Banco de Tejidos CIEN: 2007 - 2023

Alberto Rábano

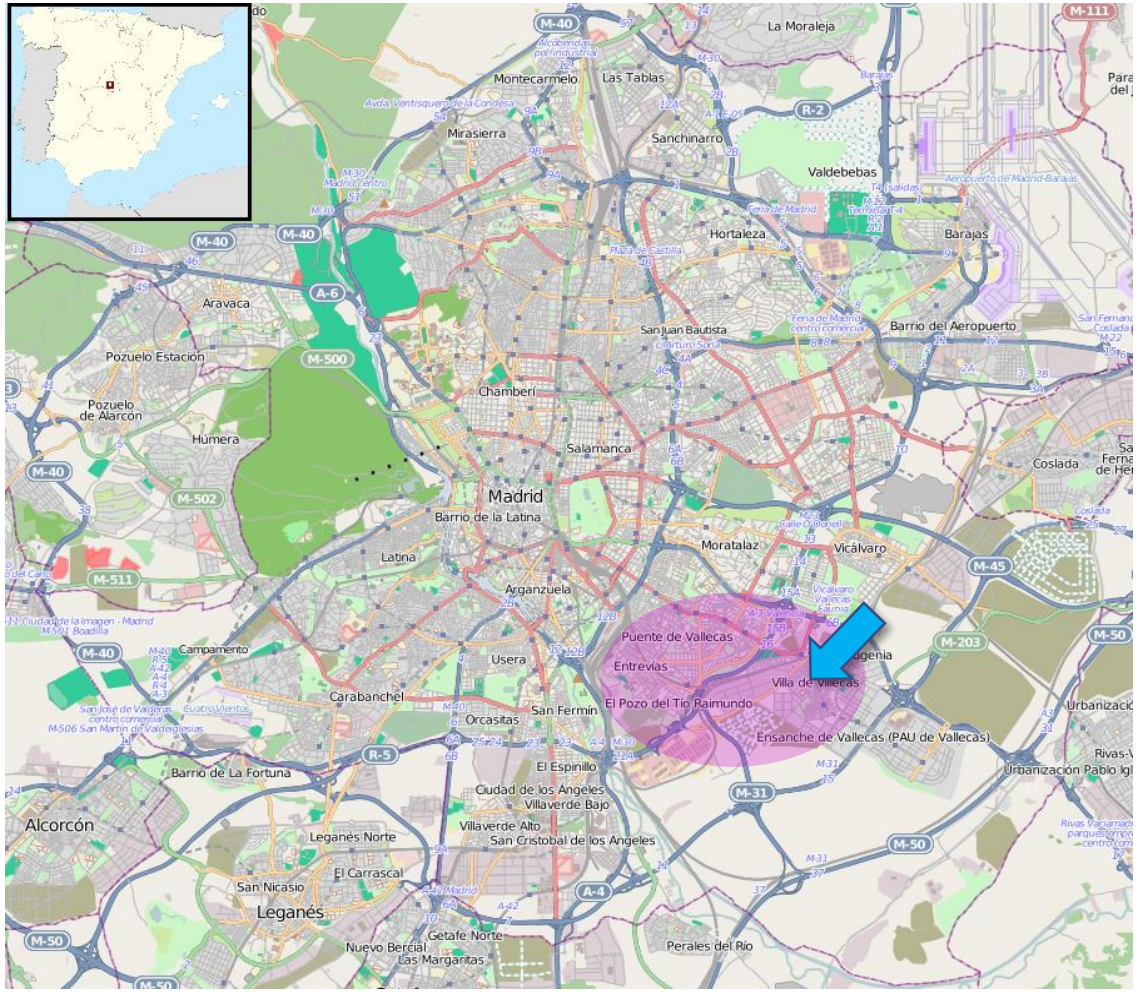
Fundación CIEN, Instituto de Salud Carlos III

Madrid

Máster en Investigación en Medicina Traslacional UCM

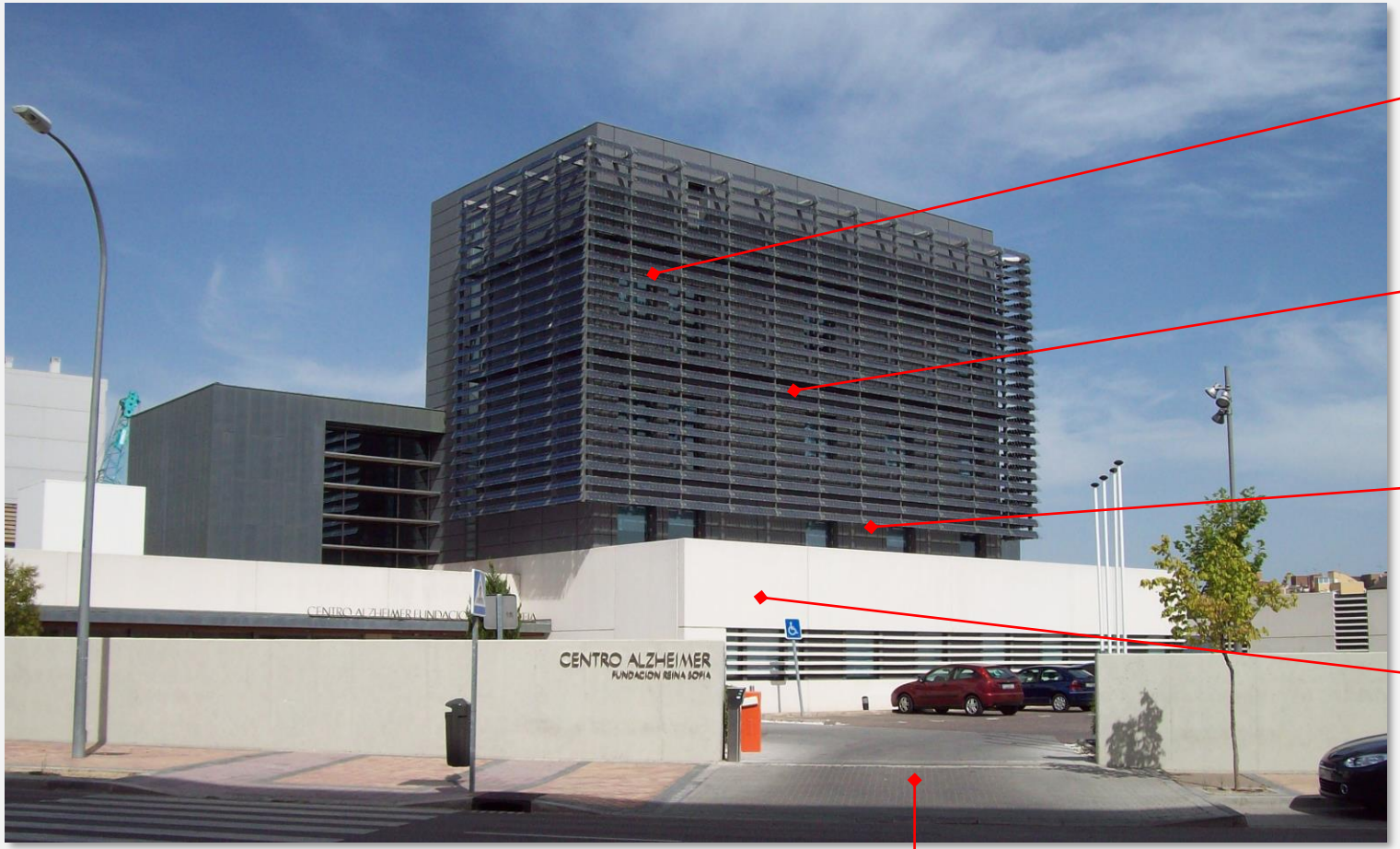
Febrero 2023

Madrid - Vallecas



Centro Alzheimer Fundación Reina Sofía

Fundación CIEN



Tercera planta:
Laboratorios

Segunda planta:
Despachos, Extracciones

Primera planta:
Despachos clínicos

Planta baja:
Dirección, Administración, Sala de seminarios

Planta sótano:
Neuroimagen, Banco de Tejidos

La iniciativa de Vallecas: programas de investigación

El Proyecto Alzheimer FRS

- Una residencia para pacientes con demencia.
- Una cohorte de pacientes institucionalizados para la investigación en demencia.

El Banco de Tejidos CIEN

- Un banco de cerebros de enfermedades neurodegenerativas.
- Muestras neurológicas de pacientes incluidas en cohortes de investigación.

El Proyecto Vallecas

- Un estudio longitudinal de envejecimiento cognitivo.
- Voluntarios para la investigación en demencia.

