

RESEARCH ARTICLE

Open Access



Gait characteristics associated with the foot and ankle in inflammatory arthritis: a systematic review and meta-analysis

Matthew Carroll^{1*}, Priya Parmar², Nicola Dalbeth³, Mark Boocock⁴ and Keith Rome¹

Abstract

Background: Gait analysis is increasingly being used to characterise dysfunction of the lower limb and foot in people with inflammatory arthritis (IA). The aim of the systematic review was to evaluate the spatiotemporal, foot and ankle kinematic, kinetic, peak plantar pressure and muscle activity parameters between patients with inflammatory arthritis and healthy controls.

Methods: An electronic literature search was performed on Medline, CINAHL, SportsDiscus and The Cochrane Library. Methodological quality was assessed using a modified Quality Index. Effect sizes with 95 % confidence intervals (CI) were calculated as the standardised mean difference (SMD). Meta-analysis was conducted if studies were homogenous.

Results: Thirty six studies with quality ranging from high to low met the inclusion criteria. The majority of studies reported gait parameters in Rheumatoid arthritis (RA). The gait pattern in RA was characterised by decreased walking speed (SMD 95 % CI -1.57 , -2.25 to -0.89), decreased cadence (SMD -0.97 , -1.49 to -0.45), decreased stride length (SMD -1.66 , -1.84 to -1.49), decreased ankle power (SMD -1.36 , -1.70 to -1.02), increased double limb support time (SMD 1.03 , 0.84 to 1.22), and peak plantar pressures at the forefoot (SMD 1.11 , 0.76 to 1.45). Walking velocity was reduced in psoriatic arthritis and gout with no differences in ankylosing spondylitis. No studies have been conducted in polymyalgia rheumatica, systemic sclerosis or systemic lupus erythematosus.

Conclusions: The review identified the majority of studies reporting gait adaptations in RA, but limited evidence relating to other IA conditions. Poor data reporting, small sample sizes and heterogeneity across IA conditions limit the interpretation of the findings. Future studies may consider a standardised analytical approach to gait analysis that will provide clinicians and researchers with objective evidence of foot function in people with IA.

Keywords: Gait, Rheumatoid arthritis, Ankylosing spondylitis, Psoriatic arthritis, Gout

Background

The term 'inflammatory arthritis' (IA) has been used to describe a number of inflammatory joint diseases including: rheumatoid arthritis (RA), ankylosing spondylitis (AS), psoriatic arthritis (PsA) and gout [1]. RA is a chronic progressive autoimmune disease characterized by joint swelling, joint tenderness and destruction of synovial joints [2]. SpA encompasses a heterogeneous group of inflammatory arthritic conditions, characterised by vertebral involvement, peripheral oligoarthritis or

polyarthritis, enthesitis, AS, PsA and undifferentiated spondyloenthesoarthritis [3, 4]. Gout is a common form of inflammatory arthritis caused by the deposition of monosodium urate crystals within joints and other soft tissue associated with hyperuricaemia [5]. IA causes lower limb and foot pain and impairment, functional disability, reduced mobility, joint deformity and altered gait strategy [6–10]. Foot pain is considered an important factor in the development of antalgic gait in IA, specifically in RA and gout [6, 11, 12]. In RA, foot pain is derived from structural and functional alterations associated with inflammatory and structural change [6, 13]. With the development of an antalgic gait, adaptations occur based upon a pain avoidance strategy. Previous

* Correspondence: matthew.carroll@aut.ac.nz

¹Department of Podiatry, Health & Rehabilitation Research Institute, Auckland University of Technology, Auckland, New Zealand

Full list of author information is available at the end of the article

studies have reported gait adaptations in RA and these include: a decrease in walking velocity and subsequent alterations to velocity related spatiotemporal parameters including, reduced cadence, increased double limb support time and decreased step length [14–18]. Changes to kinematic parameters including, reduced sagittal plane ankle ROM and increased peak rearfoot eversion have also been reported [7, 14, 17, 18]. Furthermore, previous studies have reported alteration to kinetic parameters including, reduced peak ankle plantarflexor power associated with reduced walking velocity, reduced ankle joint ROM, reduced ankle joint angular velocity, reduced ankle plantarflexor moments and decreased strength of the ankle plantarflexor muscles [16, 17, 19]. An increase in peak forefoot plantar pressure parameters has also been reported in RA [16].

Gait analysis provides information about spatial-temporal parameters, kinetics, kinematics and muscle activity to further delineate the relationship between joint disease, joint impairments and compensatory gait strategies adopted to overcome painful and disabling deformities [15, 20]. Gait analysis has been reported as a useful clinical tool to quantify foot function in both early and established RA [7, 8, 14, 15]. However, less common IA conditions, such as AS, PsA, gout, polymyalgia rheumatica, systemic sclerosis and systemic lupus erythematosus, also have various consequences

for the lower limb such as changes in foot function, and extra articular complications involving the skin and vascular integrity [9, 10, 12, 21–24]. A recent systematic review of studies investigating walking abnormalities associated with RA, Baan [25] identified changes in gait such as a slower walking, longer double support time, and avoidance of extreme positions. These changes were in relation to the frequently found static features in RA, for instance, hallux valgus, pes planovalgus and rearfoot abnormalities. However, Baan [25] only reported gait parameters in RA and did not consider other IA conditions. However, recently there has been an interest in evaluating gait patterns in other IA conditions that includes gout [12], PsA [21] and AS [10]. No previous systematic review has conducted meta-analysis of gait parameters in IA compared to healthy control population. The aim of the systematic review was to evaluate spatiotemporal, foot and ankle kinematic, kinetic, peak plantar pressure and muscle activity parameters in people with IA and healthy controls.

Methods

Identification of studies

Four electronic databases were searched (Medline, CINAHL, SportsDiscus and The Cochrane Library). The search was completed in March 2015. The search

