In SpA pts with lower activity by SPARCC score significant correlation between Dkk-1 and inflammatory lesions in SIJ (r=0.400, p=0.043) and a negative correlation between Dkk-1 and BASDAI (r=-0.513, p=0.017) were found.
There was no correlation between Scl and Dkk-1 levels among all pts and different subgroups.
Conclusions: Scl level is significantly higher in pts with lower disease activity by SPARCC MRI SIJ score and ASDAS-CRP. Dkk-1 significantly positively correlates with disease activity due to CRP level and SPARCC score, but not to BASDAI.
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SAT0405 CLINICAL, BIOLOGICAL AND GENETIC FACTORS, PREDICTORS OF TREATMENT NONRESPONSE TO TNF INHIBITORS (TNFI), IN ANKYLOSING SPONDYLITIS (AS) AND PSORIATIC ARTHRITIS (PSA)

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Background: TNF inhibitors (TNFi), are effective in controlling the activity of spondyloarthritis. But, there is a proportion of patients, who have to stop treatment due to its ineffectiveness or to the appearance of adverse events. In addition, these therapies imply high economic costs. To identify predictors of response, would help us to make decisions and to improve the risk/benefit ratio, in patients candidates who are candidates to initiate TNFi
Objectives: To determine clinical, biological and genetic predictors of nonresponse to treatment with TNFi in patients with AS and PsA.
Methods: We analyzed 118 patients [49 AS and 69 PsA (24 axial and peripheral involvement and 45 only peripheral)], under treatment or who were to start treatment with TNFi. Data were collected, prior to the start of the TNFi and at the last scheduled visit to the Rheumatology Service of the Hospital Puerta de Hierro, during the period 2013–2014. A clinical response was defined as the reduction ≥50% of the initial BASDAI, in patient with axial involvement, and if the final DAS 28 PCR was <2.6, in those patients with only peripheral involvement. A total of 73 men and 45 woman, mean age 53±11.2 years, and a median duration of illness of 15 years (IQR 10–23) were included. The baseline ESR and CRP were (10mm/hr IQR 5.0–27.0 and 2mg/l IQR 0.0–9.0) respectively. The mean and SD of BASDAI, DAS28 CPR and BASFI were (6.0±1.9, 3.0±0.6 and 5.4±2.5) respectively. A univariate analysis was performed using a Cox proportional hazard regression model which included: Smoker status, axial pain, peripheral arthritis, sacroiliitis, IBD, uveitis, psoriasis, HLA B27, VSG, PCR, BASDAI, BASFI, VGP, the number of TNFi and 45 single nucleotide polymorphism (SNPs) previously reported to be associated with response to TNFi. SNP genotyping was performed using de Sequenom MassARRAY plataform.Variables with a *P*-value <0.1 were included in a multivariate analysis. The discrimination capacity of the model was assessed using the Harrell C index. *P*-values <0.05 were considered statistically significant. Statistical analysis was performed with the SPSS v.17 software.
Results: The median duration of treatment was 62.9 months (IQR 40.7–96.5), the response to TNFi was 79.7% of patients, with mean and SD of BASDAI, BASFI and DAS 28 PCR (2.7±2.2, 4.2±2.8, 1.5±0.6) respectively. The factors that increased the non-response rate, were: the group of peripheral PsA versus AS (HR 2.94, *P*=0.023), VGP (HR 1.47, *P*<0.001), BASDAI (HR 1.80, *P*=0.001), BASFI (HR 1.52, *P*=0.001) and the number of TNFi used (*P*<0.001). There was a trend of significance (*P* <0.10) for females, with a 2.13-fold lower response rate than males *(P*=0.065). The SNPs associated were: rs4240847 of the *MAPKAPK2* gene (allele A, HR 1.63, *P*=0.019), rs11096957 of the *TLR-10* gene (T allele, HR 1.49, *P*=0.011), rs11541076 of the *IRAK-3* (allele T, HR 1.47, *P*=0.050), rs916344 of the *MAPK14* gene, in a recessive form,since CC alleles against CG or GG increased 10.12 times the non-response rate (HR 10,12; *P*=0.027) and rs11591741 of the *CHUK* gene (GG+GC/CC; HR 8.3, *P*=0.035).The mutivariable analysis is shown in the following table:

Table. Multivariate analysis and factors independently associated with the nonresponse rate

Variable	*P*-value	Hazard ratio	95% IC HR	
			Lower	Upper
Sex (Female/Male)	0,007	4,46	1,49	13,35
BASFI at the start of treatment	0.000	1,75	1,31	2,35
rs11591741 (GG/GC o CC)	0,022	3,83	1,21	12,12

Discriminatory capacity of the model, Harrell Index C 0.92

Conclusions: Female gender, basal BASFI elevated and SNP rs11591741 (GG) of *CHUCK* gene were identified as predictors of nonresponse to TNFi treatment in these patients.
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SAT0406 PREVALENCE OF VERTEBRAL FRACTURES IN AXIAL SPONDYLOARTHRITIS. A SYSTEMATIC REVIEW OF OBSERVATIONAL STUDIES

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Background: Some studies have described a higher rate of osteoporosis in axial spondyloarthritis (AxSpA). However, there are still some doubts about whether vertebral fractures (VF) should be a concern in AxSpA patients
Objectives: To evaluate the prevalence and incidence of VF in AxSpA
Methods: A systematic review was performed in Medline, Embase and Cochrane Library databases limited to studies published from Jan/2006 to Dec/2015) in Spanish, Italian and English. Search strategy combined synomyms of AxSpA, fractures, plus a filter study type. We selected cross-sectional or longitudinal studies estimating the prevalence and/or incidence of VF in adult AxSpA patients
Results: The search retrieved 3944 references which after screening by title and abstract ended in 90 studies to study in depth. Finally, 12 estudies were included. The majority of the studies evaluated the VF prevalence, and only 2 studies evaluated the incidence of VF. Prevalence estimates depended on VF definitions, varying between 4.1% (clinically diagnosed VF) and 32.4% (morphometric fracture by Genant definition). Table 1 shows all studies included and their data
Conclusions: The published studies that focus on VF in AxSpA are very heterogeneous, but in general showed a slight increase in the VF prevalence. More studies are need focused on VF incidence in AxSpA
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Abstract SAT0406 – Table 1

Author, year	Country/ies	Design	N	Population	Primary objective	Vertebral fracture	Incidence or Prevalence
Kang 2014	South Korea	Prospective	298	AS	VF incidence	Clinically diagnosed VF	4.1% at 2 years 13.6% at 4 years
Robinson 2013	Sweden	Cross-sectional (survey)	17764	AS	VF prevalence	Clinically diagnosed VF	4.1%
Feldtkeller, 2006	Germany Holand Belgium	Retrospective survey	1080	AS	VF prevalence and incidence	Clinically diagnosed VF	5.7% (4.3% after trauma)
Jun 2006	South Korea	Cross-sectional	68	AS	VF prevalence	Genant	16.2%
Klingberg 2013	Sweden	Cross-sectional	69	AS	VF prevalence	Genant	12%
Montala 2011	Spain	Cross-sectional	176	AS	VF prevalence	Genant	32.4% (25.5–39.3%)
Rossini 2015	Italy	Cross-sectional	71 AS 71 healthy	AS	VF and relation with bone remodeling	Genant	29%
Van der Weijden 2012	Holand	Cross-sectional	113 AxSpa 80 AS	EspAx	VF prevalence	Genant	15%
Mitra 2000	England	Cross-sectional	66 AS 39 healthy	AS (Males)	VF prevalence and relation with BMD	Mc Closkey	16.7%
Mehmet 2007	Turkey	Cross-sectional	59 AS 40 healthy	AS	VF prevalence	Tourissot	31%
Ulu 2013	Turkey	Cross-sectional	86 AS 50 Healthy	AS	VF prevalence	Tourissot	28%
Ulu 2014	Turkey	Cross-sectional Case y controls	59	AS	VF prevalence	Tourissot	32.5%