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Biopsychosocial Determinants of Hand Function and Its Trajectories Over Five Years in Patients With Hand Osteoarthritis

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Objective. This study aimed to investigate hand function trajectories over five years in primary hand osteoarthritis (OA). Additionally, determinants of baseline and longitudinal hand function were assessed.

Methods. A total of 538 patients with both baseline and five-year study visits were analyzed. Annual data of the Australian/Canadian Osteoarthritis Hand Index (AUSCAN) function subscale (range 0–36) from the Hand Osteoarthritis in Secondary Care study were used. Latent Class Growth Analysis (LCGA) identified hand function trajectories. Baseline and longitudinal associations with hand function were assessed using multivariable linear and multinomial logistic regression.

Results. There were 538 patients (86.1% women; mean age 61.0 years) with a mean AUSCAN function 15.6 (SD 8.4; range 0–36; higher scores indicate worse hand function) included. Multivariable analysis showed that hand function was cross-sectionally associated with AUSCAN pain, grip strength, hand mobility, and the illness perception consequences. In 301 patients with five-year follow-up data, four hand function trajectories emerged. The worst trajectories were defined by baseline function scores of 23.69 (95% confidence interval [CI] 22.04–25.61) with a slope of 0.36 (95% CI 0.03–0.69) and 16.87 (95% CI 15.41–18.34) with a slope of 0.38 (95% CI 0.01–0.74) and deteriorated over five years (mean decrease of respectively 1.82 and 1.88). Deteriorating trajectories were associated with structural damage and the illness perception chronicity of disease.

Conclusion. Hand function varies greatly in patients with hand OA. LCGA identified four longitudinal patient groups, of which two showed an unfavorable course of hand function over five years. Hand function was characterized by disease characteristics, including structural damage and psychosocial determinants.

INTRODUCTION

Hand osteoarthritis (OA) is a prevalent disease that affects nearly 200 million individuals worldwide.[1] It increases with age and affects one in four men and one in two women by the age of 85 years.[2] Hand OA leads to a variety of symptoms, of which loss of hand function and disability are among the most important, as they impact activities of daily living and participation in society.[3,4] Often, loss of hand function attracts less attention in comparison to hand pain,[5] but earlier studies showed that it is at least equally important for quality of life of patients with hand OA.[3,6]

Which factors contribute to impaired hand function are not clear, but it is considered to be multifactorial. Female sex was associated with disability, reduced hand performance, and less grip strength regardless of radiographic hand OA severity.[7] Importantly, female sex was positively associated with hand pain,[8] which in turn negatively influenced hand function.[9,10] Regarding coping styles, comforting cognitions, decreasing physical activity, and pacing were positively associated with disability.[11] Evidence about the association of structural damage of the hand joints, such as bony swelling, deformity, and radiographic osteoarthritic signs with hand function remained

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SIGNIFICANCE & INNOVATIONS

- Disease-specific, psychological factors, and coping styles are associated with worse baseline hand function.
- Patients with a relatively worse baseline hand function show further deterioration over time.
- Structural damage and perceiving hand osteoarthritis as chronic are associated with worse longitudinal hand function.

inconclusive,[12,13] although erosive hand OA has shown stronger associations with loss of hand function than nonerosive hand OA.[14] Thumb-base OA (first carpometacarpal [CMC-1]) is especially associated with impaired hand function.[14–16]

In addition to loss of function, hand OA results in other unfavorable outcomes in the hands,[17] such as loss of strength (i.e., grip and pinch strength), loss of fine force control, and loss of mobility.[18–20] How these outcomes relate to each other is unclear,[21] although it is shown that hand pain and loss of function are closely intertwined.[5,8,22]

Previously, we have shown that the course of hand pain remained stable over time on the group level.[8] Whether this is similar for the course of hand function over time is unknown. Hand function is considered to gradually decline over time,[5] as is expected of a chronic disease with structural damage that increases over time. This was also observed on the group level in patients with hand OA after midterm follow-up of six to seven years.[13,23] However, on the individual level, the disease course can be rather heterogenous. Approximately 50% of patients reported a decrease in function, whereas the rest improved or remained stable.[23] Identification of specific patient groups experiencing different hand function trajectories over time and their determinants could guide information for patients about their prognosis and tailor management options. Therefore, this study aimed to investigate hand function trajectories in patients with hand OA over a five-year follow-up and to identify determinants associated with baseline hand function and hand function trajectories.

METHODS

Study population. Data from the observational Hand Osteoarthritis in Secondary Care (HOSTAS) cohort consisting of patients from the Leiden University Medical Center Rheumatology Outpatient Clinic were used. Between June 2009 and October 2015, 538 patients with primary hand OA as diagnosed by their treating rheumatologist were included. Full details on the cohort have been published previously.[24]

All patients were included at baseline. For the latent class growth analysis (LCGA), patients with baseline and five-year study visits with complete Australian/Canadian Osteoarthritis Hand Index (AUSCAN) function subscale were included (n = 301).

The HOSTAS study protocol was approved by the Medical Ethics Committee Leiden Den Haag Delft (NL26201.058.08). All patients provided written informed consent.

