ACTUALIZACION EN AIJ 5.1
VALENCIA 13 MAYO 2016

FACTORES PREDICTIVOS DE RECAIDA

Pablo Mesa del Castillo Bermejo

Hospital Clínico Universitario Virgen de la Arrixaca.

ARTRITIS IDIOPATICA JUVENIL

DIAGNOSTICO

I.L.A.R.

HLA B27
FACTOR REUMATOIDE
Nº ARTICULACIONES
CLINICA
HISTORIA FAMILIAR
(AC. ANTINUCLEARES)

	ACR	EULAR (1977)	ILAR (1993)	ILAR-PRINTO
CATEGORIAS	3	6	7	?
EDAD DE INICIO	<16	<16	<16	?
DURACIÓN ARTRITIS	6 sem	3 meses	6 sem	?
INCLUYE ESPONDILITIS	No	Si	Si	?
INCLUYE ARTRITIS PSORIASICA	No	Si	Si	?
INCLUYE ENFERMEDAD INFLAMATORIA INTESTINAL	No	Si	Si	?
EXCLUSION DE OTRAS ENFERMEDADES	Si	Si	Si	?

TRATAMIENTO

SISTEMICA
OLIGO
POLI FR –
POLI FR +
PSORIASICA
ENTESITICA
INDIFERENCIADA

AINES
CORTICOIDES LOCALES
CORTICOIDES SISTEMICOS
F.A.R.A.L
BIOLOGICOS

RESPUESTA

ACTIVIDAD

INACTIVIDAD

RECAIDA

REMISION

FACTORES PREDICTIVOS DE RECAIDA

FACTORES CLÍNICOS

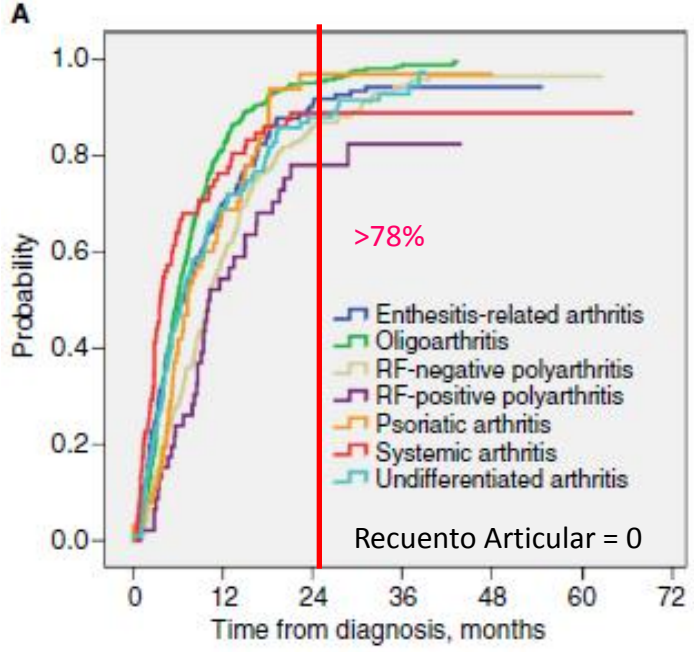
EXTENDED REPORT

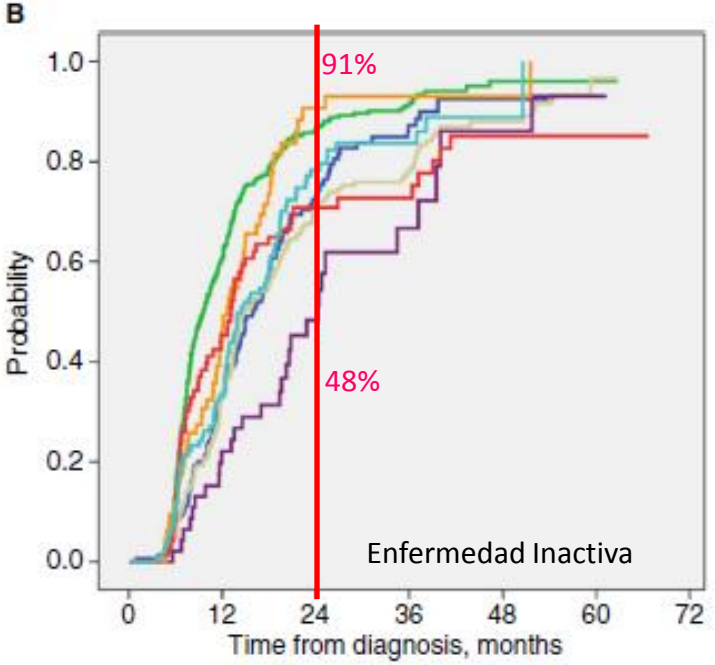
The outcomes of juvenile idiopathic arthritis in children managed with contemporary treatments: results from the ReACCh-Out cohort

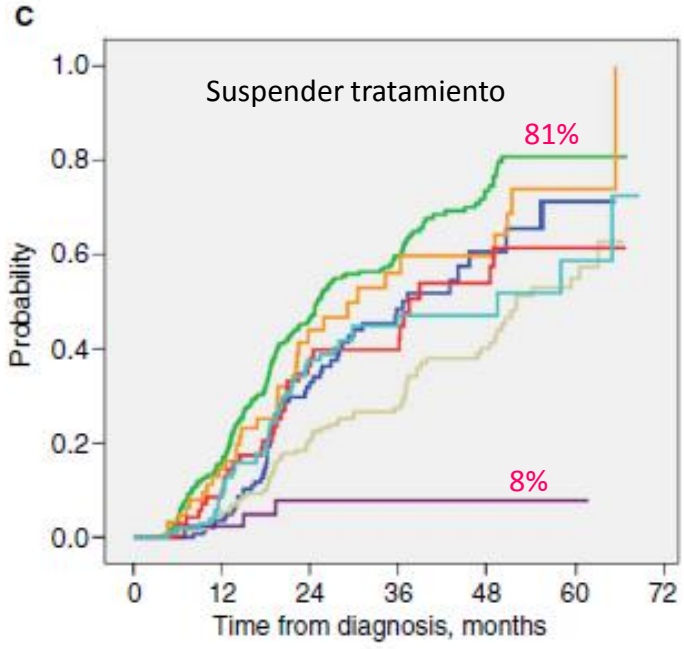
Ann Rheum Dis 2015;**74**:1854–1860. doi:10.1136/annrheumdis-2014-205372

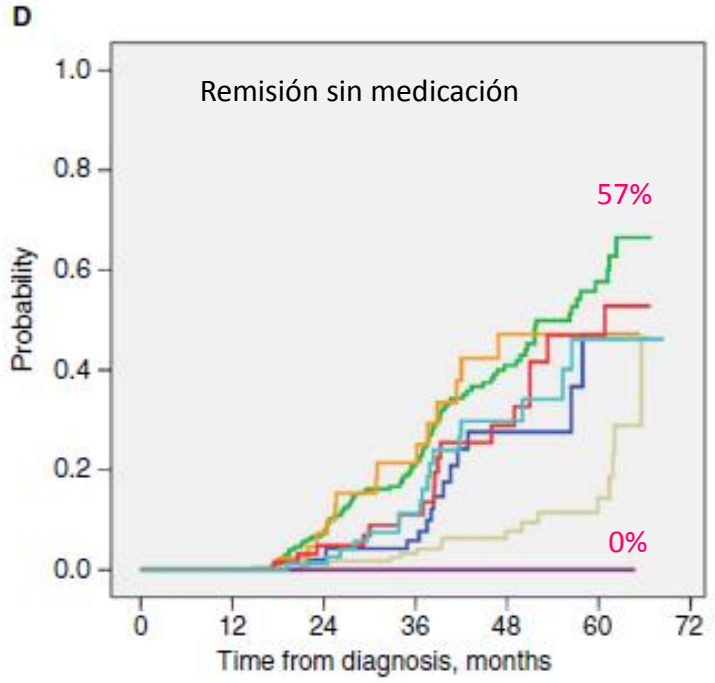
Jaime Guzman,[1] Kiem Oen,[2] Adam M Huber,[3] Karen Watanabe Duffy,[4] Gilles Boire,[5] Natalie Shiff,[6] Roberta A Berard,[7] Deborah M Levy,[8] Elizabeth Stringer,[3] Rosie Scuccimarri,[9] Kimberly Morishita,[1] Nicole Johnson,[10] David A Cabral,[1] Alan M Rosenberg,[6] Maggie Larché,[11] Paul Dancey,[12] Ross E Petty,[1] Ronald M Laxer,[8] Earl Silverman,[8] Paivi Miettunen,[10] Anne-Laure Chetaille,[13] Elie Haddad,[14] Kristin Houghton,[1] Lynn Spiegel,[8] Stuart E Turvey,[1] Heinrike Schmeling,[10] Bianca Lang,[3] Janet Ellsworth,[15] Suzanne E Ramsey,[3] Alessandra Bruns,[5] Johannes Roth,[4] Sarah Campillo,[9] Susanne Benseler,[10] Gaëlle Chédeville,[9] Rayfel Schneider,[8] Shirley M L Tse,[8] Roxana Bolaria,[16] Katherine Gross,[16] Brian Feldman,[8] Debbie Feldman,[17] Bonnie Cameron,[8] Roman Jurencak,[4] Jean Dorval,[13] Claire LeBlanc,[9] Claire St. Cyr,[14] Michele Gibbon,[4] Rae S M Yeung,[8] Ciarán M Duffy,[4] Lori B Tucker,[1] for the ReACCh-Out investigators

- Articulaciones activas = 0
- No sintomas sistémicos
- No entesitis
- No uveitis activa
- PGA < 10 mm

