NOVEDADES (MUNDIALES) EN MIOPATÍAS INFLAMATORIAS

Dr. Albert Selva O'Callaghan

Áreas de interés

1. Etiopatogenia
2. Técnicas de imagen
3. Cáncer
4. Autoanticuerpos
5. Pulmón
6. Tratamiento

Etiopatogenia

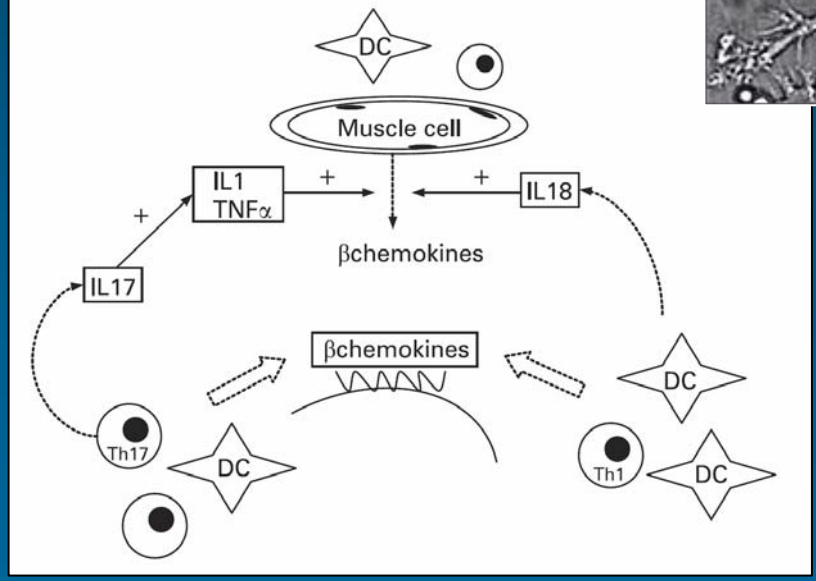
Review

Chemokines and dendritic cells in inflammatory myopathies

A Tournadre,[1,2] P Miossec[1]

Ann Rheum Dis 2009;**68**:300–304. doi:10.1136/ard.2008.095984

Review



PDCs como APC y producción IFN α I

Th17 (IL-17)

¿La célula muscular como APC?

Journal of Neuroimmunology 209 (2009) 131–138

Contents lists available at ScienceDirect

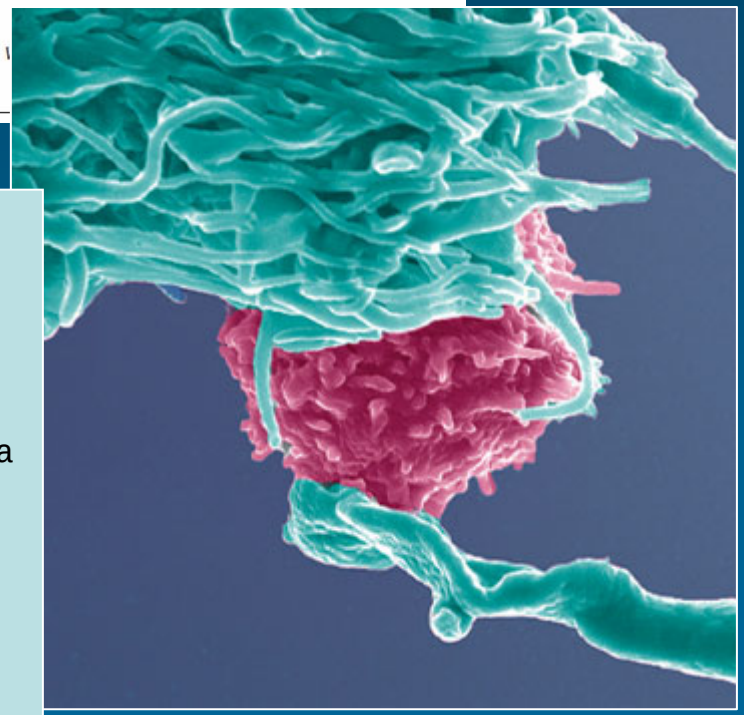
Journal of Neuroimmunology

journal homepage: www.elsevier.com/locate/jneuroim

Intercellular exchanges of membrane fragments (trogocytosis) between human muscle cells and immune cells: A potential mechanism for the modulation of muscular immune responses