Assessment of hand function. The AUSCAN function questionnaire was completed annually. Higher scores indicate worse hand function.[25] The questionnaire contains nine items. Each question was scored on a 0 to 4 Likert scale (total score 0–36). We considered the minimal clinical important improvement (1.4) as clinically relevant change over time.[26]

Other clinical determinants. Sex, age, body mass index, educational level, marital status, 18 self-reported comorbidities with the Modified Charlson Comorbidity Index[27] and osteoporosis, symptom duration in years, and fulfillment of American College of Rheumatology (ACR) criteria for hand OA[28] were collected with standardized questionnaires. Hand pain was assessed with the AUSCAN pain subscale (range 0–20).[25]

The health-related quality of life (HRQoL) was measured with a Dutch translated short-form 36.[29] Mental component summary (MCS) and physical component summary (PCS) scales using norm-based scores from the Dutch population (mean ± SD 50 ± 10; higher scores indicate better quality of life) were calculated.[24,30]

The Hospital Anxiety and Depression Scale (HADS) subscales depression and anxiety (added in January 2011) were calculated (range 0–21 per subscale).[31] With the illness perception questionnaire (IPQ, added in January 2011) cognitive representations of illness were collected (identity [range 0–14], timeline [range 6–30], consequences [range 6–30], personal control [range 6–30], treatment control [range 5–25], illness coherence [range 5–25], cyclical timeline [range 4–20], and emotional representations [range 6–30]).[32]

The Coping with Rheumatic Stressors (CORS) questionnaire (added in January 2011) was used to assess the coping strategies with pain (comforting cognitions [range 9–36], decreasing activity [range 8–32], and diverting attention [range 8–32]), with limitations (optimism [range 5–20], pacing [range 10–40], and creative solutions [range 8–32]), and with dependency (accepting [range 6–24] and consideration [range 7–28]).[33]

Physical examination. Standardized physical examination of the distal interphalangeal (DIP), proximal interphalangeal (PIP), metacarpophalangeal (MCP), first interphalangeal (IP-1) and CMC-1 joints for bony swellings (range 0–30) and of the DIP, PIP, IP-1, first MCP, and CMC-1 joints for deformity (range 0–22) was performed by trained research nurses.

A Seahan pinch grip gauche was used for pinch grip strength, and a Seahan hand dynamometer was used for cylindrical grip strength; the average of two hands was calculated. Hand mobility was assessed by the Modified Kapandji Index (range 0–50; lower scores indicate worse mobility) and Hand Mobility in

Scleroderma Test (HAMIS) score (range 0–54; higher scores indicate worse mobility). Interobserver reliability of these mobility tests was good.[20]

Radiography. Dorso-volar radiographs of the hands were taken at baseline. Scoring of the DIP, PIP, IP-1, MCP, and CMC-1 joints was performed with the Kellgren-Lawrence (KL) Scoring Scale (range 0–120).[34] Furthermore, DIPs and PIPs were assessed with the Verbruggen-Veys anatomic phase score, in which erosive OA was defined as at least one joint having an "E" (eroded) or "R" (remodeled) phase.[35] Intraobserver reliability of both scores was good.[24]

Missing data. For the AUSCAN function subscale, one to two missing values were allowed and could be substituted by the average value for the subscale.[25] If more than two values were missing, the AUSCAN function questionnaire at that time point was considered missing. For the IPQ questionnaire, this was dependent on the specific domain (>2 missing components).[32] If more than one of the components of the HRQoL PCS and MCS were missing, the total scale was considered missing. Other summed scores were defined as missing when any component was missing.

Statistical analysis. A total of 538 patients was included at baseline. Baseline demographic and disease-specific characteristics were assessed using mean and SD for normally distributed data and median with interquartile range (IQR) for non-normally distributed data. Cross-sectional associations between determinants and baseline AUSCAN function were assessed with univariable and multivariable regression (adjusted for age, sex, AUSCAN hand pain, HAMIS, cylindrical grip strength, HADS depression, and the IPQ domains identity and consequences). The selection of included variables in this model was based on clinically known and statistical association to AUSCAN hand function at baseline.[20]

Patients included in the LCGA (n = 301) were compared to the patients without sufficient study data (n = 237) using unpaired Student's *t*-tests for normally distributed data and Mann-Whitney U tests for non-normally distributed data. A confidence interval (CI) of 95% ($P < 0.05$) was used. LCGA was used to determine subgroups within the cohort with a different AUSCAN function at baseline and AUSCAN function development over time.

Various LCGA models were run, with two to six classes (trajectories) and with linear ($y = ax + b$), quadratic ($y = ax^2 + b$), and cubic formulas ($y = ax^3 + b$). The y is the dependent (AUSCAN function), x is the time in years, *a* is the mean AUSCAN function change over time (slope), and *b* is the baseline AUSCAN function (intercept). In total, 18 LCGA models were fitted, consisting of linear, quadratic, and cubic models with one (reference model) to six classes. The best-fitted model was selected by the smallest Bayesian information criterion value, a high entropy value, a significant Vuong-Lo-Mendell-Rubin likelihood ratio test (LRT), and a significant bootstrap LRT. These LRTs predict whether the number of trajectories run in the model describe the study data better than the model with n – 1 trajectories ($P < 0.05$). Furthermore, ≥300 patients were required per model, and ≥50 patients were required per trajectory, according to best practice for the use of LCGA.[36]

As a sensitivity analysis, we performed an LCGA model with patients that had two or more study visits (n = 485), with an average follow-up time of 4.2 years (range 1–5 years). Average posterior probabilities of the trajectories (ie, the possibility of belonging to a trajectory) were evaluated. The posterior probabilities are a measure of trajectory stability. Values >0.80 indicate good classification quality.[37]

To determine the mean AUSCAN change over five years, we multiplied the slopes of the trajectories by five years. Potential trajectory-associated determinants were assessed with a multinomial regression model. Associations were expressed in odds ratios (ORs) with 95% CIs. The multinomial models were performed unadjusted and adjusted for baseline AUSCAN function. Utilized statistical programs were SPSS version 29.0 and MPlus version 8.11.

Data availability statement. No data are available. The data underlying this article cannot be shared publicly due to the protection of the privacy of individuals who participated in the HOSTAS study. Anonymized aspects of the data can be shared on reasonable request to the corresponding author.

RESULTS

Patient characteristics. Baseline characteristics of the 538 patients included at baseline are presented in Table 1. Mean AUSCAN function at baseline was 15.6 (SD 8.4 [range 0–36]). Of patients, 86.1% were women, with a mean age of 61.0 years (SD 8.6). A total of 90.1% of patients fulfilled the ACR hand OA criteria (Table 1).