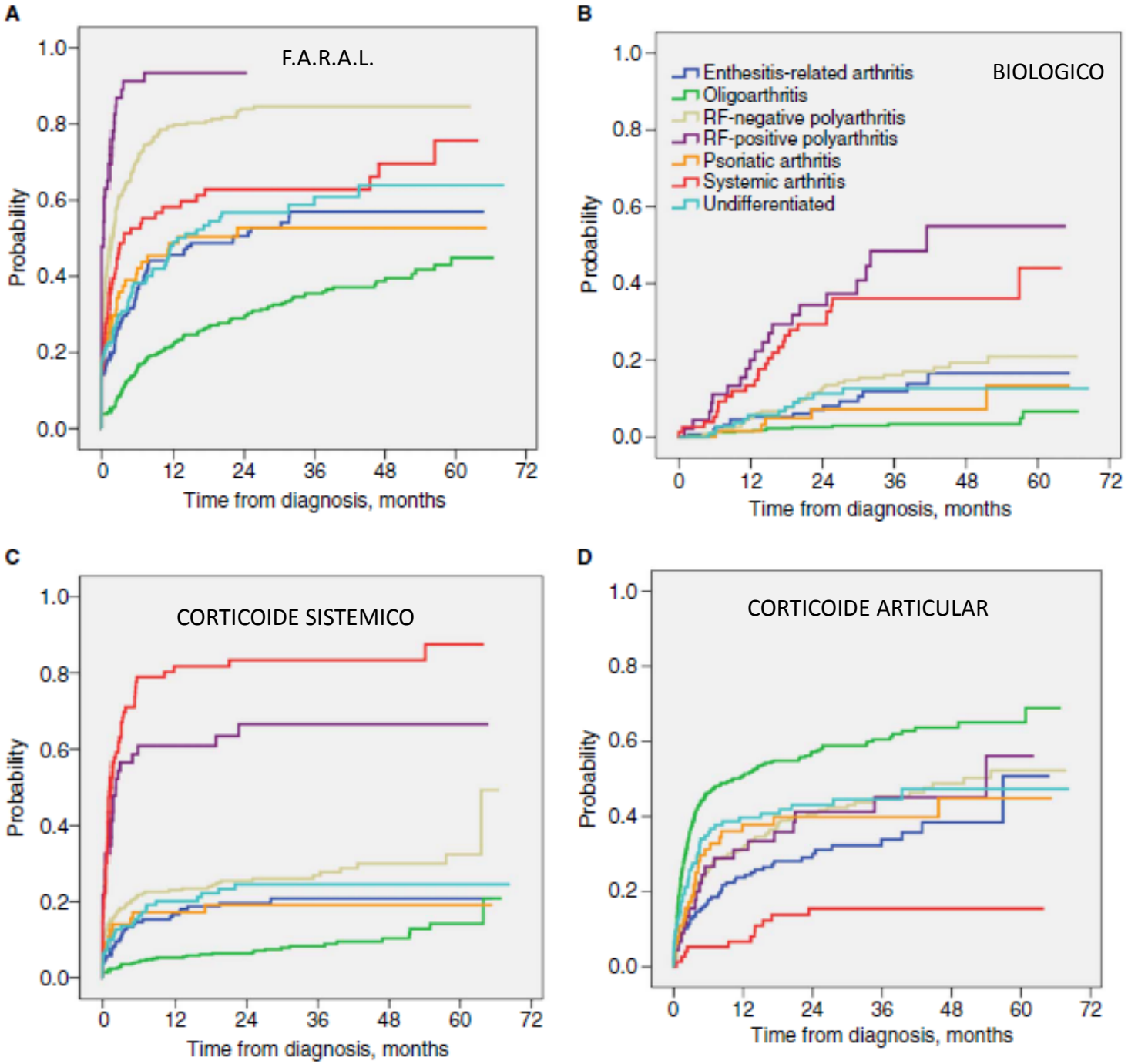








Guzman J, *et al*. *Ann Rheum Dis* 2015;**74**:1854–1860. doi:10.1136/annrheumdis-2014-205372



Guzman J, *et al*. *Ann Rheum Dis* 2015;**74**:1854–1860. doi:10.1136/annrheumdis-2014-205372

EXTENDED REPORT

The risk and nature of flares in juvenile idiopathic arthritis: results from the ReACCh-Out cohort

Ann Rheum Dis 2015;**0**:1–7. doi:10.1136/annrheumdis-2014-207164

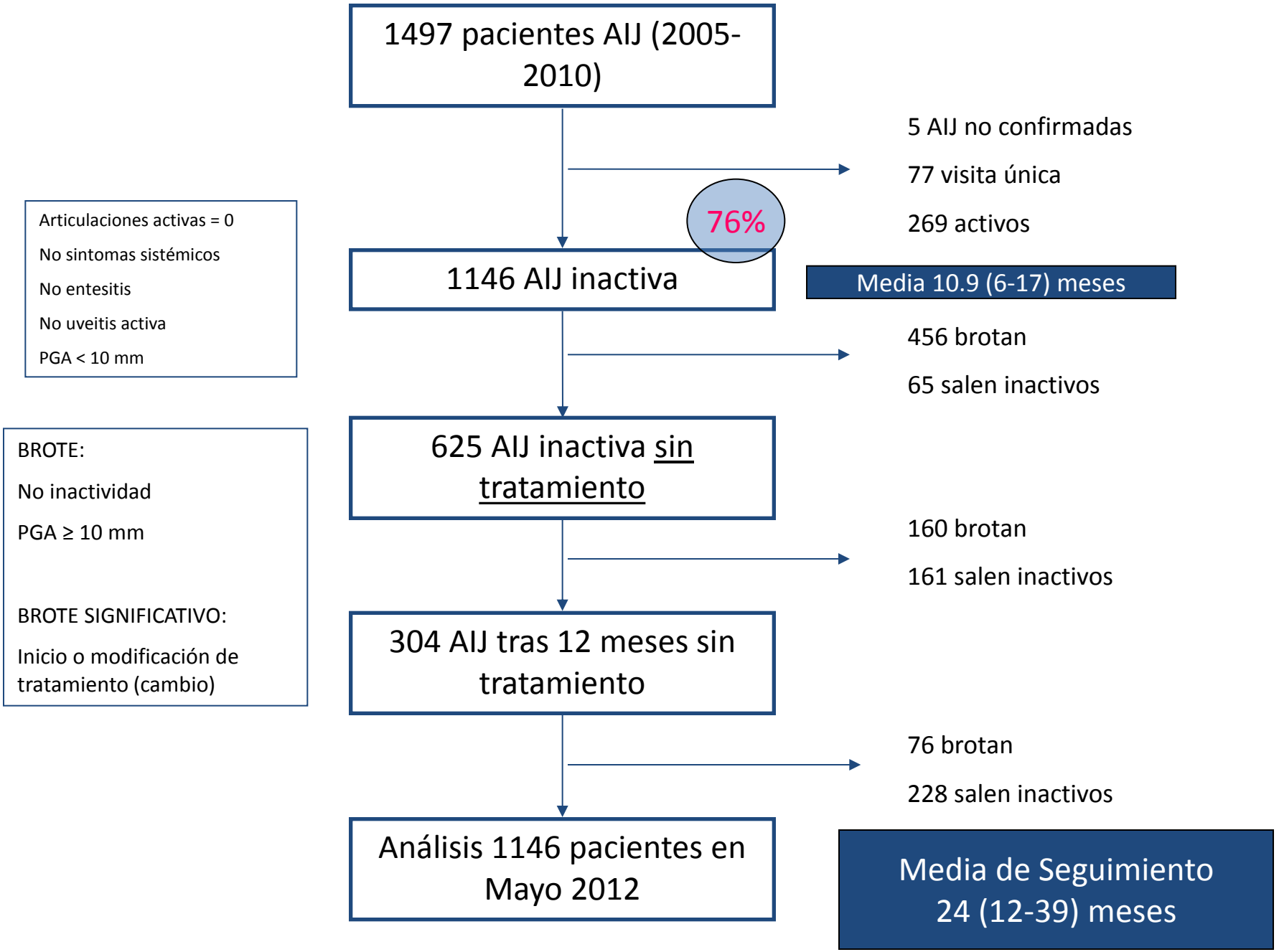
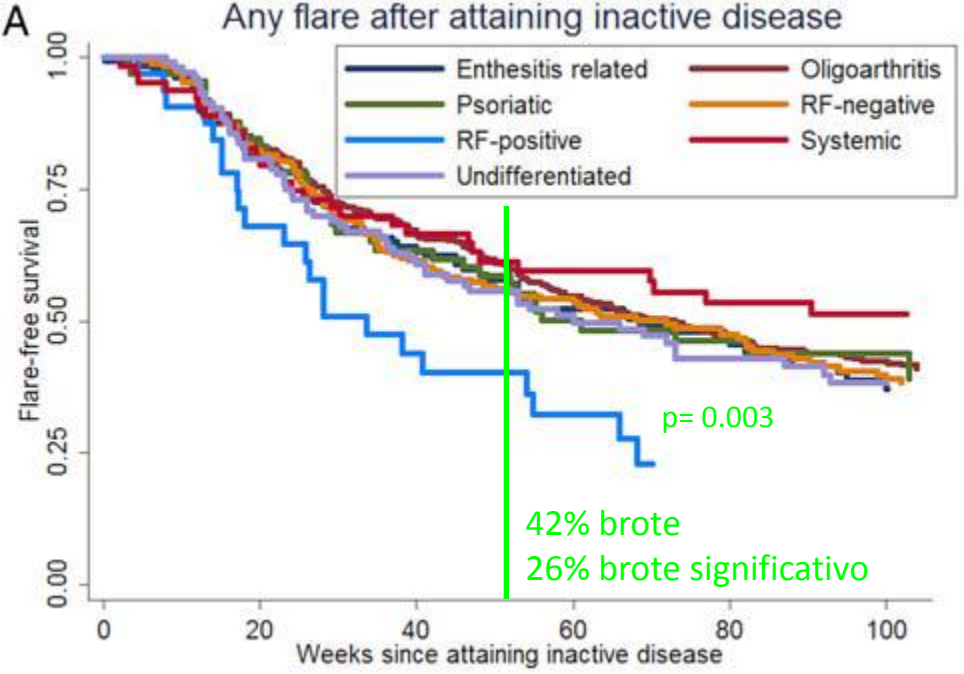
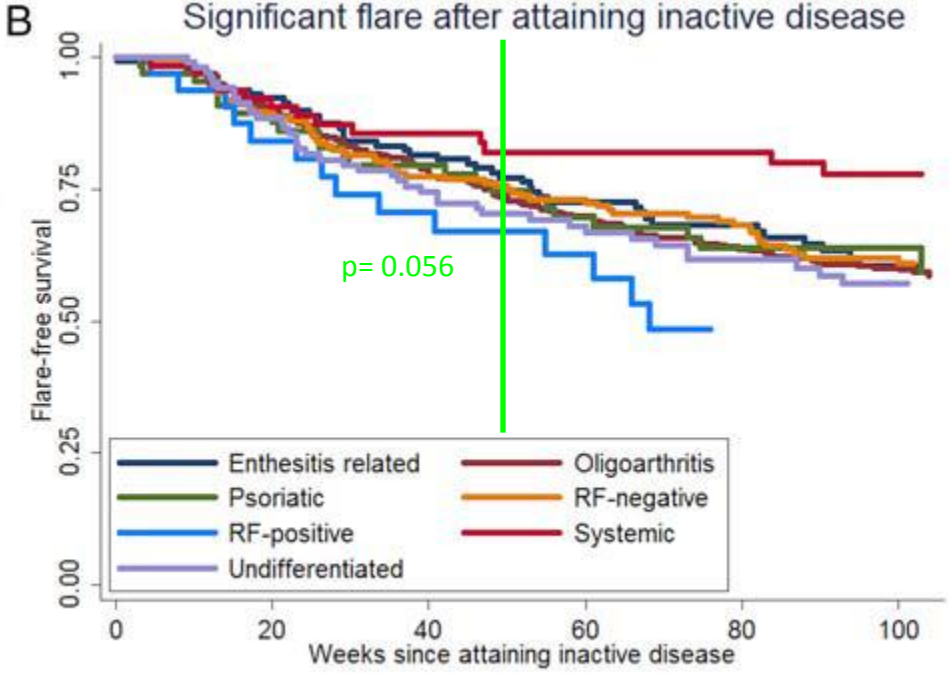