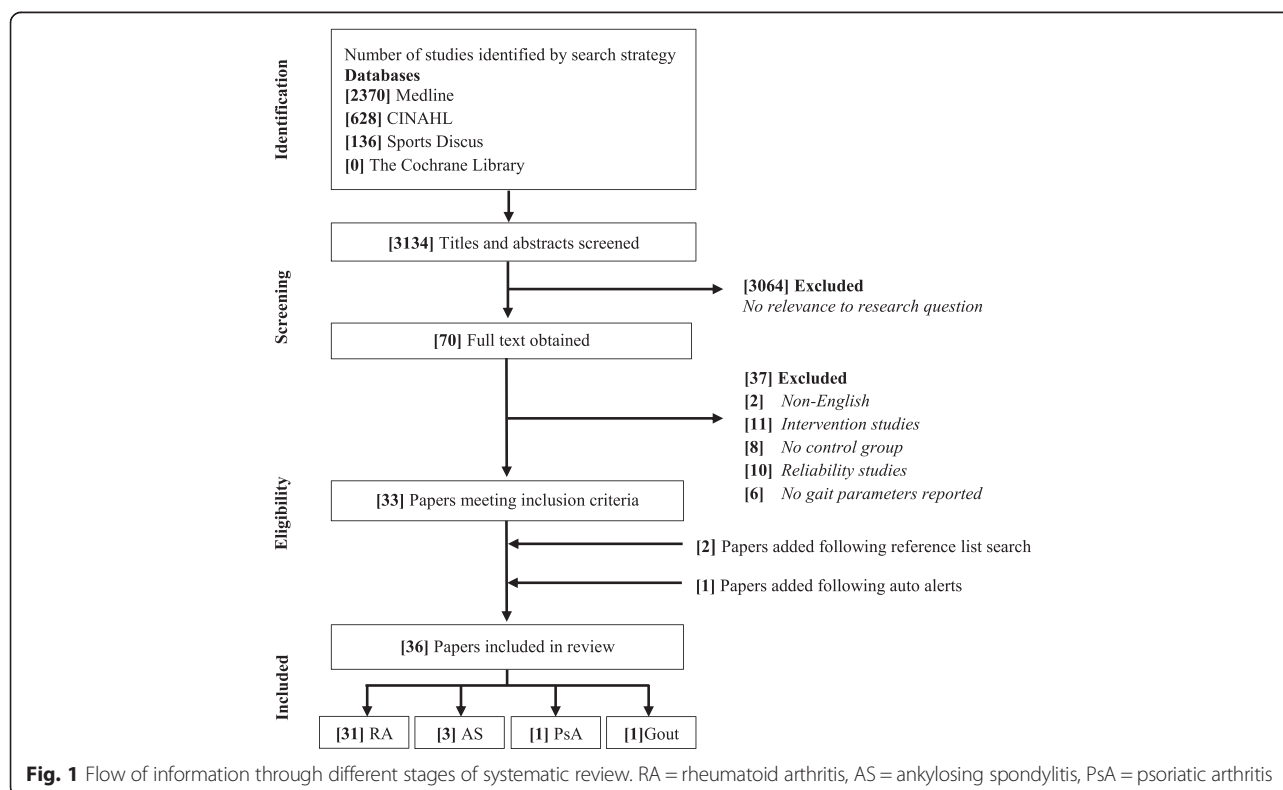


Table 1 Characteristics of included studies

Author	IA	Case demographics			Control demographics			Gait parameters investigated				
		Number	Gender (F:M)	Age mean (SD)	Number	Gender (F:M)	Age mean (SD)	ST	KIM	KIN	PP	EMG
Turner [7]	RA	23	14:9	49.4 (10.5)	23	14:9	49.5 (13.6)	■	■		■	
Woodburn [8]	RA	11	9:2	59.6 (12.0)	5	NR	NR	■	■			
O'Connell [11]	RA	10	8:2	54.0	7	5:2	34.0 ^a	■	■	■	■	
Woodburn [13]	RA	50	34:16	54.0 (11.8)	45	29:16	51.8 (12.4)		■			
Turner [14]	RA	12 (FF) 10 (RF) 6 (COMB)	9:3 8:2 4:2	7.9 (9.3) 53.8 (13.2) 64.7 (6.9)	53	33:20	55.2 (11.7)	■	■			■
Turner [15]	RA	74	58:16	56.4 (12.0)	53	33:20	55.2 (11.7)	■	■	■	■	
Turner [16]	RA	12	12:0	46.0 ^a	12	12:0	47.0 ^a	■	■	■	■	
Weiss [17]	RA	50	43:7	55.0 (14.0)	37	22:15	51.0 (14.0)	■	■	■		
Khazzam [18]	RA	22	20:2	54.0 ^a	25	12:13	41.0 ^a	■	■			
Barn [19]	RA	10	6:4	50.0 (9.0)	5	3:2	47.0 (6.0)		■	■		■
Woodburn [34]	RA	10	NR	52.3 ^a	10	NR	27.9 ^a		■			
Bowen [36]	RA	114	93:21	59.6 (12.0)	49	37:12	33.2 ^a				■	
Dubbeldam [37]	RA	21	17:4	46.6 (12.8)	14	11:3	41.6 (8.5)	■	■			
Yavuz [38]	RA	9	8:1	53.2 (12.3)	14	9:5	53.6 (18.7)				■	
Rome [39]	RA	19	15:4	56.1 (11.1)	21	12:9	51.0 (8.9)	■				
Eppeland [40]	RA	17	7:10	51.1 (6.2)	20	8:12	50.4 (5.3)	■				
Schmiegel [41]	RA	21	NR	57.1 (10.2)	16	NR	50.8 (9.4)				■	
Schmiegel [42]	RA	112	NR	55.0 (11.0)	20	NR	53.2 (12.3)				■	
Laroche [43]	RA	9	6:3	60.0 (7.0)	9	7:2	60.0 (7.0)	■	■			
Laroche [44]	RA	9	6:3	60.6 (6.8)	7	5:2	58.5 (7.4)	■	■	■		
Semple [45]	RA	74	58:16	54.6 (12.0)	53	33:20	55.2 (11.7)	■			■	
Rosenbaum [46]	RA	25	23:2	55.0 (9.9)	21	20:1	50.8 (9.3)				■	
Tuna [47]	RA	50	38:12	50.0 (9.0)	50	39:11	49.8 (7.6)				■	
Otter [48]	RA	25	21:4	45.3 (12.7)	25	22:3	48.0 (8.6)				■	
Woodburn [49]	RA	102	76:26	63.5 ^a	42	31:11	61.0 ^a				■	
Siegel [50]	RA	4	3:1	56.5 (7.2)	2	2:0	28.0 (11.0)		■	■		
Fransen [51]	RA	113	76:37	60.0 (5.5)	102	67:35	58.7 (5.3)	■				
Isacson [52]	RA	17	17:0	40.0 (5.0)	11	11:0	29.0 (7.0)	■	■			
Minns [53]	RA	124	104:20	56.6 ^a	67	32:35	50.2 (10.2)	■			■	
Simkin [54]	RA	18	11:7	58.0 ^a	20	10:10	51.0 ^a	■		■		
Stauffer [55]	RA	30	18:12	NR	29	15:14	NR	■	■			
Del Din [10]	AS	12	4:8	49.4 (10.5)	12	4:8	55.75 (3.2)	■	■	■		
Mangone [56]	AS	17	2:15	47.0 (21.9)	10	1:9	38.7 (14.5)	■	■			
Zebouni [57]	AS	12	4:8	46.5 ^a	11	NR	39.5 ^a	■	■			
Woodburn [21]	PsA	42	25:17	45.3 (12.7)	29	18:11	40.0 (10.5)	■	■	■		
Rome [12]	GT	25	6:19	61.2 (11.7)	25	6:19	57.3 (12.2)	■			■	

SD standard deviation, NR not reported, IA inflammatory arthritis, RA rheumatoid arthritis, GT gout, AS ankylosing spondylitis, PsA psoriatic arthritis, ST spatiotemporal, KIM kinematic, KIN kinetic, PP plantar pressure, EMG electromyography, FF group severe forefoot deformity group, RF group severe rearfoot deformity group, COMB group severe fore- and rearfoot deformity group