Univariable and multivariable adjusted associations with baseline hand function. At baseline, female sex, age, symptom duration, at least one comorbidity, lower educational level, HAMIS score, AUSCAN pain score, HADS anxiety and depression scores, the IPQ domains identity, consequences, and emotional representations, and the CORS domains decreasing activity, pacing, creative solutions, accepting, and consideration were positively associated with worse hand function. The Modified Kapandji Index, pinch grip strength, cylindrical grip strength, the HRQoL MCS and PCS scales, and IPQ illness perception treatment control showed negative associations with hand function (Table 1).

In multivariable models (Table 2), the first model included age and sex, but associations did essentially not change (data not

Table 1. Baseline patient characteristics and associations with baseline hand function (N = 538)*

Patient characteristics	Value	Crude β (95% CI)
Demographics		
Female sex, n (%)	463 (86.1%)	4.34 (2.28–6.41)
Age, years, mean (SD)	61.0 (8.6)	0.08 (−0.00 to 0.17)
Symptom duration, years, median (IQR)	5 (2–12)	0.17 (0.86–0.25)
BMI, kg/m^2, mean (SD)	27.1 (4.8)	0.00 (−0.16 to 0.15)
Presence of comorbidity, n (%)	222 (42.8%)	2.45 (0.99–3.96)
Low educational level, n (%)	297 (61.2%)	1.89 (0.35–3.40)
Living together, n (%)	427 (79.4%)	1.54 (−0.31 to 3.39)
AUSCAN function, mean (SD)	15.6 (8.4)	
Hand mobility, median (IQR)		
Modified Kapandji Index score	44 (39–46)	−0.36 (−0.50 to −0.22)
HAMIS	4 (3–6)	0.56 (0.35–0.77)
Hand strength, kg/m^2, median (IQR)		
Pinch grip strength	3 (2–4)	−1.70 (−2.35 to −1.05)
Cylindrical grip strength	22 (17–28)	−0.37 (−0.44 to −0.30)
Structural damage		
Number of joints with bony swelling, median (IQR)	11 (7–15)	0.08 (−0.60 to 0.21)
Number of joints with deformity, median (IQR)	3 (1–6)	0.20 (−0.18 to 0.42)
KL-sum score, median (IQR)	17 (8–29)	0.04 (−0.01 to 0.82)
Erosive OA, n (%)	154 (28.6%)	1.51 (−0.09 to 3.12)
AUSCAN pain, mean (SD)	9.3 (4.3)	1.50 (1.39–1.61)
HRQoL SF-36, mean (SD)		
MCS	51.7 (8.7)	−0.22 (−0.30 to −0.14)
PCS	44.7 (8.2)	−0.58 (−0.65 to −0.50)
HADS (range 0–21), median (IQR)		
Depression	3 (2–4)	0.74 (0.49–0.99)
Anxiety	4 (2–7)	0.55 (0.31–0.78)
IPQ domains, median (IQR)		
Identity (range 0–14)	4.9 (2.2)	1.49 (1.15–1.84)
Timeline, chronic (range 6–30)	26.2 (3.6)	0.09 (−0.14 to 0.32)
Consequences (range 6–30)	16.5 (4.3)	0.67 (0.49–0.85)
Personal control (range 6–30)	18.6 (3.6)	−0.02 (−0.25 to 0.21)
Treatment control (range 5–25)	13.9 (2.7)	−0.26 (−0.57 to 0.05)
Illness coherence (range 5–25)	18.6 (3.8)	−0.26 (−0.47 to −0.04)
Timeline, cyclical (range 4–20)	13.2 (3.2)	−0.01 (−0.27 to 0.25)
Emotional representations (range 6–30)	14.4 (4.9)	0.39 (0.23–0.56)
CORS domains, median (IQR)		
Pain		
Comforting cognitions (range 9–36)	26 (24–30)	−0.13 (−0.30 to 0.05)
Decreasing activity (range 8–32)	16 (14–20)	0.60 (0.41–0.78)
Diverting attention (range 8–32)	18 (15–21)	0.20 (0.02–0.38)
Limitations		
Optimism (range 5–20)	16 (14–17)	−0.08 (−0.36 to 0.21)
Pacing (range 10–40)	24 (20–29)	0.39 (0.26–0.52)
Creative solutions (range 8–32)	20 (17–23)	0.39 (0.23–0.56)
Dependence		
Accepting (range 6–24)	13 (10–16)	0.41 (0.21–0.60)
Consideration (range 7–28)	20 (17–22)	0.34 (0.12–0.56)

* Missingness did not exceed 5%, except for working status (n = 471), symptom duration (n = 500), HADS (n = 382), IPQ (n = 383), and CORS (n = 327 to n = 380). HRQoL SF-36 norm-based scores with a mean of 50 and an SD of 10 using Dutch population norms. AUSCAN, Australian/Canadian Osteoarthritis Hand Index; BMI, body mass index; CI, confidence interval; CORS, Coping with Rheumatic Stressors questionnaire; HADS, Hospital Anxiety and Depression Scale; HAMIS, Hand Mobility in Scleroderma Test; HRQoL SF-36, health-related quality of life short-form 36; IPQ, illness perceptions questionnaire; IQR, interquartile range; KL, Kellgren-Lawrence; MCS, mental component score; OA, osteoarthritis; PCS, physical component score.

shown). Subsequently, we added hand pain in the model, and the associations with comorbidity, lower educational level, pinch grip strength, HADS anxiety, and the IPQ domain emotional regulation were lost. After further adjustment for disease signs, the associations with female sex, age, and symptom duration disappeared. After adjustment for HADS depression and the IPQ domains identity and consequences, no associations were attenuated. Only hand pain, the disease signs including hand mobility (HAMIS) and grip strength, and the IPQ domains identity and consequences remained associated with hand function at baseline.

