Acute Changes in Hamstring Injury Risk Factors After a Session of High-Volume Maximal Sprinting Speed Efforts in Soccer Players

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Background: Maximal sprinting speed (MSS) overexposure is associated with increased risk of injury. This study aimed to describe changes in sprint performance-related factors and hamstring strain injury (HSI) risk factors after a high-volume sprinting session in soccer players.

Hypothesis: A high-volume sprinting session can induce acute changes in several sprint performance-related factors (sprint time and mechanical properties) and HSI risk factors (posterior chain muscle strength, hamstring range of motion, and dynamic lumbo-pelvic control [LPC], measured as changes in anterior pelvic tilt [APT] during maximal speed sprinting).

Study Design: Prospective observational case series.

Level of Evidence: Level 4.

Methods: Fifteen active male amateur soccer players participated. Changes in sprint performance-related factors and HSI risk factors were examined for 72 hours after high-volume MSS efforts (H-VMSSE) using a soccer-contextualized multifactorial approach. Muscle damage proxy markers (hamstring perceived soreness and creatine kinase) were also examined.

Results: H-VMSSE induced decrements in sprint performance-related factors. Significant reductions in theoretical maximal horizontal velocity ($P < 0.01$; effect size [ES], -0.71) and performance ($P = 0.02$; ES, -0.59) were observed for 48 and 72 hours after H-VMSSE. Small but significant reductions in posterior chain muscle force-generating capacity were detected for 48 and 72 hours after H-VMSSE for the nondominant ($P < 0.03$; ES, -0.60) and dominant ($P < 0.04$; ES, -0.40) leg. Finally, players exhibited persistent small, albeit nonsignificant ($P = 0.06$; ES, 0.53), decreases in dynamic LPC (APT increases) for 72 hours after H-VMSSE.

Conclusion: H-VMSSE induced declines in both sprint performance-related factors and HSI risk factors. Sprinting can alter a player's anatomic structure by increasing APT during the maximum speed phase of the sprint.

Clinical Relevance: A soccer-contextualized multifactorial approach might allow for the regulation of MSS dosage depending on individual HSI risk factor status, thereby serving as a tailored "vaccine" for sprinting needs.

Keywords: hamstring injury risk factors; hamstring ROM; hamstring strength; pelvic tilt; sprint kinetics; sprint performance

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Hamstring muscle strain injuries (HSIs) are the most prevalent injuries in soccer, accounting for 24% of all injuries, and have not shown signs of a decrease over the last few decades.[9] Sprinting, which leads to excessive strain on the hamstring muscles during the late swing phase of the running cycle, has been identified as the primary determinant of tissue failure.[7] This mechanism has been widely recognized as the major cause of injury in soccer[9] and should serve as the fundamental basis and starting point for any preventive interventions. Moreover, it has been shown recently that the majority of hamstring injuries occur when players run >25 km h^{-1} and at >80% of their maximal sprinting speed (MSS).[1]

On the other hand, the integration of consistent MSS into elite sports training is crucial for effectively conditioning players for high-speed scenarios during match-play.[20,21] Coaches should include training scenarios that involve high percentages of MSS to potentially prevent future soft tissue injuries, similar to a "vaccine" effect.[20,21] In this regard, sprinting stands out as the only exercise capable of eliciting a sprint-specific activation of the hamstring muscles, both in terms of magnitude and timing. This distinct activation pattern cannot be replicated through conventional strengthening exercises, which typically achieve only 18% to 75% of the electromyographic activity observed in the hamstrings during MSS.[42]

A U-shaped relationship has been found between the number of exposures to MSS and the risk of injury both in Gaelic football and soccer players.[20,21] Players who consistently reach ≥95% of their MSS have a reduced risk of injury compared with those who achieve lower relative maximal velocities; however, under- and overexposure of players to MSS events increases the risk of injury.[21] Specifically, there is an increased risk of injury associated with acute increases in the total distance covered at ≥95% of players' MSS.

Recently, a new soccer-based screening test battery covering a wide spectrum of modifiable HSI risk factors (strength, range of motion [ROM], lumbo-pelvic control [LPC], and MSS mechanical properties) has been proposed.[17,25,26,28,34] This approach takes into account the multifactorial nature of HSI and allows the player to be classified according to their individual risk factor(s) profile. This individualized, multifactorial approach might help in understanding the effect that acute increases in the total distance covered at ≥95% of players' MSS has on HSI risk factors in soccer players. We believe that understanding the changes in HSI risk factors resulting from a passage of MSS overexposure, which means the systematic repetition of the injury mechanism (where hamstring muscle-tendon tissues face the highest mechanical strain), is crucial for understanding HSI etiology and effectively regulating the dosage of MSS as a distinct and singular "vaccine" or "poison."[7]

Sprint-related interventions (simulated soccer match) in previous studies induced centrally mediated hamstring function impairments in semiprofessional soccer players[22] and alterations in sprint kinematics (greater anterior pelvic tilt [APT]) in amateur soccer players.[40] Although providing valuable insights, these studies aimed to explain the effects of sprint-related interventions only by unifactorial means, describing the acute changes of a single isolated HSI risk factor (knee flexor strength or LPC [APT]). Moreover, while providing a globally and ecologically stressful stimulus to the lower limbs by combining sprints with decelerations and short recovery periods, sprint-related interventions (simulated soccer match) also reduce the players' capacity to repetitively reach ≥95% of their MSS due to the fatigue accumulated. To the best of our knowledge, no studies have focused on analyzing the responses to high-volume MSS efforts (H-VMSSE) where the impact of fatigue is reduced, allowing players to repeatedly reach ≥95% of their MSS. Implementing a H-VMSSE protocol would involve exposing the players to MSS overexposure, which has been associated previously with increased risk of injury.[20,21] Moreover, this type of protocol would systematically reproduce one of the primary mechanisms for hamstring injuries in soccer—the sprint.[7]

Therefore, the aim of this study was to describe the time course and magnitude of acute changes in sprint performance-related factors and HSI risk factors after a H-VMSSE protocol in amateur soccer players, using a soccer-contextualized multifactorial approach along with muscle damage proxy markers.