Anne Waschbisch [a,1], Sven G. Meuth [a], Alexander M. Herrmann [a], Barbara Wrobel [a], Nicholas Schwab [a], Hanns Lochmüller [b,2], Heinz Wiendl [a,*]

[a] Clinical Research Group for Multiple Sclerosis and Neuroimmunology, Department of Neurology, University of Wuerzburg,
[b] Friedrich-Baur-Institute and Department of Neurology, Ludwigs-Maximilians University, Munich, Germany



Células T “Comen a bocados” Mb de células musculares (“aquí te pillo aquí te mato”)

· se estimula la proliferación de células T autoreactivas (Círculo vicioso)

· la adquisición MHC I, transforma los linfocitos T en diana (Círculo virtuoso)

Sinapsis inmunológica

APC y linfocitos T

Regulación de la respuesta inmune

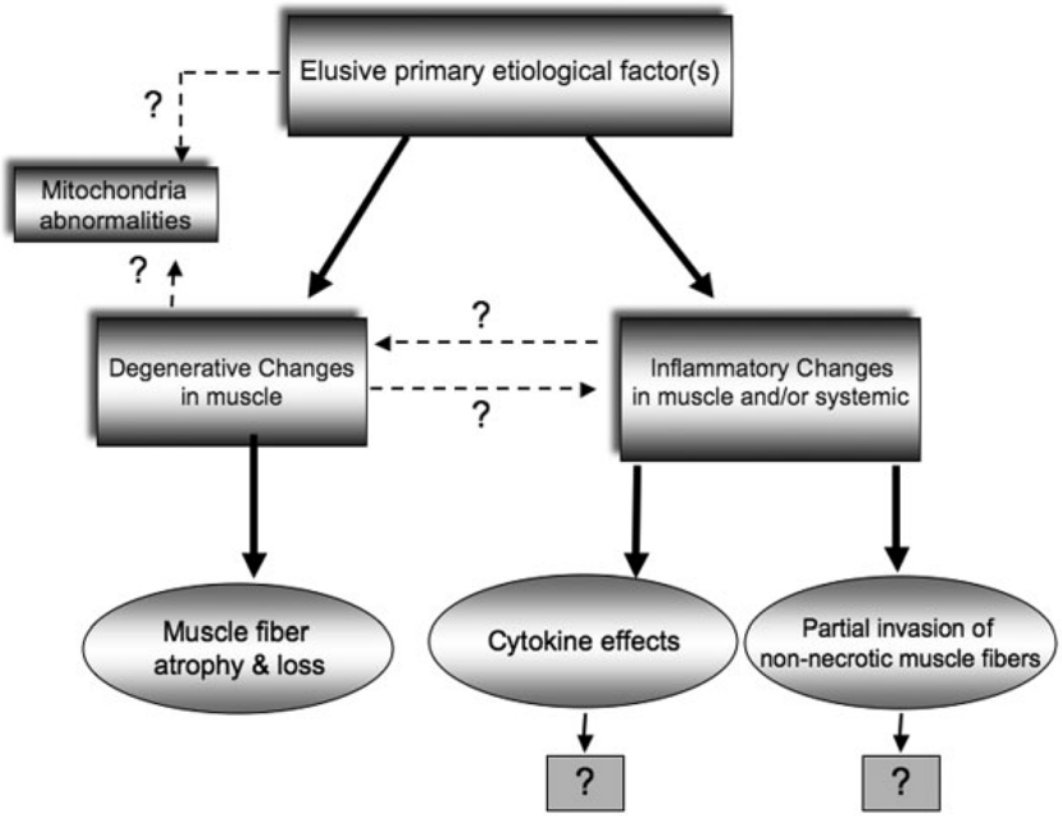
POINT OF VIEW

Sporadic Inclusion Body Myositis: Pathogenic Considerations

George Karpati, OC, CQ, MD, FRCP(C), and Erin K. O'Ferrall, MD

Sporadic inclusion body myositis is the commonest acquired disease of skeletal muscles after 50 years of age, and as such it has commanded a great deal of attention of investigators over the past 25 years. As a result, a large amount of information has accumulated concerning its clinical profile, myopathology, and immunopathology. In the myopathology and immunopathology, there is general agreement that the characteristic features could be divided into a degenerative and an inflammatory group. ogenesis of muscle fiber damage. In se two groups of changes, and if so, ntroversial observations and critically versus degeneration.