El Proyecto Alzheimer Fundación Reina Sofía

Cohorte Alzheimer de Vallecas (CAV – CAFRS)

Comunidad de Madrid

CONSEJERÍA DE FAMILIA, JUVENTUD Y POLÍTICA SOCIAL

Instituto de Salud Carlos III

Centro Alzheimer FRS

Fundación CIEN

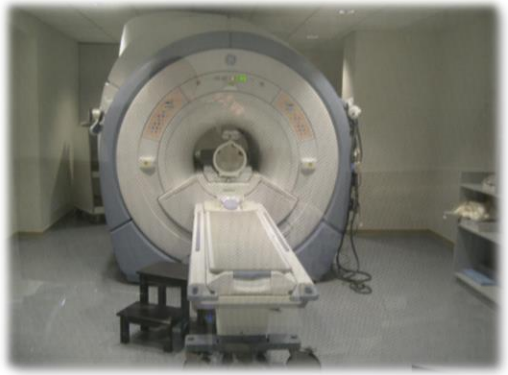
Unidad Multidisciplinar de Apoyo

Seguimiento Cohorte CAV - CAFRS

Datos de seguimiento

Resonancia Magnética 3T

Muestras sangre, cerebro



Journal of Alzheimer's Disease 28 (2012) 211–222
DOI 10.3233/JAD-2011-110875
IOS Press

Promoting Research in Advanced Dementia: Early Clinical Results of the Alzheimer Center Reina Sofía Foundation

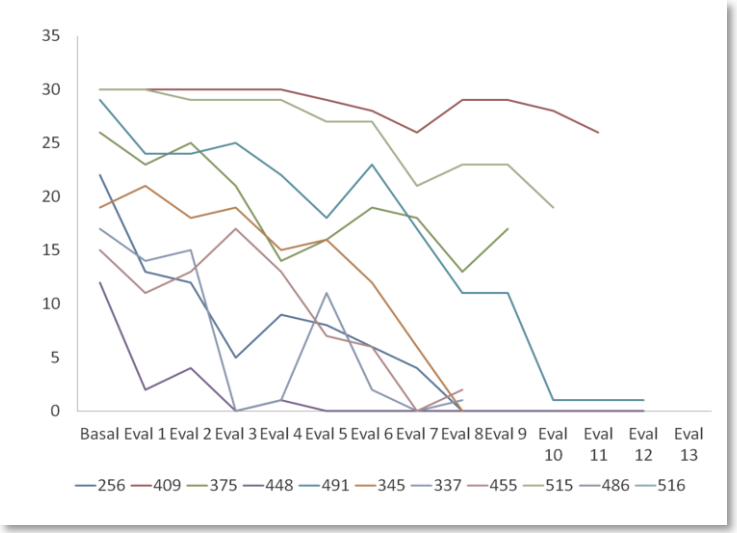
Javier Olazarán[a,*], Luis Agüera-Ortiz[b], Ricardo S. Osorio[a], Beatriz León-Salas[a], José Luis Dobato[a], Isabel Cruz-Orduña[a], Belén González[a], Meritxell Valentí[a], Nuria Gil-Ruiz[a], Belén Frades[c], M.I. Ramos-García[a] and Pablo Martínez-Martín[c]
[a]*Alzheimer Disease Research Unit, CIEN Foundation, Carlos III Institute of Health, Alzheimer Center Reina Sofía Foundation, Madrid, Spain*
[b]*CIBERSAM, Carlos III Institute of Health, Madrid, Spain*
[c]*CIBERNED, Carlos III Institute of Health, Madrid, Spain*

Table 3
Scale measures in the final clinical protocol of the ACRSF

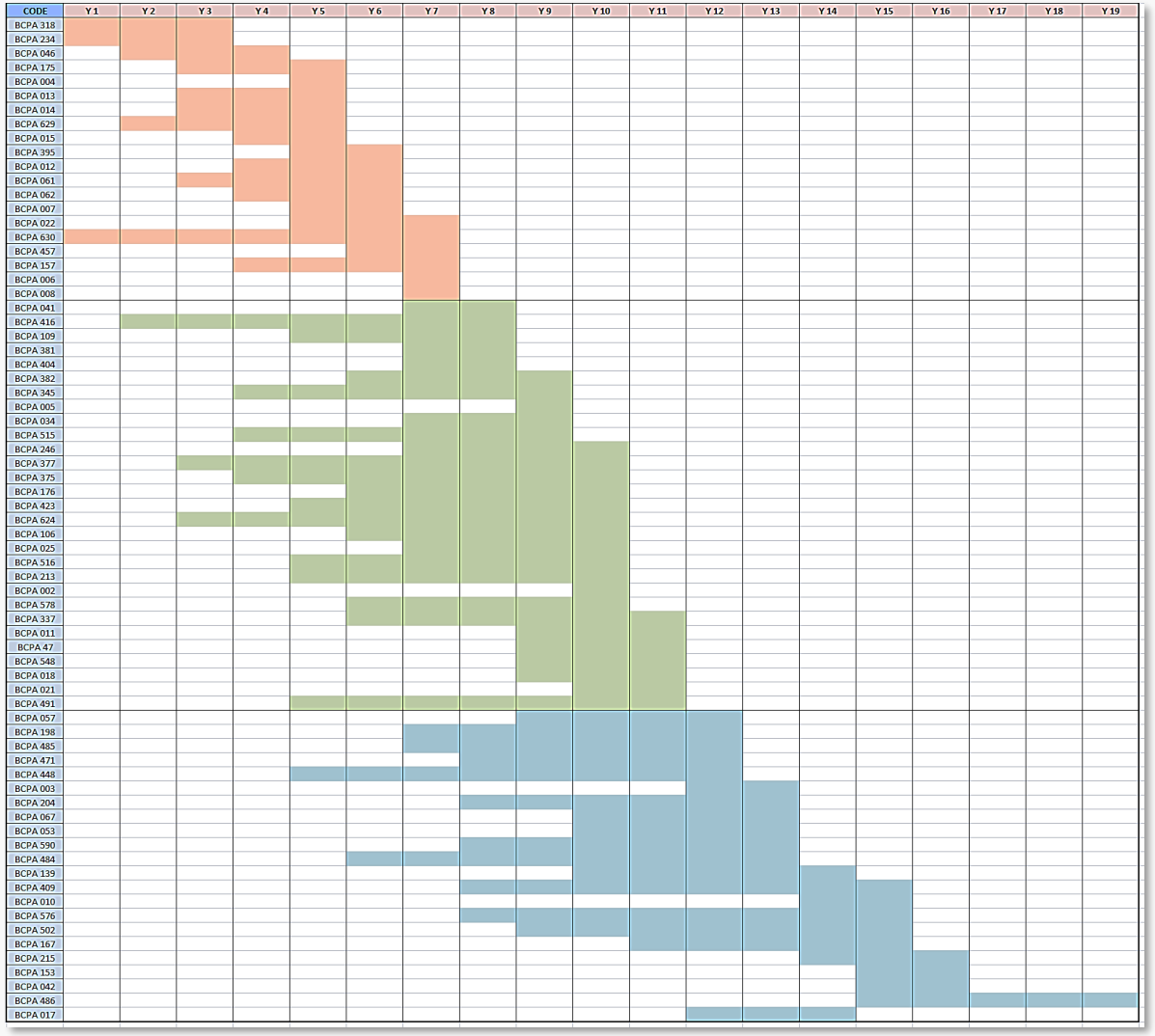
Area	Scale	Objective/Rationale	Observations[1]	References[2]
Cognition	MMSE	General cognition, universal measurement	B, 6	[17, 18]
	sMMSE	General cognition, advanced dementia	B, 6	[19, 20]
	Animals	Verbal fluency, frontotemporal functions	B, 6	[22, 23]
	SIB	General cognition, avoid floor effect	B, 6	[46, 47]
Behavior and mood	NPI	Overall picture of behavior problems	B, 6	[14–16]
	APADEM	Apathy in advanced dementia	B, 6	[48]
	CMAI	Agitation, detailed assessment	B, 12	[49, 50]
	CSDD	Depression, using both informant and patient observation	B, 12	[51, 52]
Personality	NEO-FFI	Premorbid personality traits, understand behavior problems	B	[56, 57]
ADL	FAST	AD specific, detailed for severe dementia	B, 6	[26, 27]
	BI	Basic ADL, sensitive to change	B, 6	[58, 59]
	IADL	Instrumental AVD	B, 6, DC	[60, 61]
Motor area	SCOPA-motor	Parkinsonism, predictor of gait dysfunction and functional dependence	B, 6	[31, 32]
	Up & Go test	Mobility, predictor of falls	B, 6	[33, 34]
	ADGS	Gait, predictor of functional dependence and QoL	B, 6	[35, 36]
	POMA	Balance, predictor of falls	B, 6	[63, 64]
QoL	QUALID	QoL in advanced dementia	B, 6, NH	[66, 67]
	QoL- AD	QoL as perceived by patient and caregiver	B, 6, DC	[41, 42]

[1]B: administered at baseline; 6: administered every six months; 12: administered every 12 months; NH: administered only to the nursing home patients; DC: administered only to the day-care patients.
[2]The original reference appears first, followed by reference of the most relevant validation studies in Spanish samples.
ACRSF: Alzheimer Center Reina Sofía Foundation; AD: Alzheimer's disease; ADL: activities of daily living; ADGS: Alzheimer's Disease Gait Scale; APADEM: Apathy in Dementia Scale; BI: Barthel Index; CMAI: Cohen-Mansfield Agitation Inventory; FAST: Functional Assessment Staging; GDS: Global Deterioration Scale; IADL: Instrumental Activities of Daily Living Scale; MMSE: Mini-mental State Examination; NEO-FFI: NEO Five-Factor Inventory; NPI: Neuropsychiatric Inventory; POMA: Tinetti Performance Oriented Mobility Assessment; QoL-AD: Quality of Life in Alzheimer's Disease Scale; QUALID: Quality of Life in Late-stage Dementia Scale; SCOPA-Motor: motor evaluation scale of the Scales for Outcomes in Parkinson's Disease; SIB: Severe Impairment Battery; sMMSE: Severe Mini-mental State Examination.

