

Multidomain gait phenotyping in knee osteoarthritis: spatiotemporal, plantar-pressure and lower-limb kinematic findings from a retrospective cross-sectional study

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Abstract

Background

Biomechanical studies of knee osteoarthritis (KOA) often examine spatiotemporal performance, plantar loading and joint motion separately, limiting multidomain functional characterisation. We characterised gait means, fixed left-minus-right signed laterality and repeated-cycle variability during self-selected walking.

Methods

Records included 246 healthy middle-aged/older (HMO) adults and 92 participants with unilateral KOA (Kellgren-Lawrence (KL) grade 2/3/4, $n = 27/31/34$). Barefoot gait yielded kinematic, plantar-pressure and centre-of-pressure (COP) data. Outcomes were means, fixed left-minus-right laterality, typical range of motion (ROM-Mean), derived absolute ROM dispersion (ROM-SD) and relative dispersion (ROM-CV). Principal-component coordinates used 368 healthy references: 246 HMO plus 122 fitting-only younger participants. Comparisons used false discovery rate (FDR) correction; ROM models adjusted for age, sex and body mass index (BMI).

Results

Only 2/13 laterality indices differed (both FDR-adjusted $p = 0.013$). In unadjusted KOA-versus-HMO comparisons, dimensionless speed (0.177 vs 0.275), normalised stride length (39.342% vs 54.235% of height), forefoot impulse share (62.422% vs 71.775%) and anteroposterior COP excursion (67.297% vs 78.925%) were lower ($p < 0.001$). After FDR correction, 14/15 ROM-Mean, 11/11 ROM-SD and 9/11 ROM-CV outcomes were lower in KOA; after age, sex and BMI adjustment, 11/15, 11/11 and 7/11 remained significant. KL4 combined lower ankle with higher hip measures.

Conclusions

KOA was characterised by unadjusted descriptive differences in walking performance and plantar loading, while adjusted ROM models showed lower knee-ankle excursion and derived absolute ROM dispersion. Limited laterality and non-uniform KL-grade patterns argue against a single biomechanical severity gradient. These domains may inform longitudinal functional phenotyping and treatment-response research.

Background

Knee osteoarthritis (KOA) is a leading cause of persistent pain, mobility limitation and adult disability, with a burden expected to rise as populations age and obesity increases. Management combines

symptom relief, exercise, weight management and long-term self-management rather than relying on a single structural marker.[1–5] Patient-reported instruments such as the Knee injury and Osteoarthritis Outcome Score (KOOS) and Western Ontario and McMaster Universities Osteoarthritis Index (WOMAC), together with performance-based tests, are indispensable for describing physical function, pain and patient-relevant limitation.[6–8] They do not, however, directly quantify how lower-limb motion and plantar loading are organised during repeated gait cycles. Objective gait measurement is therefore best viewed as a complement to, not a replacement for, clinical and patient-reported assessment.

The history of KOA classification helps explain the need for a broader functional description. The Kellgren-Lawrence (KL) system, introduced in the 1950s, remains familiar, yet structural grade, pain and disability do not always move together, partly because pain mechanisms extend beyond radiographic change.[9–12] Knee instability and buckling add another dimension because they are linked to reduced balance confidence, fear of falling and subsequent functional decline even when radiographic grade alone provides an incomplete account.[13, 14] Instrumented gait analysis now quantifies progression, support timing, joint motion and foot-ground interaction beyond visual examination.[15–18] Methods range from laboratory and plantar-pressure systems[19, 20] to ambulatory wearable sensors.[21–23] For KOA, the unresolved question is therefore not whether walking is altered, but how several altered domains relate when they are measured together under the same walking condition.

Previous KOA studies consistently report slower walking and altered spatiotemporal performance, although some kinematic differences are attenuated when speed is controlled.[24, 25] A systematic review describes altered regional plantar loading in KOA.[26] Separate mechanics studies report abnormal or cumulative knee loading.[27–29] A dynamic-pressure study provides further plantar evidence,[30] and additional mechanics work addresses knee loading during walking.[31] Review and multijoint work show adaptations extending to the hip, ankle and trunk.[32, 33] More recent work has used multivariable or clustering approaches to identify gait profiles and candidate phenotypes.[34, 35] Much of the literature nevertheless isolates one measurement family, making it difficult to tell whether slower walking coexists with less forefoot-directed transfer, whether the largest motion differences remain confined to the knee, and whether repeated-cycle variability mirrors the change in mean excursion. Self-selected speed preserves habitual walking but makes speed part of, rather than independent from, the measured phenotype.

Mean and dispersion are related but non-equivalent: the mean describes typical performance, loading or excursion, whereas dispersion describes repeated observations. Kinematic variability has been studied in relation to gait stability in older adults and KOA, but its meaning depends on the variable, population and task.[36, 37] In this study, ROM-SD was algebraically derived from ROM-Mean and ROM-CV; it was therefore treated as derived absolute ROM dispersion, not as an independent measure of gait stability. Functional data analysis has been used to summarise time-varying movement data,[38] and principal component analysis (PCA) has identified KOA-related gait waveform features.[39] Restricting each three-variable block to one joint and plane was our design choice to preserve anatomical traceability. We used full-cycle, loading-response and single-support values and analysed typical joint range of motion (ROM-

Mean), derived absolute ROM dispersion (ROM-SD) and scale-relative dispersion (ROM-CV) in parallel, placing mean gait, signed left-right laterality, plantar measures and variability in one framework.

The primary objective was to characterise the multidomain gait phenotype associated with KOA during barefoot self-selected walking by comparing asymptomatic middle-aged and older reference participants with patients who had unilateral KOA and a definite affected side. We examined spatiotemporal and plantar-pressure/COP means and variability, fixed left-minus-right signed laterality, and lower-limb ROM-Mean, ROM-SD and ROM-CV. KL-grade, sex and affected-versus-contralateral comparisons, covariate-adjusted ROM models, KL associations and same-cohort rankings of the area under the receiver-operating-characteristic curve (AUC) for individual indicators were secondary and exploratory. We expected KOA to be accompanied by slower walking and reduced knee–ankle excursion, with domain-specific rather than uniformly increased variability.

Methods

Study design and setting

This single-centre cross-sectional study retrospectively analysed existing gait-biomechanics records. KOA records were collected between October 2022 and October 2023. KOA records came from patients assessed through the orthopaedic outpatient and inpatient services of the Second Hospital of Shanxi Medical University. Healthy-reference participants were recruited from the community and a university campus and underwent the same gait protocol.

Participants, reference sample and eligibility

The healthy-reference sample comprised 368 participants recruited from community and university-campus sources: 122 aged < 40 years, 112 aged 40–60 years inclusive and 134 aged > 60 years. The middle-aged and older strata formed the HMO group ($n = 246$) used in inferential comparisons with 92 participants with unilateral KOA (KL2/3/4, $n = 27/31/34$). For the present analysis, the 122 younger participants contributed only to fitting the fixed healthy-reference PCA coordinates and were excluded from HMO–KOA tests and AUC rankings. Their median (IQR) age, height, body mass and BMI were 26 (25–29) years, 170 (160–176) cm, 66.0 (57.625–76.65) kg and 22.848 (20.062–25.267) kg/m². Women accounted for 54 (44.3%) participants.

KOA eligibility required a clear clinical diagnosis, complete imaging records, a definite affected side and unilateral disease; all participants had anteroposterior and lateral knee radiographs. Three physicians experienced in osteoarthritis care or imaging independently assigned KL grades while blinded to gait results; disagreements were resolved by joint review. Bilateral KOA was not included, so no bilateral index-knee hierarchy was applied. The affected side was the recorded involved side; the contralateral side was asymptomatic but was not treated as an independently verified healthy control.

Healthy-reference eligibility required age within one of the three strata and independent natural walking with repeated valid passes. Participants could not depend on assistive devices and had no known lower-limb musculoskeletal disease or current lower-limb pain affecting walking. Exclusions were major lower-limb trauma, surgery or structural deformity, or neurological, vestibular or other disorders materially affecting balance or gait. Further exclusions were evident low-back, hip, knee or ankle pain at testing; inability to walk independently or complete valid passes; or insufficient kinematic, plantar-pressure or COP data quality.

Only retained records meeting the documented eligibility and data-quality criteria were included. Figure 1 presents the composition of the final analytic cohorts rather than a conventional enrolment cascade. Analyses requiring bilateral data were restricted to participants with the requisite bilateral measurements, and outcome-specific denominators are reported in the corresponding tables. Within the retained records, the same eligibility, acquisition-quality and participant-level side rules were applied, and cycles were aggregated before analysis. Blinded KL grading reduced measurement-related classification bias.