Ann Neurol 2009;65:7–11



Técnicas de imágen

MRI AND ULTRASOUND IN DIAGNOSIS AND MANAGEMENT

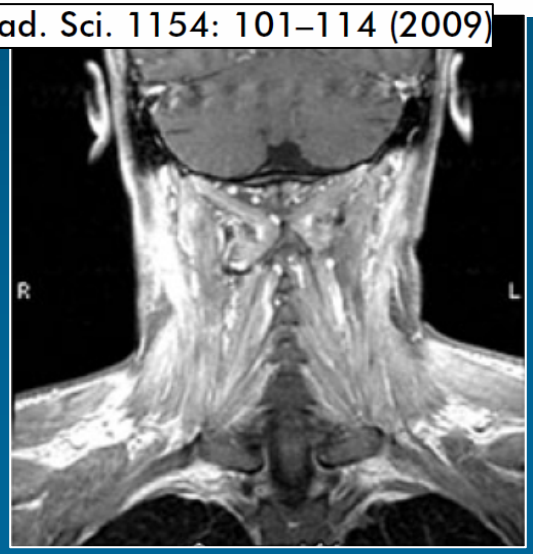
Magnetic Resonance Imaging of the Idiopathic Inflammatory Myopathies

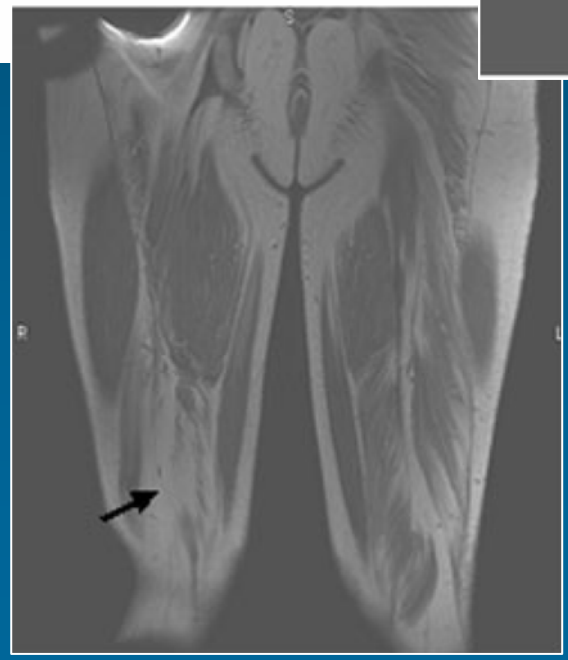
Structural and Clinical Aspects

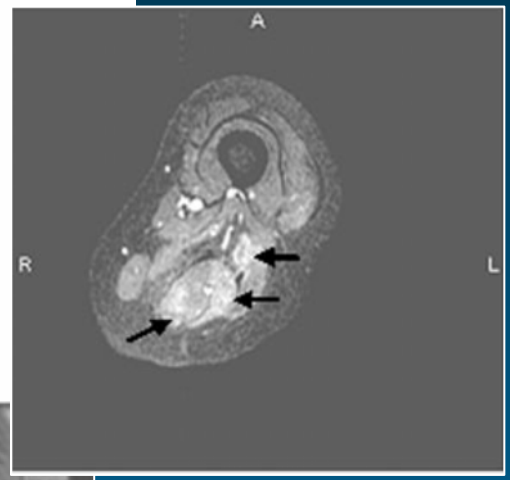
Rodolfo Victor Curiel,[a] Robert Jones,[b] and Kathleen Brindle[c]

Departments of [a]Medicine, [b]Pathology, and [c]Radiology, The George Washington University, Washington, DC, USA

Ann. N.Y. Acad. Sci. 1154: 101–114 (2009)