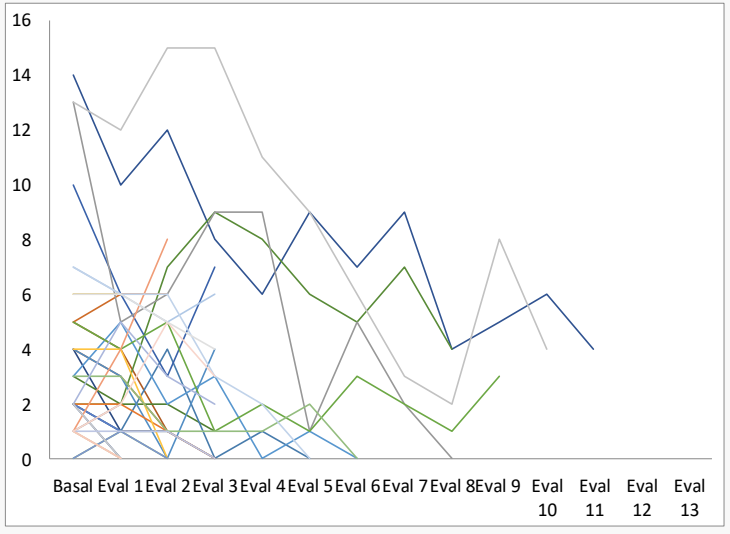
Severe Mini-mental State Examination

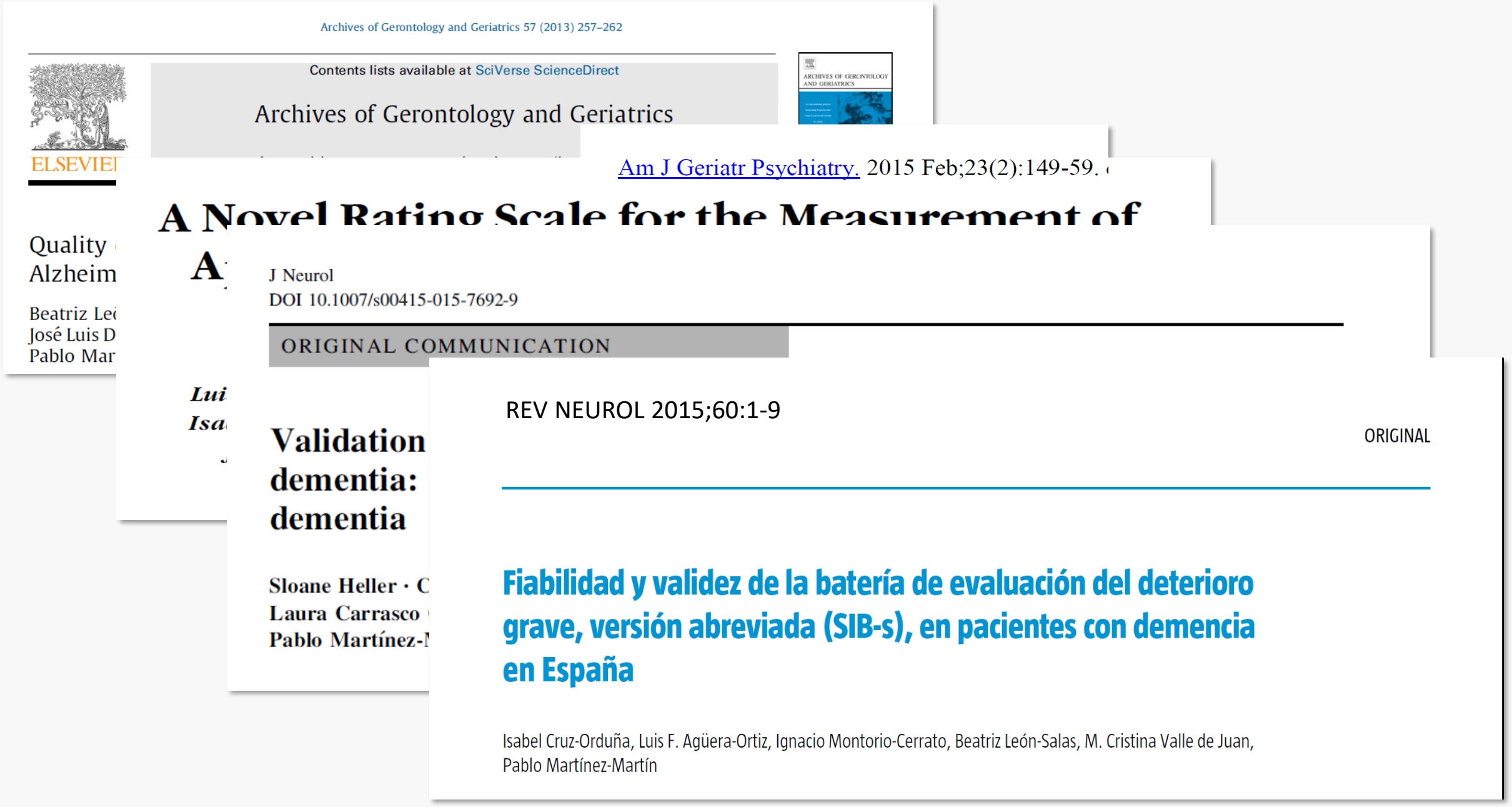


Survival time



Semantic fluency: animals





Archives of Gerontology and Geriatrics 57 (2013) 257–262

Contents lists available at SciVerse ScienceDirect

Archives of Gerontology and Geriatrics

ELSEVIER

Quality
Alzheim

Beatriz Le
José Luis D
Pablo Mar

Am J Geriatr Psychiatry. 2015 Feb;23(2):149-59.

A Novel Rating Scale for the Measurement of
A

Lui
Isa

J Neurol
DOI 10.1007/s00415-015-7692-9

ORIGINAL COMMUNICATION

Validation
dementia:
dementia

Sloane Heller · C
Laura Carrasco
Pablo Martínez-

REV NEUROL 2015;60:1-9

ORIGINAL

Fiabilidad y validez de la batería de evaluación del deterioro grave, versión abreviada (SIB-s), en pacientes con demencia en España

Isabel Cruz-Orduña, Luis F. Agüera-Ortiz, Ignacio Montorio-Cerrato, Beatriz León-Salas, M. Cristina Valle de Juan, Pablo Martínez-Martín

American Journal of Alzheimer's

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ELSEVIER

Alzheimer's & Dementia ■ (2015) 1-9

Alzheimer's & Dementia

C

Cerebral Microbleeds in
Clinical and Pathologica

Inmaculada Boyano, MD, PhD[1], Ar
Jorge López-Alvarez, MD[2,3], Carolin
Emma Osa-Ruiz, BSc[2], Irene Rodrígu
Almudena Pérez, BSc[2], Eva Alfayate
Laura Fernández, PsyD[2], Luis Agüer
Alberto Rábano, MD, PhD[2], and Javi

Journal of Alzheimer's Disease xx (2020) x–xx
DOI 10.3233/JAD-200600
IOS Press

Pathological Correlations of Neuropsychiatric Symptoms in Institutionalized People with Dementia

Ester Esteban de Antonio[a], Jorge López-Álvarez[b], Alberto Rábano[c], Luis Agüera-Ortiz[b,d], Antonio Sánchez-Soblechero[a], Laura Amaya[a], Sofía Portela[a], Carlos Cátedra[a] and Javier Olazarán[a,e,*]

[a]*Neurology Service, University Hospital Gregorio Marañón, Madrid, Spain*
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[e]*Memory Disorders Unit, HM Hospitals, Madrid, Spain*

Algunas cifras de la CAV-CAFRS…

540 pacientes con demencia incluidos en la cohorte.

3738 evaluaciones realizadas por la UMA.

68% de pacientes con RM (3T).

3058 muestras de sangre (**52812** alícuotas de hemoderivados).

168 cerebros extraídos (**50%** con RM seriadas previas).

26 exitus (17%) en la primera ola.

64 pacientes con COVID-19 sintomático o asintomático (cohorte actual, 43%).