1497 pacientes AIJ (2005-2010)
5 AIJ no confirmadas
77 visita única
269 activos
76%
Articulaciones activas = 0
No sintomas sistémicos
No entesitis
No uveitis activa
PGA < 10 mm
1146 AIJ inactiva
Media 10.9 (6-17) meses
456 brotan
65 salen inactivos
BROTE:
No inactividad
PGA ≥ 10 mm
BROTE SIGNIFICATIVO:
Inicio o modificación de tratamiento (cambio)
625 AIJ inactiva sin tratamiento
160 brotan
161 salen inactivos
304 AIJ tras 12 meses sin tratamiento
76 brotan
228 salen inactivos
Análisis 1146 pacientes en Mayo 2012
Media de Seguimiento 24 (12-39) meses

Table 1 Cumulative probabilities of flare within 6, 12 and 24 months after attaining a state of inactive disease for different JIA categories

		Probability of flare after attaining inactive disease (95% CI)					
		Within 6 months		Within 12 months		Within 24 months	
	N*	Any flare	Significant flare	Any flare	Significant flare	Any flare	Significant flare
Whole cohort	1146	0.254 (0.229 to 0.282)	0.154 (0.133 to 0.178)	0.425 (0.394 to 0.456)	0.266 (0.240 to 0.295)	0.603 (0.570 to 0.636)	0.400 (0.368 to 0.434)
Systemic arthritis	68	0.268 (0.176 to 0.396)	0.127 (0.065 to 0.238)	0.387 (0.278 to 0.520)	0.179 (0.103 to 0.300)	0.486 (0.366 to 0.621)	0.222 (0.135 to 0.354)
Oligoarthritis persistent	457	0.226 (0.189 to 0.269	0.143 (0.113 to 180)	0.402 (0.356 to 0.451)	0.279 (0.238 to 0.325)	0.594 (0.542 to 0.646)	0.412 (0.362 to 0.466)
Oligoarthritis extended	31	0.337 (0.192 to 0.549)	0.226 (0.108 to 0.435)	0.415 (0.255 to 0.624)	0.269 (0.138 to 0.484)	0.537 (0.343 to 0.757)	0.467 (0.268 to 0.717)
RF-negative polyarthritis	212	0.251 (0.196 to 0.319)	0.163 (0.117 to 0.224)	0.445 (0.376 to 0.521)	0.263 (0.205 to 0.334)	0.618 (0.542 to 0.694)	0.390 (0.318 to 0.471)
RF-positive polyarthritis	35	0.389 (0.242 to 0.583)	0.192 (0.091 to 0.379)	0.598 (0.429 to 0.772)	0.329 (0.192 to 0.526)	0.770 (0.595 to 0.908)	0.515 (0.338 to 0.719)
Psoriatic arthritis	75	0.266 (0.174 to 0.393)	0.171 (0.098 to 0.287)	0.432 (0.319 to 0.564)	0.253 (0.163 to 0.380)	0.609 (0.483 to 0.738)	0.408 (0.292 to 0.549)
ERA	155	0.259 (0.192 to 0.345)	0.126 (0.079 to 0.197)	0.429 (0.347 to 0.522)	0.237 (0.171 to 0.323)	0.628 (0.532 to 0.725)	0.394 (0.306 to 0.497)
Undifferentiated arthritis	113	0.290 (0.213 to 0.388)	0.194 (0.129 to 0.284)	0.444 (0.353 to 0.547)	0.297 (0.218 to 0.397)	0.617 (0.515 to 0.720)	0.429 (0.333 to 0.540)

*Number of subjects who attained inactive disease.
ERA, enthesitis-related arthritis; JIA, juvenile idiopathic arthritis; RF, rheumatoid factor.





1er BROTE

627 (57%) pacientes brotaron

- 82% con artritis
- 18% sin artritis

PGA>10 mm (58)
Uveítis activa (21)
Entesitis (25)
Síntomas sistémicos (5)

401 (35%) 1 brote
151 (13.2%) 2 brotes
75 (6.5%) > 2 brotes

Varía entre categorías AIJ
53% vs 95.8% (p>0.001)

60.3% de los brotes fueron significativos:

- AINEs 41%
- Infiltración HT 19.8%
- F.A.R.A.L 17.7%

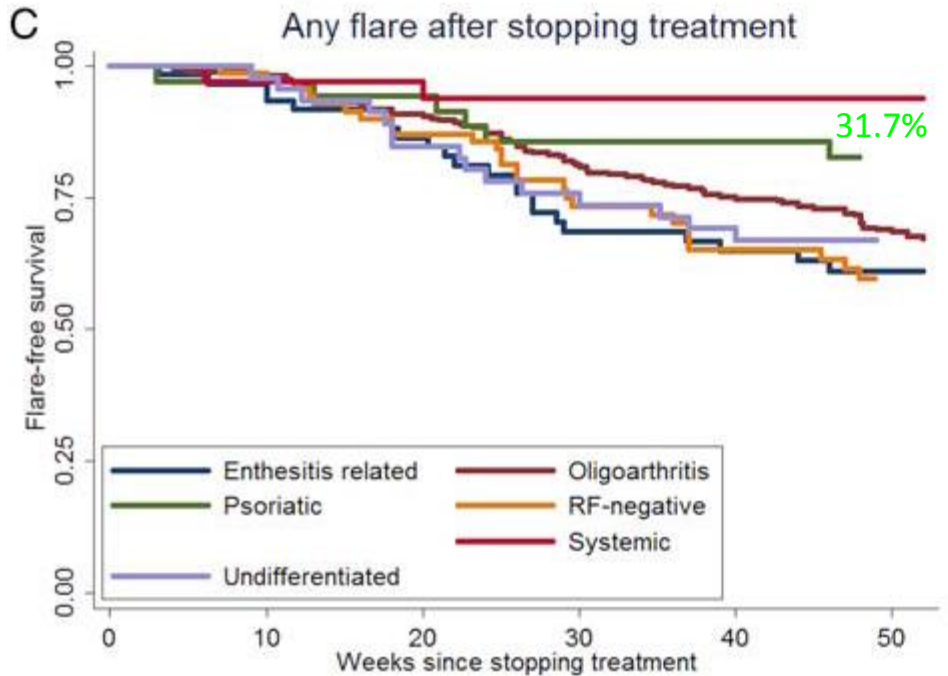
AIJ Poliarticular FR +:

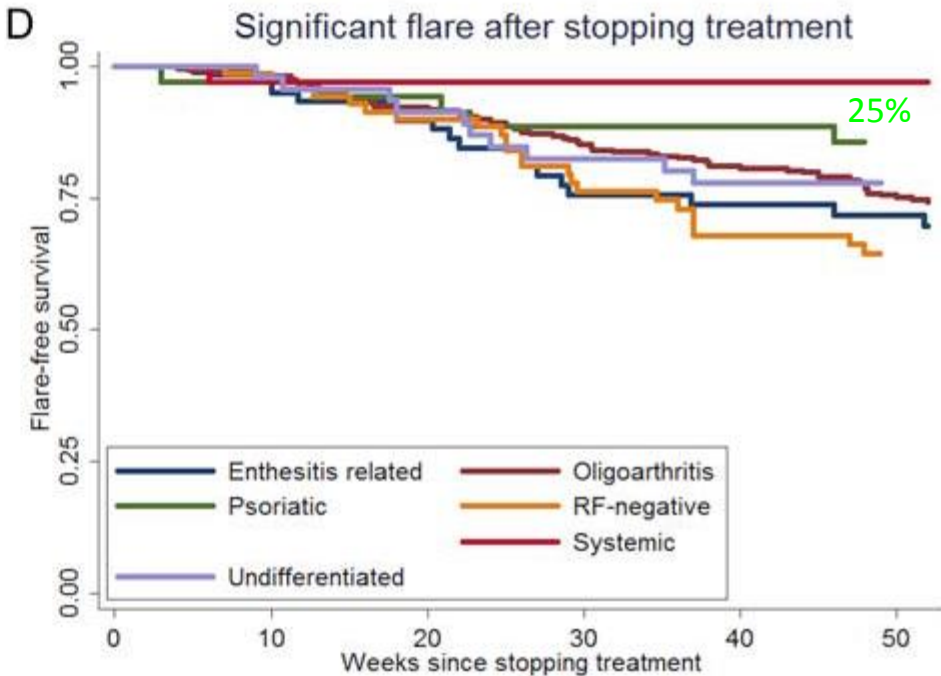
- Corticoides sistémicos (19%)$^{p=0.07}$
- Tratamiento biológico (20%)$^{p<0.001}$

Guzman J, *et al. Ann Rheum Dis* 2015;**0**:1–7. doi:10.1136/annrheumdis-2014-207164

Table 2 Cumulative probabilities of flare within 3, 6 and 12 months after stopping all antirheumatic treatments

