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Etiology-Specific Ankle Dorsiflexion Limitation Reorganizes Stance-Phase Biomechanics during Barefoot Gait: A Time-Resolved SPM1D and EMG Study

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Abstract

Purpose: This study aimed to comparatively analyze the lower-limb biomechanical differences between two populations with ankle dorsiflexion limitation during the stance phase of gait, thereby providing a scientific basis for clinical rehabilitation and athletic training. **Methods:** We recruited 12 males with congenital ankle dorsiflexion limitation and 12 males with acquired ankle dorsiflexion limitation, along with 12 healthy male controls. Group differences in lower-limb kinematics, kinetics, and surface electromyography (sEMG) during barefoot walking were subjected to statistical analysis using one-dimensional Statistical Parametric Mapping (SPM1D). **Results:** The Congenital Ankle Dorsiflexion-Limited Cohort (CDFL) had greater ankle dorsiflexion during the support phase (22–42%) than the Acquired Ankle Dorsiflexion-Limited Cohort (ADFL) (p=0.003), with increased terminal plantar flexion torque and positive power, and higher average activation intensity of the gastrocnemius medialis (GM), while the power trajectory was close to that of the control group. The ADFL had higher plantar flexion torque during 0–83% of the gait cycle (p=0.001), increased knee flexion throughout the gait cycle (p=0.014), and elevated negative knee power and positive hip power in the middle and late stages, with increased average activation intensity of the rectus femoris (RF) and decreased activation intensity of the GM and tibialis anterior (TA). **Conclusion:** This study reveals phenotype-specific gait adaptations associated with different etiologies of ankle dorsiflexion limitation, ADFL predominantly exhibit a proximal-compensation pattern, whereas those with CDFL favor a distal strategy. These findings argue for etiology-tailored rehabilitation—strengthening ankle push-off and distal–proximal coordination in ADFL, and prioritizing terminal push-off and lateral stability in CDFL.

Keywords: Barefoot walking; gait biomechanics; surface EMG; congenital ankle dorsiflexion limitation; acquired ankle dorsiflexion limitation.

Introduction

Ankle dorsiflexion (DF) range of motion (ROM) exerts a foundational influence on gait mechanics. Functioning as the central hub for impact attenuation, elastic-energy storage and return, and terminal push-off during stance, the ankle–foot complex and lower-leg musculature jointly shape and regulate the lower limb's multi-joint spatiotemporal coordination. Any restriction of dorsiflexion ROM cascades proximally along the kinetic chain, altering the timing and magnitude of sagittal-plane angle–moment–power profiles at the knee and hip, thereby affecting energy transfer and locomotor efficiency. Clinically, ankle dorsiflexion limitation is frequently observed in the context of chronic Achilles–gastrocnemius tightness, sequelae of ankle sprain, prolonged immobilization, and scar adhesions; it may also arise from congenital soft-tissue or osseous morphological variants. Importantly, ankle dorsiflexion limitation (DFL) is not a unitary phenotype but comprises at least two clinically meaningful subtypes: congenital ankle dorsiflexion limitation (CDFL) and acquired ankle dorsiflexion limitation (ADFL). These subtypes likely impose different constraints on distal ankle–foot structures and, in turn, elicit distinct proximal compensatory pathways. CDFL often co-occurs with developmental distal features—such as idiopathic toe walking and congenital clubfoot following Ponseti treatment—and is characterized by reduced dorsiflexion, restricted multi-segment foot motion, and altered push-off power. By contrast, ADFL is commonly associated with chronic ankle instability, prolonged immobilization, or stiffness of the Achilles–triceps surae complex, and frequently presents with modified knee–hip mechanics. Because existing gait studies often treat DFL as a homogeneous entity, without etiological stratification, stance-phase deviations arising from distal preservation (more typical of CDFL) versus proximal substitution (more typical of ADFL) are easily conflated, risking misinterpretation of gait mechanics and misallocation of therapeutic focus, subtype-specific compensation spectra and intervention targets can be obscured; differentiating CDFL from ADFL is therefore essential [4], [7], [14], [32], [34]. Differentiating CDFL from ADFL at the outset is biomechanically and clinically relevant because stance-phase support, energy absorption, and push-off may be reorganized in subtype-specific ways that directly affect gait interpretation and targeted rehabilitation.

Recent evidence indicates that dorsiflexion limitation is common across populations: approximately one-third of community-dwelling older adults meet established thresholds for limitation, the proportion is higher among inpatients or foot–ankle clinic cohorts (reaching ≥70%), and even among young competitive athletes about 10% exhibit reduced dorsiflexion mobility [1], [22], [23], [29]. Although ankle dorsiflexion limitation has been frequently described as a phenotype, whether different etiologies correspond to distinct inter-joint compensation strategies and muscle-recruitment profiles remains insufficiently established; systematic, head-to-head comparative evidence is lacking. This knowledge gap constrains the development of phenotype-informed assessment and targeted intervention strategies. Mechanistically, early–mid stance depends on tibial progression and energy absorption, whereas terminal stance relies on elastic energy return and push-off. Ankle push-off provides a decisive burst of positive power, contributing both to redirection of whole-body dynamics and to swing initiation. The plantar flexors and intrinsic foot muscles regulate longitudinal-arch stiffness and participate in elastic energy storage and recoil, thereby shaping distal-to-proximal energy coupling. When distal function is constrained, joint power and moments are reallocated proximally [17], [19]. Given that early–mid stance primarily entails controlled motion, energy absorption, and tibial progression, whereas terminal stance relies on elastic recoil and push-off, different etiologies are likely to exhibit distinct patterns of joint-moment and joint-power redistribution across the stance phase. Accordingly, contrasting CDFL and ADFL within a stance-phase framework helps identify etiology-specific compensatory pathways and intervention targets. Recent evidence shows that reduced passive dorsiflexion range of motion is associated with greater limb stiffness and increased knee–ankle joint moments during walking, consistent with a model in which distal restrictions drive proximal reorganization [2]. As a prototypical acquired form of dorsiflexion limitation, chronic ankle instability exerts mechanical effects that extend beyond the ankle to the knee and hip—especially during functional tasks—underscoring that ADFL cannot be interpreted solely at the ankle level. Therefore, establishing the etiological subtype of DFL and interpreting gait accordingly are prerequisites for sound biomechanical inference and clinical decision-making [2], [14], [34]. On the congenital side, individuals with idiopathic toe walking or post-treatment clubfoot often exhibit persistent deviations in the ankle rocker and push-off power, indicating more durable distal constraints and a tendency toward distal-driven strategies; by contrast, acquired forms (e.g., chronic ankle instability, immobilization, or tendon–fascicle remodeling) more commonly shift compensation proximally to the knee and hip. This etiologic bifurcation implies different time-resolved joint-power and EMG signatures during level walking which, if not stratified and instead analyzed in pooled fashion, are easily diluted or overlooked [4], [7], [32], [34].