Study size and ethics

Study size was determined by all available records in the retained dataset meeting the eligibility and data-quality criteria; no target or post hoc adequacy threshold justified the final cohort. The approval of the study protocol by the Ethics Committee of the Second Hospital of Shanxi Medical University on 30 September 2022 (No. (2022) YX No. 213) .

Gait acquisition and cycle quality control

Three-dimensional lower-limb kinematics were acquired at 200 Hz with a Codamotion ODINx64 system and the laboratory's 28-marker cluster-and-pelvic-wand lower-limb protocol. Barefoot plantar-pressure and COP data were acquired at the protocol-specified 125 Hz using Footscan 7 gait second-generation software and a 2 m pressure plate in the centre of a 6 m walkway. An external hardware trigger started both systems, which then sampled on their own clocks rather than in frame-by-frame synchrony. Participants walked barefoot at a comfortable self-selected pace; speed was therefore an outcome rather than a routine adjustment covariate. Figure 1 summarises acquisition, side selection and the analysis sequence.

A complete gait cycle extended from one ipsilateral initial contact to the next. Loading response extended from ipsilateral initial contact to contralateral toe-off, and single support from contralateral toe-off to contralateral initial contact. Cycle and phase boundaries used initial-contact and toe-off events stored in ODIN. Participants repeated self-selected walking until at least 10 quality-accepted complete cycles were available per side. Cycles were accepted when footprints and pressure curves were complete, markers remained visible, the participant did not target the plate, and no measurement-region

interruption, acceleration/deceleration, loss of balance, marker occlusion or equipment error occurred. Accepted cycles were aggregated within participant and side before group analysis. All spatiotemporal, plantar-pressure/COP and ROM means and variability measures were calculated from cycle-level values; trials and cycles were not treated as independent observations.

Side rules and multidomain outcomes

One participant-level side rule was used across analyses. HMO mean, variability, association and AUC analyses used bilateral averages calculated after side-specific processing. KOA group, KL-grade, sex, association and AUC analyses used the recorded affected side. Affected-versus-contralateral comparisons retained both side-specific records and used paired statistics. Signed laterality indices were calculated from bilateral measurements with a fixed left-minus-right formula. Their sign therefore represents fixed anatomical side, not affected-minus-contralateral direction or asymmetry magnitude; pooling left- and right-affected cases may attenuate a disease-oriented signal.

Spatiotemporal outcomes described progression, step geometry and support timing. Step length, stride length and step width were divided by height and multiplied by 100 and are reported as % of height. Lower-limb length (L) was estimated as $0.53 \times \text{standing height}$.^[40] Dimensionless speed was calculated as $v/\sqrt{gL}$, where v is walking speed and $g = 9.81 \text{ m/s}^2$.^[41] Plantar outcomes comprised peak pressure-derived vertical force divided by body mass (N/kg); software-derived rearfoot, midfoot and forefoot contact-area and impulse shares, each expressed as a percentage of whole-foot totals; and COP anteroposterior excursion and path length divided by foot length or mediolateral excursion divided by foot width. Means and coefficients of variation (CVs) were calculated within participant and side from all quality-accepted cycle-level values. Seven spatiotemporal, three plantar-pressure/COP and three sagittal-plane joint-ROM signed laterality indices were analysed. Complete definitions, units and formulae are given in Supplementary Tables S1–S6 (see Additional file 1).

Joint ROM channels and phase-specific PCA

Hip, knee and ankle angle waveforms were summarised in frontal, sagittal and transverse planes over the full gait cycle, loading response and single support. For each participant, side, joint, plane and phase, ROM-Mean represented the typical excursion across accepted cycles. ROM-CV represented dispersion relative to that excursion. ROM-SD represented derived absolute ROM dispersion and was calculated from the unrounded participant-side summaries as $\text{ROM-Mean} \times \text{ROM-CV}/100$ before healthy bilateral averaging.

Separate three-variable PCA blocks were formed for each of nine joint-plane combinations and each ROM channel, using full-cycle, loading-response and single-support values. Parameters were fitted in all 368 healthy-reference participants; KOA records were projected into the fixed coordinates and did not contribute to refitting. ROM-Mean values were standardised directly, with log transformation examined as a sensitivity analysis, whereas positive ROM-SD and ROM-CV values were log transformed before

healthy-reference standardisation. The first principal component (PC1) orientation was fixed so that the full-cycle loading was positive. A component was retained only when all analysis-specific empirical retention criteria were satisfied: Kaiser–Meyer–Olkin (KMO) sampling adequacy at least 0.50; Bartlett's test of sphericity $p < 0.05$; Horn parallel analysis with 5000 simulations indicated retention of one component;[42] PC1 explained at least 50% of variance; every absolute loading was at least 0.40, the fifth percentile of 1000 participant-bootstrap Tucker congruence values was at least 0.90, and the minimum leave-one-participant-out congruence was at least 0.90. Six ROM-Mean and eight each of the ROM-SD and ROM-CV modules passed. Failed modules were represented by their three phase-specific variables. For retained modules meeting the family-level significance criterion, the three source phases were followed with module-level multiplicity correction. Diagnostics, loadings and phase contributions are in Supplementary Tables S7–S9 and Supplementary Figures S1–S3 (Additional file 1).

Statistical analysis

Continuous variables are reported as median (interquartile range) and categorical variables as count (percentage). Independent two-group comparisons used the Mann–Whitney U test; affected-versus-contralateral comparisons used the Wilcoxon signed-rank test; four-group comparisons used the Kruskal–Wallis test; and categorical variables used chi-squared tests. For outcomes with a significant four-group omnibus result after within-family correction, all six pairwise Mann–Whitney comparisons were reported with Benjamini–Hochberg adjustment.[43] Effect-size magnitude was $|r|=|Z|/\sqrt{N}$, where N was $n_1 + n_2$ for independent comparisons and the number of non-zero paired differences for paired comparisons; forest plots assigned the sign from the stated group or side direction. Kruskal–Wallis effect size was $\eta^2_H = \max[0, (H - k + 1)/(N - k)]$; negative untruncated values were reported as 0. Categorical effect sizes were Cramer's V. Baseline characteristics were Benjamini–Hochberg adjusted in separate HMO–KOA and KL-grade families ($m = 6$ each). Other analysis-defined families were: signed laterality $m = 13$; spatiotemporal mean $m = 9$; plantar-pressure/COP mean $m = 10$; spatiotemporal CV $m = 8$; plantar-pressure/COP CV $m = 10$; ROM-Mean $m = 15$; ROM-SD $m = 11$; and ROM-CV $m = 11$. Each selected three-phase analysis used $m = 3$. Supplementary Table S16 reports Wilcoxon location-shift estimates and 95% confidence intervals as group 2 minus group 1 (affected minus contralateral for paired contrasts); these are not confidence intervals for $|r|$. Statistical significance was a two-sided FDR-adjusted $p < 0.05$ based on unrounded values.

Covariate robustness analyses used ordinary least-squares regression with HC3 heteroscedasticity-robust standard errors.[44] With HMO and female as references, group, age, sex and body mass index were entered together; the group coefficient estimated the adjusted KOA–HMO ROM difference. Diagnostics included residual plots, Cook distance, condition number, variance-inflation factors and leave-one-participant-out sign stability. These models covered ROM outcomes only. Within KOA, exploratory associations with ordinal KL grade used Spearman ρ with separate FDR families for mean/signed-laterality ($m = 47$) and variability ($m = 40$). For descriptive single-indicator ranking, KOA was the positive class and the area under the receiver-operating-characteristic curve (AUC) was reported as $\max(\text{AUC}_{\text{raw}}, 1 - \text{AUC}_{\text{raw}})$. Homologous ROM-Mean versus ROM-SD or ROM-CV differences used

independently oriented scores. PCA fitting used the 368-person healthy-reference sample; AUC direction selection and ranking used the 246 HMO and 92 KOA inferential cohort. No confidence intervals, p values or paired tests were calculated for these descriptive same-cohort rankings. Available complete cases were analysed using IBM SPSS Statistics 31.0.1.0.

Use of generative AI and AI-assisted technologies

No generative AI or AI-assisted tools were used to generate scientific data, references, figures, analyses, or conclusions. Any editorial or formatting assistance used during manuscript preparation was reviewed and verified by the authors, who take full responsibility for the content of the article.

