allele (p=0.03) compared to FDRs without the *0405 allele. Being positive for the SE was not significantly associated with sICAM, sVCAM, or E-selectin levels.
Conclusions: In RA-free FDRs, having an HLA-DRB1*0404 or HLA-DRB1*0405 allele was associated with markers of endothelial injury. Therefore, the genetic predisposition to RA could contribute to parallel development of atherosclerosis during the preclinical period of RA.
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FRI0707 DRUG LEVELS AND ANTIDRUG ANTIBODIES IN THE DEVELOPMENT OF PARADOXICAL PSORIASIS AND PALMOPLANTAR PUSTULOSIS

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Background: The pathogenesis of psoriasis and palmoplantar pustulosis induced by Tumor Necrosis Factor inhibitors (TNFi) is largely unknown. Only one study, in 9 inflammatory bowel disease patients, investigated the relation with infliximab drug levels in the development of psoriasis or palmoplantar pustulosis and demonstrated no relation with of trough concentrations in these events (1). However, psoriasis and palmoplantar pustulosis were not studied separately.
Objectives: To study the differences in drug levels and antidrug antibodies (ADA) of TNFi in rheumatoid arthritis (RA) and ankylosing spondylitis (AS) patients who developed de novo psoriasis, palmoplantar pustulosis and those who did not develop skin adverse events.
Methods: In this retrospective study data was collected from the observational cohorts of Reade of consecutive RA and AS patients in whom TNFi was started. At every visit, serum samples were collected. We quantified the samples before/on time of event of the patients who developed psoriasis or palmoplantar pustulosis, for the patients with no skin adverse events (control group) at 24 or 28 weeks. Drug levels and ADA were measured with an Enzyme-linked immunosorbent assay and antibody binding test respectively.
Results: A total of 830 TNFi naive patients with RA and AS were included, of whom 21 developed psoriasis (n=11) or palmoplantar pustulosis (n=10). These patients were only observed in the adalimumab and etanercept cohorts. Sixteen patients with an event and 585 patients in the control group had serum samples available to quantify drug levels and ADA. No statistical significant differences were found in drug levels of adalimumab and etanercept for both RA and AS patients (table 1). Moreover, no statistical significant differences were observed in the detection of ADA between the three groups. However, no ADA were detected in patients who developed psoriasis or palmoplantar pustulosis compared to the overall 13.9% of the RA patients and 25.5% in AS patients.

Table 1. Differences in drug levels and detection of anti-drug antibodies between palmoplantar pustulosis, psoriasis and control group

		Druglevels adalimumab (μg/ml; median (IQR)		Drug levels etanercept (μg/ml); median (IQR)		Anti-drug antibodies adalimumab, no. (%)
RA						
Palmoplantar pustulosis	n=3	7,6 (0,01–12,0)	n=1	4,4	n=3	0 (0)
Psoriasis	n=3	8,5 (6,5–10,0)	n=2	2,3 (1,5–3,1)	n=2	0 (0)
Control group	n=153	7,4 (4,0–10,0)	n=89	2.7 (1.9–3.9)	n=151	21 (13,9)
p-value		0,992		0,380		0,674
AS						
Palmoplantar pustulosis	n=3	9,0 (6,5–10,0)	n=1	1,7	n=3	0 (0)
Psoriasis	n=1	10, 0	n=2	1,3 (0,8–1,7)	n=1	0 (0)
Control group	n=46	8,5 (3,7–11,3)	n=99	2.6 (1.4–4.0)	n=47	12 (25,5)
p-value		0,754		0,406		0,754

RA: rheumatoid arthritis; AS: ankylosing spondylitis; Control group: patients who did not develop skin adverse events; IQR: interquartile range; no. number of patients. p-value <0,05 was considered statistically significant.

Conclusions: Patients who develop paradoxical psoriasis and palmoplantar pustulosis have adequate drug levels and no ADA were detected.
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FRI0708 NEUROPATHIC PAIN IS A WEAK PREDICTOR OF NEW ONSET CHRONIC WIDESPREAD PAIN

