Journal of Trainology 2026;15(2):21-25

SHORT COMMUNICATION

Individualized high-velocity running exposure and lower-body soft-tissue injury in professional rugby: A within-athlete matched analysis

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Objectives: To examine whether individualized high-velocity running (HVR) exposure metrics distinguish lower-body soft-tissue injury periods from matched within-athlete control periods in a single professional rugby team, and to evaluate whether such an analysis is feasible with the data one club holds.

Design: Retrospective within-athlete matched case-control analysis.

Methods: Forty-seven male professional rugby union players were monitored across a 20-week competitive window in 2025 using 10-Hz GPS units; 35 had preseason body-composition records. Fifteen lower-body soft-tissue injuries were identified, and six had monitoring data at both the injury anchor and a within-athlete control anchor 28 days earlier. HVR was defined at 85%, 90%, and 95% of individual maximum velocity. Recency and 7- and 14-day frequency were compared using conditional logistic regression, with Firth-penalized estimation and minimum detectable odds ratios (MDOR).

Results: No metric was associated with injury. At 85%, recency odds ratio (OR) was 1.08 (95% confidence interval [CI] 0.88-1.32; 4 pairs), 7-day frequency 1.00 (95% CI 0.14-7.10; 6 pairs), and 14-day frequency 3.00 (95% CI 0.31-28.84; 6 pairs). Four models showed quasi-complete separation and one had no informative strata. MDOR ranged from 1.33 to 25.41.

Conclusions: Individualized HVR metrics did not separate injury from control periods. The findings indicate uncertainty rather than absence of effect and illustrate why single-team datasets are poorly suited to individualized HVR injury-risk inference.

(*Journal of Trainology* 2026;15(2):21-25)

Key words: ■ sprint exposure ■ muscle strain ■ athlete monitoring ■ conditional logistic regression ■ sport science

INTRODUCTION

High-velocity running (HVR) exposure is routinely monitored in professional rugby because near-maximal running places substantial mechanical demands on the lower limbs and may influence soft-tissue injury risk. Rugby union involves repeated accelerations, decelerations, and sprints, and lower-body injuries account for much of the overall injury burden.[1-3] Translating routinely collected HVR data into injury-risk decisions, however, remains difficult for coaches, sport scientists, and medical staff.

Evidence from field sports suggests the HVR-injury relationship is complex. Regular near-maximal running may help maintain tissue tolerance, whereas abrupt increases in high-speed running may elevate injury risk.[4-7] Much of this literature uses absolute speed thresholds and between-athlete designs, which may not account for differences in speed, position, or training history.[8-10] Individualized velocity thresholds offer an athlete-specific alternative, because a fixed absolute speed may represent a near-maximal effort for one player but a moderate effort for another.[9] Within-athlete matched designs are also appealing because they compare injury and non-injury periods in the same player, reducing confounding from stable individual characteristics.[11,12]

A practical question is whether these approaches work on the data a single professional team holds. Such datasets typically contain few injury events and sparse near-maximal exposures. The purpose of this short communication was therefore not to build an injury-prediction model, but to use a worked within-athlete analysis to examine whether individualized HVR metrics produce clear, interpretable injury-control contrasts in a single-team dataset, and to draw out the implications for practitioners attempting the same analysis.

METHODS

This retrospective matched case-control analysis used deidentified athlete-monitoring and injury data from one professional rugby union team. Forty-seven male players were monitored during the study window; 35 had preseason body-com-

Received June 26, 2026; accepted August 10, 2026

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position records and form the descriptive sample (age 27.0 ± 3.8 y, height 187.8 ± 7.3 cm, body mass 106.4 ± 14.7 kg). The study was determined exempt by the Loyola University Chicago Institutional Review Board (Project #4258; consent waived for retrospective deidentified data) and conducted in accordance with the Declaration of Helsinki.

