number of tender joints at palpation was 3/30 (IQR [2; 6]). Results of models for humidity are presented in Table 1. There was an association between humidity percentage and AUSCAN pain ≥ 20 for third quartile vs. first quartile (OR [79-85%] vs. [43-68%] = 1.99 (1.08 to 3.68), p = 0.03) and between humidity and VAS pain during activity ≥ 42 for second quartile vs. first quartile (OR [68-79%] vs. [43-68%] = 1.97 (1.06 to 3.63) p = 0.03). Considering patients with at least 1 spontaneous hand joint pain, an association was found for all 3 higher quartiles of humidity ([68-79%], [79-85%] and [85-96%]) compared to the lower one ([43-68%]), but without dose-effect. There was no effect of humidity on AUSCAN stiffness or function, or number of tender joints at palpation. Temperature was not associated with any clinical outcomes (pain, function or stiffness).

Conclusion: This is the first study about impact of meteorological factors in HOA. While we did not find any association between temperature and symptoms, we found an association between humidity and pain. Although we cannot conclude on any causality between humidity and pain, our results support patient's belief about influence of humidity on joint pain.

REFERENCE:

[1] Timmermans EJ, J Rheumatol 2015;42:1885–92.

Table 1: Association between humidity and HOA symptoms: logistic regression models with systematic adjustment on sex, age, sum of Kellgren-Lawrence score for all hand joint and HAD score.

	OR (IC 95%)	p-value
AUSCAN-pain subscore ≥ 20		
Humidity (%)		0.14
1. [43 ; 68[	1	
2. [68 ; 79[	1.76 (0.96;3.25)	0.07
3. [79 ; 85[	1.99 (1.08;3.68)	0.03
4. [85 ; 96[	1.52 (0.83;2.79)	0.18
At least 1 spontaneous tender joint		
Humidity (%)		0.02
1. [43 ; 68[	1	
2. [68 ; 79[	1.74 (0.94;3.21)	0.08
3. [79 ; 85[	2.67 (1.44;4.94)	0.002
4. [85 ; 96[	1.95 (1.06;3.61)	0.03
VAS-scale for pain during activity ≥ 42		
Humidity (%)		0.1832
1. [43 ; 68[	1	
2. [68 ; 79[	1.97 (1.06;3.63)	0.03
3. [79 ; 85[	1.58 (0.86;2.91)	0.14
4. [85 ; 96[	1.56 (0.85;2.86)	0.15

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Disclosure of Interests: None Declared.
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POS1361 THE INFLUENCE OF BASELINE DEMOGRAPHICS ON VARIABILITY OF OA PAIN ASSESSED BY WOMAC PAIN CHANGE FROM BASELINE IN INTERVENTIONAL TRIALS

Keywords: Biomarkers, Osteoarthritis, Randomized control trial

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Background: Development of new products for OA treatment is difficult, partly due to excessive placebo response and high variability in patient-reported pain outcomes, and exclusion of trial subjects with potentially confounding pain from non-target joints may increase the effect size. This may in part be due to reductions in variability, and hence in standard deviation (SD) of the mean changes in pain scores, which improves the statistical power to detect differences between study groups. The impact of refining the study population in terms of added benefit to the SD and statistical power vs. added screen failures is poorly described.

Objectives: To investigate the impact of common demographic and pain characteristics on the SD of mean pain change from baseline in a large interventional OA trial database.

Methods: Data from 2 randomized controlled trials of oral salmon calcitonin in OA (1) were analyzed post-hoc. The SD of the mean change from baseline (SD-CFB) to Year 2 in the WOMAC knee pain score (0-100) was calculated. The SD-CFB was also calculated for subgroups (e.g. demographics, pain of the target and non-target knee). The sample size required to identify a statistically significant difference of at least 8 out of 100 between groups (with 80% power) was calculated, and the additional proportion of eligible subjects to be screen-failed if the particular subgroup was excluded was calculated to quantify the potential impact on the study feasibility.

Results: A total of 1,487 subjects had WOMAC pain data throughout the trial period. Results are shown in Table 1. Few clinical characteristics influenced the SD and hence the required sample size, except for Asian race, associated with a lower SD compared to Caucasians (19.71 vs. 21.85), to detect a significant difference. Exclusion of those with high BMI (≥ 35 kg/m^2) led to a reduced sample size required of 113. Subjects with non-target knee pain above the median had higher pain variability and thus a larger sample sizes required to detect differences vs. those with pain scores below the median. However, exclusion of these groups required screen failing approx. extra 50 % of the eligible population to achieve this. A less exclusive method was excluding subjects with non-target knee pain not exceeding that of the target knee. Radiographic characteristics did not influence the SD, except for those with a KL grade 3 or 4 of the non-target knee (Table 1). This observation requires further scrutiny.