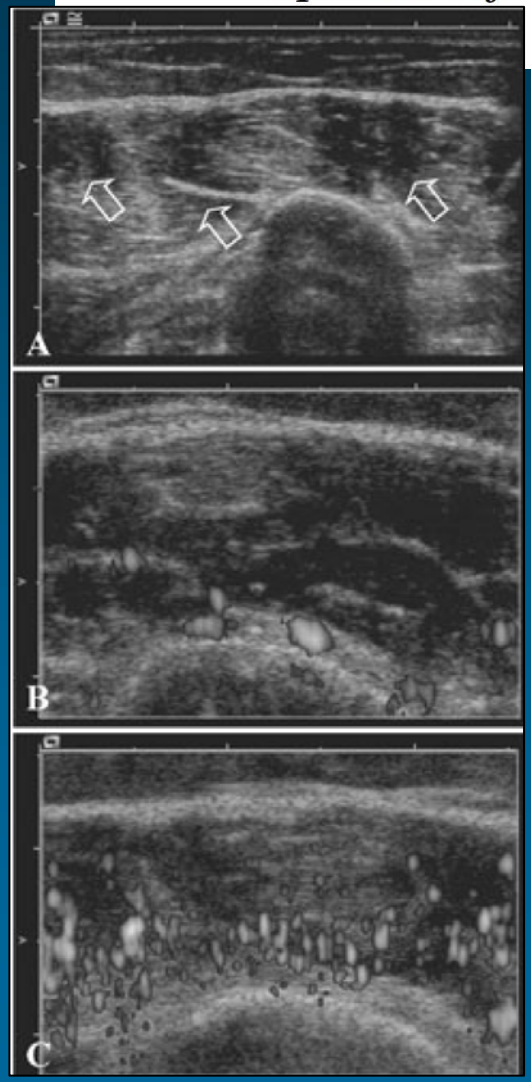


Ultrasound in the Inflammatory Myopathies

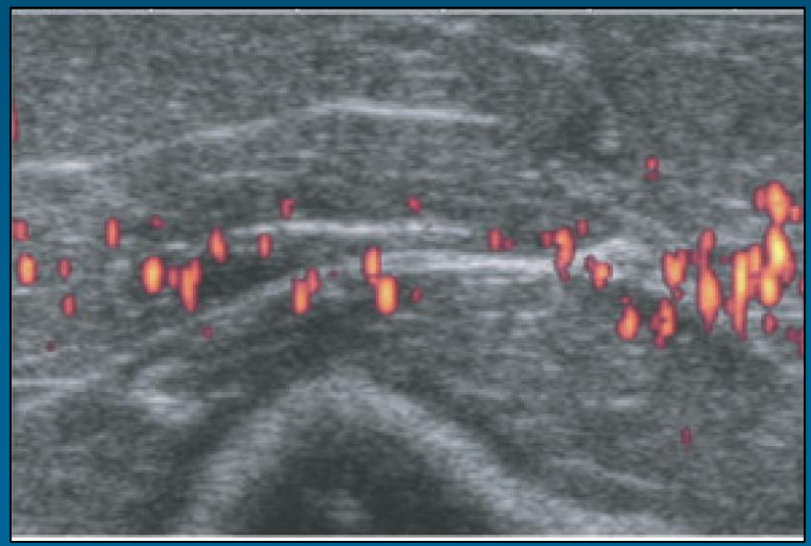
Marc-André Weber

Department of Radiology, German Cancer Research Center, Heidelberg, Germany

Ann. N.Y. Acad. Sci. 1154: 159–170 (2009)



Aumento de tamaño y perfusión y baja ecogeneicidad



Cáncer

Clinical dermatology • **Review article**

Clinical and Experimental Dermatology

2009

Defining cancer risk in dermatomyositis. Part I

V. Madan, H. Chinoy,* C. E. M. Griffiths and R. G. Cooper*

*Dermatology and *Rheumatic Diseases Centres, Salford Royal Hospital NHS Foundation Trust, University of Manchester, Manchester, UK*

Clinical and Experimental Dermatology, **34**, 451–455

Table 2 Summary of population-based epidemiological studies.

References	Patients, *n*			Diagnostic verification	Malignancies, *n*		SIR (95% CI)	
	Total	PM	DM		PM	DM	PM	DM
Manchul *et al.*[7]	71	40	31	Bohan and Peter's criteria[2,3]	7	9	N/A	N/A
Lakhanpal *et al.*[8]	115	65	50	N/A	18	11	N/A	N/A
Lyon *et al.*[9]	104	64	40	Clinical, histology in 75%	4	1	PM/DM 1.6 (0.27–13.0)	–
Sigurgeirsson *et al.*[10]	788	396	392	Bohan and Peter's criteria[2,3]	58	94	Males 1.8 (1.1–2.7); females 1.7 (1.0–2.5)	Males 2.4 (1.6–3.6); females 3.4 (2.4–4.7)
Airio *et al.*[11]	311	175	71	Bohan and Peter's criteria,[2,3] histology in 84%	26	63	1.0 (0.5–1.8)	6.5 (3.9–10)
Chow *et al.*[12]	539	336	203	Nil	26	26	3.8 (2.6–5.4)	1.7 (1.1–2.4)
Stockton *et al.*[13]	705	419	286	Nil	71	77	2.1 (1.5–2.9)	7.7 (5.7 –10.1)
Buchbinder *et al.*[15]	537*	321	85	Histological review	58	36	2.0 (1.4–2.7)	6.2 (3.9–10.0)

PM, polymyositis; DM, dermatomyositis; CI, confidence interval; IBM, inclusion body myositis; CTD, myositis associated with connective tissue disease; CM, childhood myositis; N/A, data not available. *Also 52 with IBM, 30 with CTD, 49 with CM.