External running load was captured during outdoor training sessions and matches using 10-Hz GPS units with integrated 100-Hz accelerometers (Catapult Vector S7, Catapult Sports, Melbourne, Australia) and processed in the manufacturer's software. The primary variable was session maximal running velocity; sessions without a usable velocity record were excluded, leaving 1,457 sessions from 47 monitored athletes across a 20-week window from 1 February to 21 June 2025. Session dates in the source export were stored in mixed text and spreadsheet-date formats, which transposed day and month in 39% of records; dates were reconstructed and validated against the club's published 2025 fixture record, matching 15 of 16 regular-season fixtures and both play-off dates exactly. Lower-body soft-tissue injuries (thigh, knee, lower leg, ankle, or foot; strain, sprain, or tendinopathy) were obtained from team medical surveillance records. Injuries were not restricted to time-loss events, but three lower-extremity records were excluded as not involving muscle, tendon, or ligament tissue.

Each athlete's individual maximum velocity was the highest maximal velocity recorded during the study period. For each session, relative exposure was session maximal velocity divided by individual maximum velocity, and binary flags identified whether the athlete reached 85%, 90%, or 95% of individual maximum. Three metrics were derived at each threshold: recency (days since the threshold was last reached before the anchor date) and frequency (sessions reaching the threshold in the prior 7 and 14 days). Recency is undefined where an athlete never reached the threshold before an anchor. Pairs undefined at both anchors share an identical exposure state and were retained as null contrasts; pairs undefined at exactly one anchor were dropped, and both counts appear in Table 1. Censoring recency at the first monitored session was rejected because it assigns such pairs a difference of exactly the anchor interval, the largest contrast the design permits, generated by elapsed time alone. Frequency is unaffected, as never reaching the threshold is a genuine zero. The injury date served as the case anchor; the matched control was anchored 28 days earlier within the same athlete. This lag was specified a priori, spanning about four microcycles and preventing overlap between the case and control exposure windows. Fifteen lower-body soft-tissue injuries were identified. One was excluded because its control anchor coincided with another injury in the same athlete, and eight were excluded because the athlete had no monitored session before the case anchor, the control anchor, or both, leaving six matched pairs.

Exposure metrics were compared using univariable conditional logistic regression stratified by injury-control pair, fitted separately for each metric and threshold. Only pairs with within-pair variation contributed; pairs with identical values were non-informative. Because sparse discordant data can produce quasi-complete separation, all models were also fitted using Firth's penalized maximum likelihood estimation with profile-likelihood intervals.[13] To distinguish absence of effect from insufficient precision, the minimum detectable odds ratio (MDOR) at 80% power ($\alpha = .05$) was derived for each estimable model from its standard error; post hoc observed power was not reported because it is a deterministic function of the obtained p-value.[14] Sensitivity analyses repeated all models with control anchors at 14 and 21 days, and with one athlete excluded whose deidentification code carried two incompatible anthropometric records. No adjustment for multiple comparisons was applied because the analysis was exploratory; correction would widen intervals further and reinforce, not alter, the conclusions. Estimates are reported as odds ratios (OR) with 95% confidence intervals (CI), with significance set at $p < 0.05$. All analyses were exploratory.

RESULTS

Of the fifteen lower-body soft-tissue injuries identified, the lower leg was most affected (n = 6), followed by the ankle (n = 4), thigh (n = 3), foot (n = 1), and knee (n = 1); eight were strains, six sprains, one tendinopathy. Nine could not be matched: one because its control anchor coincided with another injury in the same athlete, eight because the athlete had no monitored session before one or both anchors. Six entered the analysis (three lower leg, two thigh, one ankle; five strains, one sprain).

Monitoring coverage before the anchors was sparse: four of six analyzed pairs had a single monitored session before the control anchor. The frequency models retained all six pairs; the recency models retained four, five, and six, three pairs in total being dropped where recency was defined at one anchor but not the other. Across the nine models, the number of pairs contributing an informative within-pair contrast ranged from four to zero.

At the 85% threshold, no metric distinguished injury from control periods: recency OR 1.08 (95% CI 0.88-1.32, p = 0.450; 4 pairs), 7-day frequency OR 1.00 (95% CI 0.14-7.10, p = 1.000; 6 pairs, 4 informative), and 14-day frequency OR 3.00 (95% CI 0.31-28.84, p = 0.341; 6 pairs, 4 informative). At 95%, recency was 1.15 (95% CI 0.61-2.16, p = 0.662; 6 pairs). Four models showed quasi-complete separation and were not interpreted, and the 14-day frequency model at 95% had no informative strata at all (Table 1, Figure 1).