Results

Participants, baseline characteristics and signed laterality

The inferential cohort included 246 HMO and 92 KOA participants. Sex distribution, median age and height were similar. KOA included more participants in the study-defined older stratum (72.8% vs 54.5%; FDR-adjusted $p = 0.007$), and median body mass (66.5 vs 64.0 kg; FDR-adjusted $p = 0.006$) and body mass index (26.602 vs 25.173 kg/m²; FDR-adjusted $p = 0.003$) were higher. The KL groups did not differ in the six recorded baseline characteristics (Table 1; Fig. 2A; Supplementary Table S10 (Additional file 2)).

Table 1
Baseline characteristics of HMO and KOA participants

Indicator	HMO (n = 246)	KOA (n = 92)	Statistic	P	FDR-adjusted P	Effect size
Sex	Female 189 (76.8%); Male 57 (23.2%)	Female 68 (73.9%); Male 24 (26.1%)	$\chi^2 = 0.173$	0.677	0.677	V = 0.023
Age category	Older 134 (54.5%); Middle-aged 112 (45.5%)	Older 67 (72.8%); Middle-aged 25 (27.2%)	$\chi^2 = 8.613$	0.003	0.007	V = 0.160
Age (years)	63 (54, 69)	64 (59, 67.25)	U = 9928.5	0.083	0.124	r = 0.094
Height (cm)	158 (155, 163)	160 (155, 165)	U = 10807.5	0.524	0.629	r = 0.035
Body mass (kg)	64 (57.125, 71)	66.5 (60, 77)	U = 8836.5	0.002	0.006	r = 0.169
BMI (kg/m ²)	25.173 (23.024, 27.344)	26.602 (24.643, 29.163)	U = 8522.0	< 0.001	0.003	r = 0.190
Data are median (IQR) or n (%). P values were FDR-adjusted by Benjamini–Hochberg. HMO, healthy middle-aged and older; KOA, knee osteoarthritis; BMI, body mass index. Mann–Whitney r gives magnitude; group summaries give direction.						

Only two of 13 fixed left-minus-right indices differed. COP mediolateral-excision laterality was 13.484 (– 1.082, 30.614) in HMO and 0.364 (– 27.890, 24.508) in KOA (|r|=0.168); knee sagittal-plane ROM laterality was 7.209 (– 0.875, 15.540) and 0.729 (– 15.792, 14.537), respectively (|r|=0.173; both FDR-adjusted p = 0.013). Only the latter differed across HMO and KL groups. The signs describe anatomical direction, not affected-side asymmetry (Fig. 2B–F; Supplementary Tables S11–S12 (Additional file 2) and S20–S21 (Additional file 1)).

Spatiotemporal performance and variability

In unadjusted group comparisons, all nine spatiotemporal means differed. Values are presented as KOA versus HMO. Dimensionless speed was 0.177 (0.138, 0.219) versus 0.275 (0.247, 0.311; |r|=0.622), and normalised stride length was 39.342% of height versus 54.235% of height (|r|=0.550; all FDR-adjusted p < 0.001). Step width, step and gait-cycle times, stance and double-support times were higher, whereas single-support time was lower (375.167 vs 421.560 ms; FDR-adjusted p = 0.002). Eight mean outcomes differed across HMO and KL strata; dimensionless speed had the largest omnibus effect ($\eta^2_H=0.389$), with all three grades below HMO and KL4 below KL3; other outcomes lacked a consistent KL2-to-KL4 order.

Values are presented as KOA versus HMO. Spatiotemporal CVs did not show a global increase. Stance-time CV was higher in KOA [23.275% (8.980, 46.280) vs 9.765% (6.270, 18.585); |r|=0.283], whereas step-

width CV [18.020% (9.812, 41.160) vs 42.145% (27.559, 58.919); $|r|=0.360$] and single-support-time CV [16.545% (9.525, 60.010) vs 28.735% (15.500, 58.796); $|r|=0.147$] were lower. In the grade analysis, step-width and stance-time CV separated HMO from all KOA grades in opposite directions (Fig. 3; Supplementary Tables S11–S12 and S20–S21).

Plantar-pressure and centre-of-pressure outcomes

In unadjusted group comparisons, nine of ten plantar-pressure/COP means differed. Values are presented as KOA versus HMO: KOA had higher peak vertical ground reaction force ($|r|=0.246$), larger heel and midfoot impulse shares, and lower forefoot contact-area and impulse shares. Forefoot impulse share showed the largest regional redistribution [62.422% (61.067, 69.532) vs 71.775% (67.462, 75.085); $|r|=0.380$]. All three COP trajectory measures were shorter: anteroposterior excursion was 67.297% versus 78.925% ($|r|=0.393$), mediolateral excursion 28.833% versus 32.755% ($|r|=0.264$), and path length 74.064% versus 84.614% ($|r|=0.347$; all FDR-adjusted $p < 0.001$). All ten means differed across HMO and KL strata without a uniform grade pattern.

Values are presented as KOA versus HMO. Five of ten plantar-pressure/COP CVs differed. Midfoot contact-area and impulse CVs were lower in KOA (7.420% vs 21.680%, $|r|=0.352$; 22.345% vs 41.105%, $|r|=0.447$), whereas anteroposterior, mediolateral and path-length COP CVs were higher ($|r|=0.267$, 0.224 and 0.296; all FDR-adjusted $p < 0.001$). Nine CV outcomes differed across HMO and KL strata (Fig. 4; Supplementary Tables S11–S12 (Additional file 2) and S20–S21 (Additional file 1)).

Lower-limb ROM mean, derived absolute dispersion and relative dispersion

Six ROM-Mean and eight each of ROM-SD and ROM-CV combinations passed the PCA gates. Fourteen of 15 ROM-Mean outcomes differed between HMO and KOA. Values are presented as KOA versus HMO. The strongest effect was lower full-cycle ankle transverse-plane ROM [15.767° (13.324, 19.874) vs 29.426° (26.249, 32.920); $|r|=0.707$], followed by knee sagittal ROM-Mean PC1 ($|r|=0.639$), ankle frontal ROM-Mean PC1 ($|r|=0.612$), single-support hip sagittal ROM ($|r|=0.605$) and knee transverse ROM-Mean PC1 ($|r|=0.592$). Loading-response hip sagittal ROM was the only significant ROM-Mean outcome higher in KOA. For knee sagittal and transverse and ankle frontal PC1 modules, all full-cycle, loading-response and single-support source measures were lower, showing that the component effects were not driven by one isolated phase.

In unadjusted group comparisons, all 11 ROM-SD outcomes were lower in KOA. The largest effects involved ankle frontal, transverse and sagittal ROM-SD PC1 ($|r|=0.678$, 0.666 and 0.632), followed by knee transverse phase-specific ROM-SD and knee sagittal ROM-SD PC1. All 24 source-phase measures underlying the eight significant modules were lower after module-level correction. Nine of 11 ROM-CV outcomes were lower in KOA after FDR correction, with the largest effects in ankle frontal, sagittal and transverse modules ($|r|=0.559$, 0.553 and 0.452). ROM-CV did not reproduce every ROM-SD result,

indicating that the strength of the dispersion contrast depended partly on the corresponding excursion magnitude (Figs. 5 and 6; Supplementary Tables S15–S16 (Additional file 2) and S20).

Radiographic grade, sex and affected-versus-contralateral patterns

Selected KL contrasts pointed in opposite distal and proximal directions. Full-cycle ankle transverse ROM-Mean was 19.379° in KL3 and 14.379° in KL4. In contrast, hip frontal/transverse ROM-Mean PC1 and full-cycle hip sagittal ROM were higher in KL4 than in KL2 and KL3. KL4 also combined lower ankle frontal/transverse ROM-SD PC1 with higher hip ROM-SD PC1, and ROM-CV showed a similar higher-hip pattern. These were selected cross-sectional contrasts, not a uniform grade sequence (Supplementary Tables S12, S15–S16 and S21).

Within KOA, men had longer normalised step length [28.348% versus 22.094% of height, men versus women; FDR-adjusted $p = 0.031$, $|r| = 0.305$] and higher double-support-time CV [61.595% vs 38.505%, men versus women; FDR-adjusted $p = 0.014$, $|r| = 0.326$]. No plantar-pressure/COP or ROM outcome differed by sex after FDR correction. Paired analysis detected no spatiotemporal or plantar-COP CV difference. Heel contact-area percentage was lower on the affected side [21.100% vs 22.492%, affected versus contralateral; FDR-adjusted $p = 0.030$, $|r| = 0.309$], and six ROM-Mean outcomes were lower, led by knee sagittal ROM-Mean PC1 ($|r| = 0.596$). No ROM-SD or ROM-CV side difference was detected (Fig. 6; Supplementary Tables S13–S16).