El Proyecto Vallecas

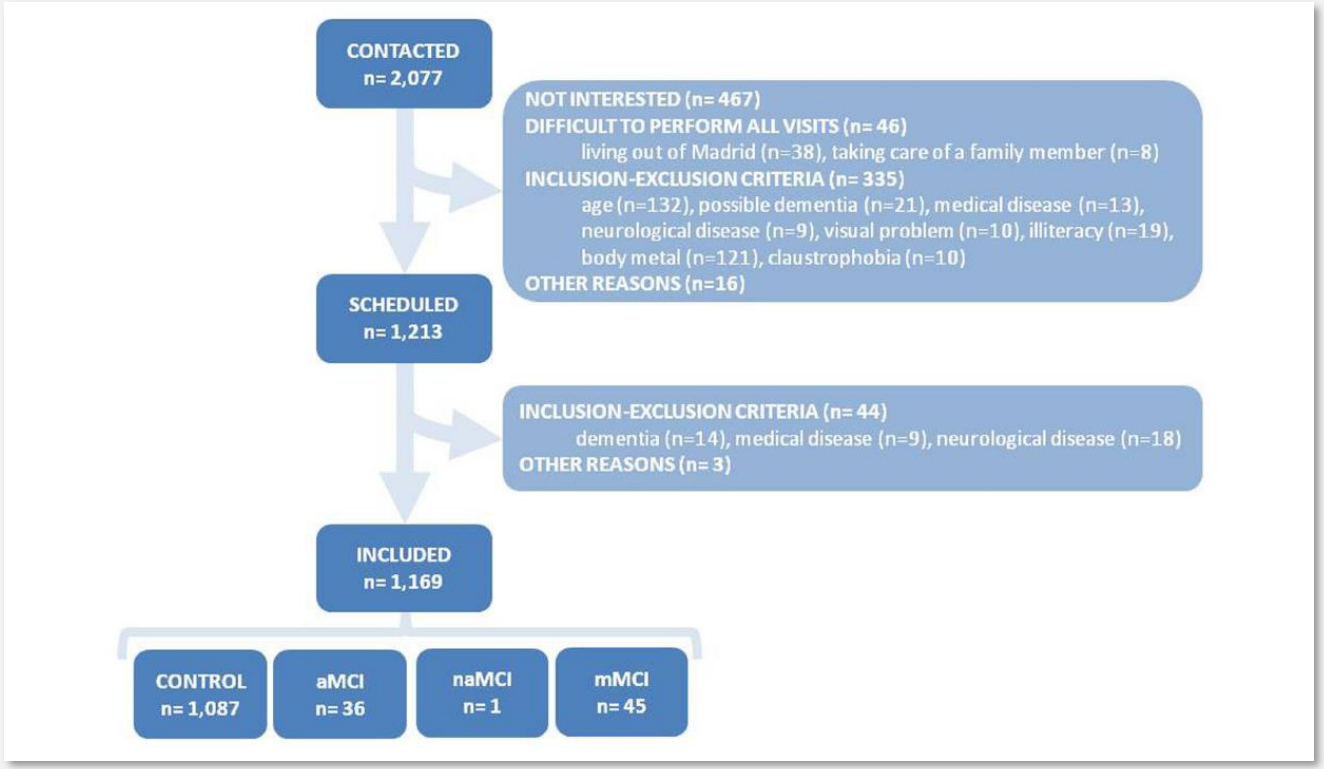
frontiers in Aging Neuroscience

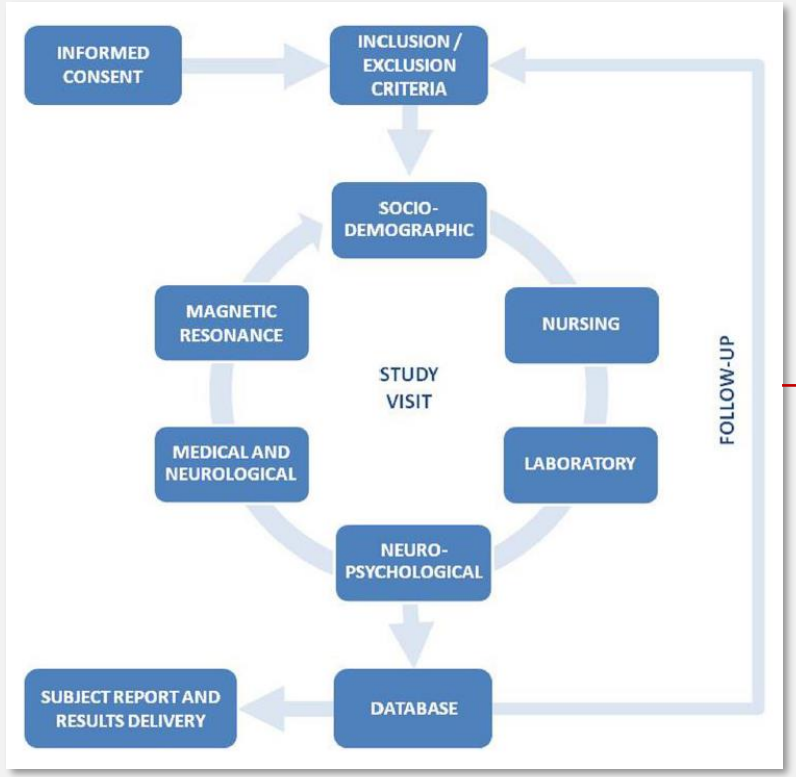
ORIGINAL RESEARCH
published: 30 September 2015
doi: 10.3389/fnagi.2015.00181

The Vallecas Project: a cohort to identify early markers and mechanisms of Alzheimer's disease

Javier Olazarán[1], Meritxell Valentí[2], Belén Frades[2], María Ascensión Zea-Sevilla[2], Marina Ávila-Villanueva[2], Miguel Ángel Fernández-Blázquez[2], Miguel Calero[2], José Luis Dobato[2], Juan Antonio Hernández-Tamames[3], Beatriz León-Salas[2], Luis Agüera-Ortiz[2], Jorge López-Álvarez[2], Pedro Larrañaga[4], Concha Bielza[4], Juan Álvarez-Linera[5] and Pablo Martínez-Martín[6]*

[1] Gregorio Marañón University Hospital, Madrid, Spain, [2] Alzheimer's Center Reina Sofía Foundation – CIEN Foundation and CIBERNED, Carlos III Institute of Health, Madrid, Spain, [3] Laboratory of Medical Imaging Analysis and Biometrics, Rey Juan Carlos University, Móstoles, Spain, [4] Department of Artificial Intelligence, Technical University of Madrid, Boadilla del Monte, Spain, [5] Department of Neuroimaging, Hospital Ruber Internacional, Madrid, Spain, [6] National Center of Epidemiology and CIBERNED, Carlos III Institute of Health, Madrid, Spain





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Gerontology

Clinical Section: Research Article

Gerontology
DOI: 10.1159/000513182

Received: July 14, 2020
Accepted: November 14, 2020
Published online: January 11, 2021

Residence, Clinical Features, and Genetic Risk Factors Associated with Symptoms of COVID-19 in a Cohort of Older People in Madrid

Teodoro del Ser[a] Miguel A. Fernández-Blázquez[a] Meritxell Valentí[a] María Ascensión Zea-Sevilla[a] Belén Frades[a] Eva Alfayate[a] Laura Saiz[a] Olga Calero[b, c] Fernando José García-López[d] Alberto Rábano[a] Miguel Medina[a, b] Miguel Calero[a, b, c]

[a]Alzheimer's Disease Investigation Research Unit, CIEN Foundation, Institute of Health Carlos III, Queen Sofia Foundation Alzheimer Research Center, Madrid, Spain; [b]Centro de Investigación Biomédica en Red sobre Enfermedades Degenerativas (CIBERNED), Instituto de Salud Carlos III, Madrid, Spain; [c]Chronic Disease Program, Institute of Health Carlos III, Madrid, Spain; [d]National Epidemiology Centre, Institute of Health Carlos III, Madrid, Spain

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Si desea recibir más información, se la enviaremos a la dirección que nos indique o entre en nuestra web www.bt.fundacioncien.es

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Instituto de Salud

Programas de donación de tejido cerebral

Programa de donación interno

- Centro Alzheimer Fundación Reina Sofía
- Seguimiento semestral, RM, muestras de sangre