METHODS

Design

The study design used observational comparison of response in a single group of amateur soccer players. During the week before the testing day (H-VMSSE protocol), the players were familiarized with the assessments. We examined changes in sprint performance-related factors and HSI modifiable risk factors using a soccer-contextualized multifactorial approach based on the screening test battery proposed by Lahti et al.[19] Specifically, changes in sprint performance (10- and 30-meter split times) and mechanical properties (theoretical relative [to body mass] horizontal maximal force [F0] and theoretical maximal horizontal velocity [V0]), modifiable HSI risk factors (posterior chain muscles strength, interaction between the hip flexor and hamstring ROM and dynamic [sprint] LPC), and muscle damage (residual fatigue) proxy markers (hamstring muscle perceived soreness and creatine kinase [CK]) were examined for 72 hours after H-VMSSE. The choice of a 72-hour recovery period was determined by the fact that, although needs can vary considerably based on the individual player, the high demands of the sprinting session might require extended recovery periods.[12] Assessments were conducted at baseline (pre) and at follow-up timepoints post, 24, 48, and 72 hours, with each test administered by the same rater. A schematic overview of the experimental design is shown in Figure 1.

Participants

A total of 15 healthy and trained male amateur soccer players (mean ± SD) (age, 21.0 ± 1.3 years; height, 170.7 ± 18.5 cm; body mass, 73.1 ± 7.4 kg) who had not suffered any myotendinous injuries for 6 months before the experiments

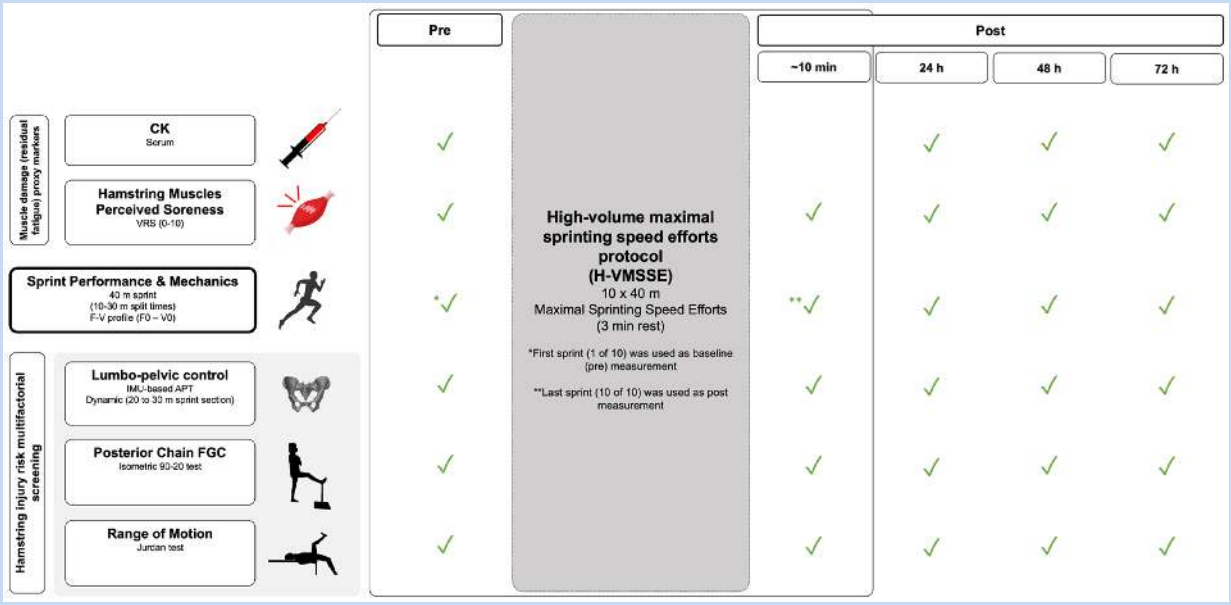

Figure 1. Schematic overview of the experimental design. Note that the first and last sprints from the repeated sprint bout were used as baseline (pre-) and postmeasures, respectively. APT, anterior pelvic tilt; CK, creatine kinase; FGC, force-generating capacity; IMU, inertial motion unit; VRS, visual rating scale.

volunteered to participate in the study. Player training status ranged from "low" to "moderate" according to Silva et al[39]; 10 were right-limb dominant and 5 were left-limb dominant (favored kicking leg). To prevent the induction of additional fatigue during the 4 consecutive days of experiments, players refrained from participating in their regular soccer training schedules. Exclusion criteria included that at least 72 hours should have passed after their respective soccer matches before participating in the first testing day of the study. All players were familiarized with the testing procedures. The experiment was conducted in accordance with the code of ethics of the World Medical Association (Declaration of Helsinki) and approval was given by the Ethics Committee of the Catalan Sports Council (037/CEICGC/2021). All participants were informed about the purpose of the study, known risks, and possible hazards associated with the experimental protocol before recruitment, and each gave written consent.

Based on a power analysis conducted using G*Power Version 3.1 (effect size [ES] 0.3, power 0.8, α probability error 0.05), it was determined that a sample size of 15 players was necessary for conducting a repeated-measures analysis of variance solely considering factors across 5 measurement timepoints.[30]

Procedures

H-VMSSE Protocol

After a standardized warm-up (see Sprint Mechanical Properties and Performance), players performed 10 × 40-meter maximal sprints interspersed with a 3-minute rest period. The purpose of the long rest periods was to mitigate fatigue and enable players to consistently achieve ≥95% of their MSS. During the protocol, players were instructed to decelerate gradually over a distance of 40 meters to minimize the neuromuscular impact of the deceleration to prevent excessive fatigue in the lower limbs.[24] Performance and mechanical properties, from each sprint, were obtained as described in the following sections. The first and last sprints were used as baseline (pre-) and postmeasures. To quantify fatigue, the percentage decrement score (S_{dec}) was calculated as follows[11]:

$$S_{\text{dec}}(\%) = \left(\frac{(S_1 + S_2 + S_3 + \ldots + S_{\text{final}})}{S_{\text{best}} \times \text{number of sprints}} - 1 \right) \times 100$$

Blood Sampling and Processing

We obtained 5 ml of blood from the antecubital vein from the volunteers at each follow-up assessment (Figure 1). Blood samples were allowed to clot for 30 minutes in a tube (SST II Advance, Becton Dickinson Vacutainer Systems) before being centrifuged at 3000*g* for 10 minutes at 4°C. Serum was aliquoted and stored at -80°C until needed for CK analysis. Biochemical analysis of CK was performed using an Advia 2400 automatic device (Siemens Healthcare Diagnostics).