Clinical dermatology • **Review article**

Clinical and Experimental Dermatology

Defining cancer risk in dermatomyositis. Part II. Assessing diagnostic usefulness of myositis serology

V. Madan, H. Chinoy,* C. E. M. Griffiths and R. G. Cooper*

*Dermatology and *Rheumatic Diseases Centres, Salford Royal Hospi*

doi:10.1111/j.1365-2230.2009.03227.x

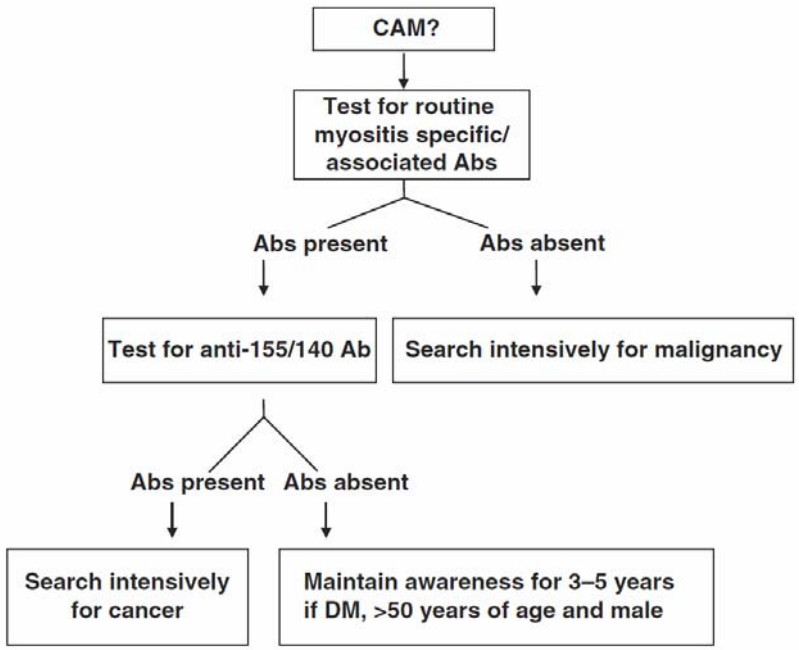


Figure 1 Possible strategy for cancer search in cancer-associated patients with myositis. Abs, antibodies; CAM, cancer-associated myositis; DM, dermatomyositis.

Autoanticuerpos

Autoimmunity Reviews xxx (2009) xxx–xxx

Contents lists available at ScienceDirect

Autoimmunity Reviews

journal homepage: www.elsevier.com/locate/autrev

Use of a commercial line blot assay as a screening test for autoantibodies in inflammatory myopathies

Johan Rönnelid [a,b,*], Sevim Barbasso Helmers [b], Helena Storfors [a], Katarina Grip [a], Lars Rönnblom [c], Karin Franck-Larsson [c], Gunnel Nordmark [c], Ingrid E. Lundberg [b]

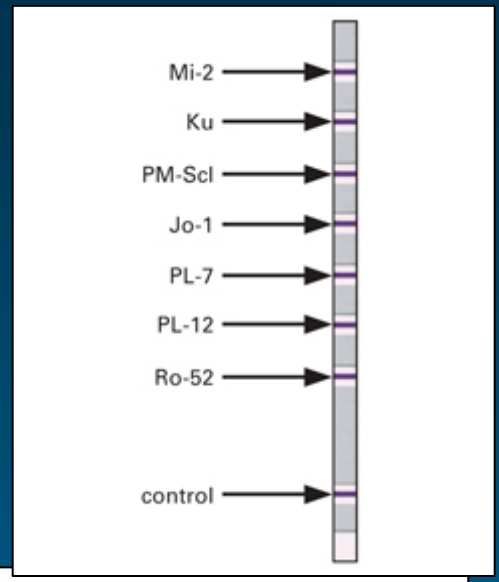


Table 2
Overall performance of the Myositis 1 line blot.