		Probability of flare after stopping treatment (95% CI)					
		Within 3 months		Within 6 months		Within 12 months	
Patients	N*	Any flare	Significant flare	Any flare	Significant flare	Any flare	Significant flare
Whole cohort	625	0.060 (0.043 to 0.084)	0.053 (0.037 to 0.075)	0.172 (0.142 to 0.207)	0.137 (0.110 to 0.170)	0.317 (0.278 to 0.360)	0.250 (0.214 to 0.290)
Systemic arthritis	37	0.030 (0.004 to 0.196)	0.030 (0.004 to 0.196)	0.062 (0.016 to 0.225)	0.030 (0.004 to 0.196)	0.062 (0.016 to 0.225)	0.030 (0.004 to 0.196)
Oligoarthritis persistent	310	0.058 (0.036 to 0.092)	0.054 (0.033 to 0.088)	0.156 (0.118 to 0.205)	0.127 (0.093 to 0.173)	0.330 (0.277 to 0.391)	0.259 (0.210 to 0.316)
Oligoarthritis extended†	8	–	–	–	–	–	–
RF-negative polyarthritis	85	0.071 (0.030 to 0.163)	0.057 (0.022 to 0.145)	0.217 (0.137 to 0.334)	0.188 (0.114 to 0.302)	0.402 (0.293 to 0.534)	0.355 (0.251 to 0.486)
RF-positive polyarthritis†	4	–	–	–	–	–	–
Psoriatic arthritis	44	0.057 (0.015 to 0.210)	0.057 (0.015 to 0.210)	0.143 (0.062 to 0.310)	0.114 (0.044 to 0.276)	0.172 (0.081 to 0.344)	0.144 (0.062 to 0.312)
ERA	77	0.083 (0.035 to 0.188)	0.066 (0.025 to 0.167)	0.242 (0.151 to 0.374)	0.190 (0.110 to 0.317)	0.389 (0.275 to 0.529)	0.302 (0.199 to 0.441)
Undifferentiated arthritis	60	0.065 (0.021 to 0.189)	0.043 (0.011 to 0.160)	0.219 (0.124 to 0.369)	0.153 (0.076 to 0.294)	0.331 (0.214 to 0.488)	0.222 (0.126 to 0.373)





FACTORES CLÍNICOS ASOCIADOS A RECAIDA

Table 3 Association of clinical features with the risk of any flare after attaining inactive disease

Characteristic	Patients with feature (%)	Crude HR*	95% CI	p Value	Adjusted HR*	95% CI	p Value
Systemic arthritis	5.9	0.67	0.46 to 0.97	0.033	0.60	0.40 to 0.91	0.015
RF-positive polyarthritis	3.0	1.92	1.29 to 2.87	0.001	1.53	0.99 to 2.39	0.057
DMARD before inactive disease	42.1	1.28	1.09 to 1.50	0.002	0.96	0.77 to 1.21	0.762
Biological agents before inactive disease	6.7	1.65	1.22 to 2.23	0.001	1.31	0.94 to 1.84	0.115
Greater than 52 weeks to attain inactive disease	33.1	1.28	1.09 to 1.50	0.002	1.15	0.95 to 1.39	0.146
Maximum joint count before inactive disease >4	35.3	1.46	1.23 to 1.74	<0.001	1.15	0.90 to 1.46	0.270
Maximum PGA before inactive disease >30 mm	50.6	1.50	1.27 to 1.78	<0.001	1.32	1.08 to 1.62	0.007
Involvement of a high-risk joint†	48.9	1.26	1.08 to 1.47	0.004	1.01	0.82 to 1.25	0.913
ANA positive‡	43.7	1.21	1.03 to 1.43	0.017	1.15	0.96 to 1.37	0.122

ARTICULACIÓN DE ALTO RIESGO

- C. CERVICAL
- MUÑECA
- CADERA
- SACROILIACA
- TOBILLO

IMPLICACIONES PRACTICAS

RIESGO RECAIDA (durante el primer año de inactividad): >40%.

- Poliartritis RF+, curso severo ó ANA + : mayor riesgo.
- 40% de esos brotes no precisarán modificar tratamiento.

SUSPENDER TRATAMIENTO

- Probabilidad de reiniciarlo: 1 de 4 (25%) en el primer año.
- Poli FR+, Oligo extendida: raramente pudieron suspender.
- AIJ sistémica: 1 de 33 (3%).

Ankle arthritis predicts polyarticular disease course and unfavourable outcome in children with juvenile idiopathic arthritis

A-C. Esbjörnsson[1], K. Aalto[2], E.W. Broström[1], A. Fasth[3], T. Herlin[4], S. Nielsen[5], E. Nordal[6], S. Peltoniemi[2], M. Rygg[7], M. Zak[5], L. Berntson[8]
on behalf of the Nordic Study Group of Paediatric Rheumatology (NoSPeR)

Clinical and Experimental Rheumatology 2015; **33**: 751-757.

Table II. Clinical characteristics for children with and without occurrence of ankle arthritis during first year of disease.

Characteristics	Total group n		Ankle arthritis first year of disease n		No ankle arthritis first year of disease n		*p*-value Ankle *vs.* no ankle
Age at disease onset, median years (IQR)	440	5.5 (2.5-9.7)	186	4.9 (2.1-8.8)	254	6.6 (2.8-10.1)	0.003[b]
Gender, female n (% of total)	440	291 (66)	186	127 (68)	254	164 (65)	0.475[a]
ANA positive, n (% of total)	391	107 (27)	160	46 (29)	231	61 (26)	0.645[a]
HLA-B27 positive, n (% of total)	410	86 (21)	173	33 (19)	237	53 (22)	0.462[a]
Assessments, first six month of disease							
ESR mm/hour, median (IQR)	333	35 (16-55)	150	45 (26-77)	183	24 (12-48)	<0.001[b]
CRP mg/litre, median (IQR)	332	14 (0-35)	143	28 (10-56)	189	10 (0-23)	<0.001[b]
Assessments, first eight years of disease							
Cumulative joints, median (IQR)*	440	6 (2-12)	186	10 (6-16)	254	3 (2-9)	<0.001[b]
Not in remission at eight years follow-up, n (%)	427	246 (42)	183	120 (66)	244	126 (52)	0.004[a]
Uveitis, n (% of total)	425	89 (21)	179	36 (20)	246	53 (22)	0.809[a]
$CHAQ_{low}/HAQ_{low}$, n (%) >0	358	35 (10)	149	18 (12)	209	17 (8)	0.279[a]
CHAQ/HAQ, n (%) >0	359	110 (31)	149	56 (38)	210	54 (26)	0.020[a]

FACTORES PREDICTIVOS DE RECAÍDA

RADIOLOGÍA

EULAR-PReS points to consider for the use of imaging in the diagnosis and management of juvenile idiopathic arthritis in clinical practice

A N Colebatch-Bourn,[1,2] C J Edwards,[1,3] P Collado,[4] M-A D'Agostino,[5,6] R Hemke,[7] S Jousse-Joulin,[8] M Maas,[7] A Martini,[9,10] E Naredo,[11] M Østergaard,[12,13] M Rooney,[14] N Tzaribachev,[15] M A van Rossum,[16,17] J Vojinovic,[18] P G Conaghan,[19] C Malattia[9,10]

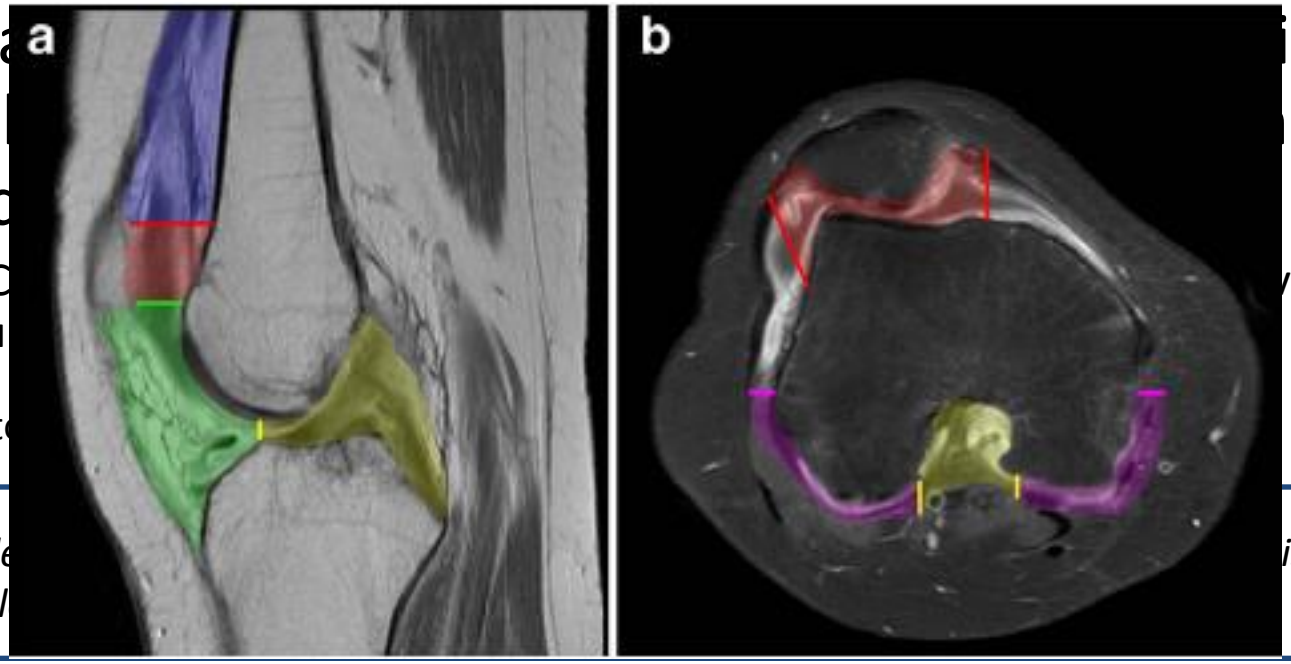
Table 1 Points to consider, level of evidence, grade of recommendation and level of agreement