Range of Motion

To assess the interaction between the hip flexor and hamstring ROM, the Jurdan test was used.[17,18] This test has been shown previously to have "excellent" test-retest reliability.[18] Players performed 2 trials with each limb at a slow pace (~3 seconds). The test was recorded from each side using a Casio Exilim EX-ZR 700 camera on a tripod situated at the height of the table

and 3 m from the center of the player's hip on the table. Two-dimensional (2-D) angles were obtained using Kinovea software Version 0.8.24, a 2-D motion analysis software that has been validated previously.[36] The best of the 2 trials from each limb was recorded for further analyses.

Muscle Soreness

A 10-point visual rating scale was used to quantify the perceived hamstring muscle soreness of each limb,[4] while players were in the position for the 90:20 (degrees of hip and knee flexion, respectively) isometric posterior chain strength test. Each number on the scale was accompanied by descriptive words for soreness, from 0 indicating no soreness to 10 indicating intolerably intense soreness.

Posterior Chain Muscle Strength

Posterior chain muscle strength was measured as the maximal voluntary contraction (MVC) using the hands-on-chest $90°_{hip}:20°_{knee}$ isometric posterior chain (90:20 IPC) strength test.[23] This test has been shown previously to have a "moderate-to-high" test-retest reliability.[23] For the 90:20 IPC, players stood with their buttocks, upper back, head, and nontested leg and heel against a wall. The heel (calcaneus) of the tested leg was placed in a custom-built support attached to a strain gauge (Force Sensor, Chronojump) with the ankle in a neutral position. The custom-built support allowed the player to adjust the height of the heel and its distance to the wall to ensure that each player was tested with 90° of hip flexion and 20° of knee flexion position (measured using a goniometer). Players performed 3 isometric MVCs of 3 to 5 seconds (with each limb) with a short rest period (~30 seconds) between contractions. The force sensor sampling frequency was 100 Hz, and MVC was then calculated as the mean force in a 1 s window when a force plateau was established.[4] The best of the 3 trials from each limb was recorded for further analyses.

Sprint Mechanical Properties and Performance

Instantaneous velocity over a 40 m sprint was measured by a linear velocity sensor (Race Analyzer, Chronojump) set at 10 pulses per sample, which implied a spatial accuracy of 3.032 cm. This means that each sample measured the elapsed time between samples with a temporal precision of 4 microseconds. The device was held by a researcher seated 2 m behind the players and was positioned at a height near the players' gravity center. Players adopted a starting position and wore their usual soccer shoes. They were instructed to remain in this static starting position, and the researcher kept the tether tensioned with the device. Moreover, to achieve a faster and more reliable start, they were instructed to start at will whenever they wanted. All tests were performed on a synthetic grass surface on an outdoor soccer pitch and conditions were standardized between repeated measures. The warm-up consisted of 5 minutes of low pace (~10 km h^{-1}) running, followed by 5 minutes of sprint-specific warm-up exercises running >30 meters and, finally, 2 progressive runs until reaching maximal speed >40 meters

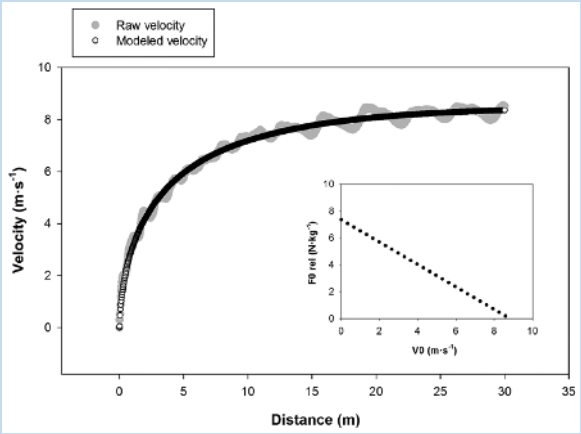

Figure 2. Actual (raw) and modeled velocity at the onset of a typical 30-meter segment from the 40-meter sprint for player 5. Inset, graphical representation of the linear force-velocity relationship ($F0 = 7.37\ N\ kg^{-1}$; $V0 = 8.82\ m\ s^{-1}$). F0 rel, theoretical relative (to body mass) horizontal maximal force. V0, theoretical maximal horizontal velocity.

interspersed by 2 min of passive rest. Then, 1 maximal sprint trial per subject was performed and recorded for further analyses. Players were instructed to gradually decelerate through a 40-meter zone to minimize the neuromuscular impact of the deceleration. The resulting data were then analyzed using Chronojump software Version 2.1.2-2 to compute sprint force-velocity (F-V) spectra using the simple field method validated by Samozino et al.[37] This method has been shown previously to have a "high" test-retest reliability to determine F-V relationships during overground sprinting.[37] The model was applied from the timepoint in which a raw acceleration of 5 m s^{-2} was detected until 30 meters because some of the players showed a loss in raw velocity right before the 40-meter mark, indicating that the sprint was not maximal in the last meters. Then, the "0" velocity was set at the moment in which the modeled velocity crossed the horizontal (time) axis from the time-velocity graph. Once the data were adjusted to the model, the modeled running velocity of each sprint was used to compute the net anteroposterior ground-reaction force (GRF) and sprint performance (defined as 10- and 30-meter sprint times). Individual linear F-V relationships were then extrapolated to calculate F0 and V0 capabilities, as described elsewhere (Figure 2).[37] Baseline reliability analyses were conducted using the initial 2 sprints from the H-VMSSE protocol.

Lumbo-Pelvic Control

Dynamic LPC was tested in parallel with sprint mechanical output and performance. A single triaxial inertial motion unit (IMU) (XSens DOT, Technologies BV) attached with double-sided tape to the S1/L5 junction and overlapped with tape was used, as previously described.[43] The sensor range was ±2000 deg/s for the gyroscope, ±16*g* for the accelerometer, and ±8 for