Methodologically, conventional discrete metrics tend to overlook when along the stance phase differences occur. Time-series Statistical Parametric Mapping (SPM1D) enables field-based inference across the entire stance-phase timeline, directly localizing the temporal windows of between-group divergence and reducing dependence on peak selection. In parallel, a barefoot paradigm minimizes footwear-induced modulation of foot deformation and electromyography, thereby revealing intrinsic control more directly; recent studies also confirm condition-dependent shifts in muscle activity between barefoot and shod walking. On this basis, the present study adopts a barefoot, time-resolved framework and integrates analyses of joint power and EMG [10], [16], [35].

Building on the above background, a comparative analysis of barefoot gait biomechanics in dorsiflexion-limited populations with different etiologies has clear theoretical and clinical significance. This study aims to compare kinematic, kinetic, and surface electromyographic (sEMG) differences during the stance phase of barefoot walking between individuals with ankle dorsiflexion limitation of different etiologies and healthy controls, to localize the temporal phases in which these differences occur, and to inform clinical rehabilitation and athletic training. We hypothesize that the CDFL cohort will exhibit greater hip flexion angle and hip extension moment during stance, along with higher early-stance ankle negative power and greater mean activation of the GM; by contrast, the ADFL cohort will show greater plantar flexion moment in mid-to-late stance, greater knee flexion angle and negative power throughout stance, and higher RF mean activation. Accordingly, the two groups are expected to display distinct time-domain characteristics.

Method

Participants

The required sample size was determined a priori using G*Power 3.1 (Franz Faul, Germany), yielding a total of 36 participants (effect size = 0.5, $\alpha = 0.05$, statistical power = 80%). Details can be found in Table 1,all participants were right-leg dominant. Inclusion criteria were as follows. For the congenital dorsiflexion-limitation (CDFL) group: medical history and clinical evaluation indicating ankle dorsiflexion limitation since childhood or adolescence; no recent acute ankle injury; and physical examination confirming restricted dorsiflexion range of motion, quantified by the weight-bearing lunge test (WBLT) or goniometry. Each measurement was repeated three times and averaged, with a WBLT tibial-inclination angle ≤ 40° adopted as the operational threshold for ankle dorsiflexion limitation [9], [20], [26], [27]. For the acquired dorsiflexion-limitation (ADFL) group, eligibility required an ankle/calf-related medical history (e.g., post-ankle sprain, post-immobilization, Achilles–posterior calf tightness) and a persistent dorsiflexion limitation lasting ≥6 months; clinical measurements followed the same procedures as above and had to meet the limitation threshold. For the healthy control group (CG), inclusion required no lower-limb injury or pain within the past 12 months and no clinical signs of dorsiflexion limitation. This study adhered to the Declaration of Helsinki, and informed consent was obtained from all participants. Ethical approval was granted by the Ethics Committee of the Research Academy of Grand Health at Ningbo University (TY2025072).

Table 1. Participant basic information

Group	Height(cm)	Weight(kg)	Age(year)	Walk speed(m/s)	WBLT(°)
CDFL	175.55(8.52)	75.68(8.32)	22.56(4.45)	1.20(0.08)	38.12(1.83)
ADFL	180.86(9.80)	77.88(7.13)	25.85(5.66)	1.17(0.12)	35.15(3.82)
CG	179.52(8.65)	70.52(8.43)	23.25(4.68)	1.15(0.09)	49.8(5.1)

Note. WBLT: the tibial inclination angle measured using the weight-bearing lunge test. There is a significant difference in WBLT, The ADFL flexion angle was significantly smaller than that of the CDFL and CG groups($p=0.012$ and $p < 0.001$).

Experimental protocol

Prior to testing, participants wore standardized form-fitting attire, and thirty-eight retroreflective markers were affixed to anatomical bony landmarks in accordance with the OpenSim 2392 musculoskeletal model (Fig. 1a) [31]. A ten-camera infrared motion-capture system operating at 200 Hz (Vicon Vero, Oxford Metrics Ltd., Oxford, UK) and two three-dimensional force plates sampling at 1000 Hz (Kistler Instrumente AG, Winterthur, Switzerland) were used to acquire kinematic trajectories and kinetic data, respectively. Surface electromyography (sEMG) signals were synchronously recorded with a Delsys system (Delsys Inc., Boston, MA, USA) at 2000 Hz. Before the test, skin was prepared by shaving, light abrasion, and cleansing with isopropyl alcohol. sEMG sensors were placed over the muscle bellies of the dominant limb’s tibialis anterior (TA), peroneus longus (PL), gastrocnemius medialis (GM), and rectus femoris (RF) (Fig. 1c). After preparation, participants warmed up by barefoot straight-line walking along a 10-m walkway at a self-selected speed. Walking speed, measured with photoelectric timing gates, showed no significant between-group differences on statistical testing; group speeds were kept essentially equivalent, with fluctuations not exceeding ±5%.Force plates were embedded at the walkway center, and participants were instructed to walk naturally so that the dominant foot contacted the first plate to yield a valid step; three valid dominant-limb strikes were collected per participant (Fig. 1b).