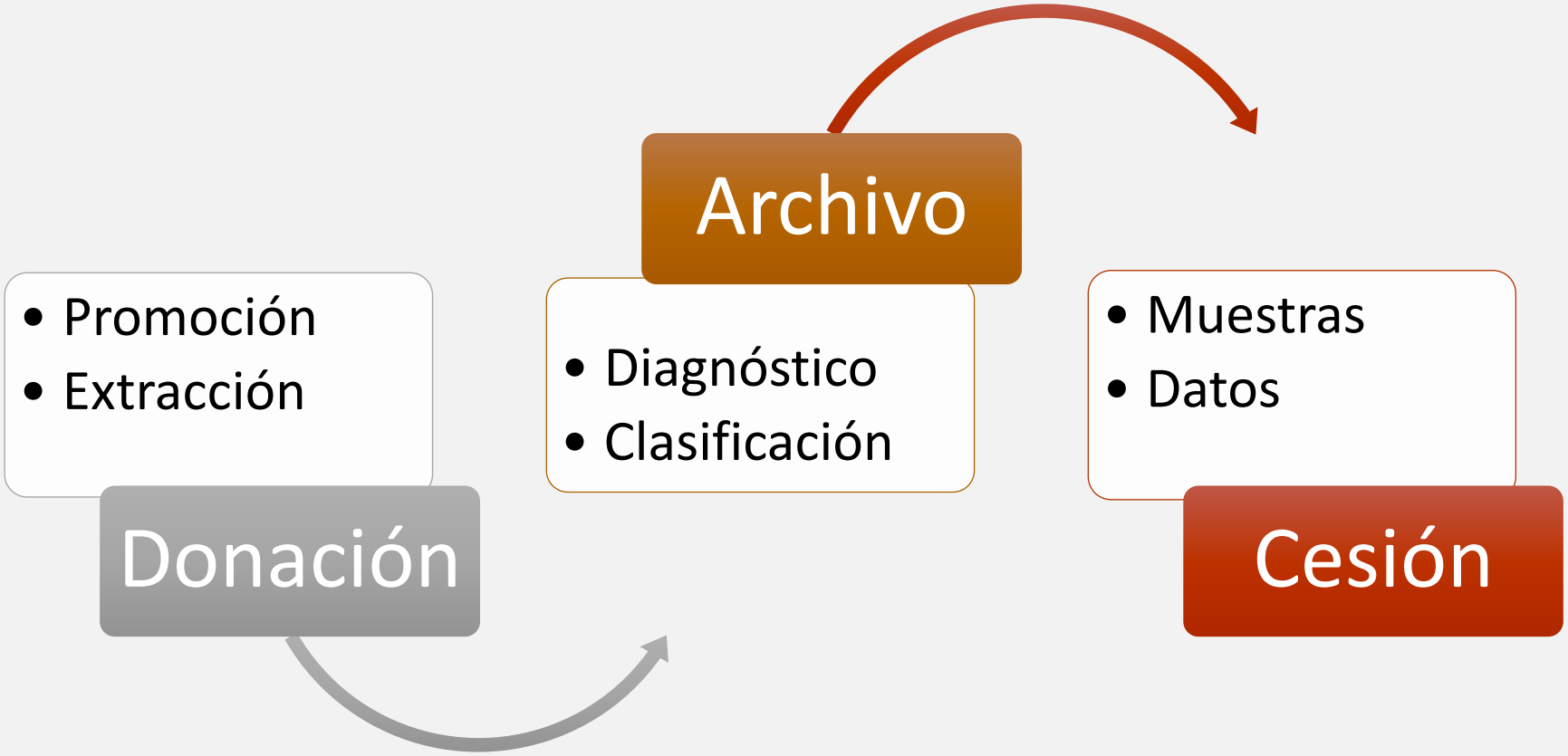
168

Programa de donación externo

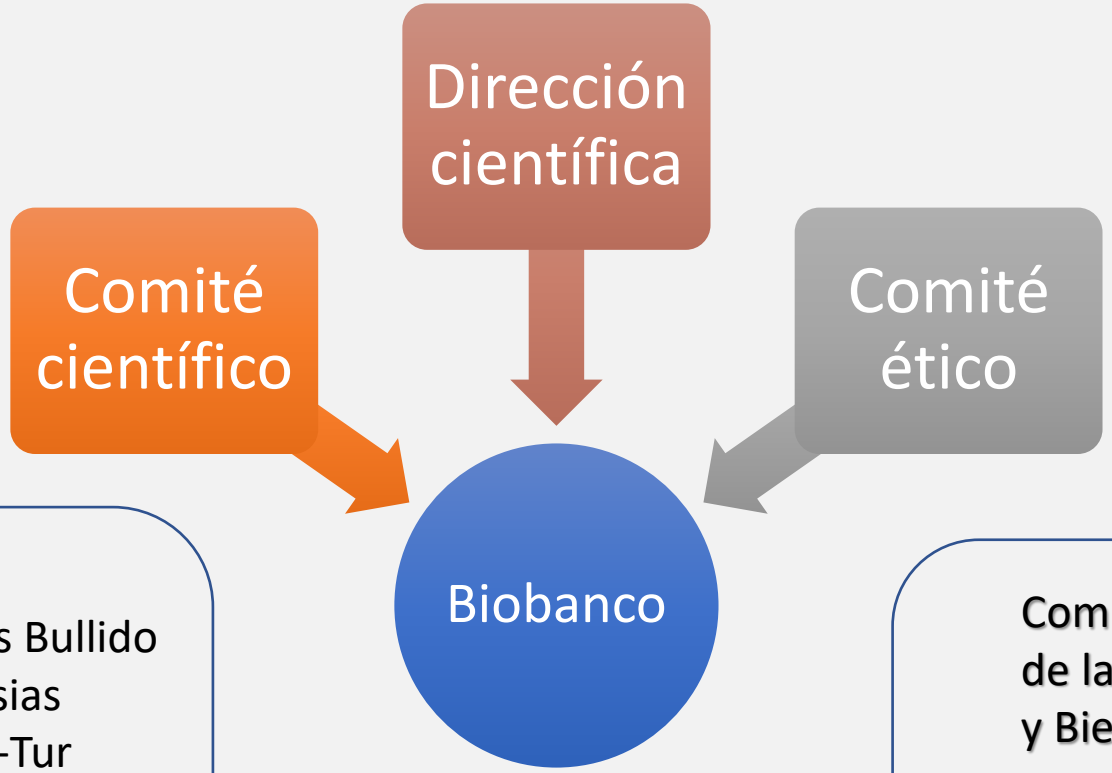
- Población general, residencias, hospitales
- No hay seguimiento de los donantes

565

Procedimientos básicos del BT-CIEN



BT-CIEN: Dirección y comités externos



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Teresa Iglesias
Jordi Pérez-Tur
Teodoro del Ser
Ramón Trullas
Meritxell Valentí

Secretaría:

Laura Saiz

Comité de Ética de la Investigación y Bienestar Animal

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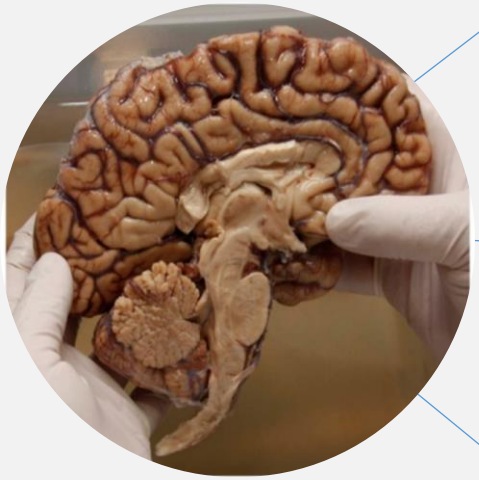
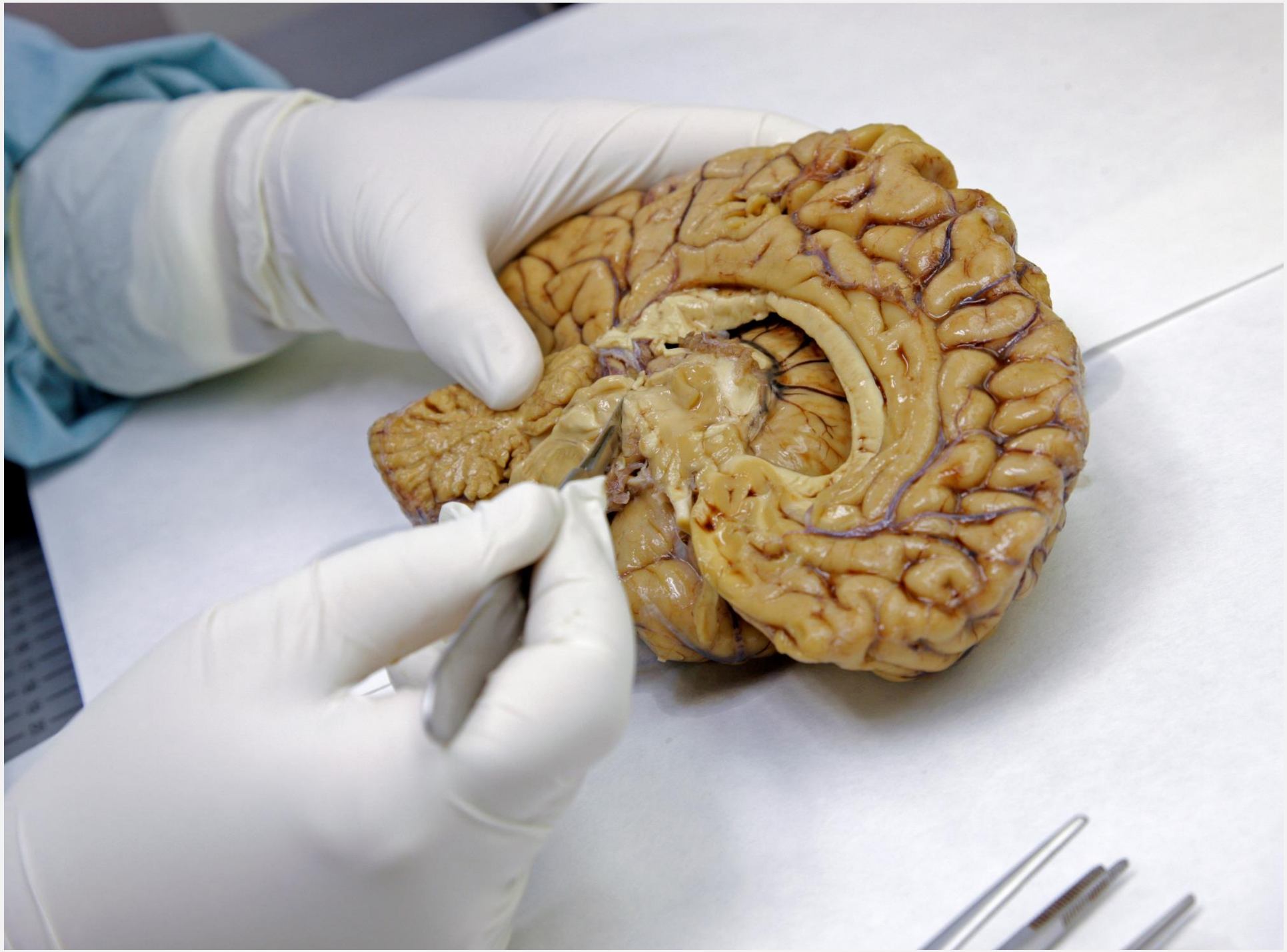
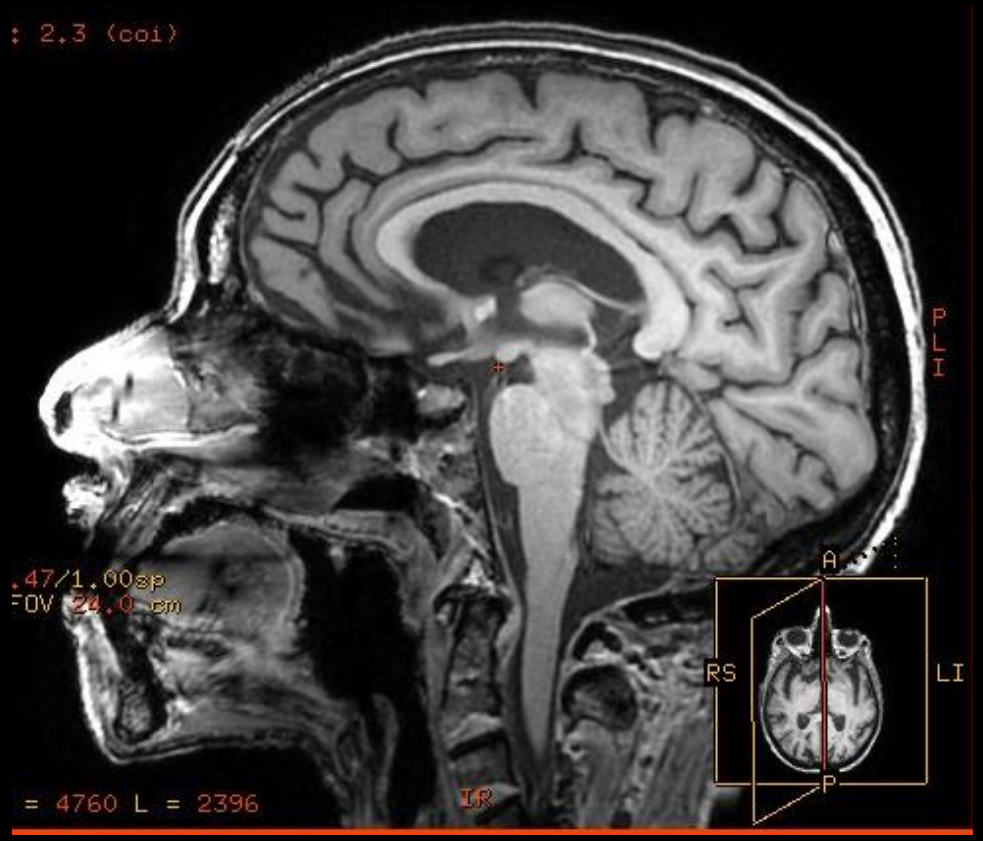
MINISTERIO DE CIENCIA E INNOVACIÓN
Instituto de Salud Carlos III