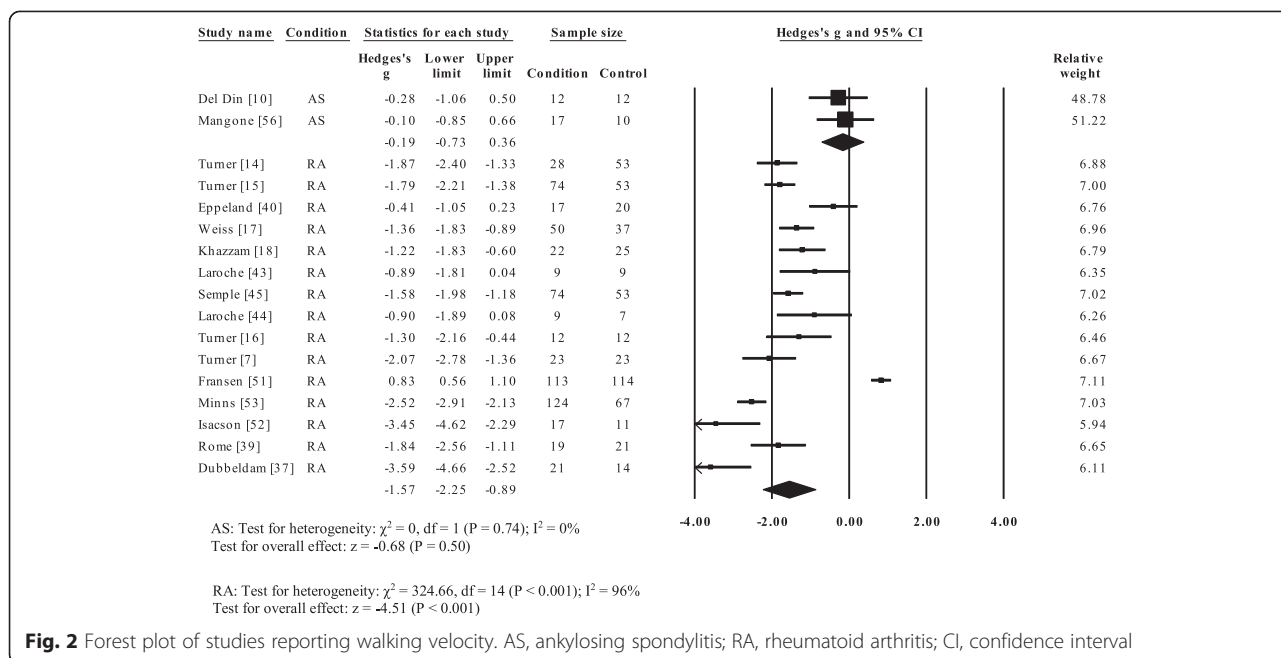
^a SD not reported

Table 2 Modified quality index

Publication	Reporting							External validity		Internal validity bias			Internal validity confounding		Quality Index score total (%)
	1. Hypothesis clearly described?	2. Main outcomes clearly described?	3. Characteristics of the patients included clearly described?	5. Distribution of principle confounder of each group clearly described?	6. Main findings clearly described?	7. Estimates of random variability provided for the main outcomes?	10. Actual probability values reported for main outcomes?	11. Were the subjects asked to participate representative of the entire population?	12. Were the subjects who were prepared to participate representative of the entire population?	16. Was it clear if the results were based on "data dredging"?	18. Were the statistical tests appropriate?	20. Were the main outcome measures valid and reliable?	21. Were all patients and controls recruited from the same population?	22. Were all patients and controls recruited over the same time period?	
Turner [7]	1	1	1	1	1	1	0	0	0	1	1	0	0	0	8 (53)
Woodburn [8]	1	1	0	0	0	1	0	0	0	1	0	1	0	0	5 (33)
Del Din [10]	1	1	1	2	1	1	1	1	0	1	1	1	1	0	13 (87)
O'Connell [11]	1	1	0	0	1	0	0	0	0	1	0	0	0	0	4 (27)
Rome [12]	1	1	1	1	1	1	1	0	0	1	1	1	1	0	11 (73)
Woodburn [13]	1	1	1	2	1	1	1	0	0	1	1	1	0	0	11 (73)
Turner [14]	1	1	1	2	1	1	1	0	0	1	1	1	1	0	12 (80)
Turner [15]	1	1	1	2	1	1	0	0	0	1	1	1	0	0	10 (67)
Turner [16]	1	1	1	1	1	0	0	1	1	1	0	1	1	0	10 (67)
Weiss [17]	1	1	0	1	1	1	1	0	0	1	1	1	1	0	10 (67)
Khazzam [18]	1	1	0	0	1	1	1	1	0	1	1	1	1	1	11 (73)
Barn [19]	1	1	1	2	1	1	1	0	0	1	1	0	0	1	11 (73)
Woodburn [21]	1	1	1	2	1	1	1	0	0	1	1	0	0	0	10 (67)
Woodburn [34]	1	1	1	2	1	0	0	1	0	1	1	1	1	0	11 (73)
Bowen [36]	1	1	1	2	1	1	1	0	0	1	1	1	0	1	12 (80)
Dubbeldam [37]	1	1	1	1	0	1	1	0	0	1	1	1	0	0	9 (60)
Yavuz [38]	1	1	0	0	0	1	0	0	0	1	1	0	0	0	5 (33)
Rome [39]	1	1	1	1	1	1	1	0	0	1	1	0	0	0	9 (60)
Eppeland [40]	1	1	1	2	1	1	1	1	1	1	1	0	1	0	13 (87)
Schmiegel [41]	1	1	1	2	1	1	0	0	0	1	1	1	0	0	10 (67)
Schmiegel [42]	1	1	1	2	1	1	0	1	1	1	1	0	1	0	12 (80)
Laroche [43]	1	1	1	0	1	0	1	0	0	1	0	0	0	0	6 (40)
Laroche [44]	1	1	1	0	1	1	1	1	1	1	1	1	0	0	11 (73)
Semple [45]	1	1	1	0	1	1	1	1	1	1	1	1	0	0	11 (73)
Rosenbaum [46]	1	1	1	1	1	1	1	0	0	1	1	1	0	0	10 (67)
Tuna [47]	1	1	1	1	1	1	0	0	0	1	1	0	0	0	8 (53)
Otter [48]	1	0	1	1	0	1	1	0	0	1	1	0	1	0	8 (53)
Woodburn [49]	1	1	1	2	1	1	1	0	0	1	1	1	0	0	11 (73)
Siegel [50]	1	1	0	0	0	0	0	0	0	1	0	0	0	0	3 (20)
Fransen [51]	1	1	1	2	1	1	1	0	1	1	1	0	1	0	12 (80)
Isacson [52]	1	0	1	0	1	0	0	0	0	1	1	0	1	0	6 (40)

Table 2 Modified quality index (Continued)

Minns [53]	0	0	1	1	1	0	0	0	0	1	0	0	0	0	4 (27)
Simkin [54]	1	1	0	0	0	0	0	0	0	1	0	0	0	0	3 (20)
Stauffer [55]	1	1	1	1	1	0	0	0	0	1	0	1	0	1	8 (53)
Mangone [56]	1	1	1	0	0	1	1	0	0	1	1	0	0	0	7 (47)
Zebouni [57]	1	1	0	0	0	0	0	0	0	1	1	0	0	0	4 (27)
														Median	10 (67)



strategy combined terms appropriate to the anatomical location; the type of gait analysis and IA condition (Additional file 1: Table S1). An initial review was undertaken of all titles and abstracts. All articles considered appropriate were read in full to establish if they met the eligibility criteria.

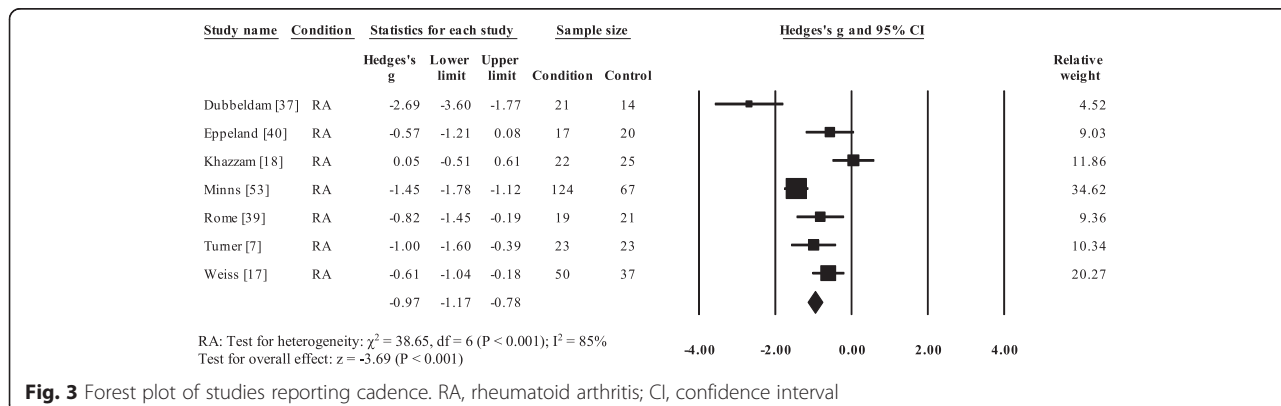
Inclusion and exclusion criteria

Studies were included if they: reported people with IA that included; RA, AS, PsA, gout, polymyalgia rheumatica, systemic sclerosis and systemic lupus erythematosus; if they assessed adults aged >18 years old; if they reported spatiotemporal, kinematic, kinetic, peak plantar pressure or muscle activity data during gait; if they were articles that included a healthy group as means of comparison. Only articles published in English

were included. Surgical and pharmacological intervention studies were excluded. No limitation was placed on the date of the publication with databases screened up to March 2015.

Data extraction

All titles and abstracts identified through database searches were downloaded into Endnote X4 (Thomson, Reuters, Carlsbad, CA). Each title and abstract was evaluated for potential inclusion by two independent reviewers (MC, KR). If there was insufficient information contained in the title to determine suitability the full text was obtained. Any discrepancies between the two reviewers (MC, KR) were resolved at a consensus meeting.



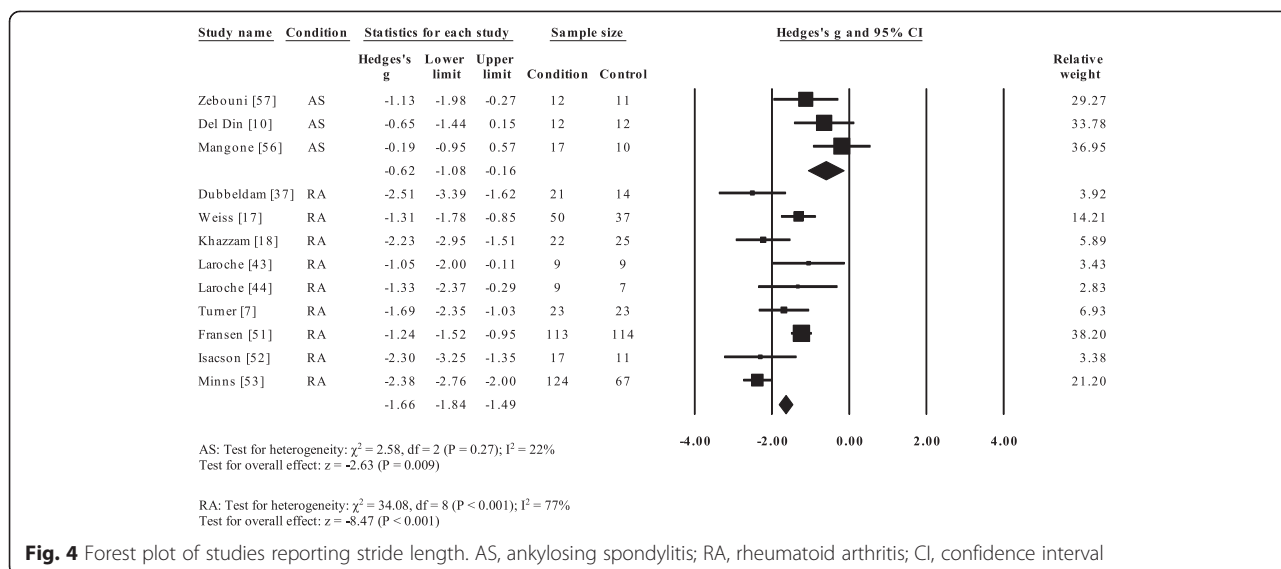


Fig. 4 Forest plot of studies reporting stride length. AS, ankylosing spondylitis; RA, rheumatoid arthritis; CI, confidence interval