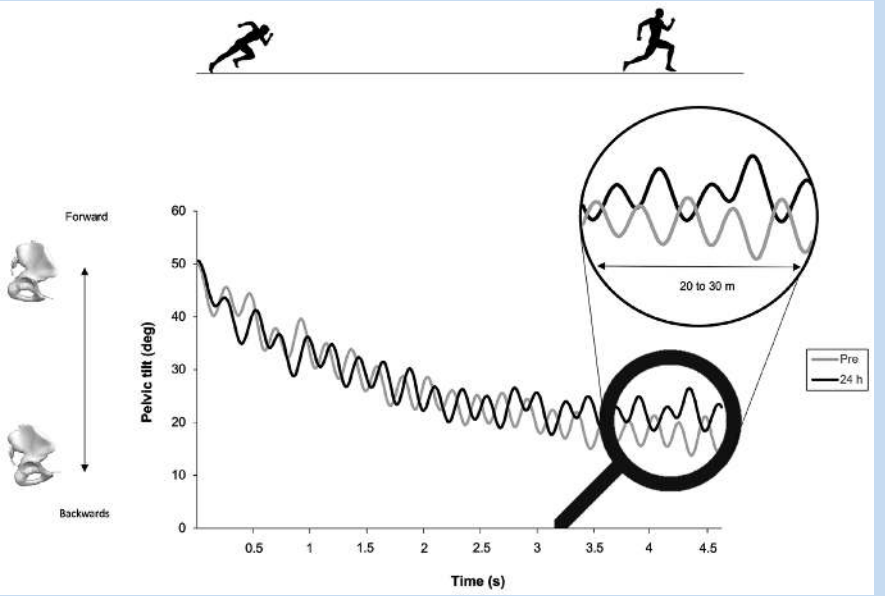

Figure 3. Representative sample of pelvic tilt-filtered signals measured during a linear sprint with an IMU attached in L5/S1 in 2 different conditions. The mean APT peaks were calculated in the region of interest shown in the figure, adding sprint times from 20 to 30 meters to $t = 0$. APT, anterior pelvic tilt; IMU, inertial motion unit.

the Gauss magnetometer. The internal sampling rate for strapdown integration and orientation through the extended Kalman filter was 800 Hz, and the output sampling data frequency was chosen at 120 Hz. APT was considered as pitch, expressed in the IMU as the orientation angle in the sagittal plane with a root-mean-square error of 0.5° in a static inclination and 1° in a dynamic inclination.

For the dynamic LPC measurement, relevant raw data from the gyroscope and orientation units were first filtered by a fourth-order Butterworth low-pass filter with a cut-off frequency of 6 Hz following the recommendation in the literature.[45] The start time of each sprint ($t = 0$) was detected using the value of 20 deg/s of the gyroscope in the sagittal plane as the threshold. To start time, sprint times from 20 to 30 meters for each subject were added in their trials. In that period, APT peaks were detected using Python software Version 3.10.9, and the mean of the peaks was calculated (Figure 3).

The use of a single inertial sensor is supported as a valid tool to measure movements of the pelvis during sprinting.[43] Baseline reliability analyses were conducted using the initial 2 sprints from the H-VMSSE protocol.

Statistics

Most of the studied variables failed all the tests for homogeneity of variance (Levene test) and normality (Shapiro-Wilk test). Therefore, a bootstrap confidence interval approach was used. This gave us 2 substantial advantages: a reliable nonparametric statistical analysis and an easy construction of the 95% CI of ESs in small samples.[13] More specifically, a bootstrapping model with 1000 samples and bias-corrected and accelerated method was used to extract the 95% CI of the Cohen *d* ES in a repeated-measures design. We compared each measurement with the baseline measures. Thresholds for ES of the Cohen *d* were trivial (ES < 0.20), small (0.20 < ES < 0.59), moderate (0.60 < ES < 1.19), large (1.20 < ES < 1.99), and very large (ES > 2.0).[14] Finally, all the reported *P* values where the likelihoods of the absolute ES being observed if the null hypothesis of zero difference was true.[13]

We evaluated baseline test-retest reliability of dependent variables using the intraclass correlation coefficient (ICC), along with a 95% CI, typical error (TE), and coefficient of variation (CV%): CV = (SD × mean) × 100.[44] Reliability thresholds for ICC values were defined as poor (<0.50), moderate (0.50-0.75), good (0.75-0.90), and excellent (>0.90).[15] All statistical analyses were conducted with Python software Version 3.10.9.

RESULTS

H-VMSSE Protocol

S_{dec} calculated from 30-meter times (performance) revealed low fatigue levels (3.67 ± 2.27%), which aligns with the expected results (players repeatedly reached ≥95% of their MSS). Moreover, no significant changes in performance and mechanical properties were found between sprints, with the only exception being V0—a variable that showed a slight decreasing trend during the last sprints (sprint 7: -3.12%; $P = 0.01$; ES, -0.49; sprint 8: -3.13%; $P = 0.01$; ES, -0.51; sprint 9: -3.96%; $P = 0.01$; ES, -0.53; sprint 10: -4.29%; $P < 0.01$; ES, -0.63).

Muscle Damage Proxy Markers

Serum CK

Every timepoint analyzed showed significant large-to-moderate increases in serum CK levels, with a peak observed 24 hours after H-VMSSE (840 ± 615 U l^{-1}; ES, 1.28) (Figure 4a).

Muscle Soreness

Perceived hamstring muscle soreness increased significantly after H-VMSSE and peaked at 48 hours (dominant leg: 3.2 ± 1.3 a.u.; ES, 1.73, large) (nondominant leg: 3.4 ± 1.5 a.u.; ES, 1.94, large) (Figure 4, b and c).

HSI Risk Factors

Range of Motion

Trivial nonsignificant ROM reductions were observed after H-VMSSE. The nondominant leg showed nonsignificant very small changes from 49.2 ± 13.7° at baseline to 44.2 ± 12.8° at 72 hours (ES, -0.37) (Figure 5, a and b).

Our baseline results showed "excellent" test-retest reliability (dominant leg: ICC, 0.94-0.99; TE, 2.51; CV%, 2.83, and nondominant leg: ICC, 0.91-0.99; TE, 2.51; CV%, 3.20).

Posterior Chain Muscle Strength

Significant small-to-moderate decreases in posterior chain muscles strength were detected until 48 hours (from 192 ± 53 N at baseline to 166 ± 34 N; ES, -0.60) and 72 hours (from 213 ± 51 N at baseline to 194 ± 47 N; ES = -0.40) after H-VMSSE for nondominant and dominant legs, respectively (Figure 5, c and d).

The 90:20 IPC strength test showed "excellent" test-retest reliability (dominant leg: ICC, 0.85-0.97; TE, 14.11; CV%, 7.21, and nondominant leg: ICC, 0.84-0.97; TE, 14.98; CV%, 7.94).

Lumbo-Pelvic Control

Dynamic LPC, measured as an increase in APT during maximal speed phase of sprint, showed nonsignificant (P = 0.06) persistent small changes from 13.8 ± 7.15° at baseline to 16.4 ± 5.17° and 17.1 ± 5.02° at 24 (ES, 0.42) and 72 hours (ES, 0.53), respectively. In addition, 11 players (most of the sample) had higher APT values at 72 hours than at baseline (increase range: 0.23° to 15.7°) (Figure 6).