	DM ($n=50$)	PM ($n=89$)	JDM ($n=4$)	IBM ($n=10$)	$\sum$ myositides ($n=153$)	Overall sensitivity	SLE ($n=26$)	pSS ($n=26$)	SSc ($n=25$)	$\sum$ disease controls ($n=77$)	Overall specificity (%)
Anti-Jo1	4(8)	14(16)	0(0)	0(0)	18(12)	11.76	0(0)	0(0)	1(4)	1(1)	98.70
Anti-Mi2	4(8)	1(1)	0(0)	0(0)	5(3)	3.27	0(0)	0(0)	0(0)	0(0)	100
Anti-Ku	1(2)	3(3)	0(0)	0(0)	4(3)	2.61	0(0)	2(8)	0(0)	2(3)	97.40
Anti-PM-Scl	4(8)	7(8)	0(0)	0(0)	11(7)	7.19	0(0)	0(0)	0(0)	0(0)	100
Anti-Pl-7	2(4)	2(2)	0(0)	0(0)	4(3)	2.61	0(0)	0(0)	0(0)	0(0)	100
Anti-Pl-12	0(0)	0(0)	0(0)	0(0)	0(0)	0.00	0(0)	0(0)	0(0)	0(0)	100
Anti-SSA/Ro52	14(28)	26(29)	0(0)	4(40)	44(29)	28.76	6(23)	16(62)	6(24)	28(36)	63.64
Any autoantibody except SRP.	22(44)	41(46)	0(0)	4(40)	67(43)	44.79	6(23)	17(65)	6(24)	29(38)	62.34
Anti-Ro52 excluded	15(30)	16(18)	0(0)	0(0)	31(20)	20.26	0(0)	2(8)	1(4)	3(4)	96.10
Anti-SRP	1(2)	7(8)	0(0)	0(0)	8(5)	5.23	1(4)	0(0)	1(4)	2(3)	97.40

Results are given as total number and percentage within parentheses. Results concerning anti-SRP is given for the antigen concentration very close to the concentration that will appear in future versions of the myositis line blot. Values for overall sensitivity and specificity concern the line blot in the present form, without anti-SRP determination. Among 127 positive responses 113 were clearly positive and 14 borderline, all of them regarded as positive in the table.
Abbreviations: DM, dermatomyositis; PM, polymyositis; JDM, juvenile dermatomyositis; IBM, inclusion body myositis; SLE, systemic lupus erythematosus; pSS, primary Sjögren's syndrome, and SSc, systemic sclerosis.

Pulmón

Original Research

INTERSTITIAL LUNG DISEASE

Clinical Profile of Anti-PL-12 Autoantibody*

Cohort Study and Review of the Literature

Meena Kalluri, MD; Steven A. Sahn, MD, FCCP; Chester V. Oddis, MD; Suzanne L. Gharib, MD; Lisa Christopher-Stine, MD; Sonye K. Danoff, MD, PhD; Livia Casciola-Rosen, PhD; Grace Hong, MS; Paul F. Dellaripa, MD; and Kristin B. Highland, MD

(CHEST 2009; 135:1550–1556)

Respirology (2007) **12**, 642–653 doi: 10.1111/j.1440-1843.2007.01140.x

ORIGINAL ARTICLE

Autoantibody to alanyl-tRNA synthetase in patients with idiopathic pulmonary fibrosis

TORU TAKAHASHI,[1] IKUO WADA,[2] YOSHINORI OHTSUKA,[3] MITSURU MUNAKATA,[3] YUKIHIKO HOMMA[4] AND YOSHIO KUROKI[1]

[1]*Department of Biochemistry, Sapporo Medical University School of Medicine, Sapporo,* [2]*Department of Cell Science, Institute of Biomedical Sciences,* [3]*Department of Pulmonary Medicine, Fukushima Medical University School of Medicine, Fukushima, and* [4]*Medical Administration Center, Hokkaido University, Sapporo, Japan*

Tratamiento

Autoimmunity Reviews xxx (2009) xxx–xxx

Contents lists available at ScienceDirect

Autoimmunity Reviews

journal homepage: www.elsevier.com/locate/autrev

Intravenous immunoglobulin as add on treatment with mycophenolate mofetil in severe myositis

Maria Giovanna Danieli [a,*], Lucia Calcabrini [a], Vincenzina Calabrese [a], Annalisa Marchetti [a], Francesco Logullo [b], Armando Gabrielli [a]