Of the 538 patients included at baseline, 301 patients had a presence of both baseline and five-year follow-up study visit and

Table 2. Multivariable regression model for associations with baseline hand function*

Baseline	Adjusted β (95% CI) (adjusted for age, sex, and hand pain)	Adjusted β (95% CI) (adding HAMIS, and grip strength)	Adjusted β (95% CI) (adding HADS depression, IPQ identity, and IPQ consequences)
Demographics			
Female sex[a]	**2.87 (1.53–4.20)**	0.62 (−0.14 to 2.68)	
Age, years	**0.07 (0.02–0.12)**	−0.04 (−0.12 to 0.04)	
Symptom duration, years	0.05 (−0.00 to 0.12)		
Presence of comorbidity[a]	0.09 (−0.88 to 1.06)		
Low educational level[a]	0.26 (−0.72 to 1.24)		
Hand mobility			
HAMIS	**0.26 (0.10–0.41)**	**0.17 (0.02–0.33)**	**0.17 (0.01–0.31)**
Hand strength, kg/m^2			
Pinch grip strength	−0.50 (−0.93 to 0.07)		
Cylindrical grip strength	**−0.18 (−0.24 to −0.16)**	**−0.12 (−0.20 to −0.04)**	**−0.10 (−0.18 to −0.02)**
AUSCAN pain (range 0–20)	**1.47 (1.37–1.58)**	**1.33 (1.19–2.68)**	**1.20 (1.05–1.36)**
HADS			
Depression	**0.23 (0.06–0.40)**	**0.28 (0.08–0.48)**	0.10 (−0.09 to 0.29)
Anxiety	0.07 (−0.09 to 0.23)		
IPQ domains			
Identity	**0.48 (0.23–0.73)**	**0.41 (0.13–0.69)**	**0.32 (0.04–0.61)**
Consequences	**0.21 (0.08–0.35)**	**0.24 (0.11–0.38)**	**0.19 (0.05–0.34)**
Illness coherence	−0.01 (−0.16 to 0.13)		
Emotional representations	0.10 (−0.01 to 0.21)		

Note: Bold values indicate statistically significant associations.
* Missingness did not exceed 5%, except for HADS (n = 382) and IPQ (n = 383). AUSCAN, Australian/Canadian Osteoarthritis Hand Index; BMI, body mass index; CI, confidence interval; HADS, Hospital Anxiety and Depression Scale; HAMIS, Hand Mobility in Scleroderma Test; IPQ, illness perceptions questionnaire.
[a] Reference categories for binary variables: female sex (male sex is the reference), presence of comorbidity (absence of comorbidity is the reference), and lower educational level (having a higher educational level is the reference).

were included in the LCGA. All six annual AUSCAN function subscale time points were used in cases of availability. For 144 patients, all six annual AUSCAN function subscale visits were present. One measurement was missing for 64 patients, two for 68, and three for 23 patients.

The 301 patients included in the LCGA (Supplementary Table S1) were more often female (83.1% in the included population [n = 301] vs 89.9% in the excluded population [n = 237]), had better AUSCAN function scores, had better mental and physical quality of life, had fewer depressive symptoms, and differed on several illness perceptions compared to the patients who were lost to follow-up (n = 237; Supplementary Table S1). For the 301 patients, the average AUSCAN function score worsened over five years, from mean 14.2 to mean 15.3 (estimated mean change over 5 years 1.1 [95% CI 0.26–1.82]).

The LCGA with the four-class linear model (four hand dysfunction trajectories) appeared to be the best fit according to statistical and clinical criteria (100 iterations; 5,000 random starts; Figure 1, Supplementary Table S2) and resulted in a severe, moderate, mild, and minimal hand dysfunction trajectory. Average posterior trajectory probabilities of this model ranged from 0.93 to 0.96. Follow-up time was five years (range 2–5 years for all patients).

The severe hand dysfunction trajectory had a baseline AUSCAN function score of 23.99 (intercept, 95% CI 22.04–25.61) and slope of 0.36 (95% CI 0.03–0.69), which corresponds to a statistical and clinically relevant estimated AUSCAN function worsening of 1.82 over five years. The moderate hand dysfunction trajectory had a baseline AUSCAN function score of 16.87 (intercept, 95% CI 15.41–18.34) and a slope of 0.38 (95% CI 0.01–0.74), which corresponds to a statistically and clinically relevant mean estimated AUSCAN function worsening of 1.88 over five years. The mild hand dysfunction trajectory had a baseline AUSCAN function score of 11.17 (intercept, 95% CI 9.94–13.50) and a slope of –0.03 (95% CI –0.32 to 0.37), which corresponds to a nonstatistically and nonclinically relevant mean estimated AUSCAN function improvement of –0.14 over five years. The minimal hand dysfunction trajectory had a baseline AUSCAN function score of 5.05 (intercept, 95% CI 3.50–6.50) and slope of –0.19 (95% CI –0.47 to 0.08), which corresponds to a nonstatistically and nonclinically relevant mean estimated AUSCAN function improvement of –0.97 over five years. A total overview of intercepts and slopes for hand dysfunction trajectories can be found in Supplementary Table S3.

As a sensitivity analysis, we performed an LCGA model with the inclusion of 485 study patients (presence of ≥2 study visits). For this model, the model was also the best statistical and clinical fit (Supplementary Figure S1, Supplementary Table S4). Posterior class probabilities of the n = 485 model ranged from 0.91 to 0.95. The model showed comparable trajectories and AUSCAN hand function changes over time as our main model with 301 patients included.