Assessment of methodological quality and diversity

The quality of studies was evaluated independently by two reviewers (MC and KR), who were blinded to author and publication details. Study quality was rated using a modified version of the Quality Index (QI) tool originally described by Downs and Black [26]. The QI tool consists of 27 items which allow for the assessment of internal and external validity, reporting of bias and power. The tool was modified to exclude thirteen questions that were not relevant to the articles assessed in this review, resulting in the retention of 14 questions. The scoring system grades each of the 14 questions either a (0 = no/unable to determine, or 1 = yes) with the exception of question five (0 = no, 1 = partially, 2 = yes). The summed score for each study was calculated, the maximum achievable being 15. No cut off scores have been described to categorise study quality for the Downs and Black quality Index [27]. In

the absence of validated cut off scores and following review of past articles that have applied the Downs and Black criteria the follow cut off values were applied: ≥ 12 was considered high quality, ≥ 7 but < 12 as moderate quality, and < 6 as poor quality [27, 28].

Data analysis and synthesis

Relevant gait parameters and information regarding overall study design, subject characteristics and gait analysis parameters were extracted from each paper by one author (MC) from those studies meeting the inclusion criteria. Data was tabulated according to the specific IA condition and gait parameters.

The clinical and methodological diversity among the studies was assessed to determine the appropriateness of data pooling for meta-analysis. Factors considered important for comparison included: mean age, sex distribution, case and comparison group size,

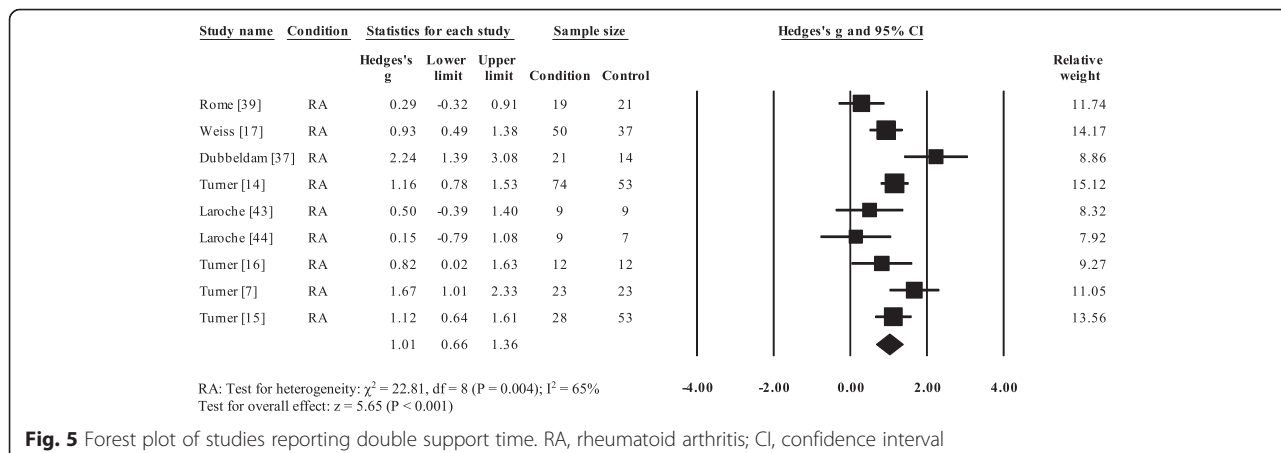


Fig. 5 Forest plot of studies reporting double support time. RA, rheumatoid arthritis; CI, confidence interval

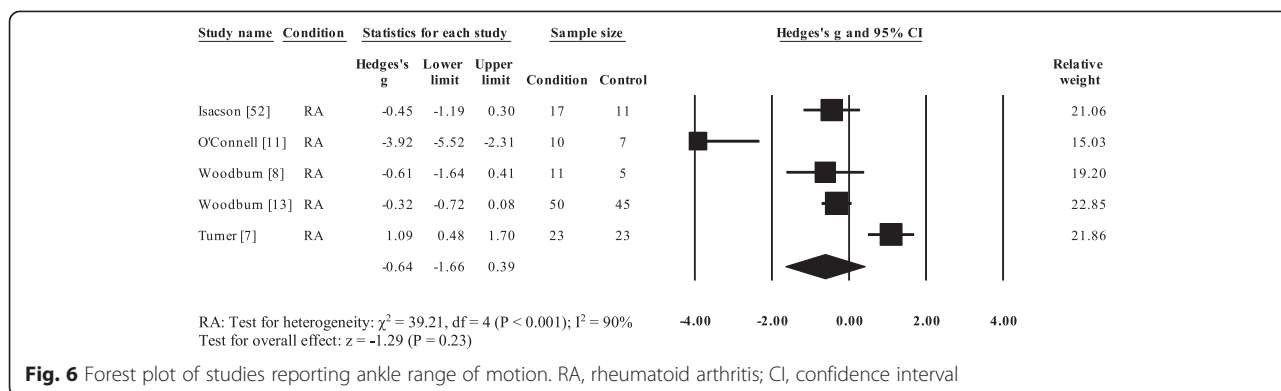


Fig. 6 Forest plot of studies reporting ankle range of motion. RA, rheumatoid arthritis; CI, confidence interval

data acquisition methodology and instrumentation. Two authors (MC and PP) reviewed the included studies and reached consensus on the appropriateness of conducting meta-analysis. Heterogeneity was considered low if the I^2 value was $\leq 25\%$, moderate if the value was $> 25\%$ and $\leq 50\%$, high if $> 50\%$ and $\leq 75\%$ and very high if greater than 75% [29]. A fixed-effect model was applied where the I^2 statistic was less than 50% and the χ^2 test indicated a non-significant degree of heterogeneity ($P > 0.1$). The random-effect model was used where the I^2 statistic was greater than 50% and the χ^2 test indicated statistically significant heterogeneity ($P < 0.1$) [30].

Where data was available from each paper a standardised mean difference (SMD) (Hedges's g) and 95% confidence interval (CI) were calculated [31]. This was calculated as the difference between cases and control group means divided by the pooled SD. Interpretation of SMDs was based on previous effect size (ES) guidelines: small effect ≥ 0.2 , medium effect ≥ 0.5 , large effect ≥ 0.8 [32]. Effect sizes were considered statistically significant if the 95% CI did not contain zero for the SMD. All data were analysed using the Comprehensive Meta-analysis, version 2 [33]. When mean and SD was not reported, the median and range were reported. Studies that met the inclusion criteria but did not report SD, or where the

SD could not be obtained were excluded from meta-analysis (Additional file 2: Table S2).