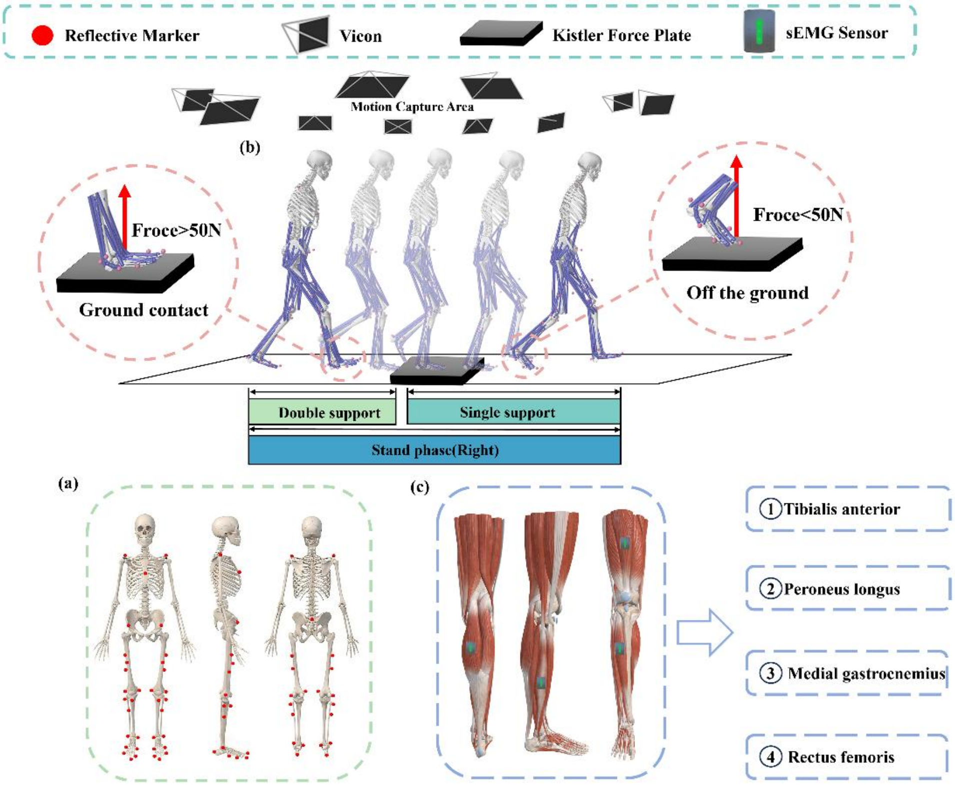

Figure 1. (a) Placement of retroreflective markers. (b) Overview of the experimental procedure. (c) sEMG electrode placement and identification of the recorded muscles.

Data processing

Data were processed in Vicon Nexus (version 2.12.1). To attenuate high-frequency noise, a zero-lag, fourth-order Butterworth low-pass filter with a 20 Hz cutoff was applied. To compare differences in gait phase distribution between the two dorsiflexion-limited groups—stance phase (STP), swing phase (SWP), and double-support phase (DSP)—we calculated each phase as a percentage of the full gait cycle. The gait cycle was defined as the interval from the right foot's initial contact to its subsequent initial contact. STP was defined as the time from right foot contact on the force plate to right toe-off. Initial contact was identified when the right-foot vertical ground reaction force (vGRF) exceeded 50 N; toe-off was identified when vGRF fell below 50 N. When the vertical GRFs on the first and second force plates each exceeded 50 N, the first and second step contacts were identified accordingly. Because no additional force plate was available, the initial contact of the third step was determined from the vertical velocity characteristics of the heel and toe markers [12], [25]. Three-dimensional kinematic and kinetic analyses were performed in Visual3D(C-Motion, Inc., Germantown, MD, USA). The analyses included sagittal-plane joint angles, moments, and powers of the hip, knee, and ankle during the stance phase. This study focused exclusively on the sagittal plane because the essence of ankle dorsiflexion limitation—and its primary functional consequences—manifests sagittally, and the associated metrics most directly address our research question. In addition, sagittal-plane variables in standard single-segment foot inverse dynamics exhibit higher measurement reliability, are less susceptible to skin-motion artifacts and modeling error, and, within the SPM1D framework, reduce the multiple-comparison burden and enhance statistical power.Joint moments and powers were normalized to body weight. Surface EMG signals were first band-pass filtered at 20–450 Hz to remove motion artefacts and external noise and ensure signal quality, then full-wave rectified to convert all values to positive for subsequent processing. The rectified signals were further smoothed with a fourth-order Butterworth low-pass filter (cutoff 20 Hz) to generate envelopes reflecting overall muscle activation. During data analysis, the integrated EMG (iEMG) for each gait cycle was obtained by numerically integrating the rectified EMG envelope over the cycle; this yields the area under the curve and serves as a composite index of neuromuscular activation [3], [15]. To reduce inter-individual variability and amplitude fluctuations under walking conditions, all EMG signals underwent peak normalization: each data point was divided by the subject-specific maximum EMG value of the corresponding muscle observed across all gait cycles under the same condition (i.e., expressed as a proportion of the maximal value for that muscle and condition) [13], [30].

Statistical analysis

All data were tested for normality using the Shapiro–Wilk test and for homogeneity of variance using Levene’s test. If the assumptions were satisfied, between-group differences were assessed with one-way ANOVA; when a significant main effect was observed, post hoc pairwise comparisons were conducted. Kinematic data were time-normalized to 100% of the stance phase; kinetic data were normalized to body weight and likewise time-normalized to 100% of the stance phase. Both were interpolated to 101 points to facilitate subsequent processing. All statistical analyses of discrete parameters were conducted using one-way ANOVA in SPSS Statistics 22 (IBM SPSS Inc., Chicago, IL, USA). For time-series parameters—i.e., the stance-phase waveforms of hip, knee, and ankle joint angles, moments, and powers—between-group comparisons were performed with one-dimensional Statistical Parametric Mapping (SPM1D). When a significant main effect was detected, planned pairwise SPM comparisons were carried out to further examine sagittal-plane kinematic and kinetic differences. Data processing was implemented in MATLAB R2024a (MathWorks, Natick, MA, USA), with statistical significance set at $p < 0.05$.