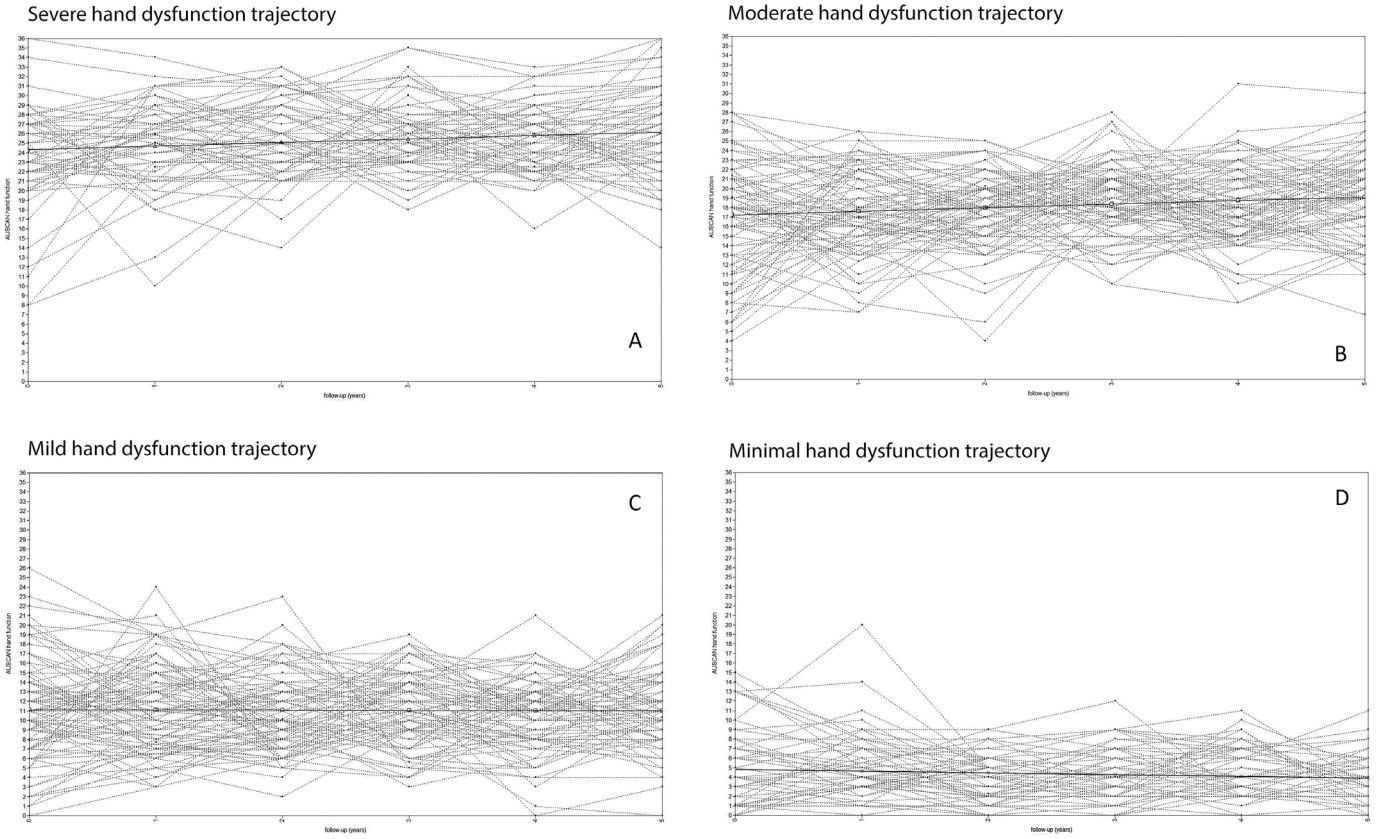

Figure 1. Latent class growth analysis of AUSCAN hand dysfunction trajectories over five years. On the y-axis, AUSCAN hand function score is depicted (range 0–36), and on the x-axis, follow-up time in years (0–5) is depicted. The linear model y = *a*x + *b* is applied, in which *a* is the estimated slope (AUSCAN hand function score change over time including 95% CIs) and *b* is the intercept (baseline AUSCAN function score including 95% CIs). For the severe hand dysfunction trajectory (A), the intercept is 23.99 (95% CI 22.04–25.61), the estimated slope is 0.36 (95% CI 0.01–0.74), and the estimated AUSCAN function score change over time is 1.82. For the moderate hand dysfunction trajectory (B), the intercept is 16.68 (95% CI 15.41–18.34), the estimated slope is 0.38 (95% CI 0.01–0.74), and the estimated AUSCAN function score change over time is 1.88. For the mild hand dysfunction trajectory (C), the intercept is 11.17 (95% CI 9.94–13.50), the estimated slope is –0.03 (95% CI –0.32 to 0.37), and the estimated AUSCAN function score change over time is –0.14. For the minimal hand dysfunction trajectory (D), the intercept is 5.05 (95% CI 3.50–6.50), the estimated slope is –0.19 (95% CI –0.47 to 0.08), and the estimated AUSCAN function score change over time is –0.97. Each trajectory shows the average trajectory of the group (solid line) and the individual hand function trajectories (dashed lines). AUSCAN, Australian/Canadian Osteoarthritis Hand Index; CI, confidence interval.

The severe hand dysfunction trajectory according to the model of the sensitivity analysis showed a baseline AUSCAN function score of 25.59 (intercept, 95% CI 24.44–26.76) and a slope of 0.12 (95% CI –0.15 to 0.39), which corresponds to a nonstatistically and nonclinically relevant estimated mean AUSCAN function worsening of 0.62 over five years. The moderate hand dysfunction trajectory worsened significantly with a baseline AUSCAN score of 17.51 (intercept, 95% CI 16.26–18.75) and a slope of 0.33 (95% CI 0.03–0.62), which corresponds to a statistically and clinically relevant estimated mean AUSCAN hand function worsening of 1.63 over five years. The mild hand dysfunction trajectory was stable with a baseline AUSCAN function score of 11.19 (intercept, 95% CI 9.92–12.51) and a slope of 0.00 (95% CI –0.28 to 0.28), which corresponds to a nonstatistically and nonclinically relevant estimated mean AUSCAN function change over time. The minimal hand dysfunction trajectory had a baseline AUSCAN function score of 5.33 (intercept) (95% CI 4.06–6.60) and a slope of –0.27 (95% CI –0.53 to –0.01), which corresponds to a statistically and clinically significant estimated mean AUSCAN function improvement of –1.36 over five years (Supplementary Table S4).