Dynamic LPC showed an "excellent" test-retest reliability (ICC, 0.85-0.98; TE, 1.91; CV%, 1.94).

Sprint Performance-Related Factors

Sprint Mechanical Properties and Performance

Statistically significant small-to-moderate increases from baseline were found in 10- and 30-meter sprint times at 48 hours after H-VMSSE (2.30 ± 0.20 seconds vs 2.48 ± 0.32 seconds; ES, 0.62, and 4.83 ± 0.35 seconds vs 5.12 ± 0.52 seconds; ES, 0.58, respectively). At 72 hours, moderate quasi-significant and significant increases from baseline were found in 10- and 30-meter sprint times (2.30 ± 0.20 seconds vs 2.49 ± 0.38 seconds; ES, 0.60, and 4.83 ± 0.35 seconds vs 5.11 ± 0.57 seconds; ES, 0.59, respectively).

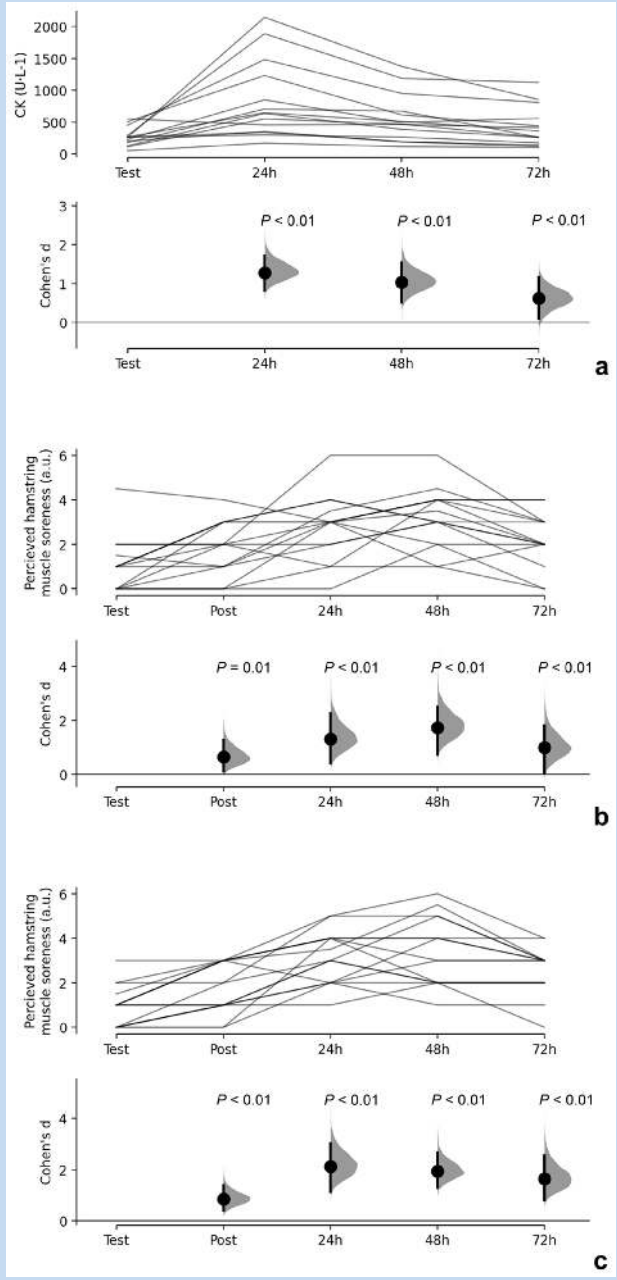

Figure 4. Cumming plot of (a) serum CK, (b) perceived hamstring muscle soreness of dominant leg, and (c) perceived hamstring muscle soreness of nondominant leg. Upper panel, individual values for each variable in different time points. Lower panel, bootstrap effect sizes (Cohen *d* difference with a 95% CI) calculated based on the comparison with the baseline (Test).

Regarding sprint mechanical properties, F0 varied widely among players and only small significant changes were found in F0 at 48 hours (from 6.40 ± 1.15 N kg^{-1} at baseline to 5.92 ± 1.51 N kg^{-1}; ES, -0.36) after H-VMSSE. (Figure 7a). On the other