Firth-penalized estimation returned finite estimates for eight of the nine models, including all four affected by separation, but no penalized interval excluded the null and the number of informative strata was unchanged. Minimum detectable odds ratios for the estimable models ranged from 1.33 (recency, 85%) to 25.41 (14-day frequency, 85%), indicating that only very large associations could have been identified. Sensitivity analyses using control anchors at 21 and 14 days retained six and seven pairs, respectively, and a further analysis excluding the athlete with duplicate anthropometric records retained five pairs. All produced the same pattern with one exception: at the 14-day anchor, penalized recency at 90% returned an interval excluding the null (OR 1.22, 95% CI

Table 1. Conditional logistic regression analyses of individualized high-velocity running exposure metrics and lower-body soft-tissue injury in professional rugby players. Six matched pairs were available; recency models use fewer because recency is undefined where the threshold was never reached.

Exposure metric	Threshold	Pairs	Informative strata	OR (95% CI)	p	Firth OR (95% CI)	Min. detectable OR
Recency	85%	4	3	1.08 (0.88-1.32)	0.450	1.04 (0.93-1.29)	1.33
Recency	90%	5	3	Unstable	—	1.06 (0.95-3.14)	—
Recency	95%	6	2	1.15 (0.61-2.16)	0.662	1.08 (0.67-2.00)	2.45
Frequency, prior 7 days	85%	6	4	1.00 (0.14-7.10)	1.000	1.00 (0.15-6.47)	16.47
Frequency, prior 7 days	90%	6	2	Unstable	—	0.20 (0.00-2.46)	—
Frequency, prior 7 days	95%	6	1	Unstable	—	0.33 (0.00-6.25)	—
Frequency, prior 14 days	85%	6	4	3.00 (0.31-28.84)	0.341	2.33 (0.38-24.07)	25.41
Frequency, prior 14 days	90%	6	2	Unstable	—	0.20 (0.00-2.46)	—
Frequency, prior 14 days	95%	6	0	Not estimable	—	Not estimable	—

Note. Estimates from univariable conditional logistic regression stratified by matched injury-control pair. Pairs denotes the matched pairs contributing to that model; informative strata denotes those with within-pair variation in the exposure metric. Unpenalized models were labeled Unstable, and their estimates and minimum detectable odds ratios withheld, where the fitted standard error exceeded 4 or the absolute log-odds exceeded 8, indicating quasi-complete separation. Pairs with recency undefined at both anchors are retained as null contrasts (difference 0), contributing to the pair count but not to estimation; pairs undefined at exactly one anchor are dropped. Firth OR denotes penalized maximum likelihood with profile-likelihood intervals. Minimum detectable OR is the smallest odds ratio detectable with 80% power at $\alpha = .05$. CI, confidence interval; OR, odds ratio.