Results

Selection and characteristics of studies

A total of 3134 citations were identified for screening with 36 articles being included for further analysis (Fig. 1). Thirty-one studies evaluated gait parameters in RA [7, 8, 11, 13–18, 34–55], three in AS [10, 56, 57] one in PsA [21] and one in gout [12]. Twenty-four studies examined spatiotemporal gait parameters, with 19 in RA, two in AS, one PsA and gout (Additional file 3: Table S3). Twenty-one studies assessed kinematic parameters, with 17 in RA, three AS and one in PsA (Additional file 4: Table S4). Ten studies examined kinetic parameters with eight in RA, one AS and one in PsA (Additional file 5: Table S5). Sixteen studies evaluated plantar pressure parameters, with 15 in RA and one in gout (Additional file 6: Table S6). Three studies assessed all gait parameters (spatiotemporal, kinematic, kinetic and plantar pressures) in the population of interest [11, 14, 15]. No studies reported gait characteristics in polymyalgia rheumatica, systemic sclerosis and systemic lupus erythematosus. The total number of participants was 2275; 1321 with IA and 954 controls. IA participants included 863 females and 312 males. The mean (SD) age of IA cases and

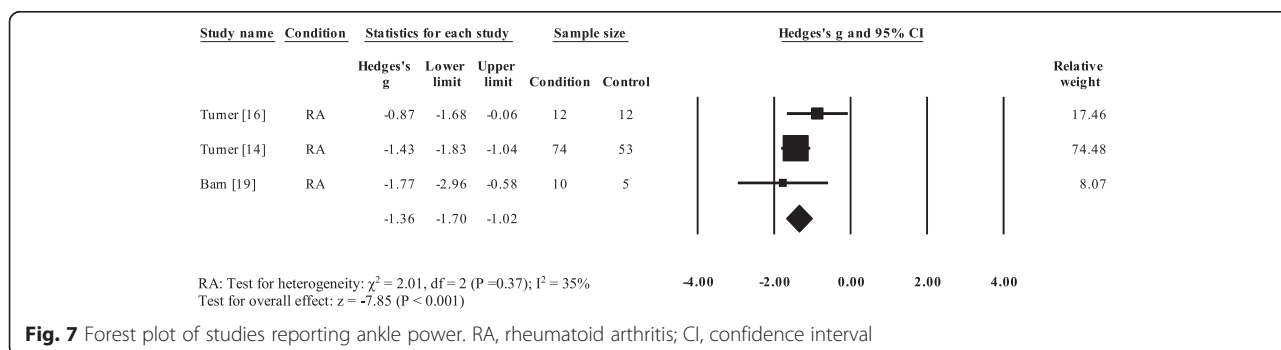
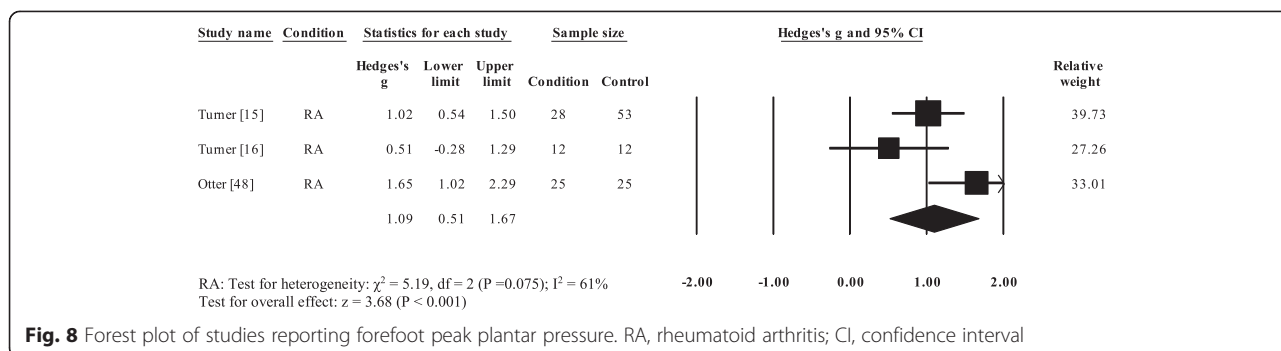


Fig. 7 Forest plot of studies reporting ankle power. RA, rheumatoid arthritis; CI, confidence interval



controls was 52.6 (9.3) and 47.8 (9.2) years, respectively (Table 1).

Methodological quality of studies

Two reviewers (MC & KR) individually scored a total of 504 items and agreed on 480 items (95 %) with an inter-rater agreement of $k = 0.90$ ($p < 0.001$). Six of the 36 articles were of high quality (quality score ≥ 12). The median (%) quality score of all articles was 10 (67 %), ranging between 20–87 % (Table 2). There was limited information on the methods of study recruitment across the majority of studies making it difficult to assess the generalisability of study results. The majority of studies investigating kinematic and kinetic parameters also reported small sample sizes.

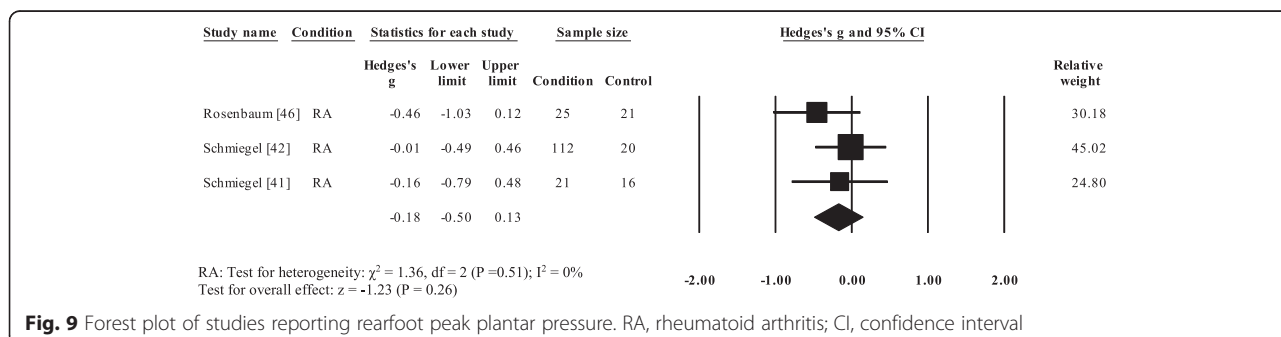
Spatiotemporal gait parameters

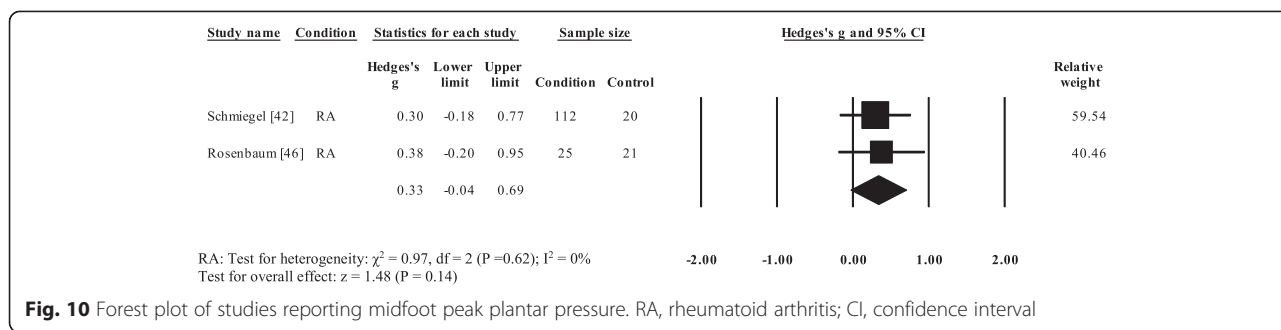
Fifteen RA [7, 14–18, 37, 39, 40, 44, 45, 51–54], one PsA [21] and one gout study [12] reported significant decreases in walking velocity. No significant differences in walking velocity were reported for AS [10, 56]. Overall pooled data (SMD, 95 % CI) (Fig. 2) for walking velocity, demonstrated a significant decreased large effect size for RA (SMD -1.55 , -2.27 to -0.83) and a non-significant decrease for AS (SMD -0.19 , -0.73 to 0.36).

Five RA studies [7, 17, 37, 39, 53] and one gout (52) reported significant decreases in cadence. Cadence was not significantly decreased in AS [57]. Overall, pooled data for cadence in RA (Fig. 3) showed a decreased but significant large effect size (SMD -0.97 , -1.49 to -0.45). Nine RA studies [7, 17, 18, 37, 44, 51–54], one AS [57] and one gout [12] reported significant decreases in stride length. Pooled data for stride length in RA (SMD -1.66 , -1.84 to -1.49) and AS (SMD -0.62 , -1.08 to -0.27) were significantly decreased with a large effect size (Fig. 4). Eight RA studies [7, 14–17, 37, 39, 51] and one gout study [12] reported significant increases in double support. Pooled data for double support in RA showed (Fig. 5) a significantly increased large effect size (SMD 1.01 , 0.66 to 1.36).

Kinematic and kinetic gait parameters

Five RA studies reported on the total ankle range of motion [7, 8, 11, 13, 52]. Three studies reported no significant differences [8, 13, 52], with one study reporting a significant increase [7] and one study reporting a significant decrease in the total ankle range of motion [11]. Results of the meta-analysis (Fig. 6) demonstrated that the overall effect size for total ankle range of motion was non-significant (SMD -0.64 , -1.66 to 0.39). Ankle power was reported in three RA [15, 16, 35] and one PsA study [21]. All four studies reported significant





reductions in ankle power. The overall effect size for ankle power in RA (Fig. 7) was significantly large (SMD -1.36, -1.70 to -1.02).