	Point to consider	Level of evidence	Grade of recommendation	Level of agreement, mean NRS 0–10 (range)
1	US and MRI are superior to clinical examination in the evaluation of joint inflammation; these techniques should be considered for more accurate detection of inflammation, in diagnosis and assessing extent of joint involvement.	3b	C	9.07 (6–10)
2	When there is clinical diagnostic doubt, CR, US or MRI can be used to improve the certainty of a diagnosis of JIA above clinical features alone.	3b	C	9.43 (9–10)
3	If detection of structural abnormalities or damage is required, CR can be used. However MRI or US may be used to detect damage at an earlier time point than CR.	3b	C	8.71 (5–10)
4	In JIA imaging may be of particular benefit over routine clinical evaluation when assessing certain joints, particularly the use of MRI in detecting inflammation of the TMJ and axial involvement.	3b	C	9.64 (8–10)
5	Imaging in JIA may be considered for use as a prognostic indicator. Damage on CR can be used for the prediction of further joint damage. Persistent inflammation on US or MRI may be predictive of subsequent joint damage.	4	C	9.07 (5–10)
6	In JIA, US and MRI can be useful in monitoring disease activity given their sensitivity over clinical examination and good responsiveness. MRI should be considered for monitoring axial disease and TMJ.	3b	C	9.07 (7–10)
7	The periodic evaluation of joint damage should be considered. The imaging modality used may be joint dependent.	3b	C	8.29 (5–10)
8	US can be used for accurate placement of intra-articular injections.	3b	C	9.64 (8–10)
9	US and MRI can detect inflammation when clinically inactive disease is present; this may have implications for monitoring.	3b	C	8.86 (5–10)

Current Status of Efforts on Standardizing Magnetic Resona…is: Report from t…d Health-e-Child…

Nusman C… N, Malattia C, van Rossu…

J. Rheumat…



"La agenda de…isición, validar escalas de val…

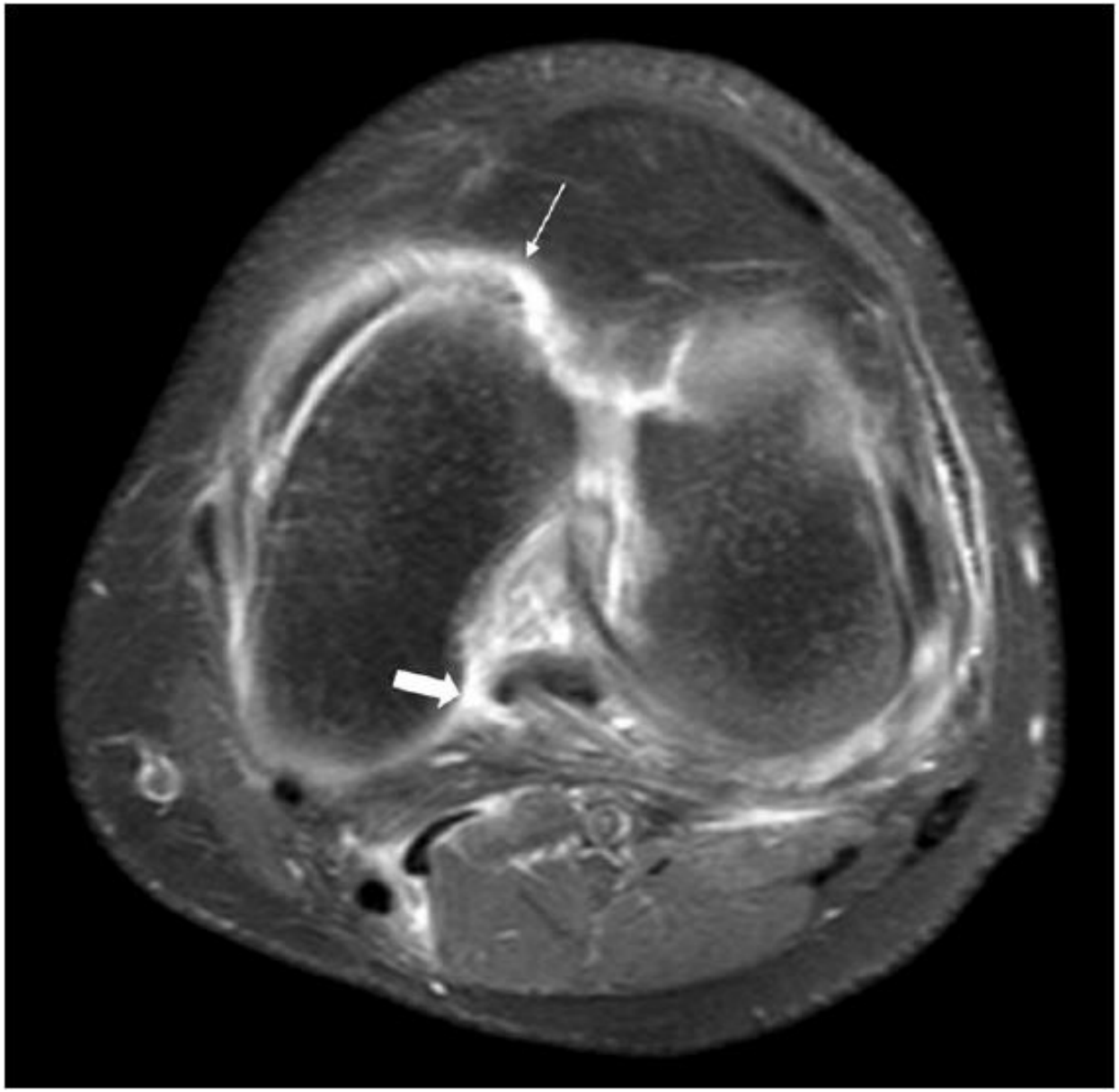
Contrast-enhanced MRI of the knee in children unaffected by clinical arthritis compared to clinically active juvenile idiopathic arthritis patients

Nusman et al. Eur Radiol (2016) 26:1141–1148

JAMRIS:

*infrapatellar (p =0.011)

*cruciate ligaments (p = 0.007)



Nusman et al. Eur Radiol (2016) 26:1141–1148

Toward Standardized Musculoskeletal Ultrasound in Pediatric Rheumatology: Normal Age-Related Ultrasound Findings

PAZ COLLADO,[1] JELENA VOJINOVIC,[2] JUAN CARLOS NIETO,[3] DANIEL WINDSCHALL,[4] SILVIA MAGNI-MANZONI,[5] GEORGE A. W. BRUYN,[6] ANNAMARIA IAGNOCCO,[7] MARIA ANTONIETTA D'AGOSTINO,[8] AND ESPERANZA NAREDO,[3] ON BEHALF OF THE OMERACT ULTRASOUND PEDIATRIC GROUP

Arthritis Care & Research
Vol. 68, No. 3, March 2016, pp 348–356

Predictive value of subclinical synovitis detected by doppler ultrasound in relation to flare in patients with juvenile idiopathic arthritis treated with biologic therapy after tapering biologic therapy; preliminary results.

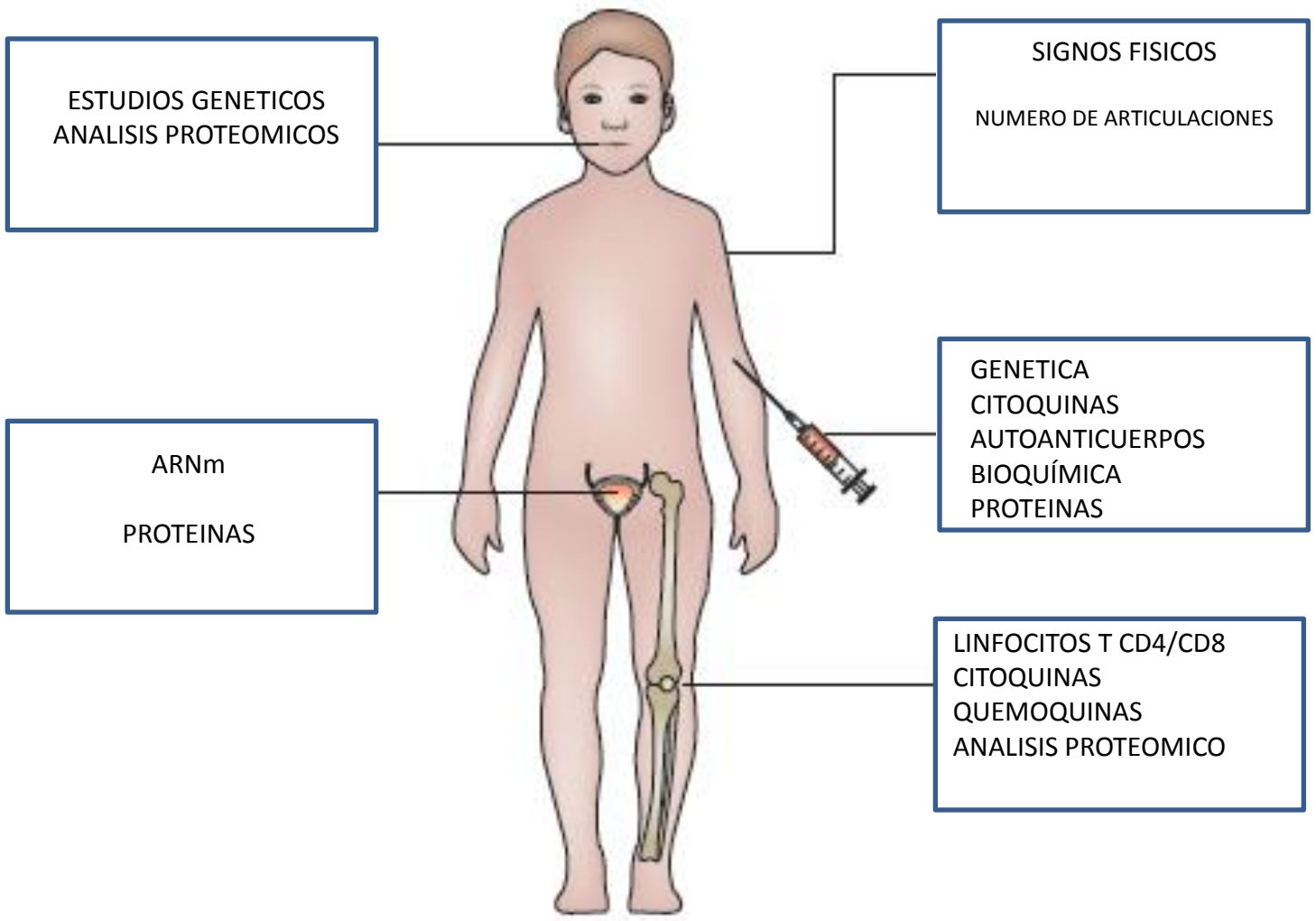
Nieto-González, J.C.; Collado, P.; Clemente, D.; López-Robledillo, J.C. ; Boteanu, A.; Rodríguez, A.; Gamir-Gamir, M.L.; Monteagudo, I. ; Naredo, E.