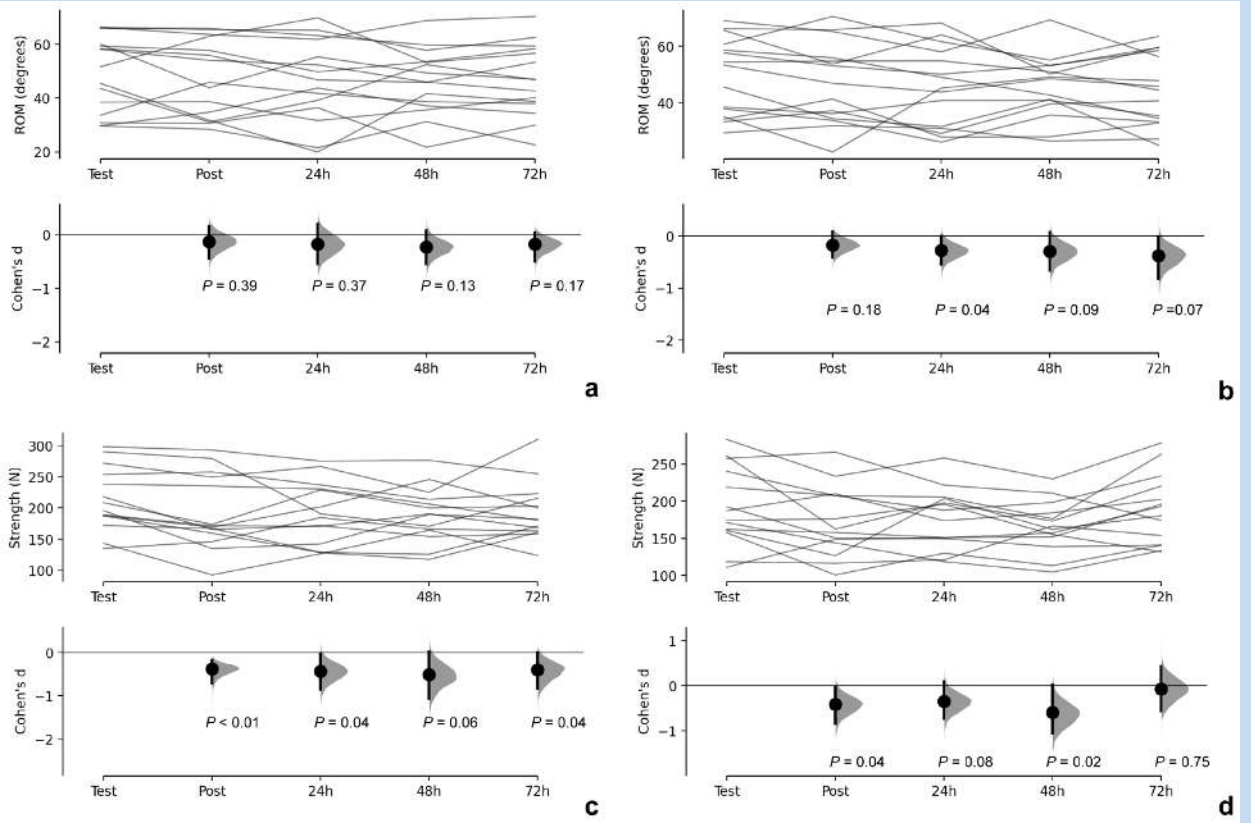

Figure 5. Cumming plot of (a) ROM measured as shin angle from the dominant leg minus the thigh angle from the nondominant leg, (b) ROM measured as shin angle from the nondominant leg minus the thigh angle from the leg, (c, d) posterior chain muscles strength measured as the maximal voluntary contraction using the hands on chest 90:20 isometric posterior chain strength test from the (c) dominant and (d) nondominant leg. Upper panel, individual values for each variable in different time points. Lower panel, bootstrap effect sizes (Cohen *d* difference with a 95% CI) calculated based on the comparison with the baseline (Test). ROM, range of motion.

hand, V0 was reduced significantly until 48 hours (from 9.05 ± 0.60 m s^{-1} at baseline to 8.65 ± 0.53 m s^{-1}; ES, -0.71, moderate) (Figure 7b).

Sprint mechanical properties showed a "good" test-retest reliability (F0: ICC, 0.66-0.94; TE, 0.48; CV%, 6.41, and V0: ICC, 0.52-0.91; TE, 0.30; CV%, 2.57).

DISCUSSION

We can assume that the H-VMSSE protocol exposed the players to MSS overexposure, which had been associated previously with an increased risk of injury.[20,21] It was found that H-VMSSE reduced sprint performance-related factors and altered HSI risk factors. Regarding sprint performance-related factors, (1) significant moderate reductions in V0 and performance after H-VMSSE were found for 48 and 72 hours, respectively. In relation to HSI risk factors, (2) small but significant decreases in posterior chain muscle strength were detected for 48 and 72 hours after H-VMSSE for the nondominant and dominant legs, respectively. Finally, (3) albeit nonsignificant, persistent small decreases in dynamic LPC, measured as an increase in APT during the maximal speed phase of sprinting, were found after H-VMSSE. However, one of the strengths of this study, which facilitated the interpretation and application of the results, is the acknowledgment that the response of the players after H-VMSSE is subject to interperson changes regarding HSI risk factors.

Decrement in Sprint Performance-Related Factors

Measurement of sprint performance (30-meter time) showed that players involved in this study performed similarly to professional players.[2,16] Moreover, the H-VMSSE protocol carried out in the present study generated slight nonsignificant performance decrements and, therefore, low fatigue levels (S_{dec} 3.62 ± 2.15%) during exercise, indicating that the players repeatedly reached ≥95% of their MSS. V0 was the only mechanical variable analyzed that showed a significant small-to-moderate decrease during the last sprints of the protocol. Most importantly, after H-VMSSE, V0 and performance (30-meter

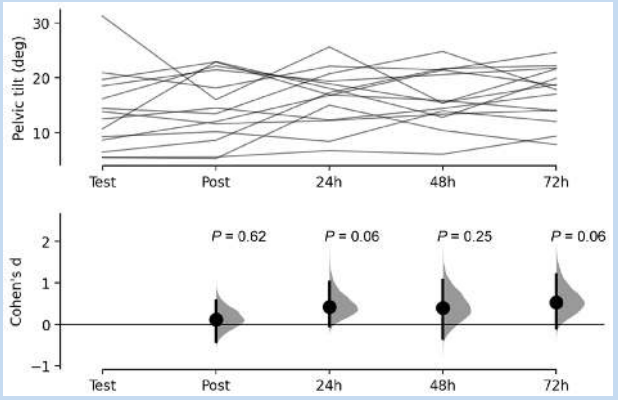

Figure 6. Cumming plot of mean peaks of pelvic tilt (degrees) measured dynamically (during 20-30-meter sprint). Upper panel, individual values for each variable in different timepoints. Lower panel, bootstrap effect sizes (Cohen *d* difference with a 95% CI) are calculated based on the comparison with the baseline (Test).

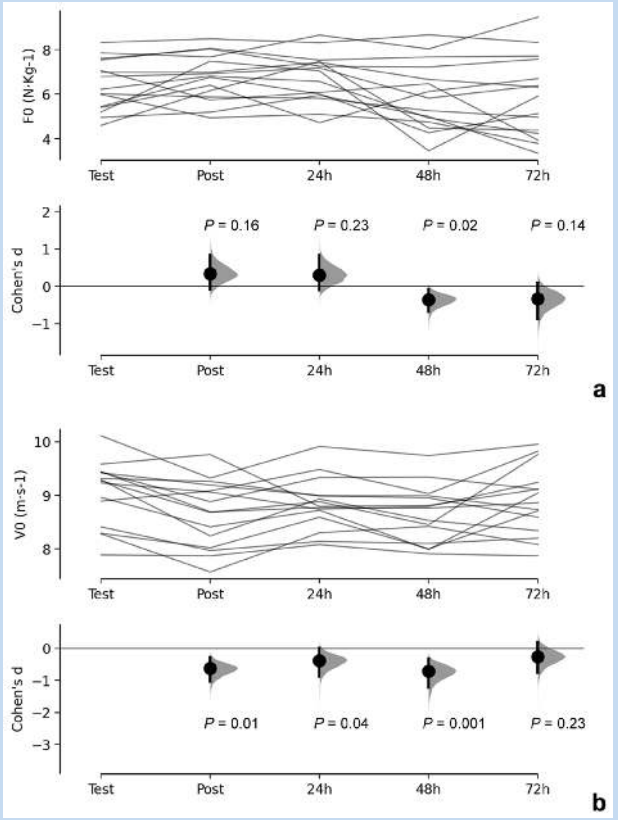

Figure 7. Cumming plot of (a) theoretical relative, to body mass, horizontal maximal force (F0) and (b) theoretical maximal horizontal velocity (V0). Upper panel, individual values for each variable in different timepoints. Lower panel, bootstrap effect sizes (Cohen *d* difference with a 95% CI) are calculated based on the comparison with the baseline (Test).

time) remained significantly reduced for 48 and 72 hours, respectively.