Results

Spatiotemporal Parameters

No significant between-group differences were observed in gait cycle duration. However, the CDFL group exhibited a significantly shorter step length than both the ADFL and control groups ($p = 0.005$ and $p = 0.017$). The percentage of the stance phase (STP) was significantly higher in the CDFL group than in the ADFL and control groups ($p = 0.001$ and $p = 0.007$), whereas the percentage of the swing phase (SWP) was significantly higher in the ADFL group than in the CDFL and control groups (both $p < 0.001$)(Table 2).

Table 2. Comparison of spatiotemporal parameters among the CDFL, ADFL, and control groups.

Variables	CDFL	ADFL	CG	*p*	*η²*
Step length(cm)	138.99(4.72)	147.24(10.30)	146.16(7.35)	**0.004**	0.189
Contact time(s),StP	0.60(0.02)	0.58(0.04)	0.58(0.05)	0.108	0.079
Contact time(s),DSP	0.08(0.01)	0.08(0.01)	0.08(0.01)	0.877	0.032
Times(s),SwP	0.43(0.02)	0.45(0.05)	0.44(0.03)	0.539	0.014
StP%	57.94(0.84)	56.28(1.33)	56.79(1.15)	**0.001**	0.287
DSP%	7.58(0.88)	7.60(0.72)	7.43(1.01)	0.816	0.008
SwP%	41.76(0.82)	43.38(1.35)	43.21(1.15)	**0.001**	0.304

Note. STP = stance phase; SWP = swing phase; DSP = double-support phase.

Kinematic Results

The SPM analysis revealed phase-specific differences. At 22–42% of stance, the CDFL group exhibited a significantly greater ankle dorsiflexion angle than the ADFL group (p = 0.003), whereas at 82–99% of stance the ADFL group showed a significantly greater dorsiflexion angle (p = 0.004). Nevertheless, both groups had dorsiflexion angles significantly smaller than those of healthy controls. At 3–8% and 16–96% of stance, the ADFL group demonstrated a significantly greater knee flexion angle than the CDFL group (p = 0.014 and p < 0.001) and the control group (both p < 0.001). The CDFL group's hip flexion angle was generally larger than that of the ADFL group during the first 50% of stance, whereas the ADFL group exhibited significantly greater hip extension angle in the latter 50% of stance (p = 0.002). Relative to the ADFL group, healthy controls showed a significantly greater hip flexion angle at 0–22% of stance (p = 0.012), while the ADFL group displayed significantly greater hip extension angle at 31–66% of stance (p = 0.008) (Fig. 2).

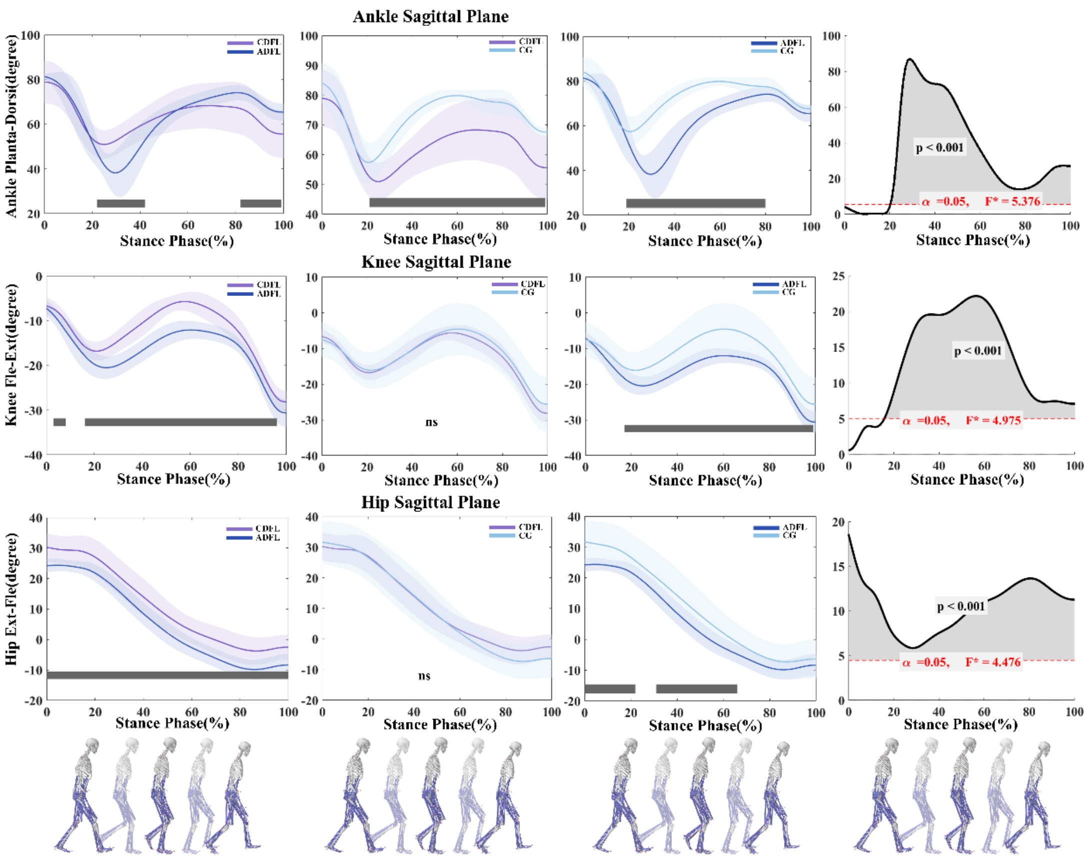

Figure 2. Comparative analysis of kinematics of lower limb hip-knee-ankle movements during the support phase.The CDFL group is shown in purple, the ADFL group in dark blue, and the healthy control group (CG) in light blue. In each panel, gray boxes indicate intervals of significant difference based on SPM results; “ns” denotes non-significant.