Adjusted ROM effects, KL associations and apparent single-indicator separation

After adjustment for age, sex and BMI, 11 of 15 ROM-Mean, all 11 ROM-SD and 7 of 11 ROM-CV group differences remained FDR-significant. The largest adjusted mean differences were full-cycle ankle transverse ROM ($\beta = -12.913^\circ$, 95% confidence interval (CI) – 14.192 to – 11.634) and single-support hip sagittal ROM ($\beta = -10.103^\circ$). All three ankle ROM-SD modules remained lower, and every FDR-significant group coefficient retained its direction during leave-one-participant-out checks (Fig. 7A; Supplementary Table S17 (Additional file 2)).

Twenty-four of 87 exploratory KL associations were FDR significant. Mean-domain associations were led by hip frontal ROM-Mean PC1 ($p = 0.513$), single-support ankle transverse ROM ($p = -0.455$), full-cycle hip sagittal ROM and hip transverse ROM-Mean PC1 (both $p = 0.432$). Variability associations were strongest for hip frontal and sagittal ROM-SD PC1 ($p = 0.543$ and 0.516), while ankle frontal and transverse ROM-SD PC1 were negatively associated with grade ($p = -0.312$ and -0.346), consistent with the selected lower-ankle/higher-hip contrasts (Fig. 7B; Supplementary Table S18 (Additional file 2)).

Direction-selected apparent AUCs were highest for full-cycle ankle transverse ROM-Mean (0.959), ankle frontal ROM-SD PC1 (0.940), ankle transverse ROM-SD PC1 (0.932), knee sagittal ROM-Mean PC1 (0.915), ankle sagittal ROM-SD PC1 (0.910) and dimensionless speed (0.904). Homologous differences

favoured ROM-SD for ankle and hip frontal PC1, whereas knee sagittal ROM-Mean exceeded both variability forms. Thus, the measurement level with the greatest apparent separation differed by joint and plane. These estimates were generated, direction-selected and ranked in the same cohort and are descriptive, not diagnostic performance measures (Fig. 7C–D; Supplementary Table S19 (Additional file 2)).

Discussion

This study integrates gait dimensions that are often reported separately. During self-selected walking, KOA was characterised by slower forward gait, prolonged support, lower forefoot impulse share, shorter COP trajectories, restricted knee–ankle excursion and reduced derived absolute ROM dispersion. Taken together, the findings showed a consistent overall pattern, although individual domains did not change uniformly: fixed left-minus-right laterality was focal, variability changed in both directions, and KL grades did not form a proportional ladder. This is a descriptive phenotype, not a causal mechanism or validated classifier.

The unadjusted descriptive spatiotemporal differences agree with evidence that people with KOA generally walk more slowly and reorganise support timing.[24, 25] Variability did not simply mirror the means: stance-time CV was higher, whereas step-width and single-support-time CVs were lower. Higher peak vertical force and greater heel/midfoot impulse shares coexisted with lower forefoot area and impulse shares and shorter COP excursions. Systematic-review and dynamic-pressure studies report altered regional plantar loading in KOA.[26, 30] Pressure-derived estimates depend on sensor hardware and testing and processing protocols.[45] Separate mechanics studies describe abnormal or cumulative knee loading during walking.[27–29, 31] Same-participant measurement shows that these features coexist, although it does not establish joint predictive value. Higher COP CVs alongside lower midfoot area/impulse CVs argue against labelling the entire phenotype ‘unstable’. Self-selected pace remains part of the phenotype.

Joint kinematics made the distal emphasis particularly clear. The largest unadjusted and adjusted differences involved ankle transverse/frontal motion, knee sagittal/transverse motion and single-support hip sagittal motion; several knee and ankle modules changed in the same direction across all three phases. This ankle–knee–hip distribution is consistent with chain-wide adaptations reported in KOA.[32, 33] The anatomically restricted PCA preserved traceability to full-cycle, loading-response and single-support ROM while reducing correlated measures; restricting each block to one joint and plane was an analysis design choice rather than a conclusion supplied by prior PCA studies.[38, 39] Variability qualified that picture. All ROM-SD outcomes and nine ROM-CV outcomes were lower in KOA, with the largest contrasts at the ankle. Lower derived absolute ROM dispersion should not be interpreted as superior stability; because ROM-SD was calculated as $\text{ROM-Mean} \times \text{ROM-CV} / 100$, it indicates a narrower absolute spread of participant-level summaries, not an independent cycle-level stability estimate. The distal concentration also cautions against treating the knee as the only relevant measurement site when describing whole-limb walking function in KOA.

Grade-, sex- and side-stratified analyses did not support a single biomechanical severity gradient. Selected KL4 contrasts combined lower ankle with higher hip measures, and exploratory correlations reproduced this distal-negative/proximal-positive pattern. Such cross-sectional configurations may reflect heterogeneous walking solutions, but they cannot establish compensation, progression or a temporal transfer of motion. That caution accords with the known discordance among radiographic grade, symptoms and function.[9–14] Sex differences were limited to normalised step length and double-support-time CV. Six ROM-Mean outcomes, but no ROM-SD or ROM-CV outcome, were lower on the affected side. Finally, only two fixed left-minus-right indices differed; because their sign encodes anatomy rather than disease side, pooled left- and right-affected cases can cancel a disease-oriented signal. Between-person and within-person contrasts therefore capture non-identical aspects of walking, and radiographic grade is not a simple biomechanical severity continuum.

The study's main strength is the common participant-level framework: an externally triggered walking protocol, fixed side rules, within-family FDR control, phase-traceable PCA modules and age-, sex- and body-mass-index-adjusted ROM models. Adjustment did not materially attenuate most measured knee–ankle ROM group differences. At present, these measures are best regarded as candidates for assessment rather than treatment targets. Dimensionless speed and stride length, forefoot impulse share, COP excursion, ankle transverse/frontal excursion, knee sagittal excursion and joint-specific dispersion are concrete domains for longitudinal validation. Loading and alignment measures have been related to later cartilage loss, radiographic change and functional decline,[46–49] and the IDEA trial showed that intensive diet and exercise can alter joint loading and clinical outcomes.[50] Future work should test reliability, responsiveness, links with pain, KOOS/WOMAC, strength, alignment and activity, and added value beyond routine measures in independent cohorts. Measurement error and clinically important change thresholds are needed before individual monitoring. Until then, observing distal as well as knee motion and distinguishing lower derived absolute ROM dispersion from excessive variability may broaden evaluation, but does not define a treatment target.

Several limitations constrain interpretation. These findings directly describe reference volunteers meeting the recorded eligibility criteria and hospital-based KL2–4 KOA patients able to complete barefoot self-selected laboratory walking; other populations, footwear, tasks and centres require validation. The retrospective cross-sectional design cannot establish temporal sequence, prognosis or treatment response, and the KL strata were modest (27/31/34). Speed remained part of the phenotype; adjusted models covered ROM only.

Conclusions

During barefoot self-selected walking, KOA showed unadjusted descriptive group differences in walking speed, stride length, support timing, forefoot impulse share and COP trajectories. ROM models adjusted for age, sex and BMI identified restricted knee–ankle excursion and lower derived absolute ROM dispersion. Mean measures described typical movement and loading; variability was domain- and joint-specific, not globally more irregular. Grade comparisons combined lower ankle with higher hip measures

and did not form a uniform severity sequence. Walking speed and stride length, forefoot impulse share, COP excursion and joint-specific ROM dispersion now require longitudinal, independent validation.

Abbreviations

AUC

area under the receiver-operating-characteristic curve

BMI

body mass index

CI

confidence interval

COP

centre of pressure

CV

coefficient of variation

FDR

false discovery rate

HC3

heteroscedasticity-consistent standard errors

HMO

healthy middle-aged and older

IQR

interquartile range

KL

Kellgren-Lawrence

KMO

Kaiser-Meyer-Olkin

KOA

knee osteoarthritis

KOOS

Knee injury and Osteoarthritis Outcome Score

PC1

first principal component

PCA

principal component analysis

ROM

range of motion

ROM-CV

relative ROM dispersion

ROM-Mean

typical ROM excursion

ROM-SD

derived absolute ROM dispersion

WOMAC

Western Ontario and McMaster Universities Osteoarthritis Index.