Sprint running and, more specifically, sprint acceleration, is a key component of performance in soccer, being common in many match-winning actions, such as winning possession of the ball, passing defending players, or gaining a position to score a goal.[10] During the acceleration phase of sprinting, forward orientation of GRF has been shown to be a stronger determinant of field sprint performance and more sensitive to sprinting-generated fatigue than the overall magnitude of vertical or resultant GRF.[31,33] Hip extensor muscles (gluteus maximus and hamstring muscles) play a key role in this horizontal force, producing a force that is directed horizontally but backward and allowing the body to propel forward during the support phase.[32] The ability to specifically produce and apply high amounts of force onto the ground in the horizontal direction as a function of running velocity has been well represented by linear F-V, describing the changes in external horizontal force generation at both low (F0) and high (V0) velocities.

In the last sprints of the H-VMSSE protocol, only small-to-moderate reductions in V0 and no significant changes in performance were observed. This can be attributed to the nature of the sprinting exercise (high recovery between sprints) and the fitness level of the participants. Therefore, high sprinting volumes, even under low fatigue conditions (H-VMSSE, 40-meter all-out sprints, interspersed with 3-minute rest periods), have the potential to induce a certain degree of acute V0 loss.

Most importantly, after the H-VMSSE protocol, V0 remained significantly reduced for 48 hours and performance was significantly lower for 72 hours. As stated earlier, because hamstring muscles play a crucial role in horizontal force production during sprint acceleration,[32] greater horizontal GRF (as averaged over an entire sprint acceleration) was found in subjects who were both able to highly activate their hamstring muscles just before ground contact and had the greatest capacity to produce eccentric knee flexor peak torque,[33] so it cannot be ruled out that hamstrings experienced a certain degree of residual fatigue (muscle damage mediated) due to the H-VMSSE. In this regard, it has been shown previously that, after a repeated sprint protocol, sprint-induced fatigue leads to a decrease in the technical ability to apply horizontal, but not vertical, force against the ground.[33] Moreover, under sprint-induced fatigue conditions, the decrement in horizontal force produced by the hamstring muscles seems to be compensated by increased function of the hip extensors and gluteus maximus, minimizing performance loss and limiting hamstring muscle damage.[8] In addition, at that time (72 hours), persistent (albeit nonsignificant [$P = 0.06$]) small decreases in dynamic LPC, measured as an increase in APT during the maximal speed phase of the sprint (20-30-meter sprint segment, where V0

occurs), were observed, and this might lead to lower limb kinematic changes in that sprint phase. Specifically, an increase in APT has been shown in semiprofessional soccer players that can induce an increased forward lean of the body during the maximum speed phase of the sprint.[40] This increased pelvic anteversion and the less upright position might alter the legs' back-to-front side mechanics by producing a less active recovery of the swing leg, which leads to a lower front side knee position that ultimately offers the player less potential to accelerate the leg toward the ground (leg retraction), given the limited ROM,[5,26] and therefore less potential to produce force at high speeds. Although lower limb kinematics were not recorded in this study, V0 and subsequent performance decrements could also be explained by the kinematic-kinetic relationship.

Sprint-Induced Changes in HSI Risk Factors

Conflicting evidence has been found for strength and hamstring length or flexibility as risk factors for the occurrence of hamstring injuries in soccer, both prospectively and retrospectively. In the present study after H-VMSSE, no significant decreases in ROM were found for either the dominant or nondominant leg. On the other hand, strength decrements in posterior chain muscles were found for 48 and 72 hours for the nondominant and dominant leg, respectively. Therefore, H-VMSSE has the potential to induce changes in an HSI risk factor such as posterior chain muscle strength,[23] considered the most valid and reliable indirect marker for assessing muscle damage.[35] The notion that the posterior chain muscles experienced a certain degree of damage was further supported by the fact that proxy markers of muscle damage, including CK and soreness,[35] remained significantly elevated for up to 72 hours. Although CK lacks specificity, it is reasonable to assume that a portion of the elevated serum CK levels originated from hamstring muscles, given the observed slight increases in soreness and persistent strength losses within that timeframe.

While hamstring strength might be a risk factor itself,[25,34] it is a dynamic risk factor that is highly sensitive to fatigue. In this regard, strength decrements after exercise are considered the best indirect marker of muscle damage and, therefore, of residual fatigue.[35] Accordingly, while the immediate hamstring strength loss is attributed to both central and peripheral fatigue, prolonged strength loss is attributed to a muscle damage process (metabolic and mechanical damage that impairs the excitation-contraction coupling and the contractile process).[3] Furthermore, in sprint mechanics, during the terminal swing phase (~90% of the running cycle), the hamstring muscles actively lengthen to absorb kinetic energy from the swing limb.[41] Moreover, the biceps femoris experienced a larger net active lengthening and strain (compared with the semitendinosus and semimembranosus) due to its greater hip and smaller knee moment arms,[41] as well as its coincidence with the contralateral iliacus maximum stretch and the second APT peak.[41] Therefore, biceps femoris mechanics during the late swing phase are consistent with conditions that are likely to induce muscle damage (or, in the worst case scenario, HSI). Therefore, the 90:20 IPC test may better reflect the extent of hamstring muscle damage after sprint-based exercises because a greater magnitude of eccentric flexion torque loss is observed in the most extended position tested due to the higher peak forces of the 2 joint biceps femoris when in a longer length position.[23,29] This might explain why the strength decrements observed for 72 hours after the 90:20 IPC test may better reflect the extent of biceps femoris damage.[23]