Kinetic results

As shown in Fig. 3, SPM analysis revealed that the ADFL group exhibited a significantly greater ankle plantar flexion moment than the CDFL group at 0–16% of stance ($p = 0.001$) and 23–81% of stance ($p < 0.001$), whereas the CDFL group showed a significantly increased plantar flexion moment at 85–99% of stance ($p = 0.001$). The ADFL group's ankle plantar flexion moment was also significantly greater than that of healthy controls at 0–83% of stance ($p = 0.004$). For the knee, the ADFL group had a significantly greater extension moment than the CDFL group at 25–42% of stance, although both patient groups were lower than controls in this interval. Conversely, the CDFL group displayed a significantly greater knee flexion moment than the ADFL group at 42–83% ($p < 0.001$) and 94–99% ($p = 0.012$) of stance. For the hip, the CDFL group showed a significantly greater extension moment than the ADFL group at 0–5% ($p = 0.008$) and 8–51% ($p = 0.001$) of stance, while the ADFL group's hip extension moment was significantly smaller than that of controls at 0–4% ($p = 0.014$) and 5–33% ($p = 0.001$).

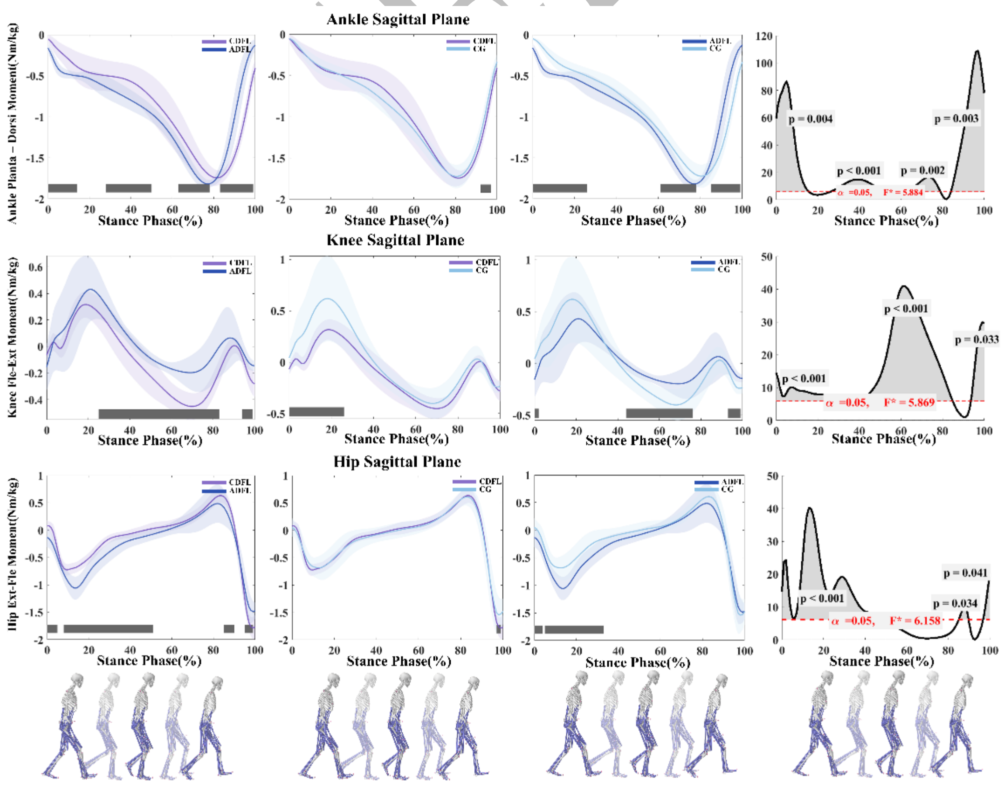

Figure 3. Comparative analysis of lower limb hip-knee-ankle dynamics during the support period using SPM.The CDFL group is shown in purple, the ADFL group in

dark blue, and the healthy control group (CG) in light blue. In each panel, gray boxes indicate intervals of significant difference based on SPM results; “ns” denotes non-significant.

Significant between-group differences in joint power across stance are shown in Fig. 4. At the ankle, negative power was greater in the CDFL group at 0–21% of stance, whereas the ADFL group exhibited greater positive power over the same interval, yielding a significant difference ($p < 0.001$). From 24–42% of stance, negative power was significantly increased in the ADFL group ($p = 0.002$). The CDFL and control groups differed significantly at 25–34% of stance, with controls showing greater negative power ($p = 0.002$). Controls also demonstrated predominantly negative power at 0–23% of stance, differing significantly from the ADFL group ($p = 0.001$). From 30–62% of stance, negative power was significantly increased in the ADFL group ($p = 0.001$). At 75–78% and 95–99% of stance, controls exhibited both greater negative power and greater terminal positive power than the ADFL group ($p = 0.009$ and $p = 0.007$).For knee power, the ADFL group showed significantly increased negative power at 61–85% of stance, while the CDFL group displayed significantly increased positive power at 92–99% of stance (both $p < 0.001$). Relative to the CDFL group, controls had greater negative power at 0–14% ($p = 0.002$) and 51–63% ($p = 0.003$) of stance, and greater positive power at 24–34% ($p = 0.001$). Compared with controls, the ADFL group exhibited predominantly increased negative power at 47–58% of stance ($p = 0.001$), but greater positive power at 68–80% ($p = 0.002$) and 92–99% ($p = 0.003$). For the hip, the ADFL group showed significantly greater positive power than the CDFL group at 0–3% ($p = 0.012$) and 15–55% ($p = 0.001$), and significantly greater positive power than controls at 22–37% ($p = 0.001$). The CDFL group’s power profile was broadly similar to that of controls.