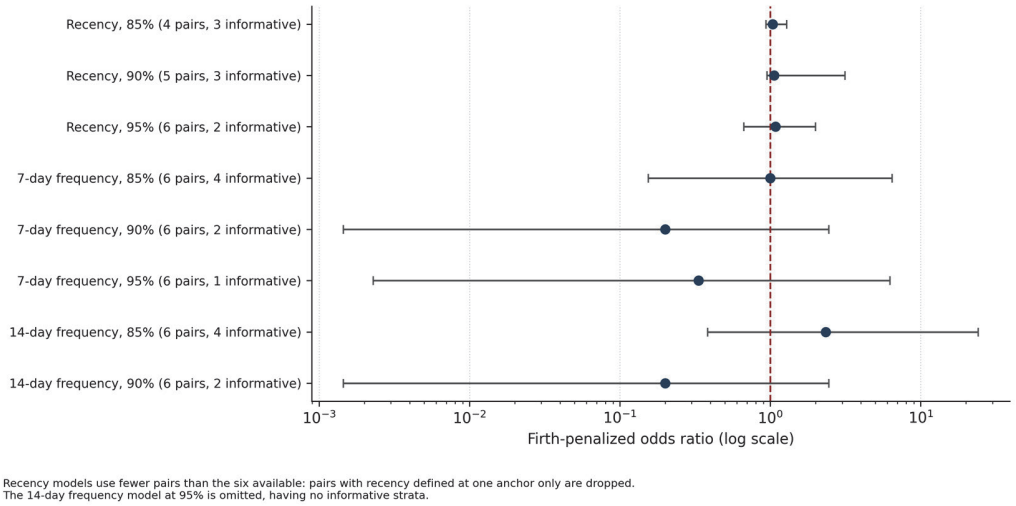

Figure 1. Firth-penalized estimates (odds ratios with 95% profile-likelihood intervals, log scale) for individualized high-velocity running exposure metrics and lower-body soft-tissue injury. Penalized estimates are shown because they are finite for every model affected by quasi-complete separation. The number of contributing pairs and of informative pairs is shown for each estimate. The 14-day frequency model at 95% is omitted, having no informative strata.

1.00-3.21). Three of its seven contrasts equaled the 14-day anchor interval exactly, and refitting without them gave OR 1.36 (95% CI 0.83-4.04). This estimate is therefore generated by elapsed time rather than by exposure. Excluding the athlete with duplicate preseason records left the frequency results unchanged but rendered all three recency models unstable.

Overall, no individualized HVR exposure metric separated injury periods from matched within-athlete control periods. The small number of injury events and, more importantly, the small number of informative discordant pairs constrained the precision and interpretability of every model, with the constraint worsening as the velocity threshold increased.

DISCUSSION

Applied to a single professional rugby team's routine data, individualized HVR exposure metrics did not produce clear or precise injury-control contrasts at 85%, 90%, or 95% of maximum velocity. The central finding is methodological rather than etiological: even with an athlete-specific threshold and a within-athlete design, a full season yielded six analyzable pairs and at most four informative strata. Penalized estimation removed the numerical failures but not their cause, and the stability of the result across 14-, 21-, and 28-day control anchors is weak evidence of robustness, because a design carrying this little information would return null, imprecise estimates under most specifications.

This should not be read as evidence that HVR exposure is unrelated to soft-tissue injury. Prior work links high-speed and sprint exposure, and abrupt changes in that exposure, to muscle injury risk, while also suggesting that regular near-maximal running may be protective when layered on adequate chronic load.[4-7, 15] Our results neither confirm nor refute them. The minimum detectable effects make the limitation concrete: for the frequency metrics only odds ratios above roughly 16 to 25 were detectable, far larger than any credible estimate in this literature, so these nulls carry almost no evidential weight.

The practical message is the contribution. Teams routinely use GPS and injury data to inform sprint-exposure decisions, and individualized thresholds are intuitively attractive. Practitioners attempting this should expect what we encountered: most injuries falling outside the window in which both anchors carry monitoring data, and few informative pairs surviving once exposure is dichotomized. Reporting informative-strata counts and minimum detectable effects alongside each estimate guards against over-interpretation. Individualized HVR monitoring remains useful for describing exposure and guiding return-to-sprint progressions, but not as a stand-alone injury-prediction tool within one team.

Strengths include the within-athlete design, controlling stable characteristics such as speed capacity and position, and individualized thresholds, which avoid the relative-intensity distortion of absolute speeds.[9,10] Limitations are central to the message. Injury surveillance covered a longer period than outdoor monitoring: three injuries preceded it entirely and a fourth preceded that athlete's first session. Individual maximum velocity was the highest value across the whole window, so it is downward-biased in athletes with fewer sessions, overstating their relative exposure, and incorporates post-injury sessions; constant within a pair, it does not bias the contrast, but threshold crossings depend partly on later data. Pooling time-loss and non-time-loss injuries may obscure an association if severity moderates the relationship with near-maximal running; stratification was infeasible. Analyzed athletes were the most continuously monitored, so peripheral squad members are underrepresented. Monitoring also lapsed for a week in late March, including a competitive fixture; this gap lies inside the exposure windows of two analyzed pairs, whose counts are undercounts. Two identifiers each carried a second preseason record with incompatible height and age; one was resolved by playing position, and the other shifts the descriptive means by under 0.2 cm. No analyzed variable depends on them. Finally, recency in days carries a deterministic elapsed-time component that no coding removes, and the date-encoding error altered 39% of session dates while remaining invisible on inspection. Both show that the obstacles to individualized injury-risk inference in one club arise as much from data structure as from sample size.