Declarations

Ethics approval and consent to participate

The approval of the study protocol by the Ethics Committee of the Second Hospital of Shanxi Medical University on 30 September 2022 (No. (2022) YX No. 213) .

Consent for publication

Not applicable

Competing interests

The authors declare that they have no competing interests.

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Author Contribution

BZ: writing-original draft, writing-review and editing, software, formal analysis and data curation. YL: writing-review and editing, methodology and formal analysis. GZ: software and writing-review and editing. MF: validation, methodology and writing-review and editing. YZ: visualisation, validation and writing-review and editing. RZ: writing-original draft, writing-review and editing, software and formal analysis. PL: validation, supervision, project administration and writing-review and editing. XW: conceptualisation, validation, supervision, project administration and writing-review and editing. All authors read and approved the final manuscript.

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Authors' information

Not applicable.

Data Availability

The de-identified participant-level data are not publicly available because consent and institutional data-governance permission for public release have not been confirmed. Requests may be directed to the corresponding author and will be considered by the responsible institution subject to institutional approval, applicable ethics requirements and a data-use agreement. Aggregate results supporting the findings are provided in the article and its additional files.

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Figures

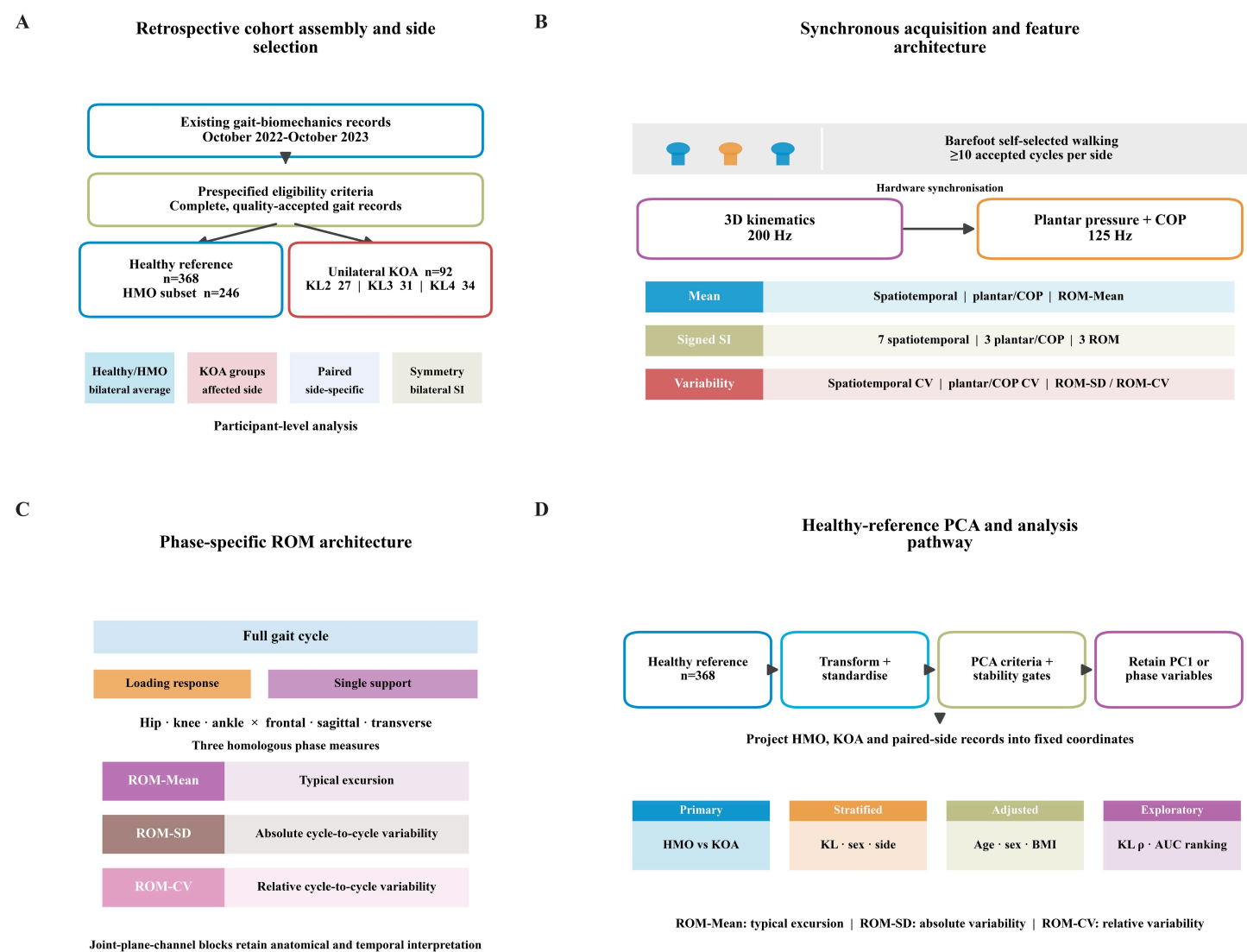


Figure 1

Final analytic cohorts, acquisition and analysis framework. (A) Final cohorts formed from retained gait-biomechanics records meeting documented eligibility and data-quality criteria. Of 368 healthy-reference participants meeting the documented eligibility criteria, 246 adults aged ≥ 40 years formed the healthy middle-aged and older (HMO) inferential group. The other 122 participants were aged < 40 years and were used only to fit the healthy-reference PCA coordinates; they were excluded from HMO–KOA tests and AUC rankings. Knee osteoarthritis (KOA) analyses included 92 participants with unilateral disease and a definite affected side across Kellgren–Lawrence (KL) grades 2–4. The available source materials do not provide the initial screening denominator, stage-specific exclusions or exclusion-by-reason counts; these quantities were not reconstructed. (B) Externally triggered acquisition and the mean, fixed left-minus-right signed-laterality and variability domains. (C) Range-of-motion mean (ROM-Mean), derived absolute ROM dispersion (ROM-SD) and relative dispersion (ROM-CV) channels formed from full-

cycle, loading-response and single-support measures. (D) Healthy-reference principal component analysis (PCA) fitting, projection and the sequence of primary, stratified, adjusted and exploratory analyses. SD, standard deviation; CV, coefficient of variation.