Annals of the Rheumatic Diseases, supl. 2 74 (Jun 2015): 616-617.

FACTORES PREDICTIVOS DE RECAIDA

BIOMARCADORES

- Factor medible y reproducible
- Refleja un proceso biológico subyacente
- Se correlaciona con la patogenia o las manifestaciones del proceso biológico subyacente
- Tiene un valor pronóstico o diagnóstico
- Fácilmente obtenible del paciente



Consolaro et al. Nature Reviews Rheumatol. 11. 265-75. May 2015.

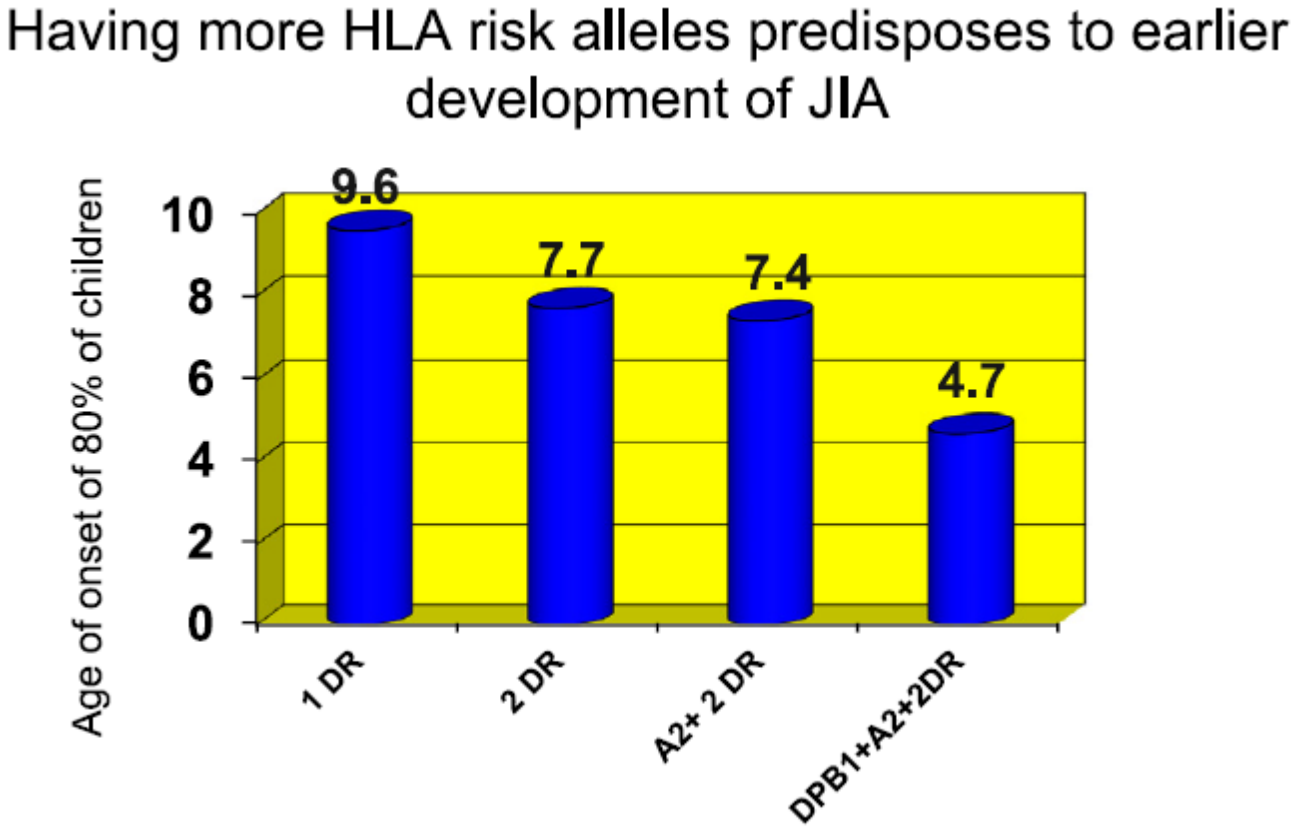
Review article

Immunogenetics of juvenile idiopathic arthritis: A comprehensive review

Aimee O. Hersh [a], Sampath Prahalad [b, c, *]

[a] University of Utah School of Medicine, Salt Lake City, UT, USA
[b] Departments of Pediatrics and Human Genetics, Emory University School of Medicine, Atlanta, GA, USA
[c] Children's Healthcare of Atlanta, Atlanta, GA, USA

A.O. Hersh, S. Prahalad / Journal of Autoimmunity 64 (2015) 113–124



*HLA-DRB1*11* and variants of the MHC class II locus are strong risk factors for systemic juvenile idiopathic arthritis

Michael J. Ombrello[a,1], Elaine F. Remmers[b], Ioanna Tachmazidou[c], Alexei Grom[d,e], Dirk Foell[f], Johannes-Peter Haas[g], Alberto Martini[h,i], Marco Gattorno[i], Seza Özen[j], Sampath Prahalad[k,l], Andrew S. Zeft[m], John F. Bohnsack[n], Elizabeth D. Mellins[o], Norman T. Ilowite[p], Ricardo Russo[q], Claudio Len[r], Maria Odete E. Hilario[r], Sheila Oliveira[s], Rae S. M. Yeung[t,u,v], Alan Rosenberg[w], Lucy R. Wedderburn[x,y], Jordi Anton[z], Tobias Schwarz[aa], Anne Hinks[bb], Yelda Bilginer[j], Jane Park[o], Joanna Cobb[bb,cc], Colleen L. Satorius[b], Buhm Han[dd,ee,ff], Elizabeth Baskin[a], Sara Signa[h], Richard H. Duerr[gg,hh], J. P. Achkar[ii,jj], M. Ilyas Kamboh[gg], Kenneth M. Kaufman[d,e], Leah C. Kottyan[d,e], Dalila Pinto[kk], Stephen W. Scherer[ll], Marta E. Alarcón-Riquelme[mm,nn], Elisa Docampo[oo,pp], Xavier Estivill[pp], Ahmet Gül[qq], British Society of Pediatric and Adolescent Rheumatology (BSPAR) Study Group[2], Childhood Arthritis Prospective Study (CAPS) Group[2], Randomized Placebo Phase Study of Rilonacept in sJIA (RAPPORT) Investigators[2], Sparks-Childhood Arthritis Response to Medication Study (CHARMS) Group[2], Biologically Based Outcome Predictors in JIA (BBOP) Group[2], Paul I. W. de Bakker[rr,ss,tt], Soumya Raychaudhuri[bb,dd,ee], Carl D. Langefeld[uu], Susan Thompson[d,e], Eleftheria Zeggini[c], Wendy Thomson[bb,cc], Daniel L. Kastner[b,1], Patricia Woo[y], and the International Chi

15970-

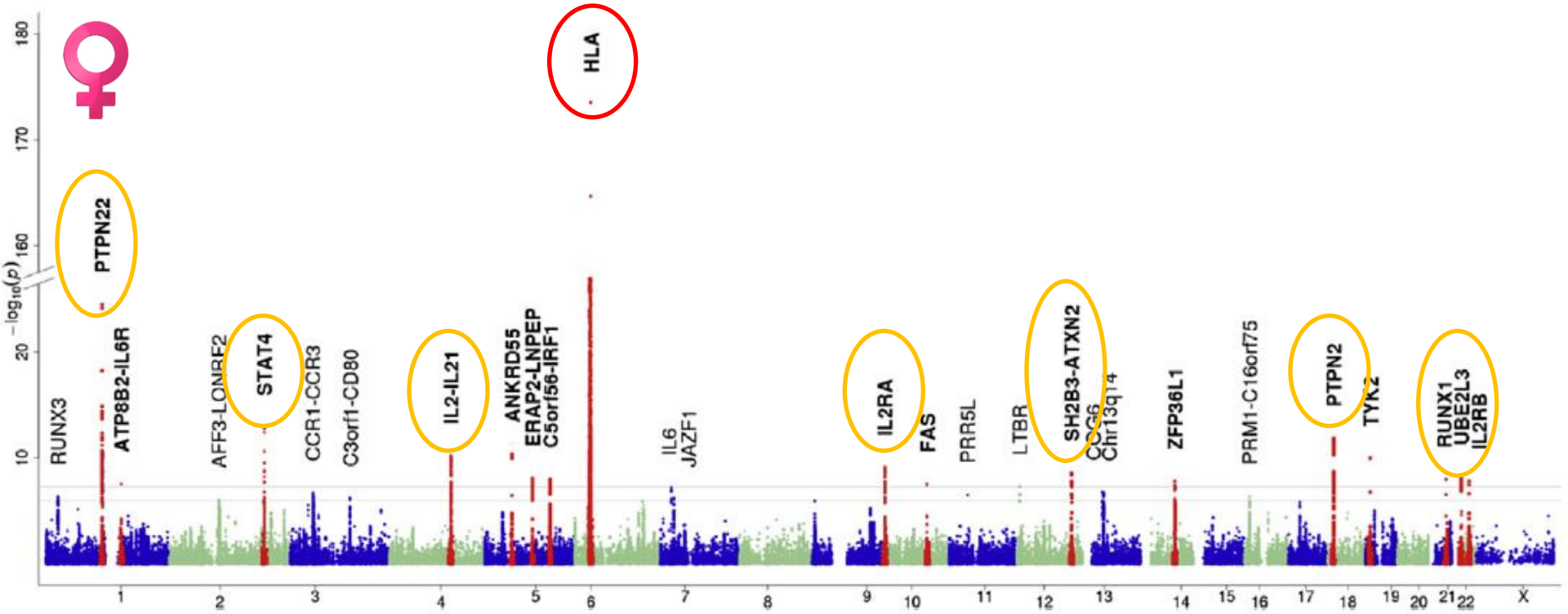
Table 1. Membership of nine geographically defined sJIA case-control strata following quality control operations