Peak plantar pressure gait parameters

Three RA studies [14, 16, 47, 48] reported significantly higher forefoot peak plantar pressures in RA. Results from the meta-analysis (Fig. 8) showed that the overall effect size for peak plantar pressure to the forefoot was significantly large (SMD 1.09, 0.51 to 1.67). Pooled results in the RA studies demonstrated no significant differences in peak plantar pressure for the rearfoot (Fig. 9), midfoot (Fig. 10), first metatarsal (Fig. 11), 2nd metatarsal (Fig. 12) and the 3–5th metatarsal heads (Fig. 13). Hallux peak plantar pressure (Fig. 14) was reported to be significantly lower in gout [12].

Muscle activity

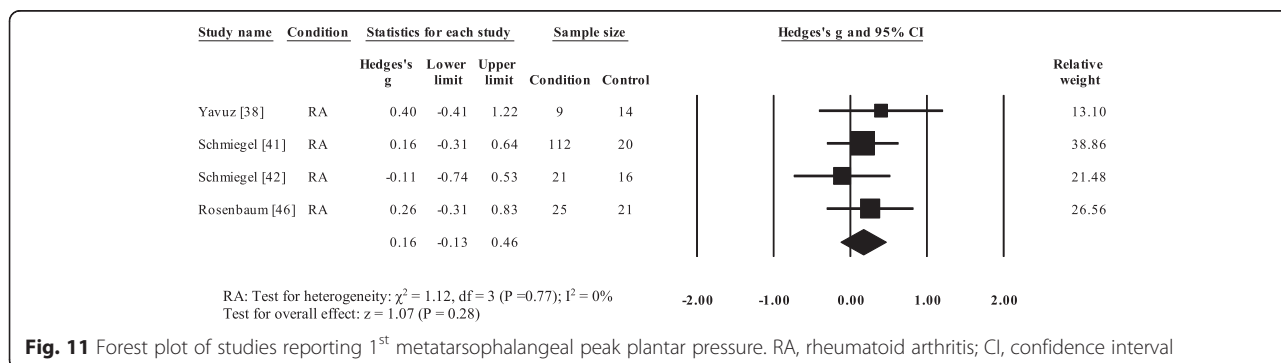
One RA study investigated muscle activity of the tibialis posterior muscle and reported increased muscle activity during the single support phase of gait [35].

Discussion

This systematic review highlights significant differences in gait variables between people with IA and

controls. The review found the majority of studies report on RA with a limited number of studies on other IA conditions. The review found similar findings to previous studies, that people with RA adopt an antalgic gait resulting from a pain avoidance pattern that contributes to a decrease in walking velocity, cadence, increased double limb support time, and decreased ankle power with increased peak plantar pressures to the forefoot [11, 15, 17, 18]. Antalgic gait was also found in gout and AS suggesting that adaptation may occur due to the disease or a compensatory mechanism to accommodate for localised foot pain and deformity [14]. Gait adaptation in PsA may relate to enthesal foot pathologies and foot pain [9, 58]. Woodburn [21] postulated a stress shielding mechanism may be the driver of gait adaptation with walking speeds decreased in attempt to lower stress at the Achilles tendon. The review found a reduction in peak plantar pressure under the first metatarsal head, suggesting that people with gout may use a pain-avoidance strategy to reduce the pain associated with the structural joint damage of the first metatarsophalangeal joint.

The chief advantage of three-dimensional (3D) motion analysis is that dynamic assessments of foot motion during functional activities, such as walking, can be performed [59]. Recent advances in motion capture



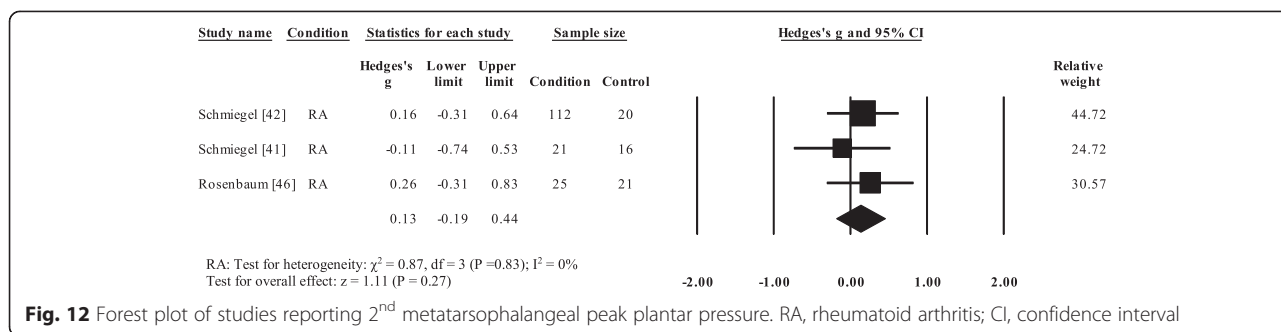


Fig. 12 Forest plot of studies reporting 2nd metatarsophalangeal peak plantar pressure. RA, rheumatoid arthritis; CI, confidence interval

technology afford improved spatial resolution and allow the definition of relatively small segments in the foot [59]. In the last decade there has been an exponential growth in the use of 3D models to explain gait strategies [60]. The development of detailed foot models is beginning to quantify the kinematics and kinetics of the foot, however there are limitations for use in people with IA. Issues related to soft tissue artefacts and the validity of skin markers to track underlying skeletal segments remains problematic. Inaccurate identification of anatomical landmarks due to the presence of foot deformity in IA may affect the estimation, interpretation and reconstruction of joint axis and ultimately the calculation of joint kinematics and kinetics [61]. The development of foot models has also increased the detail and variety of 3D motion analysis variables used to explain gait strategies in people with IA. In comparison to spatiotemporal gait parameters and plantar pressure variables there appears to be no consensus as to the most important gait variables that relate to overall functional status.

This review has some limitations. There was a large variation in the disease activity, disease duration and level of deformities across all studies. Many studies used relatively small samples that were underpowered and the heterogeneous nature of the IA population makes interpretation of the data difficult. A number of

studies were included in the review but excluded from data pooling due to a lack of data reporting of standard deviations and mean values of gait parameters. Previous studies have described a wide range of methodologies to acquire and define gait parameters and this complicates the synthesis of data across different studies. The review was restricted to case-control studies and did not consider findings from intervention studies. We only analysed the foot and ankle characteristics in IA, with no consideration given to data from the knee, hip and pelvis.

Two key pathways have been postulated to contribute to the development of foot pain and deformity in IA: inflammatory and/or mechanical [59]. However, limited objective evidence exists to comprehensively examine inflammatory and mechanical markers in the context of foot pain and deformity across IA conditions. Given the limited data across all IA conditions, future directions should include analysis of muscle activity; this will provide information on the forces producing movements and patterns of muscle activation. Future research is required to understand the combined effects of spatiotemporal, kinematic, kinetic and plantar pressure impact on foot function. This will allow for relationships to be investigated across the differing gait parameters and may further define the mechanism of gait adaptation with IA conditions.

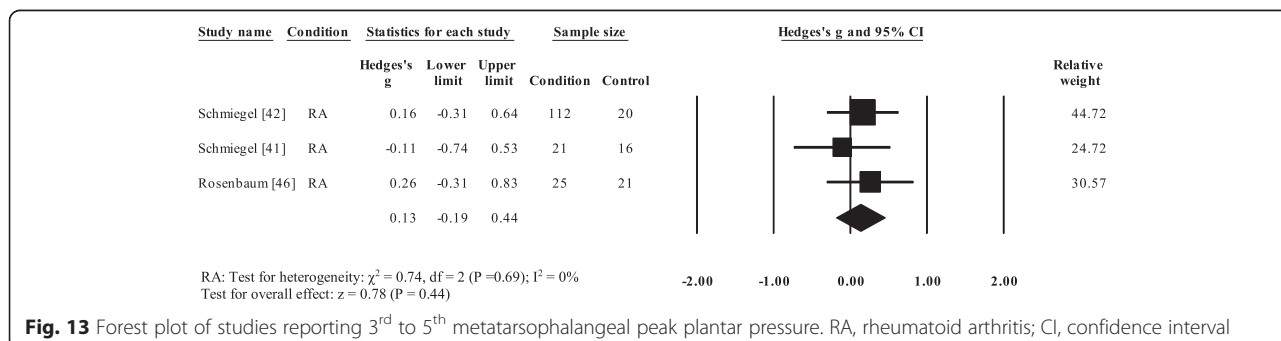
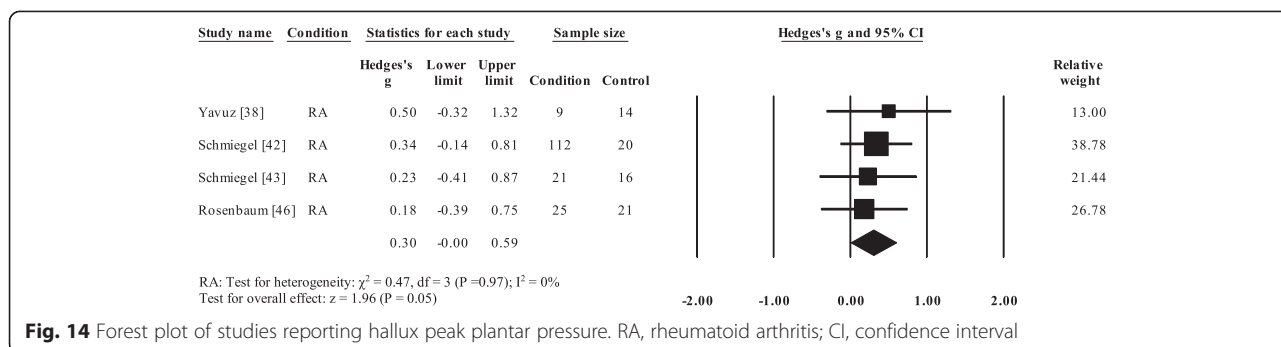