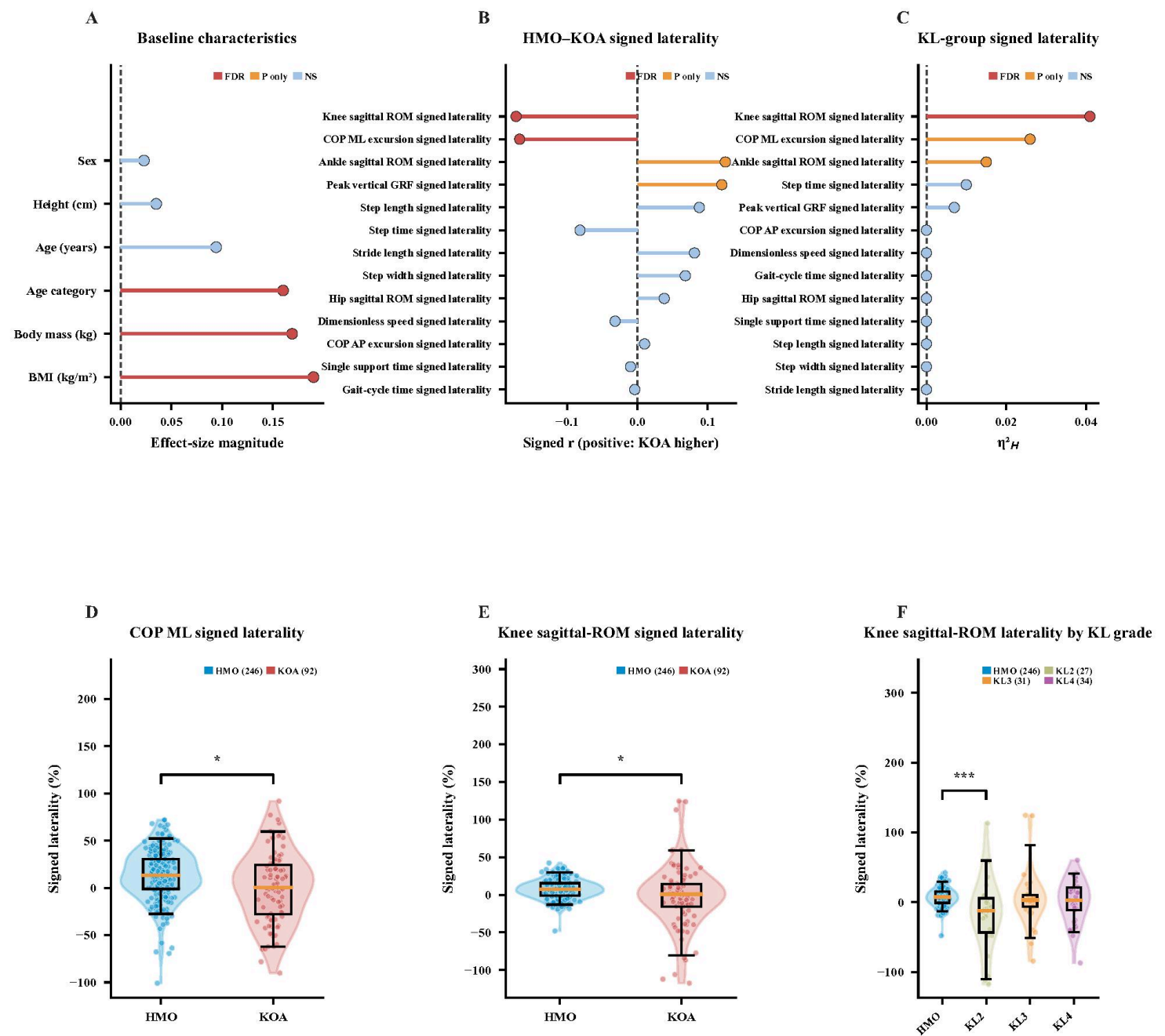


Figure 2

Baseline characteristics and fixed left-right laterality. (A) Baseline effect-size magnitudes for HMO versus KOA. (B) All 13 HMO–KOA signed-laterality effects. (C) Signed-laterality effects across HMO, KL2, KL3 and KL4. (D) Centre-of-pressure (COP) mediolateral-excursion signed-laterality-index distribution. (E) Knee sagittal-plane ROM signed-laterality-index distribution. (F) Knee sagittal-plane ROM signed laterality across HMO and KL grades. Indices use fixed left-minus-right coding and therefore describe anatomical side, not affected-versus-contralateral direction. The plot legend identifies FDR-significant, nominal-only

and non-significant evidence categories. Violin panels show raw observations, kernel density, interquartile boxes, medians and FDR-significant comparisons.

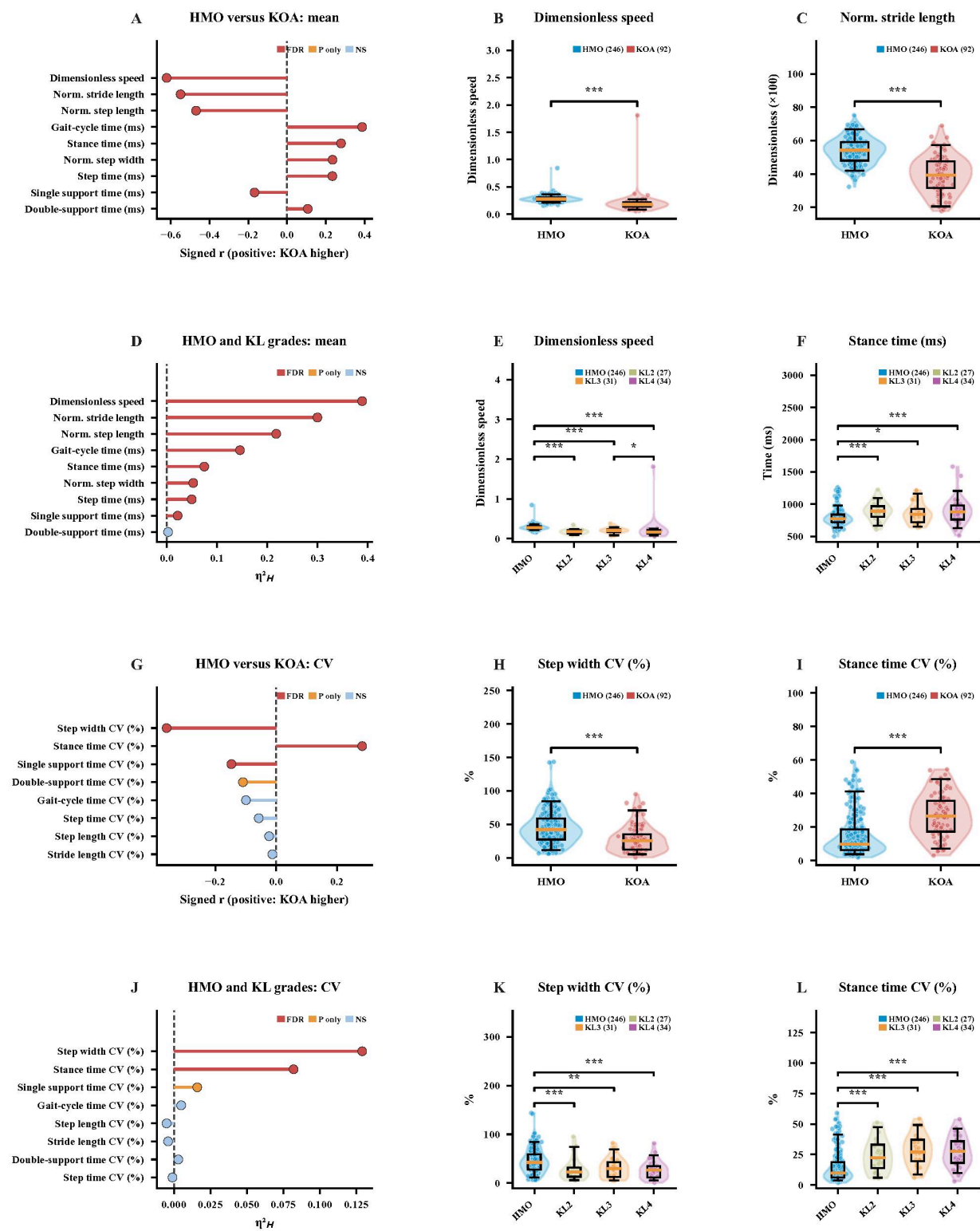


Figure 3

Spatiotemporal gait means and variability. (A) Complete HMO–KOA mean effect-size profile. (B–C) HMO–KOA distributions of dimensionless speed and normalised stride length. (D) Complete mean

effect-size profile across HMO and KL grades. (E–F) Grade distributions of dimensionless speed and stance time. (G) Complete HMO–KOA coefficient-of-variation (CV) effect-size profile. (H–I) HMO–KOA distributions of step-width and stance-time CV. (J) Complete CV effect-size profile across HMO and KL grades. (K–L) Grade distributions of step-width and stance-time CV. Positive signed r values indicate higher values in KOA. Evidence categories and violin elements are defined as in Figure 2.