Determinants of hand dysfunction trajectories. The associated determinants per hand dysfunction trajectory are found in Table 3. Baseline characteristics of the four trajectories can be found in Supplementary Table S5. The minimal hand dysfunction trajectory was used as the reference category.

A reduced grip strength, a high AUSCAN pain score, a low HRQoL PCS, perception of the illness as chronic, and usage of the coping styles decreasing activity and creative solutions were positively associated with the mild hand dysfunction trajectory.

Table 3. Crude associations (ORs with 95% CIs) of determinants and outcomes of interest with hand dysfunction trajectories*

Baseline	Minimal (n = 59)	Mild (n = 89)	Moderate (n = 91)	Severe (n = 62)
Mean baseline AUSCAN function, mean (SD)	4.9 (4.3)	11.1 (5.4)	16.9 (5.6)	23.6 (5.2)
Demographics, OR (95% CI)				
Female sex	1	1.20 (0.53–2.69)	1.43 (0.63–3.30)	2.64 (0.93–7.49)
Age	1	0.98 (0.94–1.03)	1.00 (0.96–1.05)	1.02 (0.97–1.07)
Symptom duration	1	1.03 (0.98–1.08)	1.07 (1.02–1.12)	1.07 (1.02–1.13)
BMI	1	1.04 (0.96–1.12)	1.06 (0.99–1.15)	1.08 (0.99–1.17)
Presence of comorbidity	1	1.42 (0.83–2.41)	1.87 (1.12–3.13)	3.17 (1.87–5.38)
Lower educational level	1	1.53 (0.79–2.99)	1.39 (0.72–2.71)	2.04 (0.97–4.30)
Living together	1	0.61 (0.26–1.42)	0.76 (0.33–1.71)	0.85 (0.35–2.05)
Hand mobility, OR (95% CI)				
Modified Kapandji score	1	0.94 (0.85–1.05)	0.89 (0.80–0.99)	0.85 (0.77–0.95)
HAMIS	1	1.02 (0.90–1.16)	1.08 (0.96–1.23)	1.19 (1.05–1.36)
Hand strength, OR (95% CI)				
Pinch strength	1	0.75 (0.56–1.01)	0.68 (0.50–0.93)	0.63 (0.45–0.89)
Grip strength	1	0.96 (0.93–0.99)	0.94 (0.91–0.97)	0.89 (0.85–0.93)
Structural damage, OR (95% CI)				
Number of joints with bony swelling	1	0.99 (0.93–1.06)	1.01 (0.95–1.08)	1.08 (1.01–1.16)
Number of joints with deformity	1	1.04 (0.93–1.16)	1.12 (0.99–1.23)	1.16 (1.03–1.29)
KL-sum score	1	1.01 (0.98–1.03)	1.02 (0.99–1.04)	1.02 (1.00–1.04)
Erosive disease on imaging	1	1.90 (0.86–4.21)	2.60 (1.19–5.68)	2.40 (1.04–5.54)
AUSCAN pain	1	1.24 (1.12–1.37)	1.49 (1.32–1.67)	1.94 (1.67–2.25)
HRQoL SF-36, OR (95% CI)				
MCS	1	0.99 (0.95–1.05)	0.99 (0.95–1.04)	0.94 (0.89–0.98)
PCS	1	0.93 (0.88–0.99)	0.86 (0.81–0.91)	0.76 (0.71–0.82)
HADS, OR (95% CI)				
Depression	1	1.09 (0.91–1.32)	1.18 (0.99–1.41)	1.35 (1.13–1.61)
Anxiety	1	1.07 (0.91–1.25)	1.15 (0.98–1.34)	1.36 (1.16–1.59)
IPQ domains, OR (95% CI)				
Identity	1	1.23 (0.93–1.63)	1.80 (1.35–2.40)	2.23 (1.64–3.02)
Timeline, chronic	1	1.12 (1.01–1.25)	1.14 (1.03–1.28)	1.20 (1.06–1.36)
Consequences	1	1.07 (0.96, 1.19)	1.13 (1.02–1.26)	1.26 (1.12–1.42)
Personal control	1	0.99 (0.89–1.11)	0.97 (0.87–1.08)	0.96 (0.85–1.07)
Treatment control	1	0.88 (0.75–1.03)	0.73 (0.62–0.87)	0.85 (0.72–1.02)
Illness coherence	1	1.07 (0.97–1.19)	1.08 (0.98–1.20)	0.96 (0.87–1.07)
Timeline, cyclical	1	0.97 (0.86–1.09)	0.92 (0.86–1.10)	1.04 (0.91–1.18)
Emotional representations	1	1.01 (0.92–1.11)	1.04 (0.95–1.14)	1.13 (1.02–1.25)
CORS domains, OR (95% CI)				
Pain				
Comforting cognitions	1	1.01 (0.93–1.09)	0.99 (0.91–1.07)	0.98 (0.90–1.07)
Decreasing activity	1	1.13 (1.02–1.25)	1.13 (1.02–1.25)	1.24 (1.11–1.38)
Diverting attention	1	1.05 (0.97–1.15)	1.05 (0.97–1.14)	1.08 (0.99–1.19)
Limitations				
Optimism	1	0.98 (0.85–1.12)	0.97 (0.85–1.11)	0.96 (0.83–1.11)
Pacing	1	1.06 (0.98–1.13)	1.08 (1.00–1.15)	1.14 (1.06–1.24)
Creative solutions	1	1.09 (1.00–1.18)	1.12 (1.03–1.22)	1.13 (1.03–1.24)
Dependence				
Accepting	1	1.09 (0.99–1.21)	1.07 (0.96–1.19)	1.15 (1.03–1.29)
Consideration	1	1.06 (0.96–1.18)	1.10 (0.99–1.23)	1.13 (1.00–1.27)

* AUSCAN, Australian/Canadian Osteoarthritis Hand Index; BMI, body mass index; CI, confidence interval; CORS, Coping with Rheumatic Stressors questionnaire; HADS, Hospital Anxiety and Depression Scale; HAMIS, Hand Mobility in Scleroderma Test; HRQoL SF-36, health-related quality of life short-form 36; IPQ, illness perceptions questionnaire; KL, Kellgren-Lawrence; MCS, mental component score; OR, odds ratio; PCS, physical component score.