Table 1.

	SD of mean WOMAC pain change	n	Sample size per group to detect mean WOMAC pain difference of 8 out of 100 (n)	Proportion of eligible subjects screen failed if subgroup was excluded (%)
All study completers	21.58	1,487	115	0
Male	21.12	516	110	34.7
Female	21.82	971	117	65.3
Age < 64 yrs	21.49	799	114	53.7
Age ≥ 64 yrs	21.68	688	116	46.3
BMI < 35 kg/m^2	21.45	1,316	113	88.5
BMI ≥ 35 kg/m^2	22.53	171	125	11.5
Subgroups by **Non-Target knee characteristics** at Baseline				
KL grade 0	20.79	60	107	4.0
KL grade I	21.19	271	111	18.2
KL grade II	20.83	766	107	51.5
KL grade III	23.80	346	139	23.3
KL grade IV	17.55	42	76	2.8
WOMAC Pain ≤ 35 (0-100)	20.08	731	99	49.2
WOMAC Pain > 35 (0-100)	22.96	722	130	48.6
Low VAS (0-100, ≤ 37)	19.39	714	93	48.0
High VAS (0-100, > 37)	23.54	722	136	48.6
Nontarget knee WOMAC pain ≥ Target knee WOMAC pain	22.64	408	126	27.4
Nontarget knee WOMAC pain < Target knee WOMAC pain	20.84	1,024	107	68.9

Conclusion: High BMI and high baseline pain of the non-target knee may contribute negatively to the study power, however, the added study cost in terms of additional screened subjects required to replace those excluded based on these parameters should be carefully evaluated. The potential impact of these parameters on the magnitude of the difference between study groups was not evaluated and may also differ, with additional statistical implications.

REFERENCE:

[1] Karsdal MA, Byrjalsen I, Alexandersen P, Bihlet A, Andersen JR, Riis BJ, Bay-Jensen AC, Christiansen C. Treatment of symptomatic knee osteoarthritis with oral salmon calcitonin: results from two phase 3 trials. CSMC021C2301/2 investigators. Osteoarthritis Cartilage 2015;23:532-543.

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POS1362 THE OA TRIAL BANK: UPDATE OF INDIVIDUAL PATIENT DATA META-ANALYSIS OF INTRA-ARTICULAR GLUCOCORTICOIDS IN PERSONS WITH KNEE AND HIP OSTEOARTHRITIS

Keywords: Osteoarthritis

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Background: Intra-articular (IA) glucocorticoid injections are often employed in the management of osteoarthritis (OA) patients, but in whom to best target treatment is unclear. OA clinical guidelines have advocated for the identification of predictors of response to different treatments, but no reliable subgroups with treatment effect have been found.

Objectives: This study aims to conduct an individual patient data (IPD) meta-analysis to evaluate the efficacy of IA glucocorticoid for knee or hip OA in specific subgroups of patients according to the baseline pain severity and inflammatory signs. This is an update on an IPD meta-analysis on IA glucocorticoid by the OA Trial Bank [1].

Methods: Randomised trials evaluating one or more IA glucocorticoid preparations in hip and knee OA, published from 2012 to May 2018, were selected from the literature. IPD of participant and disease characteristics and outcome measures were acquired. The primary outcome was pain severity at short-term follow-up (around 4 weeks). A two-stage IPD analysis was performed. Potential interaction effect of baseline severe pain (≥70 points, 0-100 scale) and signs of inflammation were studied using linear mixed-effects models. Analysis of trend was conducted, assessing if a baseline pain cut-off was associated with the threshold for clinically important treatment effect of IA glucocorticoid compared to placebo.

Results: Four out of 16 eligible randomised clinical trials (n=641) were combined with the existing OA Trial Bank studies (n=620), yielding n=1261 from eleven studies. Participants with severe baseline pain compared to those with less severe pain had greater pain reduction at mid-term (around 12 weeks) (mean reduction: -6.90 (95%CI -10.91; -2.90)), but not at short- or long-term follow-up. No interaction effects were found between inflammatory signs and IA glucocorticoid injections compared to placebo at all follow-up time-points (Table 1). Analysis of trend showed treatment response to IA glucocorticoid injections from baseline pain levels >50 (0-100 scale) and above (Figure 1).

Conclusion: This IPD meta-analysis demonstrated that in participants with severe baseline pain, clinically relevant response is seen at mid-term follow-up, suggesting that sustained response to IA glucocorticoid injection may be seen in a subgroup of OA participants. As baseline pain score increases, treatment response is seen in participants from moderate pain levels onwards. No concrete conclusions can be made on baseline inflammatory signs. Ongoing IPD studies with an increased number of studies are required.