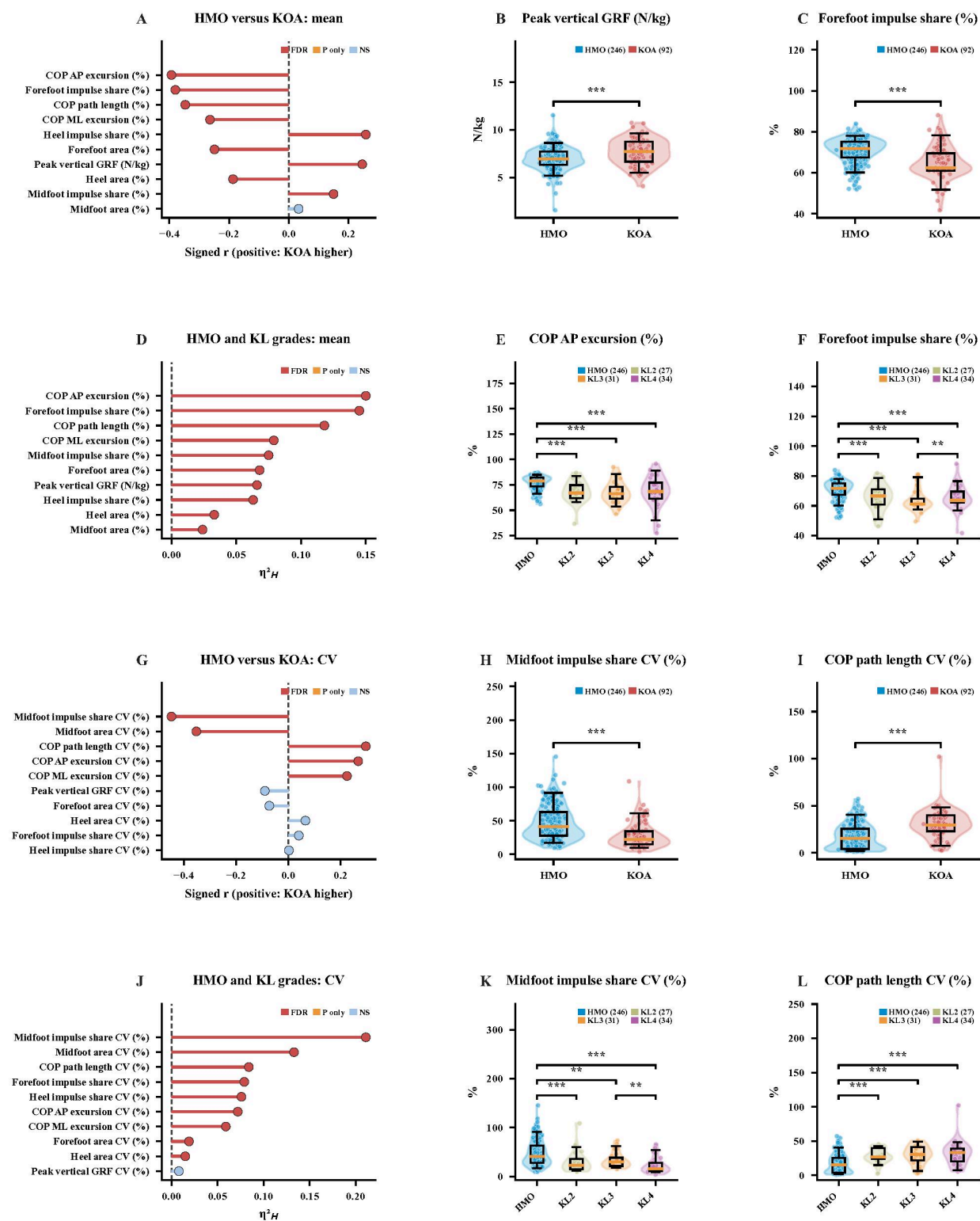


Figure 4

Plantar-pressure and centre-of-pressure means and variability. (A) Complete HMO–KOA mean effect-size profile. (B–C) HMO–KOA distributions of peak vertical ground reaction force and forefoot impulse share. (D) Complete mean effect-size profile across HMO and KL grades. (E–F) Grade distributions of COP anteroposterior excursion and forefoot impulse share. (G) Complete HMO–KOA CV effect-size profile. (H–I) HMO–KOA distributions of midfoot contact-impulse CV and COP path-length CV. (J) Complete CV effect-size profile across HMO and KL grades. (K–L) Corresponding grade distributions. Positive signed r values indicate higher values in KOA. Evidence categories and violin elements are defined as in Figure 2.

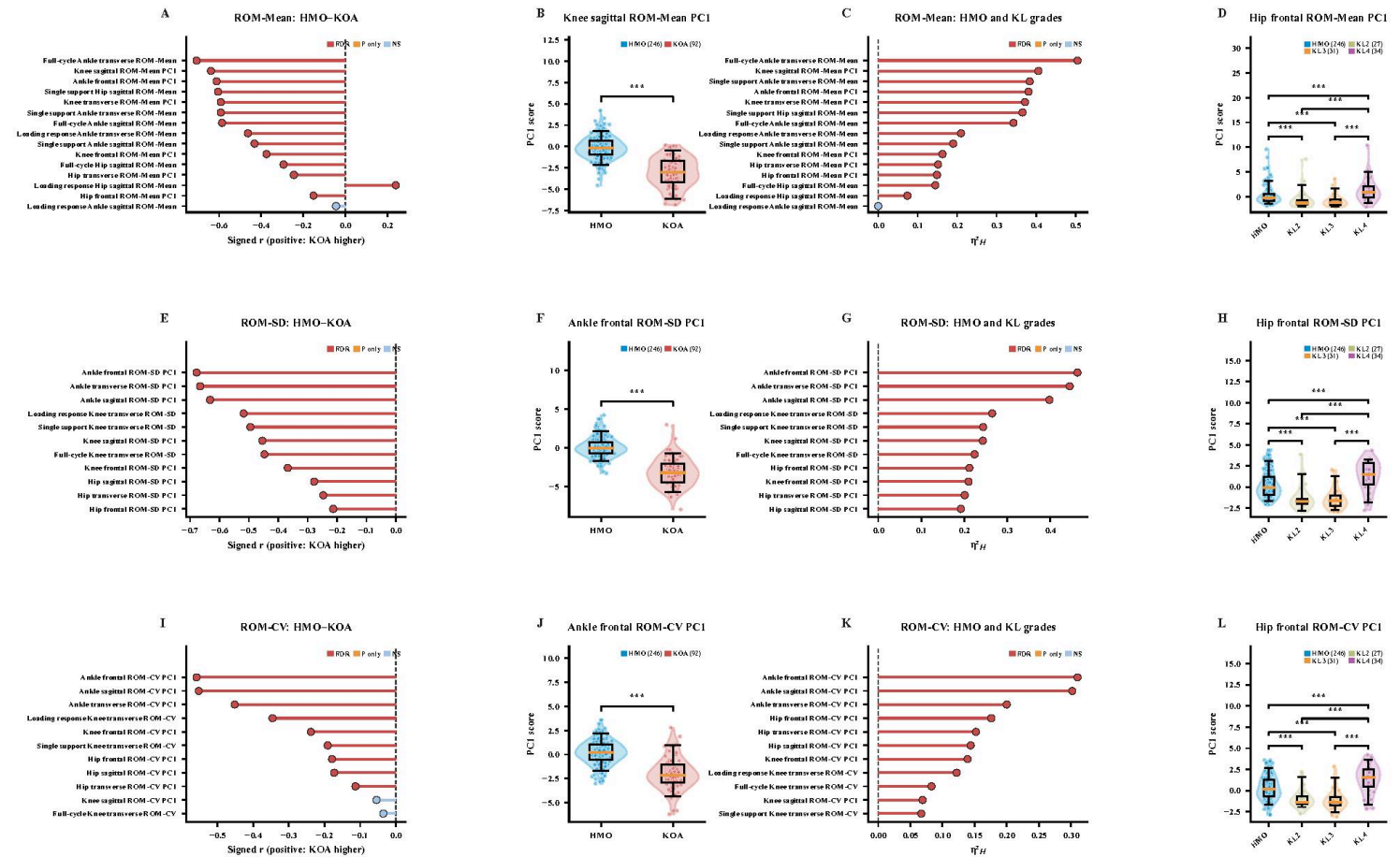


Figure 5

Group and KL-grade differences in joint ROM channels. (A) Complete HMO–KOA ROM-Mean effect-size profile. (B) Knee sagittal-plane ROM-Mean PC1 distribution. (C) Complete ROM-Mean effect-size profile across HMO and KL grades. (D) Hip frontal-plane ROM-Mean PC1 grade distribution. (E) Complete HMO–KOA ROM-SD effect-size profile. (F) Ankle frontal-plane ROM-SD PC1 distribution. (G) Complete ROM-SD effect-size profile across HMO and KL grades. (H) Hip frontal-plane ROM-SD PC1 grade distribution. (I) Complete HMO–KOA ROM-CV effect-size profile. (J) Ankle frontal-plane ROM-CV PC1 distribution. (K) Complete ROM-CV effect-size profile across HMO and KL grades. (L) Hip frontal-plane ROM-CV PC1 grade distribution. Positive signed r values indicate higher values in KOA; η^2_H denotes the Kruskal–Wallis effect size.