A longer symptom duration, at least one comorbidity, less hand mobility (a lower modified Kapandji score), less grip and pinch strength, erosive hand OA, a higher AUSCAN pain score, and a lower HRQoL PCS and attributing more complaints and consequences to the illness, perceiving the illness as chronic, perceiving less treatment control, and usage of the coping style decreasing activity, pacing and creative solutions were associated with the moderate hand dysfunction trajectory.

Female sex, longer symptom duration, at least one comorbidity, less hand mobility (a lower modified Kapandji score and a higher HAMIS score), lower grip and pinch strength, a higher degree of structural damage, the presence of erosive hand OA,

a higher AUSCAN pain score, a lower HRQoL, and several psychological factors and coping styles were associated with the severe hand dysfunction trajectory.

When adjusted for baseline function, most associations attenuated, except for the associations with symptom duration, number of joints with bony swelling or deformity, structural damage, and the illness perception of perceived chronicity and disease consequences (Supplementary Table S6).

Integration of both baseline and longitudinal associated determinants. Figure 2 shows that structural damage and the IPQ domain perceived chronicity of disease were solely associated with unfavorable five-year hand dysfunction trajectories. All other factors were associated with both baseline hand function and trajectories.

DISCUSSION

We investigated the course of hand function over five years including cross-sectional and longitudinal associated determinants with hand function. To our knowledge, we were the first to use LCGA to identify different hand function trajectories over time in patients with hand OA. We identified four distinct groups of patients, of which the severe and moderate trajectory showed a clinically and statistically relevant worsening over time on average.

Previous OA cohorts described a gradual loss of hand function over the short to midterm on the group level with large individual heterogeneity, as is in line with our results.[13,23,38] We further specified hand function over time into four different hand dysfunction trajectories.

In knee and hip OA, dysfunction trajectories have been investigated with group-based trajectory modeling. A study identified three dysfunction trajectories over a 10-year follow-up period, during which the low and high disability trajectories remained stable, but the moderate disability trajectory showed a slight worsening over time, and another study also showed deterioration of function for the groups with worse baseline functional limitation.[39,40] We now add for hand OA that there are different hand dysfunction trajectories that represent different patient groups and show both worsening and stable trajectories. This is especially relevant because hand joints are non–weight bearing osteoarthritic joints, in contrast to hip and knee OA. Furthermore, hand OA involves multiple joints with varying involvement, which is especially suitable for LCGA that can group individuals according to similar overall disease courses.

Trajectory-associated determinants fell into two categories: determinants that were solely cross-sectionally associated with

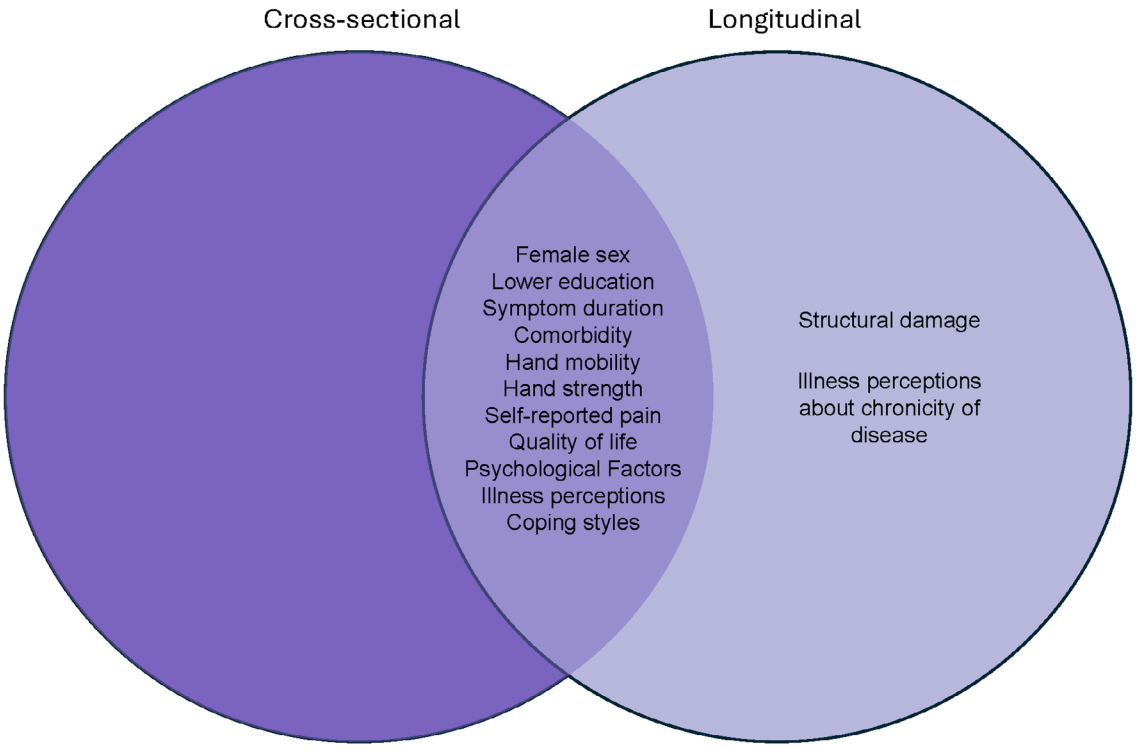

Figure 2. Cross-sectionally and longitudinally associated determinants with hand function. The darkest circle (left) shows determinants associated solely with hand function cross-sectionally. The medium light circle (middle) represents the determinants associated with hand function cross-sectionally and longitudinally with the worst AUSCAN dysfunction trajectories. The lightest circle (right) consists of the determinants solely associated with hand function longitudinally.