Stratum	Cases	Controls	MHC-region SNPs*
United States	243	1,718	34,992
United Kingdom	202	4,097	32,105
Germany	115	193	34,458
Turkey	49	94	33,115
Italy	49	59	34,434
Brazil	48	62	31,814
Argentina	33	115	32,870
Canada	17	427	31,988
Spain	14	182	27,005
Total	770	6,947	

Table 2. sJIA-associated classical HLA alleles and haplotypes identified by meta-analysis of six independent populations

Allele or haplotype	P_{meta}	OR (95CI)	I^2
*HLA-DRB1*11*	2.7×10^{-16}	2.3 (1.9, 2.8)	0.17
*HLA-DRB1*11:01*	7.1×10^{-14}	2.3 (1.9, 2.9)	0.00
*HLA-DQA1*05:01*	2.6×10^{-9}	1.7 (1.4, 2.0)	0.00
*HLA-DQA1*05*	2.6×10^{-9}	1.7 (1.4, 2.0)	0.00
*HLA-DQB1*03:01*	9.2×10^{-9}	1.7 (1.4, 2.0)	0.11
*HLA-DRB1*11:04*	7.8×10^{-5}	2.0 (1.4, 2.7)	0.00
*HLA-DQB1*03*	1.5×10^{-4}	1.4 (1.2, 1.7)	0.40
*HLA-DQA1*02*	5.4×10^{-4}	0.7 (0.6, 0.8)	
*DRB1*11–DQA1*05–DQB1*03*	6.4×10^{-17}	2.3 (1.9, 2.9)	0.32
*DRB1*11:01–DQA1*05:01–DQB1*03:01*	3.1×10^{-11}	2.2 (1.7, 2.8)	0.00
*DRB1*11:04–DQA1*05:01–DQB1*03:01*	1.5×10^{-6}	2.3 (1.6, 3.1)	0.22

Manhattan plot of associations in oligoarticular and RF-negative polyarticular JIA.

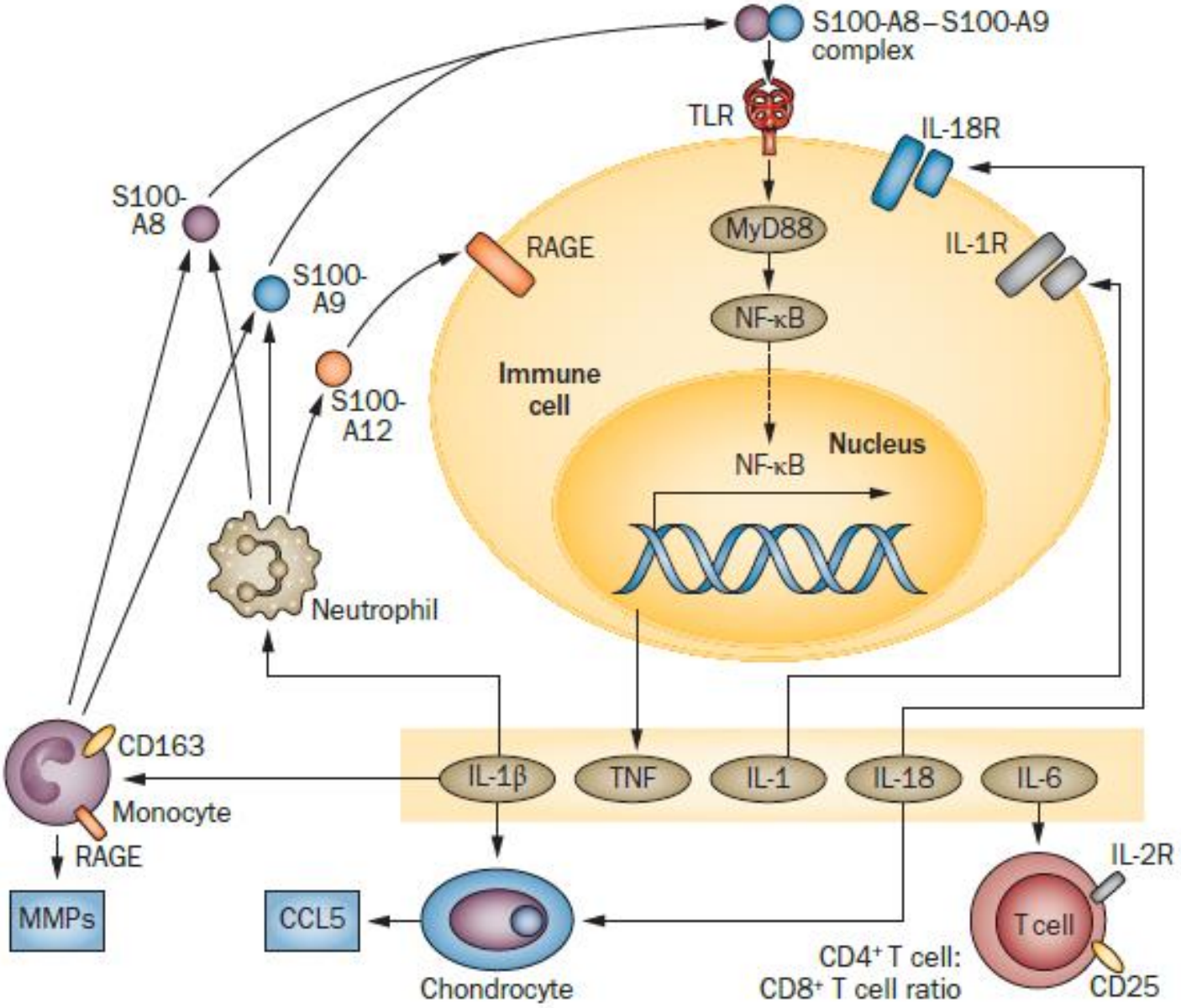


The association of PTPN22 rs2476601 with juvenile idiopathic arthritis is specific to females. Genes Immun. 2015 Oct;16(7):495-8. doi: 10.1038/gene.2015.32. Epub 2015 Aug 20. Association of juvenile idiopathic arthritis with PTPN22 rs2476601 is specific to females in a Greek population. Pediatr Rheumatol Online J. 2016 Apr 23;14(1):25. doi: 10.1186/s12969-016-0087-3.

Table 1 | Biomarkers for JIA

Biomarker	Direction of trend	Clinical relevance
Serum or plasma		
S100-A8, S100-A9 (MRP-8, MRP-14)	Increased	Indicates increased disease activity in systemic JIA Predicts therapeutic response in systemic JIA Predicts relapse after achievement of remission
S100-A12 (MRP-6)	Increased	Indicates increased disease activity in systemic JIA Predicts relapse after achievement of remission
IL-18	Increased	Correlates with disease activity Predicts therapeutic response and development of MAS in systemic JIA
sIL-2Rα (CD25)	Increased	Detects subclinical MAS and predicts development of MAS in systemic JIA
sCD163	Increased	Detects subclinical MAS and predicts development of MAS in systemic JIA
Follistatin-like protein 1	Increased	Detects subclinical MAS and predicts development of MAS in systemic JIA
MMP-3	Increased	Correlates with disease severity in enthesitis-related arthritis Correlates with progression of structural joint damage in systemic JIA
Synovial fluid		
$CD4^+$ T cell: $CD8^+$ T cell ratio	Decreased	Predicts risk of arthritis extension in oligoarthritis
CCL5	Increased	Predicts risk of arthritis extension in oligoarthritis
IL-18	Increased	Correlates with disease activity in systemic arthritis

Abbreviations: CCL5, C-C chemokine ligand 5 (also known as RANTES); JIA, juvenile idiopathic arthritis; MAS, macrophage activation syndrome; MMP-3, matrix metalloproteinase-3; MRP, myeloid-related protein; sCD163, soluble CD163; sIL-2Rα, soluble IL-2 receptor α.



Consolaro et al. Nature Reviews Rheumatol. 11. 265-75. May 2015.

Interleukin-18 for predicting the development of macrophage activation syndrome in systemic juvenile idiopathic arthritis

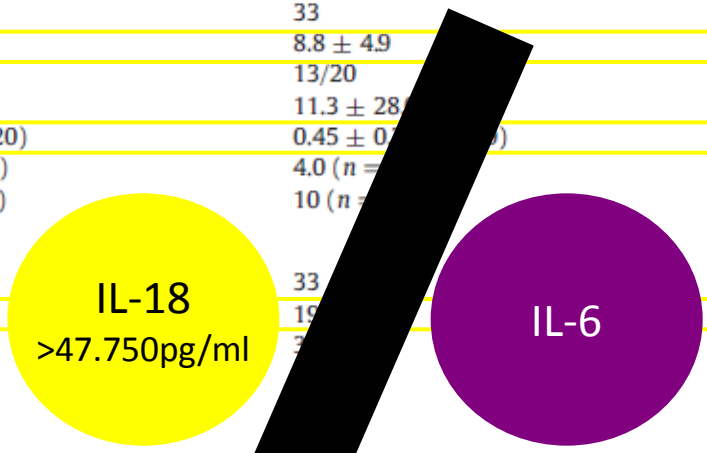
Masaki Shimizu [a,*], Yasuo Nakagishi [b], Natsumi Inoue [a], Mao Mizuta [b], Giyo Ko [c], Yutaka Saikawa [c], Tomohiro Kubota [d], Yuichi Yamasaki [d], Syuji Takei [d], Akihiro Yachie [a]

[a] Department of Pediatrics, School of Medicine, Institute of Medical, Pharmaceutical and Health Sciences, Kanazawa University, Kanazawa 920-8641, Japan
[b] Department of Pediatric Rheumatology, Hyogo Prefectural Kobe Children's Hospital, Kobe, Hyogo, Japan
[c] Department of Pediatrics, School of Medicine, Kanazawa Medical University, Uchinada, Japan
[d] Department of Pediatrics, School of Medicine, Faculty of Medicine, Kagoshima University, Kagoshima, Japan

/ Clinical Immunology 160 (2015) 277–281

Table 1
Clinical features of the 2 systemic juvenile idiopathic arthritis subsets based on serum IL-6 and IL-18 levels.