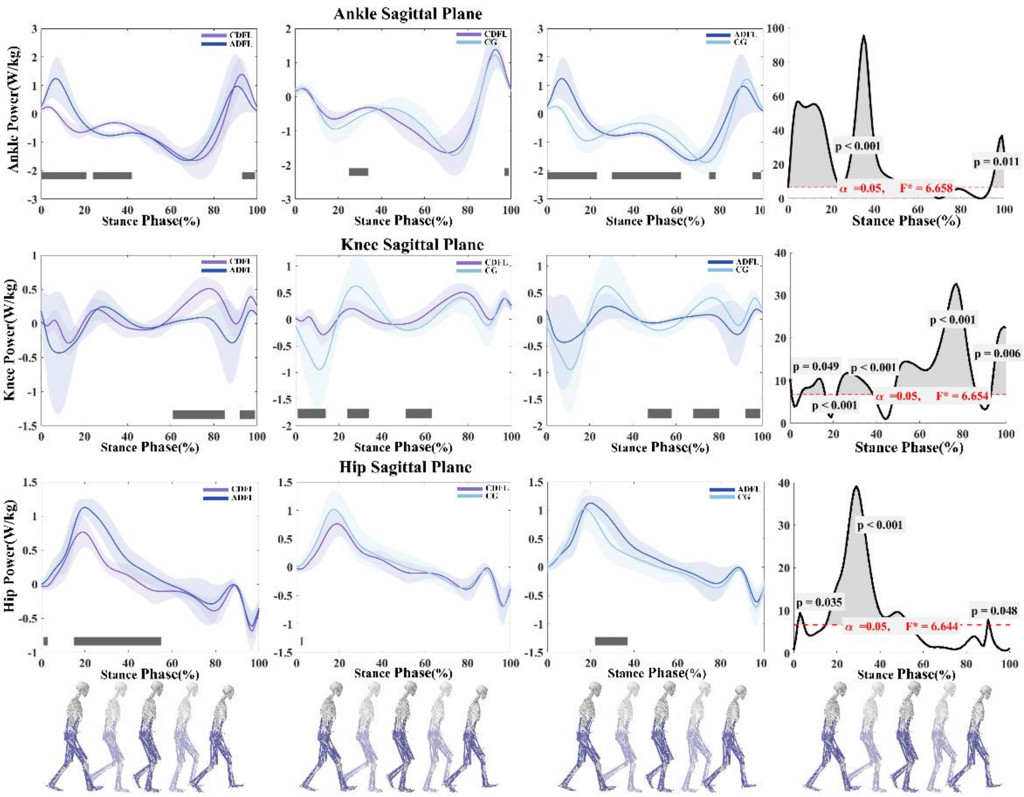

Figure 4. Comparative analysis of lower limb hip-knee-ankle dynamics during the support period using SPM.The CDFL group is shown in purple, the ADFL group in dark blue, and the healthy control group (CG) in light blue. In each panel, gray boxes indicate intervals of significant difference based on SPM results; “ns” denotes non-significant.

Muscle Activation results

As shown in Fig. 5, during the stance phase the iEMG of the TA differed significantly between the ADFL group (14.5%) and the control group (CG; 17.2%) ($p = 0.028$). For the PL, the difference between the CDFL group (22.1%) and the ADFL group (29.6%) approached significance ($p = 0.070$), and a significant difference was also observed relative to the healthy controls (31.6%) ($p = 0.014$). In addition, GM activation differed significantly among the CDFL (38.4%), ADFL (20.1%; $p < 0.001$), and control (32.4%; $p < 0.003$) groups. Similarly, during barefoot stance, RF activation differed significantly among the CDFL (19.4%), ADFL (32.2%; $p < 0.001$), and control (16.7%; $p = 0.006$) groups.

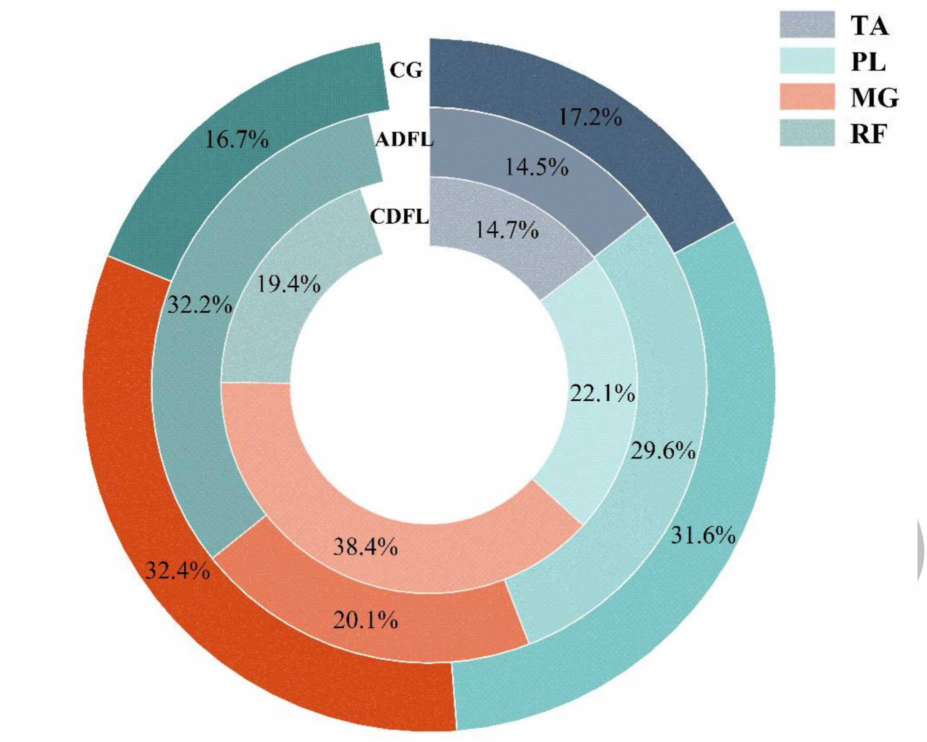

Figure 5. Proportion of each muscle iEMG contributing to total activation during the stance phase across the three groups.