the trajectory of hand dysfunction and determinants that were associated with both cross-sectional and longitudinal hand function. Regarding longitudinal determinants, structural damage showed a dose-response relationship with hand dysfunction trajectories over time. Current literature does not report an association of structural damage with hand function cross-sectionally.[13,23] However, previous studies were inconclusive with regard to longitudinal associations of structural damage with hand function.[13]

In hip and knee OA, the association of a higher KL score with functional decline was also reported.[40] The different results between our study and earlier hand OA studies[13,23] could be explained by the fact that we used a different statistical method (LCGA) enabling the identification of different trajectory-based patient groups and were able to identify these patient groups by specific determinants, such as structural damage. The longitudinal association of hand function with structural damage fits with the concept of increased structural damage over time in patients with hand OA,[41] which can imaginably lead to functional limitations possibly mediated through both mechanical limitations and hand pain.[42] Erosive hand OA, in which structural damage, pain, and inflammation is present, was also positively associated with worse hand function over time in our cohort.

Hand pain and hand function remain closely intertwined, as reported previously,[5,8] which is reflected in our multivariable analysis at baseline in which hand pain emerged as an independent determinant of hand function. Pain has also been reported as an important associated factor for functional impairment in hip and knee OA.[43] Earlier, we showed stable hand pain trajectories over time.[8] One could hypothesize that increased hand pain might lead to a loss of hand function because patients avoid using their painful hand to minimize discomfort.[9,10]

Specific illness perceptions were associated with worse hand function over time. In our cohort, the illness perception chronicity of disease was only longitudinally associated with worse hand function. The illness perception chronicity of disease was previously reported in a two-year[44] and a six-year follow-up study as being associated with worsening of hand function over time.[45] The association could be explained by the hypothesis that patients who experience a greater loss of hand function over time perceive their disease more as chronic, as is suggested in these studies.

The perceived consequences of impaired hand function could be dependent on the used coping styles.[11] This also seemed to be reflected in our cohort.

Baseline hand function appeared to be the most important determinant of the LCGA trajectories, and univariate analysis revealed associations with demographic, disease, and psychosocial factors. However, in the multivariable analysis, only hand pain, grip strength, hand mobility, and the illness perceptions identity and consequences appeared to be independently associated with baseline hand function. Regarding cross-sectionally associated determinants, decreasing activity, pacing, creative solutions, accepting, and consideration were associated with having a worse cross-sectional and longitudinal hand function in our cohort. We previously reported these CORS domains to be cross-sectionally associated, with decreasing activity and pacing also longitudinally associated over a one-year follow-up period.[11] We add to this that the coping styles creative solutions, accepting, and consideration also influence hand function over five years of follow-up. The coping styles decreasing activity, pacing, and creative solutions are in line with expectations about utilized coping mechanisms regarding functional limitation of the hands.

Our study has certain limitations. The patients in our cohort showed a clinically and statistically significant better AUSCAN function in contrast to the baseline cohort.

The loss to follow-up might have attenuated the observed trajectories in an unknown direction. The loss to follow-up of patients with more severe hand function impairment could have skewed the observed trajectories for the moderate and severe hand dysfunction trajectories, for which a slope closer to zero is present because the observed effect of a worsening hand function over time is diluted. Imputation could not be performed because missing data were nonrandom. However, for future direction, joint modeling approaches could be used because they are suitable for data such as ours, which is missing not-at-random, under the assumption of an informative dropout process.

We performed a sensitivity analysis, running an LCGA model with 485 patients (≥2 study visits present). This model achieved comparable results and demonstrated a comparable good statistical performance. The most severe hand dysfunction trajectories showed a deterioration over time, whereas the mild hand dysfunction trajectory showed a stabilized trajectory, and the minimal hand dysfunction trajectory even showed an improvement in hand function. Remarkably, there were no signs of regression to the mean. We chose the model with 301 patients because this model ensured optimal data density and the most representative trajectories.

Another limitation of this study is that our patient population predominantly consists of women. The amount of men per hand dysfunction trajectory is sparse and could therefore hamper the association of female sex with longitudinal hand function in reaching statistical significance.

The last limitation of this study is generalizability, in two ways. First, the discontinuation of patients with the worst hand function poses a challenge in the generalizability of the results to patients with more severe disease courses. Second, the study population consists of patients referred through an outpatient rheumatology clinic, which implies that they might have presented themselves with significant complaints and may have been further along in the disease course, which limits the applicability of the findings to the broader population of individuals with hand OA, especially those in primary care.

In conclusion, we identified four groups of patients with different hand dysfunction trajectories over time, in which the moderate and severe groups significantly deteriorated and the mild and minimal groups remained stable and slightly improved. These specific patient groups were characterized by demographic, disease, and psychosocial determinants. Our findings can be used to inform patients with hand OA about the prognosis of their hand function. Furthermore, we believe hand function is an equally important outcome parameter as hand pain in clinical trials and should be considered in future trial design. The identification of determinants that are associated with hand function over time is important because they may be modifiable. Psychological determinants, illness perceptions, and certain coping styles have the potential to be additional treatment targets to improve hand function over time.

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AUTHOR CONTRIBUTIONS

All authors contributed to at least one of the following manuscript preparation roles: conceptualization AND/OR methodology, software, investigation, formal analysis, data curation, visualization, and validation AND drafting or reviewing/editing the final draft. As corresponding author, Dr Olde Meule confirms that all authors have provided the final approval of the version to be published and takes responsibility for the affirmations regarding article submission (eg, not under consideration by another journal), the integrity of the data presented, and the statements regarding compliance with institutional review board/Declaration of Helsinki requirements.

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