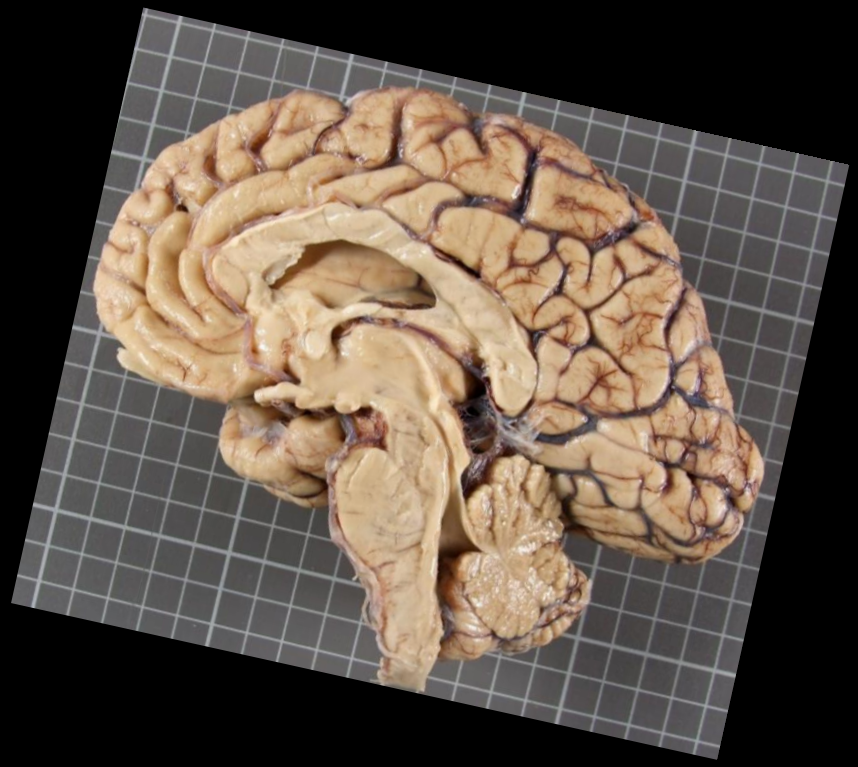


- **Familiares**
  - Informe Neuropatológico
  - Diagnóstico definitivo
  - ¿Enfermedad genética?
- **Clínicos**
  - Informe Neuropatológico
  - Correlación clínico-patológica
  - Formación (sesiones clínicas)
  - Investigación clínica
- **Investigado-res**
  - Muestras biológicas
  - Clasificación de los casos
  - Datos clínicos asociados
  - Control de calidad (muestras y datos)

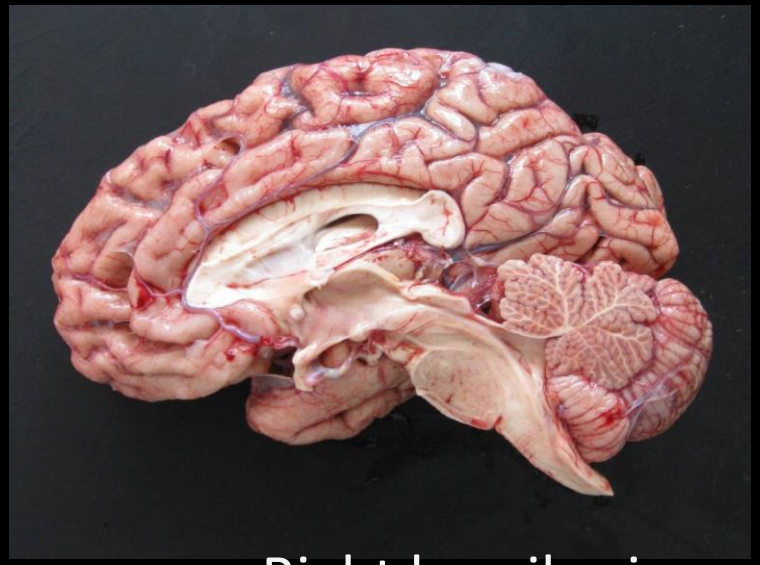




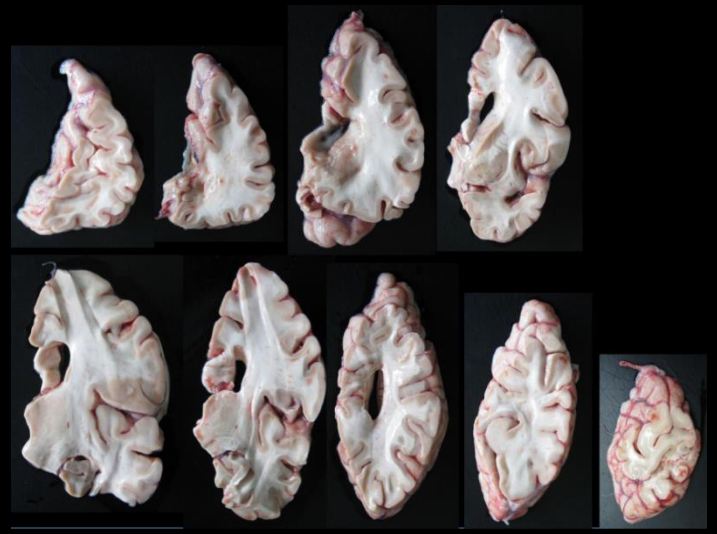
3T magnetic resonance
post mortem pre-extraction



Macroscopy of fixed brain

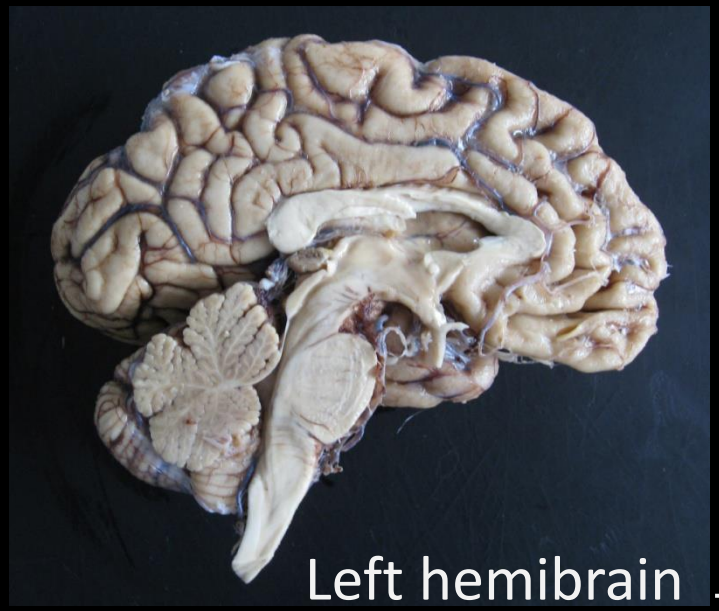


Right hemibrain

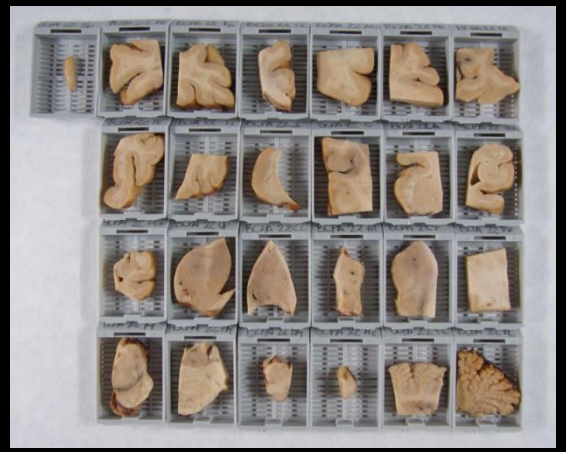


Freezing

Storage



Left hemibrain

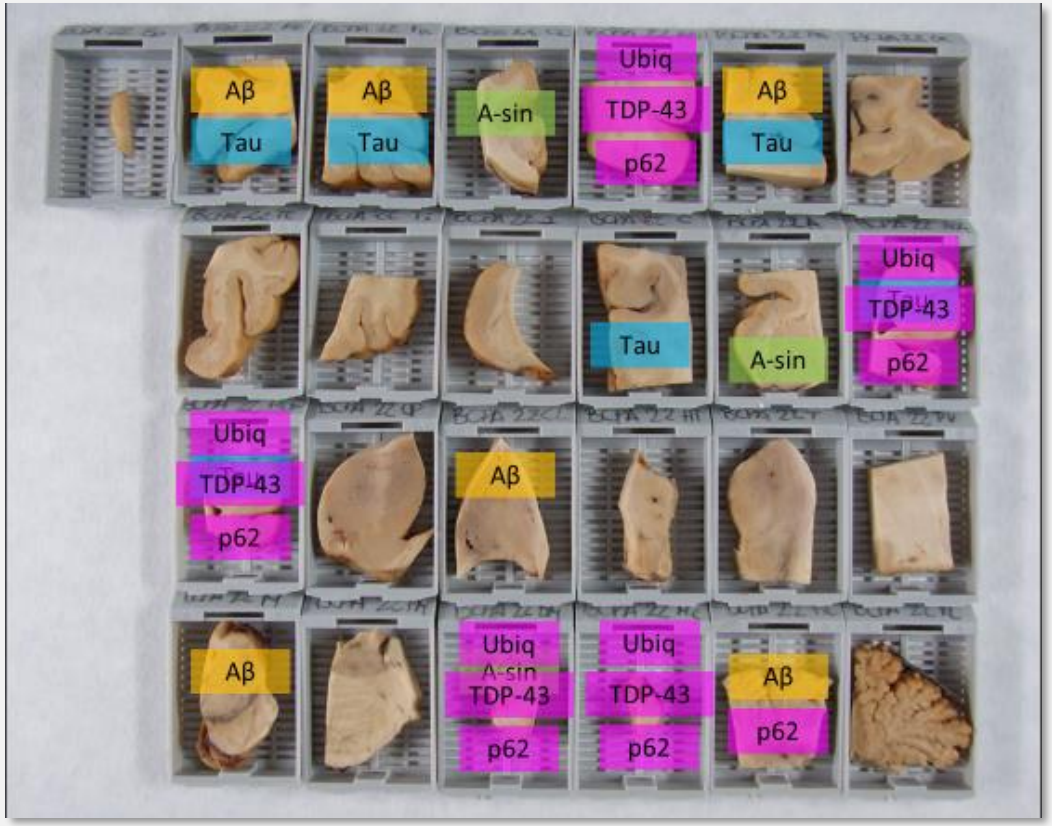


Neuropathology

Table 6. Illustrating the blocks routinely taken from fixed post-mortem brains and the stains employed in a suspected case of Alzheimer's Disease.