As shown in Table 3 and Fig. 6, during the entire stance phase, the three groups differed significantly in the mean activation of the TA, PL, GM, and RF. Tukey post hoc comparisons indicated that the ADFL group exhibited a significantly different mean TA activation compared with healthy controls (p = 0.015). Notably, both dorsiflexion-limited groups showed lower mean TA activation than controls. For PL, the mean activation level in both the CDFL and ADFL groups was significantly lower than in controls (p = 0.001 and p = 0.031). For GM, the ADFL group's mean activation was significantly lower than both the control and CDFL groups (both p = 0.001), whereas the CDFL group showed significantly higher GM activation than controls (p = 0.002). For RF, the ADFL group demonstrated higher mean activation than controls (p = 0.001), and the ADFL group also exceeded the CDFL group (p = 0.005).

Table 3. Main effects and post hoc pairwise comparisons of mean muscle activation across the three groups during the stance phase.

Stance Phase											
	One-way ANOVA					**Tukey HSD (p-adjusted)**					
Muscle	**CDFL Mean(SD)**	**ADFL Mean(SD)**	**CG Mean(SD)**	***p***	***η²***	**CDFLvsADFL** ***p***	**Hedges' g**	**CDFLvsCG** ***p***	**Hedges' g**	**ADFL vsCG** ***p***	**Hedges' g**

TA	0.07(0.02)	0.06(0.03)	0.08(0.02)	**0.020**	0.095	0.335	0.356	0.324	-0.495	**0.015**	-0.691
PL	0.11(0.04)	0.12(0.03)	0.15(0.04)	**0.001**	0.161	0.736	-0.209	**0.001**	-0.899	**0.031**	-0.829
GM	0.18(0.03)	0.07(0.04)	0.14(0.05)	**0.001**	0.639	**0.001**	3.752	**0.002**	1.134	**0.001**	-1.750
RF	0.09(0.02)	0.12(0.02)	0.08(0.06)	**0.001**	0.219	**0.005**	-1.781	0.385	0.301	**0.001**	1.044

Note. Statistical significance set at $p < 0.05$; bold indicates significant differences.

Abbreviation: SD, standard deviation.

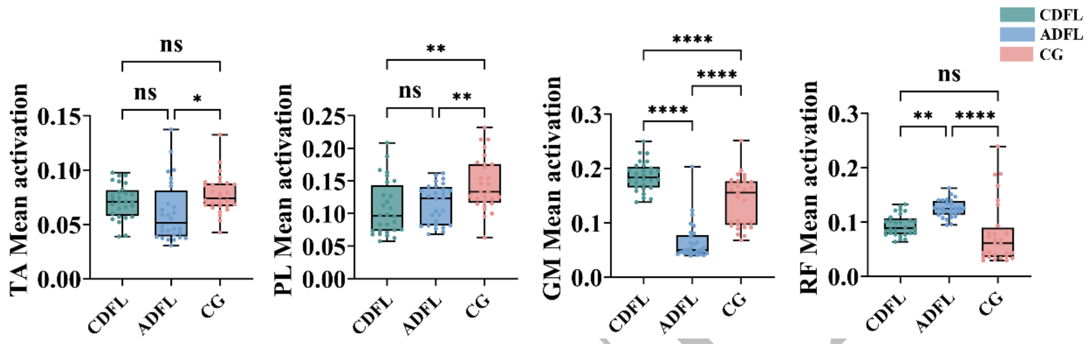

Figure 6. Between-group comparisons of mean muscle activation during the stance phase. “ns” denotes non-significant; * $p < 0.05$, ** $p < 0.01$, **** $p < 0.0001$.

Discussion

We conducted a comparative evaluation of stance-phase lower-limb kinematics, kinetics, and surface electromyography during barefoot gait among participants with congenital (CDFL) or acquired (ADFL) ankle dorsiflexion limitation, with healthy controls as the reference group. We prespecified that CDFL and ADFL would exhibit distinct time-resolved compensation profiles. Overall, the findings support this differentiation: in CDFL, early stance was characterized by a more flexed hip posture, higher hip extension moments in early–mid stance, more pronounced early-stance ankle energy absorption, and greater activation of the medial gastrocnemius (GM), collectively indicating a more distal-driven strategy that preserves terminal push-off [8], [18], [24], [36]. These observations are consistent with and support our a priori hypotheses for CDFL. For ADFL, we posited larger ankle plantar flexion moments in mid-to-late stance, higher knee flexion angles and greater knee negative power across stance, and higher mean activation of the rectus femoris (RF). The results were largely supported, with the negative-power timing refined to a mid-to-late stance concentration rather than persisting throughout stance (partially supported for this component): ankle plantar flexion moments were broadly elevated in early-to-mid stance (with partial carry-over into late stance), knee flexion angles remained higher, and mean RF activation was increased; however, negative power at the knee and ankle was concentrated in mid-to-late stance rather than persisting throughout the entire stance phase. Taken together, ADFL exhibited a more proximalized compensation pattern, maintaining forward progression via greater energy absorption at the knee and energy generation at the hip [2], [8], [21]. These mechanical profiles were accompanied by distinct spatiotemporal differences—a greater stance-phase proportion and shorter step length in CDFL, and a greater swing-phase proportion in ADFL—further indicating that etiology shapes the temporal organization of support and propulsion [6], [11]. In sum, the CDFL hypotheses were supported; the ADFL hypotheses were largely supported with the above timing modification.

From a joint-mechanics perspective, CDFL and ADFL exhibit two distinguishable mechanical profiles. The stance-phase behavior of CDFL can be summarized as an "early control–late release" pattern: during early–mid stance, more pronounced negative ankle power (energy absorption) together with greater hip extension moments facilitates controlled tibial progression while preserving distal function; in terminal stance, plantar flexion moments and positive ankle power are concentrated to produce a propulsion peak, a pattern consistent with the group's higher stance-phase proportion and shorter step length [8], [18], [24], [36]. By contrast, ADFL exhibits a pattern of proximal substitution with a broadly elevated plantar flexion demand: ankle plantar flexion moments are generally higher in early–mid stance, while a more sustained knee-flexed posture together with greater mid-to-late stance negative power indicates heavier reliance on proximal energy absorption to maintain roll-over. Subsequently, late-stance hip extension increases, yielding a larger window of hip positive power that compensates—via a hip-dominant strategy—for reduced distal mechanical contribution (we did not measure metabolic cost; this remains a mechanistic inference); this is consistent with a shorter stance window (i.e., a greater swing-phase proportion) [8], [21], [37]. Overall, CDFL tends to preserve the ankle-rocker's storage–return function and is supported by hip moments, whereas ADFL reallocates mechanical work to the knee and hip, most prominently during mid-to-late stance [2], [24], [36].