[a] *Clinica Medica, Dipartimento di Scienze Mediche e Chirurgiche, Italy*
[b] *Clinica Neurologica, Università Politecnica delle Marche, Ancona, Italy*

doi:10.1093/brain/awp104 Brain 2009: Page 1 of 9 | 1

BRAIN
A JOURNAL OF NEUROLOGY

Effect of Alemtuzumab (CAMPATH 1-H) in patients with inclusion-body myositis

Marinos C. Dalakas,[1] Goran Rakocevic,[1] Jens Schmidt,[1] Mohammad Salajegheh,[1] Beverly McElroy,[1] Michael O. Harris-Love,[2] Joseph A. Shrader,[2] Ellen W. Levy,[2] J[illegible] and Allan D. Kirk[4]

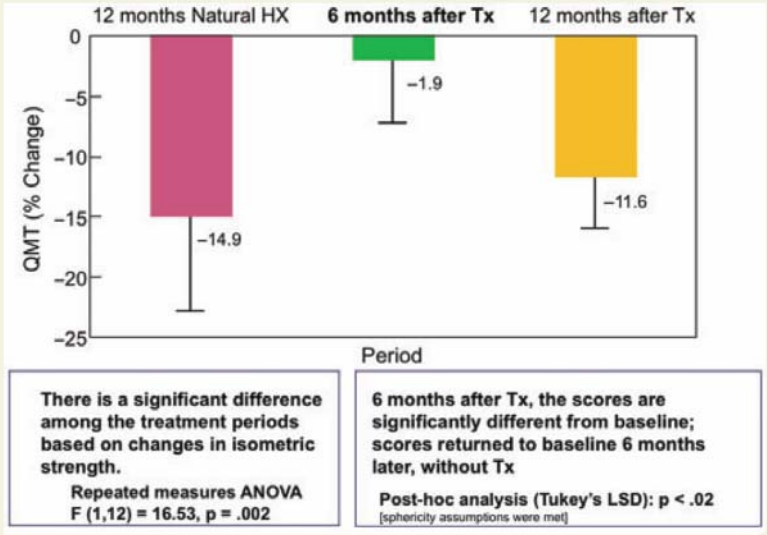


Figure 1 Changes in muscle strength using QMT. During a 12-month natural history period there is a decline in muscle strength based on QMT measurements by a mean of −14.9%. After 6 months of CAMPATH treatment the mean total muscle strength scores changed by a −1.9% from baseline ($P = 0.002$). At month 12 from CAMPATH initiation (6 months of follow-up without therapy), the patients' strength had declined by a mean of −9.7% ($P < 0.01$) reaching almost the baseline.

Anti-CD52

Depleción linfocitos T (B) y monocitos

Leucemias de células T

INHIBITION OF MYOSTATIN WITH EMPHASIS ON FOLLISTATIN AS A THERAPY FOR MUSCLE DISEASE

LOUISE R. RODINO-KLAPAC, PhD,[1,2] AMANDA M. HAIDET, BS,[1,2] JANAIAH KOTA, PhD,[1,2] CHALONDA HANDY, BS,[1,2] BRIAN K. KASPAR, PhD,[1,2] and JERRY R. MENDELL, MD[1–3]

[1] Center for Gene Therapy, Research Institute at Nationwide Children's Hospital, 700 Children's Drive, Columbus, Ohio 43205 USA
[2] Department of Pediatrics, Ohio State University, Columbus, Ohio USA
[3] Department of Neurology, Ohio State University, Columbus, Ohio USA

Muscle Nerve **39:** 283–296, 2009

Antagonistas de la miostatina

International Myositis Classification Criteria Project

Contact information of Lead Investigator:
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Fax : +46- 8- 5177 3080

GENOME-WIDE ASSOCIATION STUDIES OF THE IDIOPATHIC INFLAMMATORY MYOPATHIES

NIAMS Grant

Figure 1. Polymorphic SNP genotypes in samples from 617 IIM cases and 1151 controls. SNPs are plotted according to chromosomal location and with the –log10 p-values by Fisher's exact testing.