Block Location	Stains
1. Middle frontal gyrus	H&E, Aβ, HP-tau, p62, pTDP-43
2. Superior and middle temporal gyri	H&E, Aβ, HP-tau, p62, pTDP-43
3. Hippocampus	H&E, Aβ, HP-tau, p62, α-syn, pTDP-43
4. Parietal lobe	H&E, HP-tau, α-syn
5. Mid-brain	H&E, Aβ, α-syn
6. Superior frontal gyrus and cingulate gyrus	H&E, α-syn
7. Occipital including calcarine and paracalcarine	H&E, Aβ, HP-tau
8. Basal Ganglia	H&E, Aβ
9 Amygdala	H&E, Aβ, HP-tau, p62, α-syn, pTDP-43
10. Thalamus	(No stains)
11. Pons	H&E, α-syn
12. Medulla	H&E, α-syn
13. Cerebellar hemisphere	H&E, Aβ, p62
14. Frontal deep white matter	H&E (LFB/N-if evidence of CVD)
15. Occipital deep white matter	H&E (LFB/N-if evidence of CVD)
16. Motor cortex	(No stains)

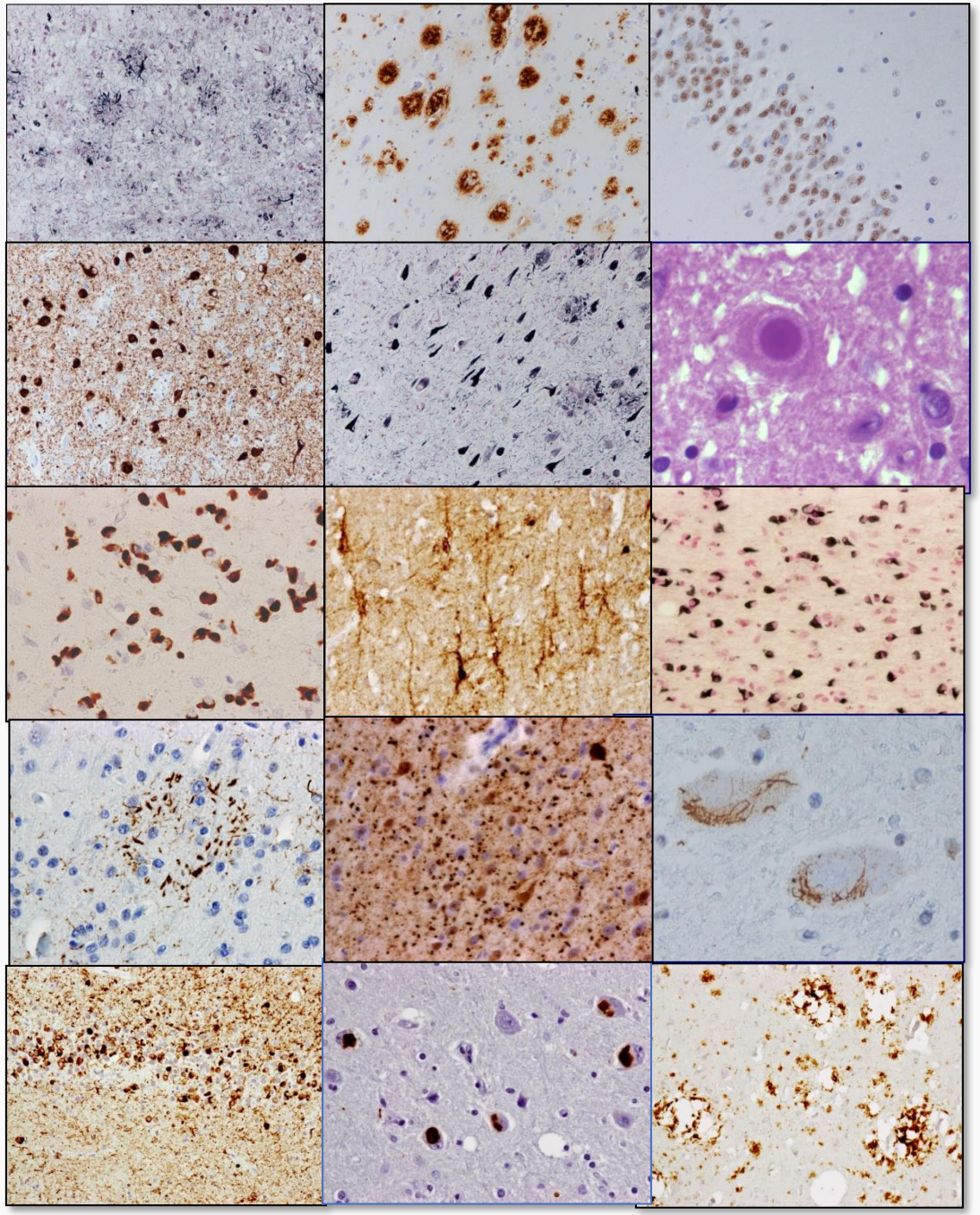
(No stains)-indicate block is taken and not routinely stained but may be if need arises. CVD-cerebrovascular disease, α-syn-α-synuclein, LFB/N-Luxol Fast Blue/Nissl.



King A *et al.*, 2020

Tinción de rutina, hematoxilina - eosina

Técnicas especiales, de plata y de inmunohistoquímica



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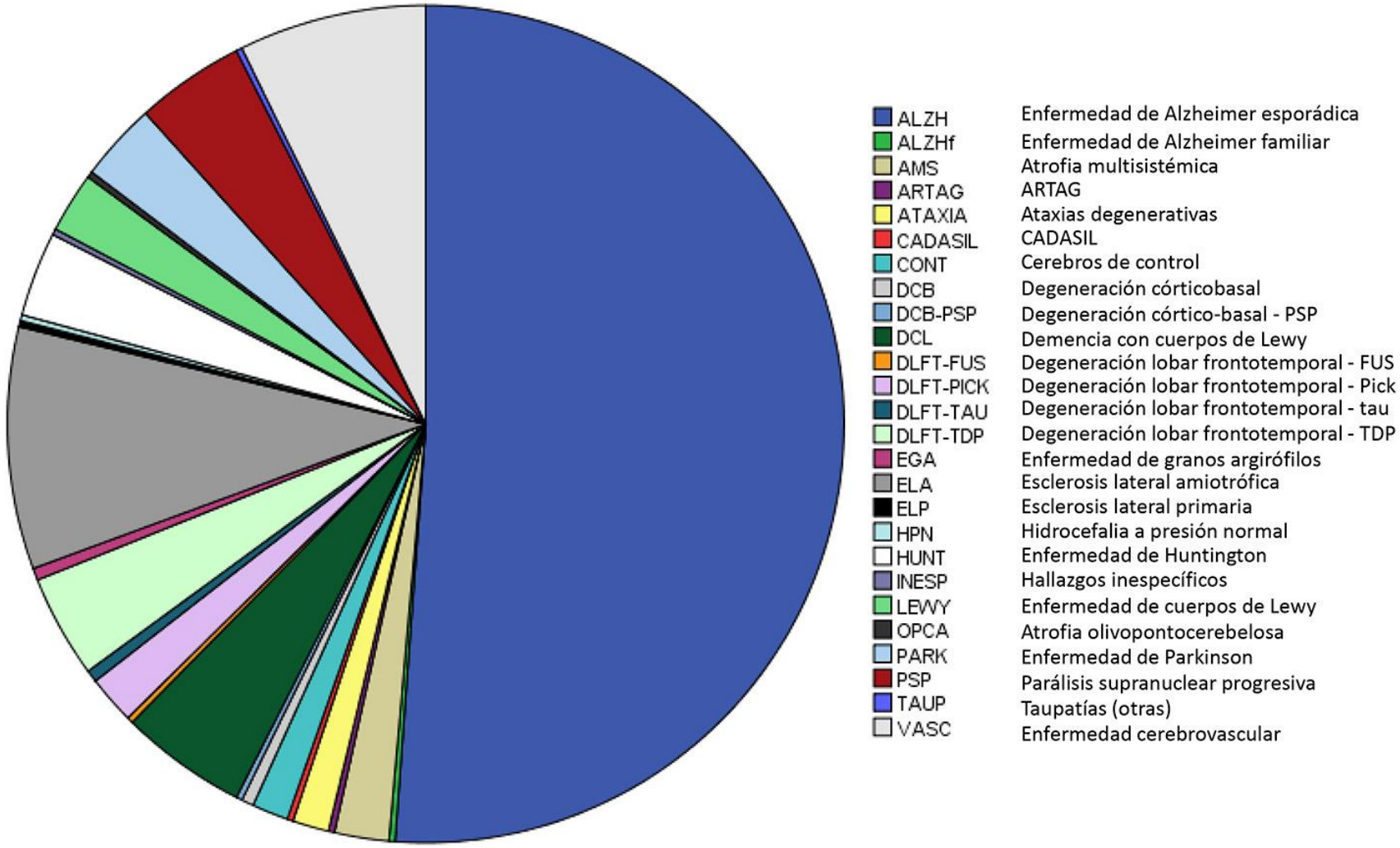
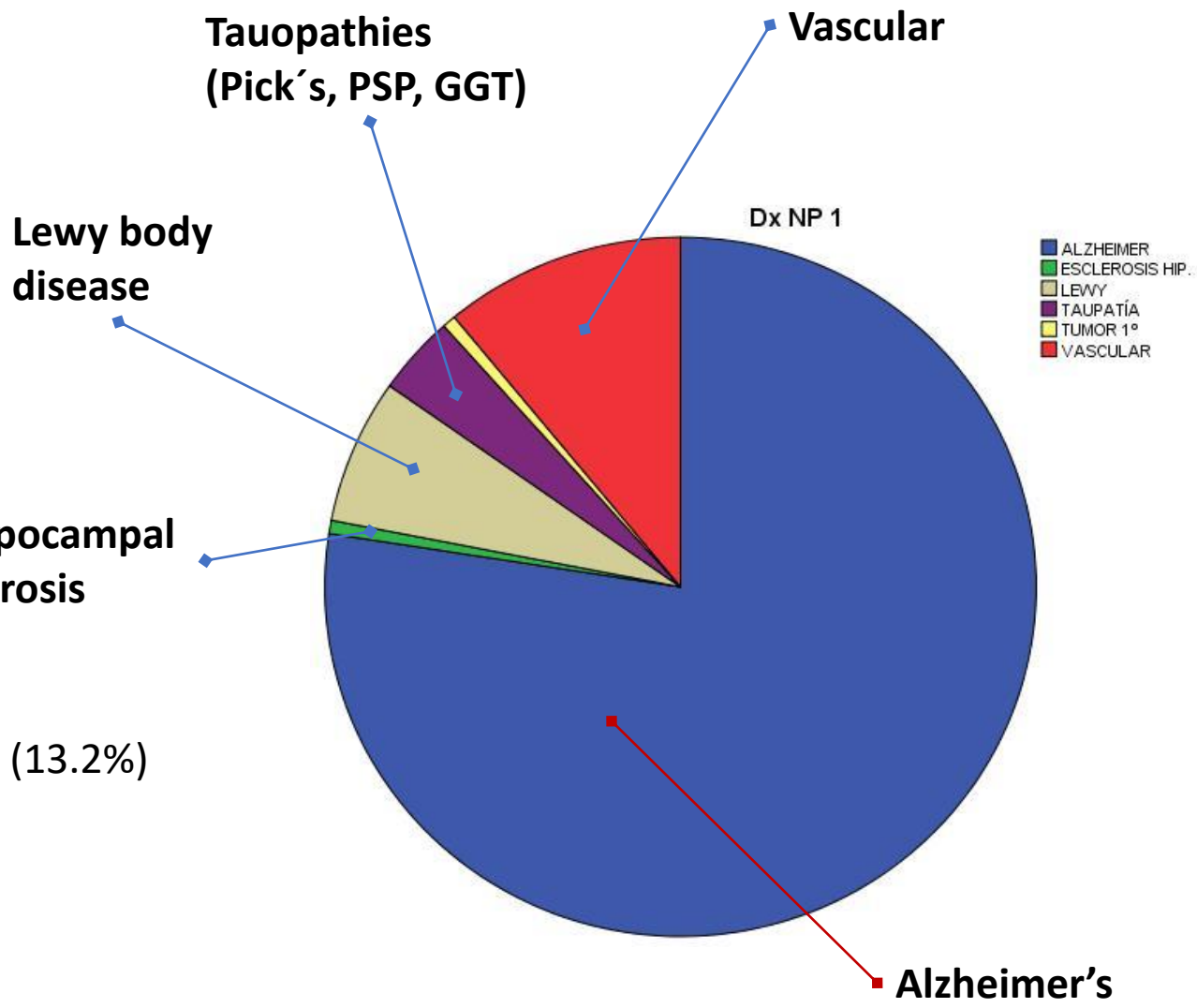
Diagnóstico neuropatológico principal