Fig. 13 Forest plot of studies reporting 3rd to 5th metatarsophalangeal peak plantar pressure. RA, rheumatoid arthritis; CI, confidence interval



Conclusion

The advancement of 3D gait analysis has given a clearer insight into the complex interaction between the underlying mechanisms of inflammation and mechanical pathways that influence the development of foot problems in people with IA. The review identified 36 gait studies with the majority of studies reporting gait adaptations in RA, but limited evidence relating to other IA conditions. Poor data reporting, small sample sizes and heterogeneity across IA conditions limit the interpretation of the findings. Future studies should consider a standardised analytical approach to gait analysis that will enable comparisons across studies and provide clinicians and researchers with objective evidence of foot function in people with IA.

Additional files

- Additional file 1: Table S1.** Description of search strategy. Details of specific search terms used and the order in which the search tool place.
- Additional file 2: Table S2.** Excluded studies. Details of the publications excluded, the reason for exclusion and the reference list of the excluded studies.
- Additional file 3: Table S3.** Spatiotemporal gait analysis parameters acquired by included studies. Details of the data acquisition method and the specific spatiotemporal parameters that were measured by studies assessing spatiotemporal gait parameters.
- Additional file 4: Table S4.** Kinematic gait parameters measured and methods of data acquisition. Details of the specific kinematic parameters, measured the biomechanical model used to determine kinematic parameters.
- Additional file 5: Table S5.** Kinetic gait analysis parameters acquired by included studies. Details of kinetic gait parameters measured.
- Additional file 6: Table S6.** Plantar pressure parameters acquired by included studies. Details of the specific regions of the foot where plantar pressure was measured and the method by which plantar pressure was acquired.

Competing interests

The authors declare that they have no competing interests.

Authors' contributions

MC proposed the study protocol, which was elaborated and specified by KR, ND & PP. MC and KR screened all the references and extracted the data. MB served as arbitrator in case of discrepancies during extraction. PP supported the data extraction process. All authors interpreted the results. All authors read and approved the final manuscript.

Acknowledgements

This work was supported by Arthritis New Zealand.

Author details

¹Department of Podiatry, Health & Rehabilitation Research Institute, Auckland University of Technology, Auckland, New Zealand. ²National Institute for Stroke and Applied Neurosciences, Auckland University of Technology, Auckland, New Zealand. ³Department of Medicine, University of Auckland, Auckland, New Zealand. ⁴Department of Physiotherapy, Health & Rehabilitation Research Institute, Auckland University of Technology, Auckland, New Zealand.

Received: 12 December 2014 Accepted: 27 May 2015

Published online: 05 June 2015

References

1. Hootman JM, Helmick CG, Brady TJ. A public health approach to addressing arthritis in older adults: the most common cause of disability. *Am J Public Health.* 2012;102(3):426–33.
2. Epstein FH, Harris Jr ED. Rheumatoid arthritis: pathophysiology and implications for therapy. *N Engl J Med.* 1990;322(18):1277–89.
3. Frizziero A, Bonsangue V, Trevisan M, Ames PRJ, Masiero S. Foot tendinopathies in rheumatic diseases: etiopathogenesis, clinical manifestations and therapeutic options. *Clin Rheumatol.* 2013;32(5):547–55.
4. Reveille JD, Arnett FC. Spondyloarthritis: update on pathogenesis and management. *Am J Med.* 2005;118(6):592–603.
5. Richette P, Bardin T. Gout. *Lancet.* 2010;375:318–28.
6. Platto MJ, O'Connell PG, Hicks JE, Gerber LH. The relationship of pain and deformity of the rheumatoid foot to gait and an index of functional ambulation. *J Rheumatol.* 1991;18(1):38–43.
7. Turner D, Woodburn J, Helliwell P, Cornwall M, Emery P. Pes planovalgus in RA: a descriptive and analytical study of foot function determined by gait analysis. *Musculoskeletal Care.* 2003;1(1):21–33.
8. Woodburn J, Nelson K, Siegel K, Kepple T, Gerber L. Multisegment foot motion during gait: proof of concept in rheumatoid arthritis. *J Rheumatol.* 2004;31(10):1918–27.
9. Hyslop E, McInnes IB, Woodburn J, Turner DE. Foot problems in psoriatic arthritis: high burden and low care provision. *Ann Rheum Dis.* 2010;69(5):928.
10. Del Din S, Carraro E, Sawacha Z, Guiotto A, Bonaldo L, Masiero S, et al. Impaired gait in ankylosing spondylitis. *Med Biol Eng Comput.* 2011;49(7):801–9.
11. O'Connell PG, Lohmann Siegel K, Kepple TM, Stanhope SJ, Gerber LH. Forefoot deformity, pain, and mobility in rheumatoid and nonarthritic subjects. *J Rheumatol.* 1998;25(9):1681–6.
12. Rome K, Survepalli D, Sanders A, Lobo M, McQueen F, McNair P, et al. Functional and biomechanical characteristics of foot disease in chronic gout: A case-control study. *Clin Biomech.* 2011;26(1):90–4.
13. Woodburn J, Helliwell P, Barker S. Three-dimensional kinematics at the ankle joint complex in rheumatoid arthritis patients with painful valgus deformity of the rearfoot. *Rheumatology (Oxford).* 2002;41(12):1406–12.
14. Turner D, Woodburn J. Characterising the clinical and biomechanical features of severely deformed feet in rheumatoid arthritis. *Gait Posture.* 2008;28(4):574–80.