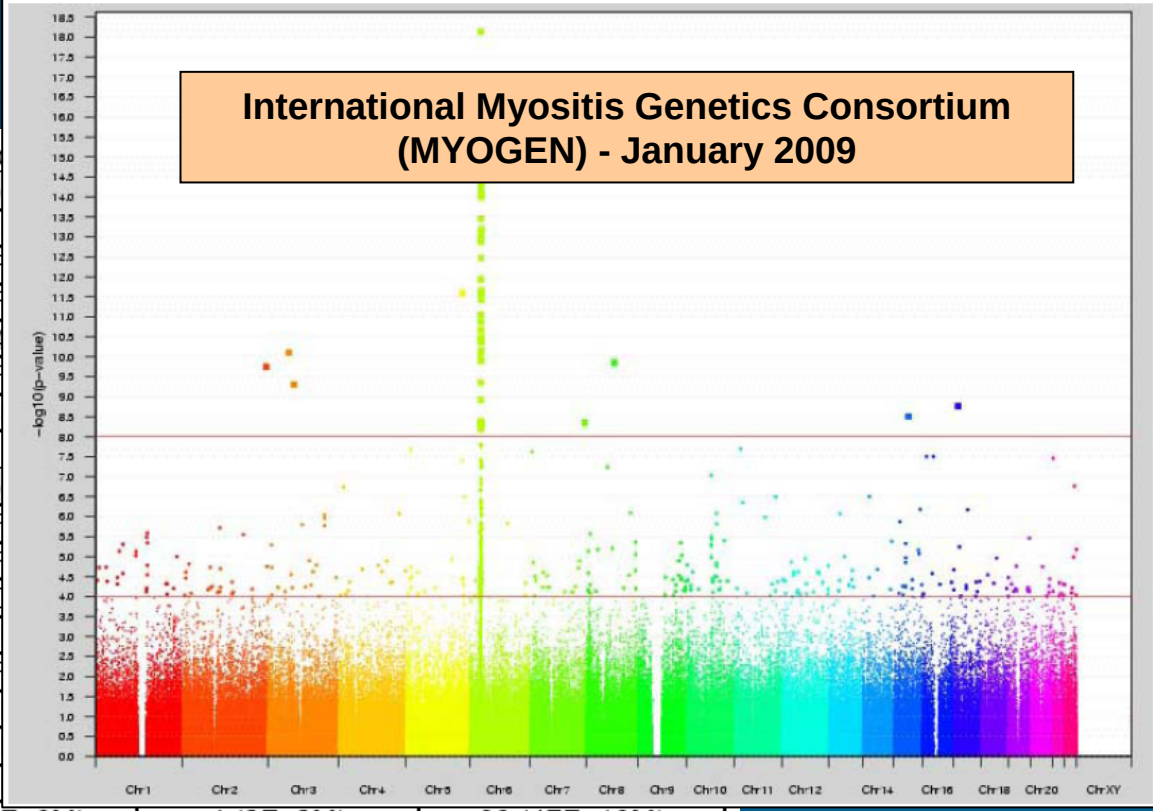

Table 2. MYOGEN Caucasian myositis subje

Aim 1 Invest.	*Polymyositis*	*Dermato*
Cooper et al.	225 (155F, 70M)	110 (78
Danko	80 (60F, 20M)	45 (30F
Lundberg	64 (48F, 16M)	42 (31F
NIH	213 (142F, 71M)	204 (164
Vencovsky	59 (41F, 18M)	80 (63F
Total Aim 1	**641 (446F, 195M)**	**481 (366**
Aim 2 Invest.	*Polymyositis*	*Dermato*
Oddis et al.	60 (35F, 25M)	82 (60F
NIH	75 (55F, 20M)	25 (15F
Pachman	0	(
Radstake et al.	20 (18F, 2M)	14 (11
Reed/Ytterberg	20 (10F, 10M)	55 (43F
Reveille	45 (37F, 8M)	34 (30
Selva*	10 (5F, 5M)	45 (38
Senécal et al.	40 (30F 10M)	31 (24
Walter et al.	32 (21F, 11M)	30 (24F, 6M)	4 (2F, 2M)	66 (47F, 19M)
Wedderburn et al.	0	0	170 (121F, 49M)	170 (121F, 49M)
Wiendl	0	8 (5F, 3M)	0	8 (5F, 3M)
Werth	0	29 (27F, 2M)	0	29 (27F, 2M)
Total Aim 2	**302 (211F, 91M)**	**353 (277F, 76M)**	**488 (337F, 151M)**	**1143 (825F, 318M)**
Total Aims 1+2	**943 (657F, 286M)**	**834 (643F, 191M)**	**714 (514F, 200M)**	**2491 (1814F, 677M)**