction plots for significant moderations, and path diagrams for supported mediations. Analyses will be performed in Jamovi 2.6 and STATISTICA 13.1.