15. Turner DE, Helliwell PS, Siegel KL, Woodburn J. Biomechanics of the foot in rheumatoid arthritis: Identifying abnormal function and the factors associated with localised disease 'impact'. *Clin Biomech.* 2008;23(1):93–100.
16. Turner DE, Helliwell PS, Emery P, Woodburn J. The impact of rheumatoid arthritis on foot function in the early stages of disease: a clinical case series. *BMC Musculoskelet Disord.* 2006;7:102–8.
17. Weiss RJ, Wretenberg P, Stark A, Palmblad K, Larsson P, Gröndal L, et al. Gait pattern in rheumatoid arthritis. *Gait Posture.* 2008;28(2):229–34.
18. Khazzam M, Long JT, Marks RM, Harris GF. Kinematic changes of the foot and ankle in patients with systemic rheumatoid arthritis and forefoot deformity. *J Orthop Res.* 2007;25(3):319–29.
19. Barn R, Turner DE, Rafferty D, Sturrock RD, Woodburn J. Tibialis posterior tenosynovitis and associated pes plano valgus in rheumatoid arthritis: EMG, multi-segment foot kinematics and ultrasound features. *Arthritis Care Res.* 2012;65(4):495–502.
20. Broström EW, Esbjörnsson A-C, von Heideken J, Iversen MD. Gait deviations in individuals with inflammatory joint diseases and osteoarthritis and the usage of three-dimensional gait analysis. *Best Pract Res Clin Rheumatol.* 2012;26(3):409–22.
21. Woodburn J, Hyslop E, Barn R, McInnes I, Turner D. Achilles tendon biomechanics in psoriatic arthritis patients with ultrasound proven enthesitis. *Scand J Rheumatol.* 2013;00:1–4.
22. Salvarani C, Cantini F, Macchioni P, Olivieri I, Niccoli L, Padula A, et al. Distal musculoskeletal manifestations in polymyalgia rheumatica: a prospective followup study. *Arthritis Rheum.* 1998;41(7):1221–6.
23. Hietaharju A, Jääskeläinen S, Kalimo H, Hietarinta M. Peripheral neuromuscular manifestations in systemic sclerosis (scleroderma). *Muscle Nerve.* 1993;16(11):1204–12.
24. Williams A, Crofts G, Teh L. 'Focus on feet'—the effects of systemic lupus erythematosus: a narrative review of the literature. *Lupus.* 2013;22(10):1017–23.
25. Baan H, Dubbeldam R, Nene AV, van de Laar MAFJ. Gait analysis of the lower limb in patients with rheumatoid arthritis: a systematic review. *Semin Arthritis Rheum.* 2012;41(6):768–88. e768.
26. Downs S, Black N. The feasibility of creating a checklist for the assessment of the methodological quality both of randomised and non-randomised studies of health care interventions. *J Epidemiol Community Health.* 1998;52(6):377–84.
27. Meyer S, Karttunen AH, Thijs V, Feys H, Verheyden G. How do somatosensory deficits in the arm and hand relate to upper limb impairment, activity, and participation problems after stroke? A systematic review. *Phys Ther.* 2014;94(9):1220–31.
28. Liu Y, Davari-Farid S, Arora P, Porhomayon J, Nader ND. Early versus late initiation of renal replacement therapy in critically ill patients with acute kidney injury after cardiac surgery: a systematic review and meta-analysis. *J Cardiothorac Vasc Anesth.* 2014;28(3):557–63.
29. Higgins JP, Thompson SG, Deeks JJ, Altman DG. Measuring inconsistency in meta-analyses. *Br Med J.* 2003;327(7414):557.
30. Deeks JJ, Higgins J, Altman DG. Analysing data and undertaking meta-analyses. In: *Cochrane handbook for systematic reviews of interventions: Cochrane book series.* 2008. p. 243–96.
31. Hedges LV. Distribution theory for Glass's estimator of effect size and related estimators. *J Educ Behav Stat.* 1981;6(2):107–28.
32. Cohen J. A power primer. *Psychol Bull.* 1992;112(1):155–9.
33. Borenstein M, Hedges L, Higgins J, Rothstein H. *Comprehensive meta-analysis version 2.* Englewood, NJ: Biostat; 2005. p. 104.
34. Woodburn J, Turner D, Helliwell P, Barker S. A preliminary study determining the feasibility of electromagnetic tracking for kinematics at the ankle joint complex. *Rheumatology (Oxford).* 1999;38(12):1260–8.
35. Barn R, Turner DE, Rafferty D, Sturrock RD, Woodburn J. Tibialis posterior tenosynovitis and associated pes Plano valgus in rheumatoid arthritis: electromyography, multisegment foot kinematics, and ultrasound features. *Arthritis Care & Res.* 2013;65(4):495–502.
36. Bowen CJ, Culliford D, Allen R, Beacroft J, Gay A, Hooper L, et al. Forefoot pathology in rheumatoid arthritis identified with ultrasound may not localise to areas of highest pressure: cohort observations at baseline and twelve months. *J Foot Ankle Res.* 2011;4(1):25.
37. Dubbeldam R, Nene AV, Buurke JH, Groothuis-Oudshoorn CGM, Baan H, Drossaers-Bakker KW, et al. Foot and ankle joint kinematics in rheumatoid arthritis cannot only be explained by alteration in walking speed. *Gait Posture.* 2011;33(3):390–5.
38. Yavuz M, Husni E, Botek G, Davis BL. Plantar shear stress distribution in patients with rheumatoid arthritis relevance to foot pain. *J Am Podiatr Med Assoc.* 2010;100(4):265–9.
39. Rome K, Dixon J, Gray M, Woodley R. Evaluation of static and dynamic postural stability in established rheumatoid arthritis: exploratory study. *Clin Biomech.* 2009;24(6):524–6.
40. Eppeland S, Myklebust G, Hodt-Billington C, Moe-Nilssen R. Gait patterns in subjects with rheumatoid arthritis cannot be explained by reduced speed alone. *Gait Posture.* 2009;29(3):499–503.
41. Schmiegel A, Vieth V, Gaubitz M, Rosenbaum D. Pedography and radiographic imaging for the detection of foot deformities in rheumatoid arthritis. *Clin Biomech.* 2008;23(5):648–52.
42. Schmiegel A, Rosenbaum D, Schorat A, Hilker A, Gaubitz M. Assessment of foot impairment in rheumatoid arthritis patients by dynamic pedobarography. *Gait Posture.* 2008;27(1):110–4.
43. Laroche D, Ornetti P, Thomas E, Ballay Y, Maillefert JF, Pozzo T. Kinematic adaptation of locomotor pattern in rheumatoid arthritis patients with forefoot impairment. *Exp Brain Res.* 2007;176(1):85–97.
44. Laroche D, Pozzo T, Ornetti P, Tavernier C, Maillefert JF. Effects of loss of metatarsophalangeal joint mobility on gait in rheumatoid arthritis patients. *Rheumatology (Oxford).* 2006;45(4):435–40.
45. Semple R, Turner DE, Helliwell PS, Woodburn J. Regionalised centre of pressure analysis in patients with rheumatoid arthritis. *Clin Biomech.* 2007;22(1):127–9.
46. Rosenbaum D, Schmiegel A, Meermeier M, Gaubitz M. Plantar sensitivity, foot loading and walking pain in rheumatoid arthritis. *Rheumatology (Oxford).* 2006;45(2):212–4.
47. Tuna H, Birtane M, Taştekin N, Kokino S. Pedobarography and its relation to radiologic erosion scores in rheumatoid arthritis. *Rheumatol Int.* 2005;26(1):42–7.
48. Otter SJ, Bowen CJ, Young AK. Forefoot plantar pressures in rheumatoid arthritis. *J Am Podiatr Med Assoc.* 2004;94(3):255–60.
49. Woodburn J, Helliwell PS. Relation between heel position and the distribution of forefoot plantar pressures and skin callosities in rheumatoid arthritis. *Ann Rheum Dis.* 1996;55(11):806–10.
50. Siegel KL, Kepple TM, O'Connell PG, Gerber LH, Stanhope SJ. A technique to evaluate foot function during the stance phase of gait. *Foot Ankle Int.* 1995;16(12):764–70.
51. Fransen M, Heussler J, Margiotta E, Edmonds J. Quantitative gait analysis—comparison of rheumatoid arthritic and non-arthritic subjects. *Aust J Physiother.* 1994;40(3):191–9.
52. Isacson J, Broström LÅ. Gait in rheumatoid arthritis: an electrogoniometric investigation. *J Biomech.* 1988;21(6):451–3. 455–457.
53. Minns R, Craxford AD. Pressure under the forefoot in rheumatoid arthritis a comparison of static and dynamic methods of assessment. *Clin Orthop Relat Res.* 1984;187:235.
54. Simkin A. The dynamic vertical force distribution during level walking under normal and rheumatic feet. *Rheumatol Rehabil.* 1981;20(2):88–97.
55. Stauffer RN, Chao EY, Györy AN. Biomechanical gait analysis of the diseased knee joint. *Clin Orthop Relat Res.* 1977;126:246–55.
56. Mangone M, Scettri P, Paoloni M, Procaccianti R, Spadaro A, Santilli V. Pelvis-shoulder coordination during level walking in patients with ankylosing spondylitis. *Gait Posture.* 2011;34(1):1–5.
57. Zebouni L, Helliwell P, Howe A, Wright V. Gait analysis in ankylosing spondylitis. *Ann Rheum Dis.* 1992;51(7):898–9.
58. Hyslop E, Woodburn J, McInnes I, Semple R, Newcombe L, Hendry G, et al. A reliability study of biomechanical foot function in psoriatic arthritis based on a novel multi-segmented foot model. *Gait Posture.* 2010;32(4):619–26.
59. Rao S. Quantifying foot function in individuals with rheumatoid arthritis: recent advances and clinical implications. *Arthritis Care Res.* 2013;65(4):493–4.
60. Deschamps K, Staes F, Roosen P, Nobels F, Desloovere K, Bruyninckx H, et al. Body of evidence supporting the clinical use of 3D multisegment foot models: A systematic review. *Gait Posture.* 2011;33(3):338–49.
61. Della Croce U, Leardini A, Chiari L, Cappozzo A. Human movement analysis using stereophotogrammetry: Part 4: assessment of anatomical landmark misplacement and its effects on joint kinematics. *Gait Posture.* 2005;21(2):226–37.