CONCLUSION

In this single-team professional rugby cohort, individualized HVR exposure metrics did not distinguish lower-body soft-tissue injury periods from matched within-athlete control periods. Estimates were null and imprecise where they could be computed, four of nine unpenalized models were unstable under separation, and minimum detectable effects confirm that only very large associations could have been identified. The findings indicate uncertainty, not absence of effect, and illustrate why single-team datasets are poorly suited to individualized HVR injury-risk inference. Larger multi-team studies with more events, severity-stratified outcomes, and richer exposure measures are needed. Practitioners should report informative-strata counts, verify exported monitoring data, and interpret internal HVR-injury models cautiously.

CONFLICT OF INTEREST

The authors received no financial support for this work and declare no conflicts of interest.

ACKNOWLEDGMENTS

The authors thank the professional rugby union team's coaching, strength and conditioning, athletic training, and sports medicine staff, and the players, whose deidentified monitoring and injury surveillance data made this study possible.

REFERENCES

1. Yeomans C, Kenny IC, Cahalan R et al. The incidence of injury in amateur male rugby union: a systematic review and meta-analysis. *Sports Med* 2018; 48: 837-848.
2. West SW, Williams S, Cazzola D et al. Injury rates, mechanisms, risk factors and prevention strategies in youth rugby union: a systematic review and meta-analysis. *Sports Med* 2023; 53: 1375-1393.
3. Rugby Football Union. England Professional Rugby Injury Surveillance Project: 2016-2017 Season Report. London: *Rugby Football Union*; 2017.

4. Gabbett TJ. The training-injury prevention paradox: should athletes be training smarter and harder? *Br J Sports Med* 2016; 50: 273-280.
5. Edouard P, Lahti J, Nagahara R et al. Sprinting: a key piece of the hamstring injury risk management puzzle. *Br J Sports Med* 2022; 57: 4-6.
6. Malone S, Roe M, Doran DA et al. High chronic training loads and exposure to bouts of maximal velocity running reduce injury risk in elite Gaelic football. *J Sci Med Sport* 2017; 20: 250-254.
7. Malone S, Owen A, Mendes B et al. High-speed running and sprinting as an injury risk factor in soccer: can well-developed physical qualities reduce the risk? *J Sci Med Sport* 2018; 21: 257-262.
8. Duhig S, Shield AJ, Opar D et al. Effect of high-speed running on hamstring strain injury risk. *Br J Sports Med* 2016; 50: 1536-1540.
9. Reardon C, Tobin DP, Delahunt E. Application of individualized speed thresholds to interpret position specific running demands in elite professional rugby union: a GPS study. *PLoS One* 2015; 10: e0133410.
10. Xie Y, Cai X. High-speed running and injury risk in soccer: a systematic review. *Front Public Health* 2026; 14: 1798241.
11. Windt J, Gabbett TJ. How do training and lifestyle factors perpetuate the injury cycle? A 2.0 smart-system approach. *Br J Sports Med* 2017; 51: 922-923.
12. Colomer P, Fortin M, Smith R et al. High-speed running and sprinting as an injury risk factor in soccer: a matched case-control study. *Sci Rep* 2020; 10: 19456.
13. Firth D. Bias reduction of maximum likelihood estimates. *Biometrika* 1993; 80: 27-38.
14. Hoenig JM, Heisey DM. The abuse of power: the pervasive fallacy of power calculations for data analysis. *Am Stat* 2001; 55: 19-24.
15. Huyghe T, Caparros T, Calleja-Gonzalez J et al. High-intensity running and sprint distance prior to hamstring injury in professional soccer players. *Front Sports Act Living* 2022; 4: 816110.