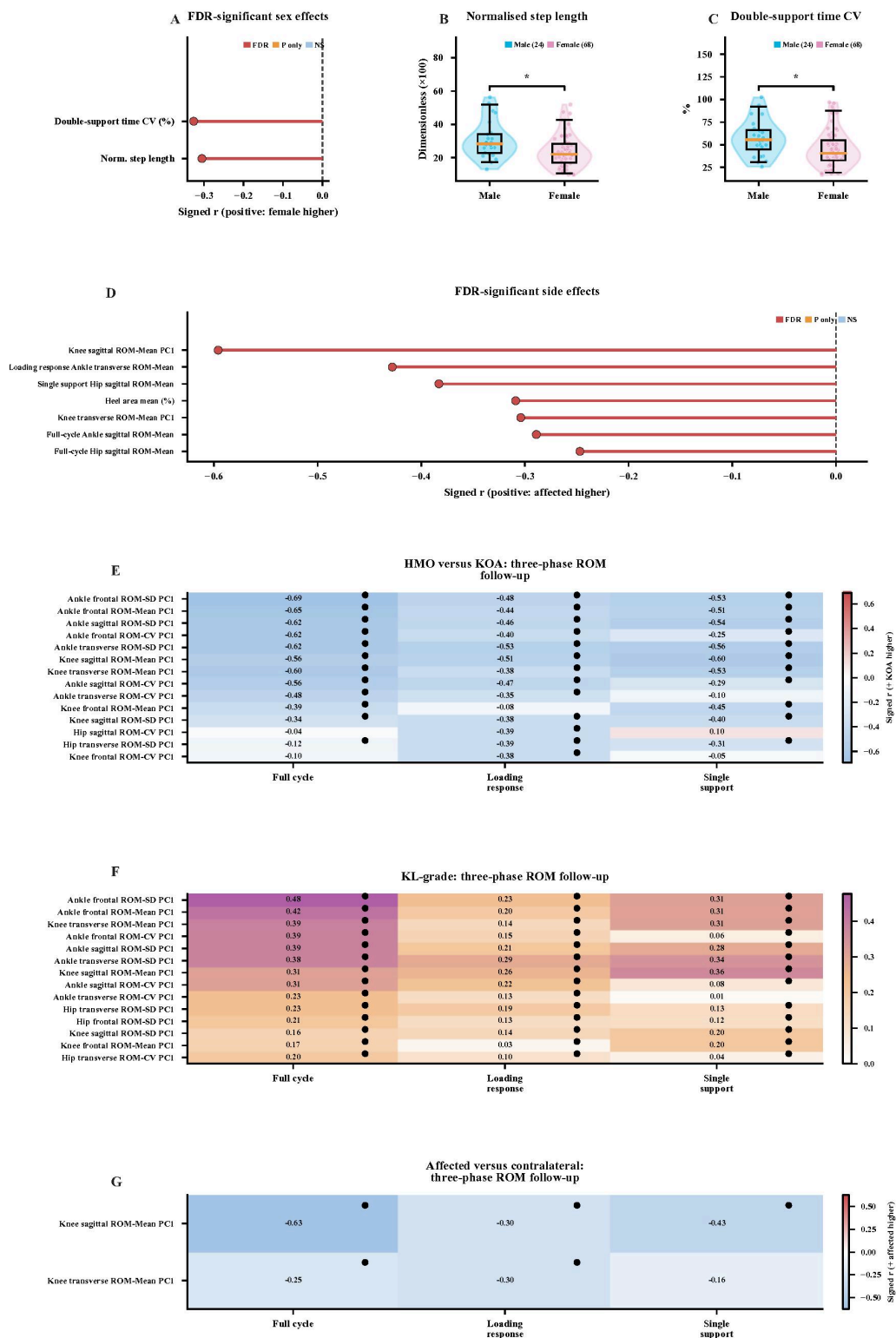


Figure 6

Sex, side and phase-specific ROM follow-up analyses. (A) FDR-significant sex effects within KOA. (B–C) Distributions of normalised step length and double-support-time CV by sex. (D) FDR-significant affected-versus-contralateral side contrasts. (E) Three-phase ROM analysis for HMO versus KOA. (F) Three-phase ROM analysis across HMO and KL grades. (G) Three-phase ROM analysis for affected versus contralateral sides. Heatmap cells show effect sizes for full-cycle, loading-response and single-support

ROM; black dots denote FDR-adjusted $p < 0.05$. In E and G, warmer red and cooler blue cells provide a secondary encoding of positive and negative signed values, respectively, while printed values give the exact direction; F uses a sequential magnitude scale. In signed panels, positive values indicate higher values in females in A, on the affected side in D and G, and in KOA in E; negative values indicate the corresponding comparison group or side in A, D, E and G.

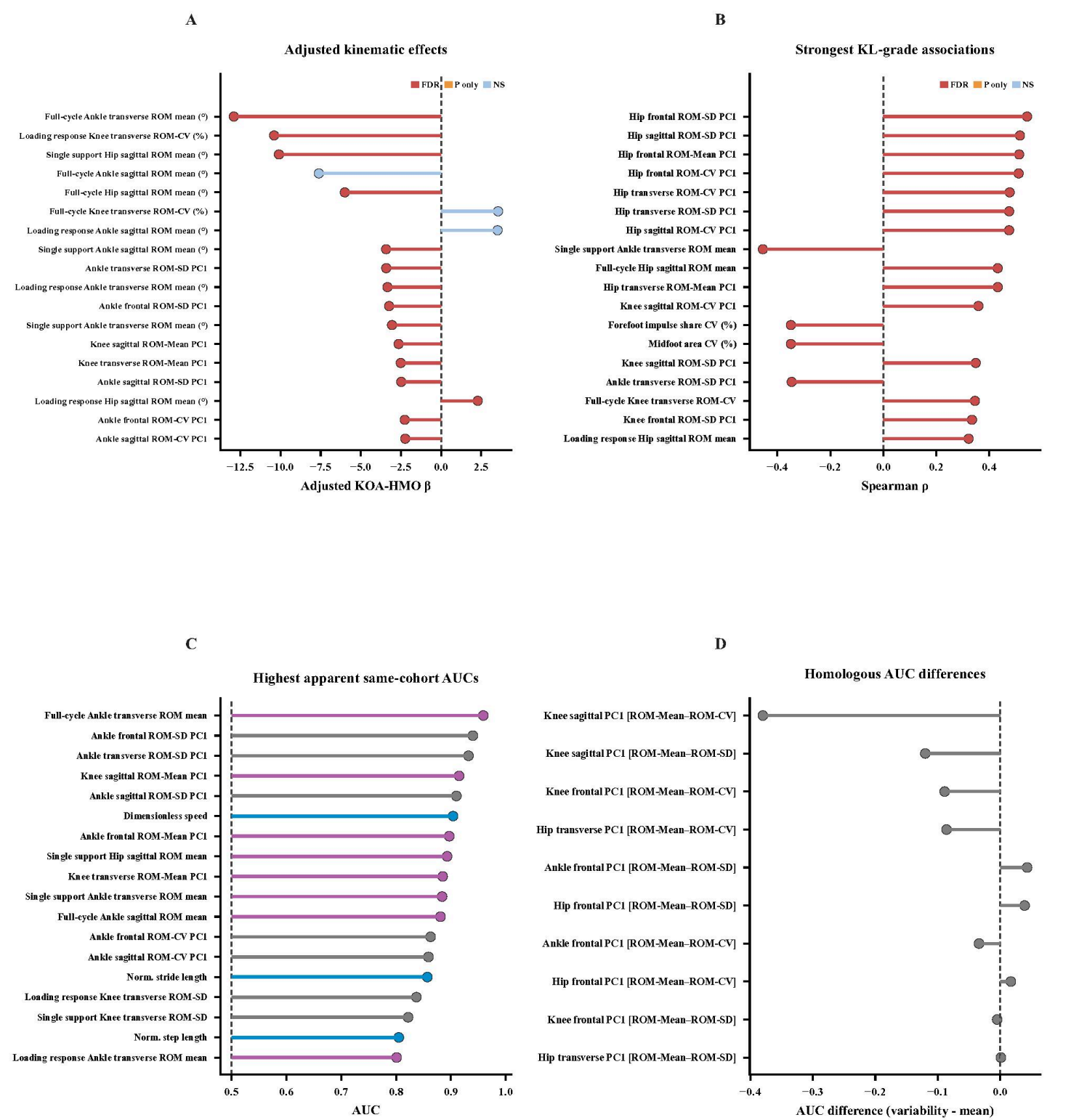


Figure 7

Adjusted ROM effects, KL associations and same-cohort AUC rankings. (A) Largest age-, sex- and body-mass-index-adjusted kinematic effects. Coefficients are in outcome-specific native units and their magnitudes are not directly comparable across outcomes. (B) Strongest Spearman associations with KL grade. (C) Highest direction-selected apparent same-cohort areas under the receiver-operating-characteristic curve (AUCs). (D) AUC differences for independently oriented homologous ROM-Mean versus ROM-SD and ROM-Mean versus ROM-CV features. Positive differences favour the variability feature; negative differences favour the mean feature. Evidence categories apply to panels A–B; panel D uses neutral markers. Score direction and ranking were selected in the same cohort, so panels C–D show no confidence intervals, p values or inferential significance and should not be read as diagnostic performance.

Supplementary Files

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- [Additionalfile1OnlineSupplement.docx](#)
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