Continuing with leg interaction as a key injury and performance indicator of sprinting, there is only 1 anatomic structure—the pelvis—that links both legs, and this can transfer mechanical energy from one leg to the other at a frequency of 4 to 5 times per second.[5] From the hamstring injury perspective, APT increases hamstring strain,[27] and, during sprinting, APT has been found to be closely related to the moments (late stance/swing) where the hamstring muscle-tendon tissues face the highest mechanical strain—the main determinant of tissue failure.[7] An increase in the APT, through a supero-anterior translation of the ischial tuberosity, would raise the eccentric load and elongation imposed on the hamstring musculature due to a greater moment arm created from reduced hip flexion.[28] The aforementioned arguments may explain the association found between APT and hamstring injury risk in both prospective and retrospective studies.[6,38] Moreover, after a sprint-related intervention (simulated soccer match combining sprints with decelerations and change of direction for 90 minutes), Small et al[40] found a significant increase in APT under fatigue (later stages of the exercise protocol). Since in the present study small, almost significant, decreases in dynamic LPC, measured as an increase in APT during maximal speed sprinting, were found until 72 hours after H-VMSSE, it seems that high sprinting volume exposure (even in the absence of fatigue) has the potential to alter the players' anatomic structure by increasing APT during the maximum speed phase of the sprint. Recently, different multimodal interventions have shown a decrease in APT both during walking and high sprint running, making this type of intervention a tool for modifying the structure of the pelvis (APT decrease) and potentially decreasing the risk of HSI derived from the consequent reduction in induced strain.[26,28]

LIMITATIONS

Although a power analysis was conducted for sample size determination (n = 15), the small cohort may not represent the broader population of soccer players, so this study should be considered a pilot study due to the limited sample size. Moreover, the observed interperson variation in response highlights the fundamental importance of understanding individual variability and demonstrates that players exhibit diverse responses to H-VMSSE. Although we believe our study significantly contributes to understanding soccer players' responses in terms of both performance and injury risk factors, statistical means may obscure patterns of individual variability

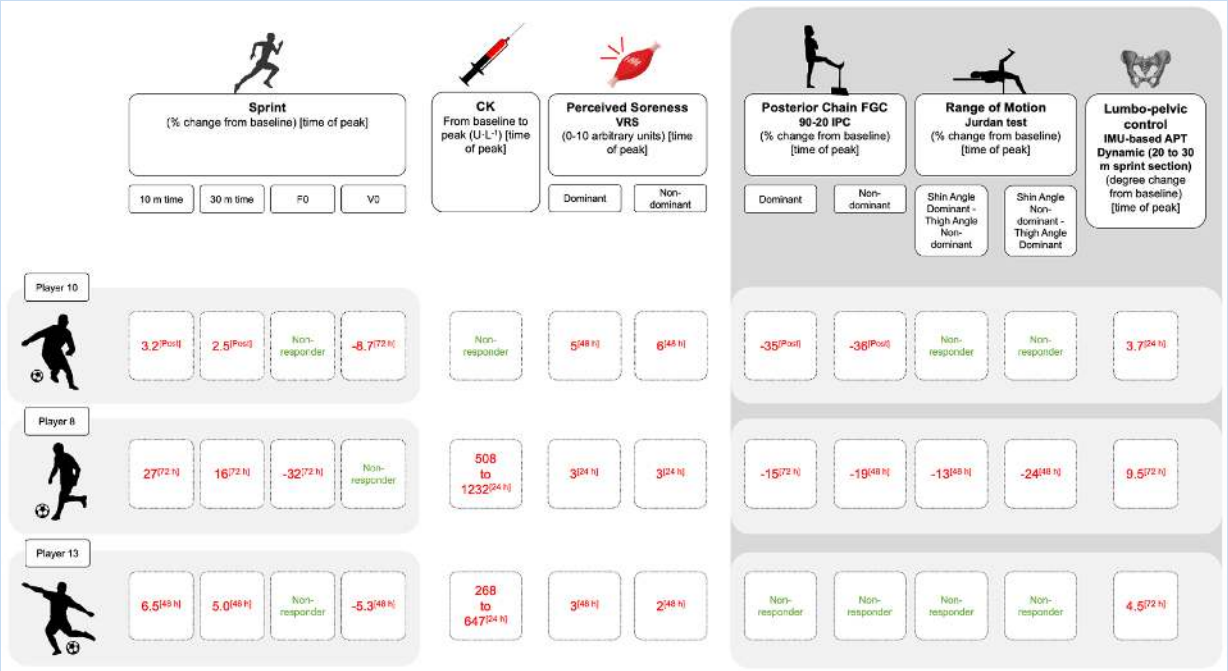

Figure 8. Individual peak values [time of peak] for main variables from 3 players as an example of the interperson variation in response to the repeated sprint bout. APT, anterior pelvic tilt; CK, creatine kinase; FGC, force-generating capacity; IMU, inertial motion unit; 90-20 IPC, 90:20 (degrees of hip and knee flexion, respectively) isometric posterior chain strength test; Non-responder, player whose peak (or lowest) values were not observed at baseline; VRS, visual rating scale.

unless individual responses are considered. In addition, assessing players based on their positions on the soccer field could have provided valuable insights into position-specific responses.

CONCLUSION

In short, this study analyzed for the first time the changes in multiple modifiable hamstring injury risk factors after a high-volume maximal sprinting speed efforts (H-VMSSE) protocol, which involved systematically reproducing one of the primary mechanisms for hamstring injuries in soccer.[7] We can assume that the H-VMSSE protocol exposed the players to MSS overexposure, which had been associated previously with increased risk of injury.[20,21] H-VMSSE reduced sprint performance-related factors and altered HSI risk factors. Specifically, decrements in sprint performance and the capacity to produce horizontal force at high velocities days after exercise were observed. In addition, posterior chain muscle strength decreased and muscle damage markers increased, suggesting that H-VMSSE caused some degree of posterior chain muscle damage. Finally, players exhibited persistent, albeit nonsignificant, small decreases in dynamic LPC, measured as an increase in APT during the maximal speed phase of the sprint after H-VMSSE. High sprinting volume exposure can therefore alter a player's anatomic structure by increasing APT during the maximal speed phase of the sprint.

PRACTICAL APPLICATIONS

Although analyses of mean values showed that H-VMSSE reduced sprint performance-related factors and altered HSI risk factors in the studied cohort, it is important to recognize that each player has a different magnitude of response. Given the critical importance of integrating consistent MSS training into elite sports training to adequately prepare players for high-speed scenarios during matches,[1,20,21] understanding individual player responses becomes imperative. A soccer-contextualized multifactorial approach informs personalized recovery and injury prevention programs, addressing both performance enhancement and injury risk reduction (Figure 8 illustrates the variability in individual responses). This approach might allow for the regulation of MSS dosage depending on individual HSI risk factor status, thereby serving as a tailored "vaccine" for sprinting needs, rather than potentially being a "poison" to each player's requirements.

Moreover, this study presents a novel, valid, and reliable methodology that is applicable both directly in teams and in field research scenarios for analyzing changes in ATP during sprinting, offering insights into its potential as an HSI risk factor.

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AVAILABILITY OF DATA AND MATERIAL

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

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