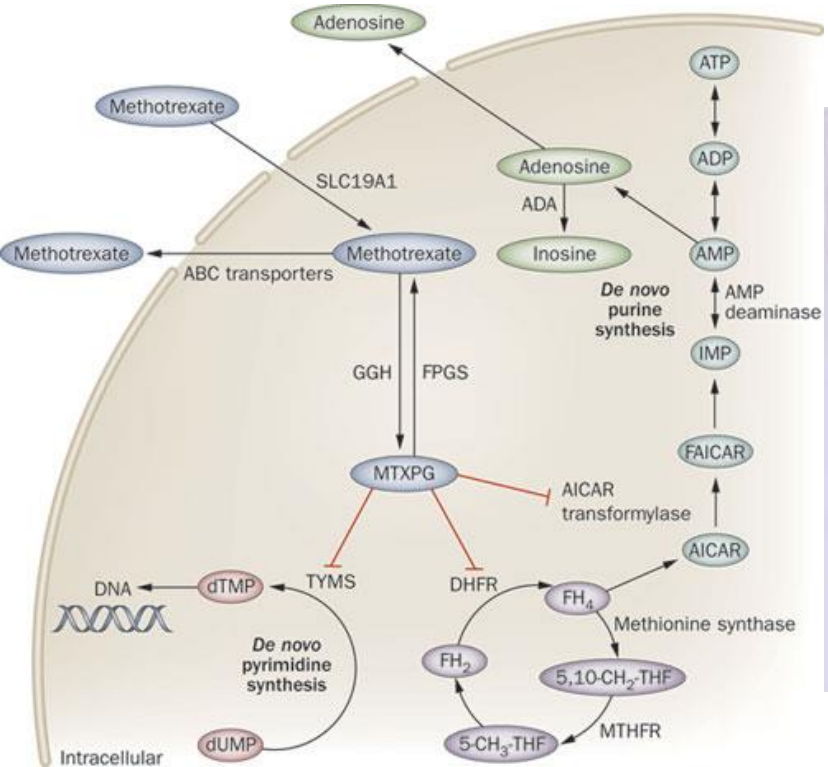
	IL-18/IL-6 < 1000	IL-18/IL-6 > 1000	
Patients	33	43	
Age	8.8 ± 4.9	6.3 ± 5.3	$p < 0.05$
Sex (male/female)	13/20	26/17	$p = 0.1046$
Disease duration (months)	11.3 ± 28	4.76 ± 16.0	$p = 0.7330$
Dosage of prednisolone (mg/kg/day) ($n = 20$)	0.45 ± 0	0.95 ± 0.69 ($n = 11$)	$p = 0.0794$
Dosage of cyclosporine (mg/kg/day) ($n = 4$)	4.0 (n =	4.33 ± 0.57 ($n = 3$)	
Dosage of metrexate (mg/m^2/week) ($n = 2$)	10 (n =		
Clinical symptoms			
Fever	33	43	
Rash	19	34	$p < 0.05$
Hepatomegaly	3	8	$p = 0.3310$
Splenomegaly		5	$p = 0.6920$
Lymphadenopathy		15	$p = 0.4504$
Pleuritis		2	$p = 0.5021$
Pericarditis		3	$p = 1.0000$
Arthritis (number of affected joints)	± 5.2	0.8 ± 1.5	$p < 0.001$
complication of MAS		15	$p < 0.0001$
Laboratory findings			
CRP (mg/dl)	13.3 ± 6.5	8.2 ± 5.7	$p < 0.01$
AST (IU/l)	36.8 ± 27.8	64.1 ± 67.6	$p < 0.01$
LDH (IU/l)	317.1 ± 105.8	531.8 ± 412.9	$p < 0.01$
Ferritin (ng/ml) ($n = 63$)	1787.4 ± 2977.3	4387.2 ± 8625.5	$p = 0.1264$
MMP-3 (ng/ml) ($n = 47$)	286.3 ± 373.8	50.5 ± 45.1	$p < 0.01$
IL-6 (pg/ml)	167.2 ± 223.9	20.6 ± 30.4	$p < 0.0001$
IL-18 (pg/ml)	33872 ± 59142	101809 ± 90128	$p < 0.0001$



RESEARCH ARTICLE Open Access

Methotrexate polyglutamates as a potential marker of adherence to long-term therapy in children with juvenile idiopathic arthritis and juvenile dermatomyositis: an observational, cross-sectional study

Hawwa *et al. Arthritis Research & Therapy* (2015) 17:295



- El pico de concentración es más elevado vía subcutánea y produce Mtx-pg de cadena más larga (más efectivos)
- El aclaramiento de Mtx-pg es menor en niños mayores
- La concentración de Mtx-pg se correlaciona con nivel VSG y PCR
- La falta de adherencia es frecuente (50%) y se asocia a pacientes mayores, en tratamiento con dosis bajas y vía oral
- 78% de aquellos con intolerancia gastrointestinal.

Anti-adalimumab antibodies in juvenile idiopathic arthritis: frequent association with loss of response

A Skrabl-Baumgartner[1], W Erwa[2], W Muntean[1], J Jahnel[1]

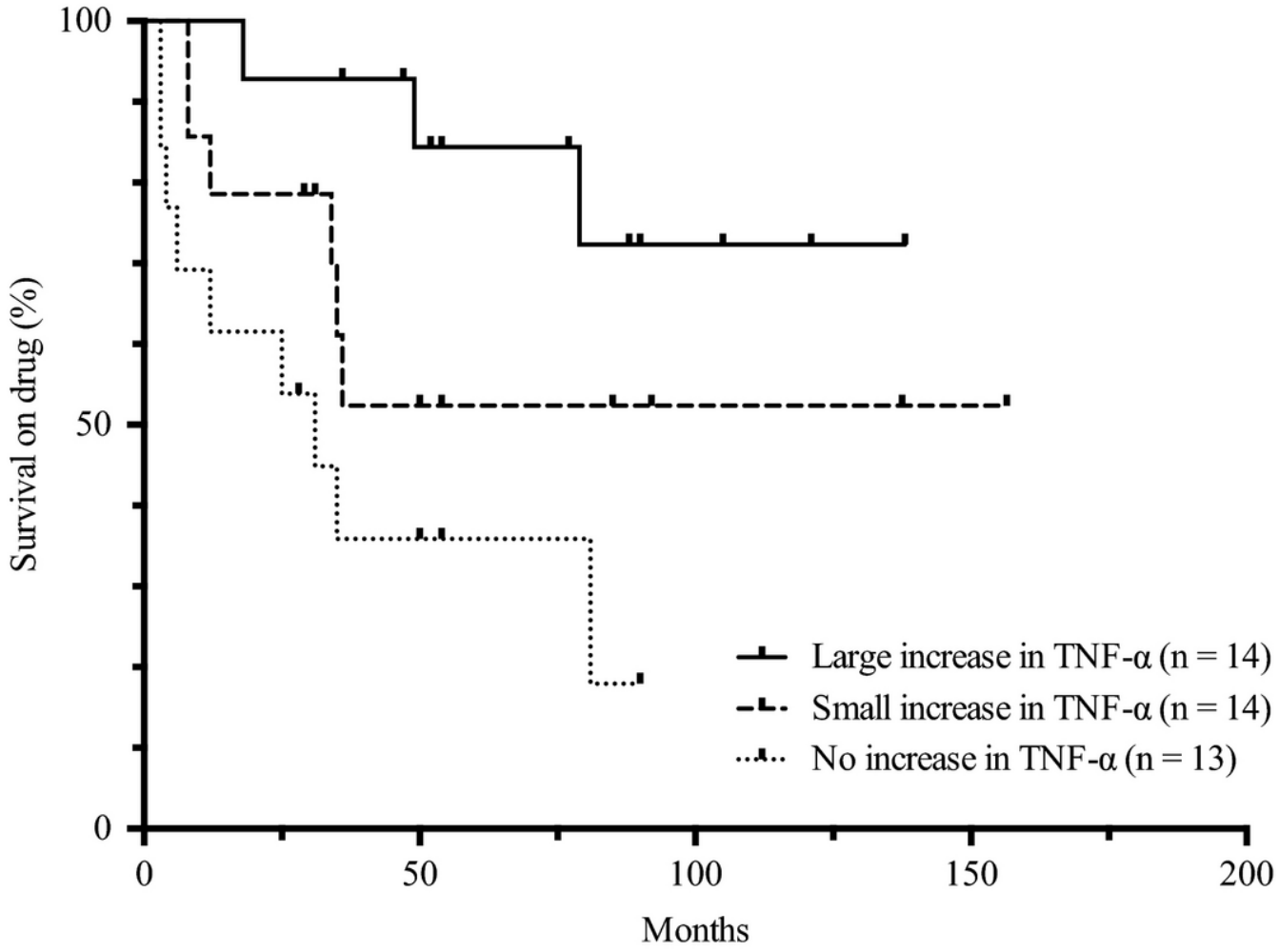
Table 1. Patients' demographic and clinical characteristics.

	Total patients (n = 23)	AAA positive (n = 6)	AAA negative (n = 17)
Age (years) at time of inclusion	14.2 (7.9–17.2)	14.3 (9.8–18.2)	14.0 (10.5–17.3)
Female	20 (87)	5 (83)	15 (88)
JIA subtype			
Oligoarthritis	14	2	12
Persistent/extended	3/11		
Polyarthritis	7	2	5
RF negative/RF positive	5/2		
Enthesitis-related arthritis	2	2	0
ANA positive	13	4	9
Associated uveitis	12 (52)	1 (16)	11 (65)
MTX	17 (74)	2 (33)	15 (88)
Prior biological treatment	6 (26)	2 (33)	4 (24)
LOR	6 (26)	5 (83)	1 (6)
JADAS-10			
At time of inclusion	1.1 (0.4–2.2)	1.2 (1.0–2.2)	1.1 (0.3–2.3)
At LOR or end of study	1.1 (0.3–9.4)	20.2 (18.2–23.7)	1.0 (0.1–2.0)
Patients with active uveitis at LOR or end of study	2	1	1
ADA serum levels (mg/L)	7.88 (3.45–17.42)	1.63 (0.25–3.09)	14.13 (9.05–17.5)

Circulating complexes between tumour necrosis factor-alpha and etanercept predict long-term efficacy of etanercept in juvenile idiopathic arthritis

Kahn R, Berthold E, Gullstrand B, Schmidt T, Kahn F, Geborek P, Saxne T, Bengtsson AA, Månsson B.

Acta Pædiatrica 2016 **105**, pp. 427–432



MUCHAS GRACIAS

FORO SERPE. Murcia.
25 y 26 de Noviembre 2016