REFERENCE:

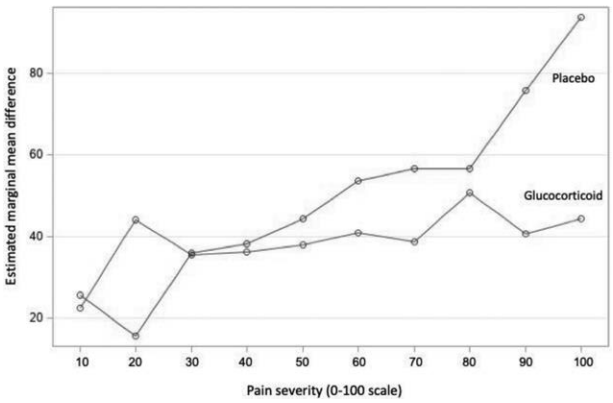
[1] van Middelkoop M, Arden NK, Atchia I, U, et al. The OA Trial Bank: meta-analysis of individual patient data from knee and hip osteoarthritis trials show that patients with severe pain exhibit greater benefit from intra-articular glucocorticoids. Osteoarthritis Cartilage 2016; 24: 1143-1152.

Table 1: Interaction effects of severe pain (≥70 points) and inflammation with IA glucocorticoid for primary outcome of pain severity

	No. of studies	No. of participants	Adjusted mean reduction (β) (95%CI)	*P*	Cochran's Q	I^2 (%)	Tau^2
Interaction effect of severe pain with IA glucocorticoid#							
IA glucocorticoid versus placebo							
Short-term	6	345	-5.98 (-18.28; 6.31)	0.266	4.47	0	0
Mid-term	5	270	**-6.90 (-10.91; -2.90)**	**0.009**	0.17	0	0
Long term	3	160	-2.84 (-62.69; 57.01)	0.857	2.88	30.5	0
IA glucocorticoid versus hyaluronic acid							
Short-term	3	513	7.50 (-56.58; 71.57)	0.665	9.41	78.8	508.88
Mid-term	3	476	6.90 (-3.39; 17.20)	0.102	0.39	0	0
Long term	2	456	8.42 (-28.03; 44.86)	0.209	0.27	0	0
Interaction effect of inflammation with IA glucocorticoid*							
IA glucocorticoid versus placebo							
Short-term	5	296	-15.01 (-51.23; 21.21)	0.314	21.83	81.7	688.97
Mid-term	4	230	-5.96 (-35.81; 23.91)	0.571	5.95	49.6	172.64
Long term	2	126	-7.43 (-125.49; 110.63)	0.571	0.64	0	0
IA glucocorticoid versus hyaluronic acid							
Short-term	2	360	**-10.82 (-20.30; -1.33)**	**0.044**	0.02	0	0
Mid-term	2	334	-9.22 (-70.10; 51.662)	0.305	0.62	0	0

#Adjusted for age and sex, *Adjusted for age, sex and baseline pain

Figure 1: Comparison of marginal means of pain scores (grouped) of IA glucocorticoid compared to placebo at short-term follow-up



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POS1363

CIRCULATING GALECTIN-1 LEVELS IN INDIVIDUALS WITH KNEE AND/OR HAND OSTEOARTHRITIS – A HALLOA STUDY

Keywords: Biomarkers, Osteoarthritis

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Background: Circulating galectin-1 levels are associated with metabolic diseases including obesity, diabetes type II, and subclinical inflammation [1]. These medical conditions are often seen as comorbidities of osteoarthritis (OA) in the knees and hands.

Objectives: The aim was to study longitudinal associations between circulating galectin-1 levels and radiographic knee OA (RKOA), radiographic hand OA (RHOA), and general OA (GOA, RKOA and RHOA).

Methods: This longitudinal study included 232 individuals from the Halland osteoarthritis cohort (HALLOA) (2) with knee radiographs at inclusion and at a two-year follow-up. Of the included individuals, 211 had hand radiographs at the two-year follow-up. Body mass index (BMI), and waist circumference (WC) were measured. Visceral fat area (VFA) was assessed by bioimpedance (InBody 770). Serum/plasma levels of HbA1c, glucose, and C-reactive protein (CRP)≥1.0 mg/L were measured according to the current laboratory standards in Sweden. CRP below 1.0 mg/L, was further analysed with a sensitive CRP ELISA method (Abnova). Insulin resistance was assessed by the triglyceride glucose index (TyG). Galectin-1, IL-1 beta, IL-6, and TNF alfa using Quantikine, ELISA (biotechne, United Kingdom). RKOA was defined according to Ahlbäck, as grade 1 or more in at least one knee at two years follow-up; RHOA was defined according