The EMG findings corroborate the mechanical patterns described above. In the CDFL cohort, the medial gastrocnemius (GM) showed higher mean activation and a greater relative contribution, coinciding with increased terminal-stance plantar flexion moments and positive ankle power—consistent with a triceps surae–centered reinforcement of late push-off. Activation of the peroneus longus (PL) was lower than in controls, which may indicate a relative down-scaling of lateral-stability contributions; however, this interpretation should be made cautiously because frontal-plane mechanics were not quantified in the present study [5], [33]. Mean tibialis anterior (TA) activation was also lower than in controls, aligning with the notion that when early-stance roll-over is governed by distal energy absorption in concert with hip-extension support, the relative dominance of TA is reduced [6]. In ADFL, mean activation of the rectus femoris (RF) is elevated while that of the medial gastrocnemius (GM) is reduced, indicating a tendency to substitute ankle push-off with the hip-flexor/knee-extensor musculature; this pattern aligns with the group's higher late-stance hip positive power and greater knee negative power. Mean tibialis anterior (TA) activation is likewise lower than in controls, suggesting adjustments in the timing and pattern of early foot control [14], [28], [34]. Integrating sagittal-plane mechanics and EMG evidence, CDFL appears to preserve distal storage–return behavior and a consolidated late push-off while stabilizing tibial progression via greater hip-extension moments in early–mid stance; by contrast, ADFL maintains roll-over through earlier and broader plantar flexion-moment demands combined with knee-based energy absorption, and then compensates for distal inefficiency with hip power generation in late stance—reflecting an etiology-specific dichotomy of distal preservation versus proximal substitution [8], [21], [24], [36].

In clinical practice, interventions for CDFL should prioritize re-establishing the ankle rocker and terminal push-off, while concurrently addressing joint range of motion and tendon–muscle conditioning to support elastic energy storage and recoil. Programs should strengthen the triceps surae with emphasis on early-stance eccentric control and late-stance power output, and incorporate intrinsic-foot–muscle training to improve early- to mid-stance energy absorption and modulation of longitudinal-arch stiffness. When patients ambulate on unstable terrain, attention may also be given to the lateral-stability function of the peroneus longus (PL), although this study did not capture frontal-plane measures and such inferences should be made cautiously [5], [16], [18], [19]. For ADFL, the rehabilitation goal is to restore distal contribution while protecting proximal joints: employ joint mobilization and soft-tissue techniques to recover dorsiflexion ROM, and implement plantar flexor re-education—particularly upregulating recruitment of the medial gastrocnemius (GM)—to reduce excessive reliance on knee-based energy absorption. Utilize technique cues and adjustments to surface and gait parameters (e.g., cadence, step length) to manage mid- to late-stance braking demands at the knee. Rebuild hip–ankle synergy by retiming hip extension and ankle push-off, while managing rectus femoris (RF) loading to prevent overdominance. When spatiotemporal metrics indicate a shortened stance duration, targeted drills can be used to modestly extend effective stance time, promoting smoother roll-over without increasing knee load [8], [21], [34], [37].

This study has several limitations. First, analyses were restricted to sagittal-plane kinematics and kinetics during stance (angles, moments, and powers), together with mean activation of four muscles; frontal- and transverse-plane mechanics and more detailed multi-segment foot modeling were not included. Consequently, the role of the peroneus longus (PL) in frontal-plane stabilization can only be inferred indirectly; future work should incorporate multi-segment foot models and multiplanar analyses [5]. Second, we did not collect metabolic measures, pain outcomes, or patient-reported outcomes (PROs). Studies using the same experimental paradigm should acquire metabolic, pain, and PRO data in parallel to more directly test the clinical significance and translational value of etiology-specific compensatory strategies [11], [16]. Additionally, EMG timing features (e.g., onset, duration, time to peak) were not analyzed; future SPM-based temporal EMG metrics would strengthen neuromuscular inferences. Third, within-group etiological heterogeneity—e.g., idiopathic toe walking and post-corrective clubfoot within CDFL, and chronic ankle instability or a history of immobilization within ADFL—may have diluted subtype-specific features; larger samples and finer etiologic stratification are warranted [7], [14], [32], [34]. Fourth, although the barefoot paradigm enhances internal validity for ankle–foot intrinsic control, extrapolation to shod conditions should be made with caution [11], [16].

Conclusion

This study compared stance-phase biomechanics between congenital (CDFL) and acquired (ADFL) ankle dorsiflexion limitation and demonstrated a clear distal–proximal differentiation in compensatory strategies. CDFL favored a distal-driven pattern that preserves the ankle-rocker's storage–return function and reinforces terminal push-off (accompanied by higher medial gastrocnemius activation). In contrast, ADFL reflected proximal substitution—maintaining forward progression through greater mid-to-late-stance knee energy absorption and hip power generation (with increased rectus femoris activation)—and exhibited a distinct spatiotemporal organization. Clinically, interventions should be etiology-stratified and phase-specific: for CDFL, prioritize restoring dorsiflexion ROM and the triceps surae's eccentric/recoil capacity to strengthen terminal push-off; for ADFL, reduce knee braking loads, re-establish hip–ankle synergy, and restore distal contribution. Notably, mechanistic interpretations concerning walking economy and frontal-plane stability remain hypothesis-generating and require confirmation via multiplanar mechanics, metabolic assessments, and patient-reported outcomes.

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