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Background: Regional pain (e.g. back pain) predicts incident chronic widespread pain (CWP), the clinical hallmark of fibromyalgia. Up to 20% of patients with CWP have neuropathic pain (NP). People with CWP and NP report similar pain characteristics including allodynia (pain in response to normal touch), have common risk factors (age, sex, body mass index, smoking and socioeconomic status) and a shared genetic predisposition. Whether NP is a risk factor for CWP is not known.
Objectives: To test the hypothesis that among persons free of CWP, NP would increase the risk of developing CWP.
Methods: In a population based study participant's pain reports were coded and those free of CWP (ACR criteria: pain lasting ≥3 months in the axial skeleton and contralateral body quadrants) identified. Participants also completed the Douleur Neuropathique 4 (DN4) (which has 7 sensory descriptors of pain (burning, painful cold, electric shocks, tingling, pins and needles, itching, and numbness), scores ≥3 indicating NP); demographics (date of birth, sex, English Index of Multiple Deprivation, occupational status); Hospital Anxiety and Depression (HAD) scale; Estimation of Sleep Problem Scale (ESPS); self-reported pain medications (summed to give a total count); and signed consent. Participants were classified as no pain, having some pain that wasn't neuropathic (NP-; DN4 score <3), or neuropathic pain (NP+; DN4 score ≥3). A follow-up questionnaire mailed 12 months later gathered pain data using the methods in the baseline survey. Based upon their pain reports at follow up participants were classified as "new CWP" for those who reported pain that satisfied the criteria for CWP, or "not CWP". Logistic regression estimated the odds of developing new CWP in the NP-, and NP+ groups compared to the no pain group. Results were expressed as odds ratios (OR) with 95% confidence intervals (CI). Population attributable fractions (PAF) estimated the % of new CWP that would be avoided if participants were not exposed to NP- or NP+.
Results: A total of 1162 participants who were free of CWP, completed the DN4 and provided pain data at follow up. Of those 523 (45.0%) had no pain at baseline, 562 (48.4%) had NP- and 77 (6.6%) had NP+. New onset CWP was reported by 153 (13.2%) participants; 19 (3.6%) of the no pain group, 108 (19.2%) of the NP- group, and 26 (33.8%) of the NP+ group. After adjusting for age and sex, compared to the no pain group, the NP- group was 3 times (OR 2.9, 95% CI (2.0, 4.2)) and the NP+ group 4 times (3.9 (2.3, 6.4)) more likely to have new CWP at follow up. These relationships were attenuated but persisted after adjustments for demographics, HAD, ESPS and medication use (NP- (2.9 (1.9, 4.3); NP+ (2.1 (1.1, 4.0)). The PAF was 41.3% (95% CI (25.2, 54.0)) for NP- and 6.0% (0.1, 11.6) for NP+. All of the individual DN4 characteristics except painful cold and itching predicted new CWP with PAF's ranging from 1.6% (0.1, 3.8) for pins and needles to 5.0% (1.1, 8.8) for burning.
Conclusions: NP predicts a small number of new onset CWP cases. CWP is highly prevalent in the general population and effective treatment of pain not of NP origin will have a significant impact on population levels of CWP.
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FRI0709 PREVALENCE OF RHEUMATIC DISEASES BASED ON COPCOPRD STUDIES: A SYSTEMATIC REVIEW

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Background: Despite many efforts, to date there has been no focused attempt to derive a robust estimate of the prevalence of rheumatic diseases (RDs) to quantify how this is influenced by other factors than them examined in every local study, however, the problems magnitude is rising and due the demographic transition and the increase in the life expectancy
Objectives: To determine, through a systematic review and meta-analysis, the prevalence of RDs in the adult general population and explore its heterogeneity
Methods: MEDLINE, EMBASE, BIREME, LILLACS and Web of Science were searched using a search strategy combining key words and related database-specific subject terms to identify relevant cross-sectional based on COPCORD methodology studies. Also was developed a manual search. Included articles were assessed for risk of bias and quality based on the STROBE statement. Prevalence figures for RDs were analyzed according to female percentage of sampled individuals, mean age and sample size. A mixed effect model was used to obtain the combined prevalence and a meta-regression to estimate the effects of other variables
Results: 44 out from 127 papers were included in English, Spanish or Portuguese. Estimates for any RDs prevalence ranged from 7.2% to 62.3% (26.9%; 95% CI 18.3%>25.6%). For rheumatoid arthritis (RA), the prevalence varied between 0.2% and 6.2% (1.04%; 95% CI 0.4%>1.6%); fibromyalgia (FM) had a mean prevalence of 2.1% (95% CI 1.0%>3.2%) and osteoarthritis: 13.5% (95% CI: 10.6%>16.4%). SLE (systematic lupus erythematosus) was the less frequent condition with average prevalence of 0.14% (95% CI: 0.005%>0.28%). The random-effects pooled prevalence for any RDs was 25% (95% CI: 18.0%, 31.1%). Prevalence was higher in studies with bigger sample size (random effect coefficient: 0.0014, p=002). There was evidence of relevant heterogeneity in the analysis (p<0,001) and for RDs, RA and FM the sample size was positively associated to the perceived heterogeneity. No effects were found for SLE
Conclusions: It was found significant variation among the prevalence across this