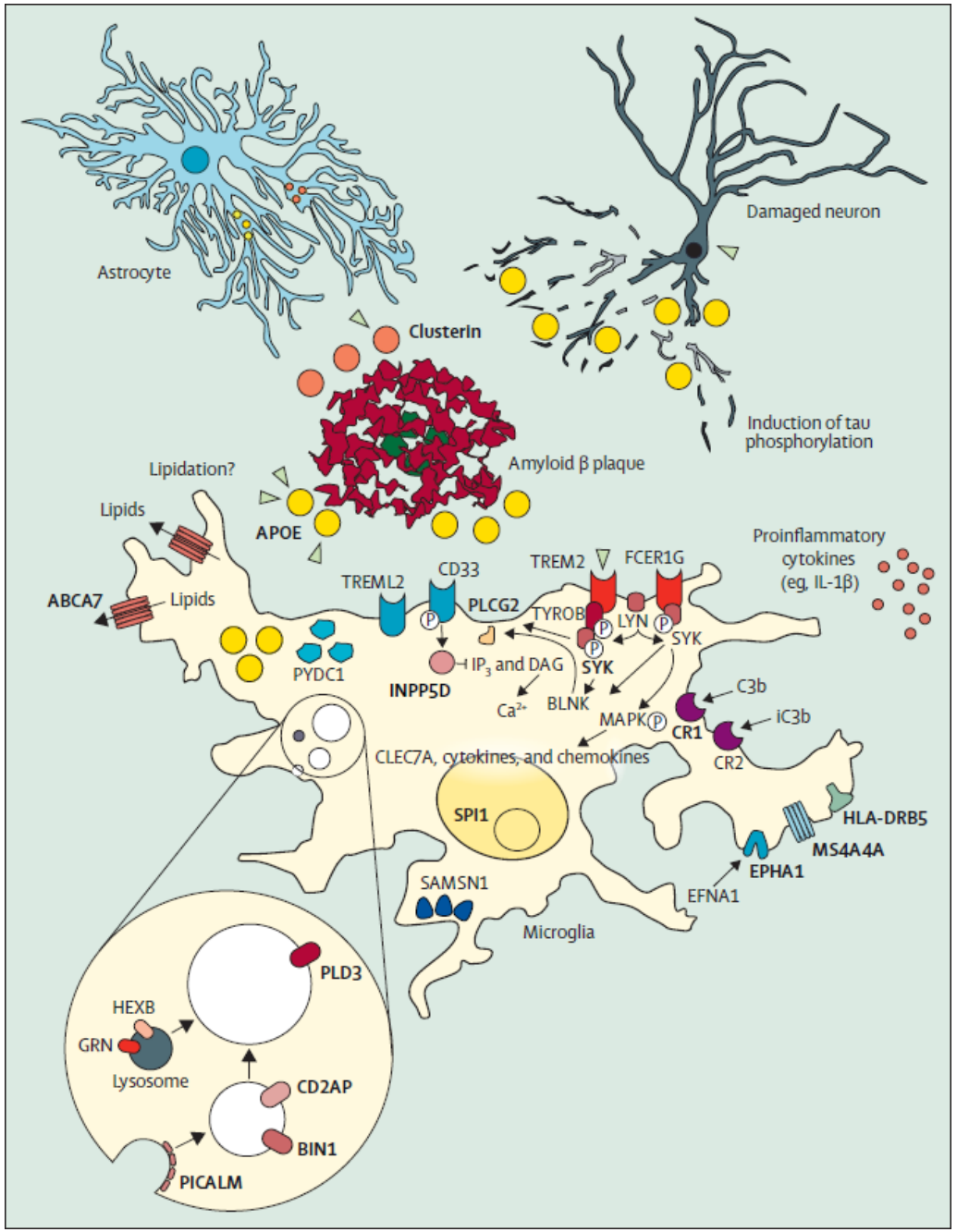
N	167
Sex	79% female
T in CAFRS (mths)	52.9 (38.6)
Age at onset	75.4 (7.3)
Age at death	87.2 (6.5)
Survival time	11.9 (4.4)
PMI (hrs.)	4.5 (2.1)
APOE e4	45.2%
High ADNC	75.8%
High vascular path.	54.5%
Lewy path. (LPC>1)*	37.8%
LATE (HS)	71.2% (45.2%)
ARTAG	52.7%
AGD	12%

*LPC =1 (13.2%)

Main neuropathological diagnosis

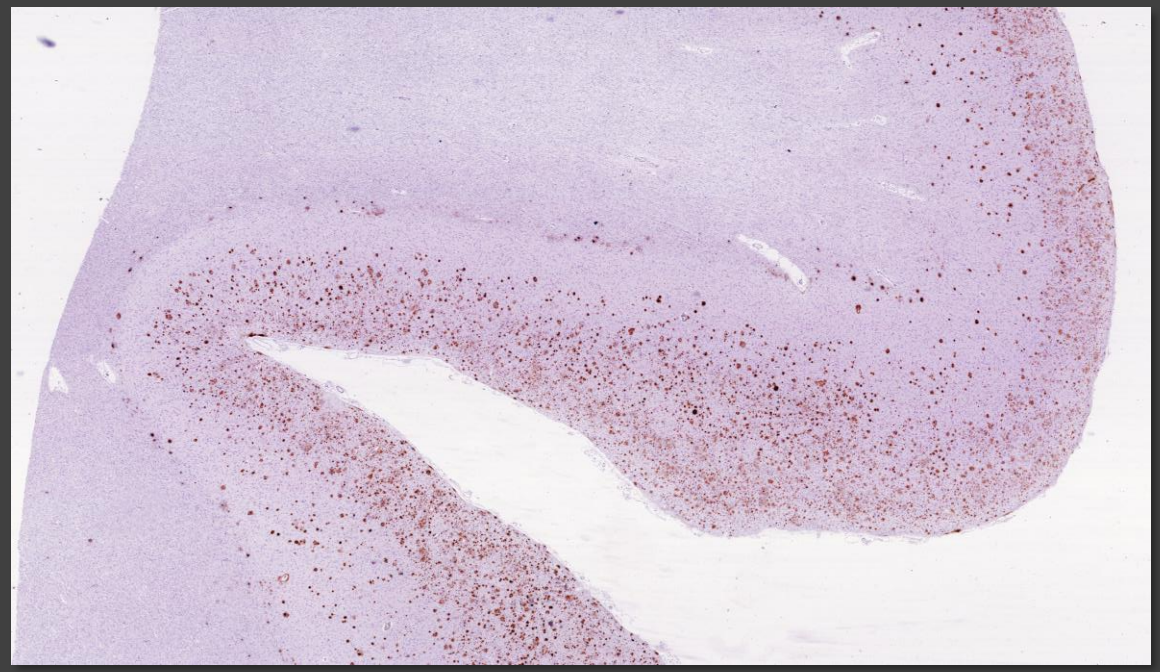


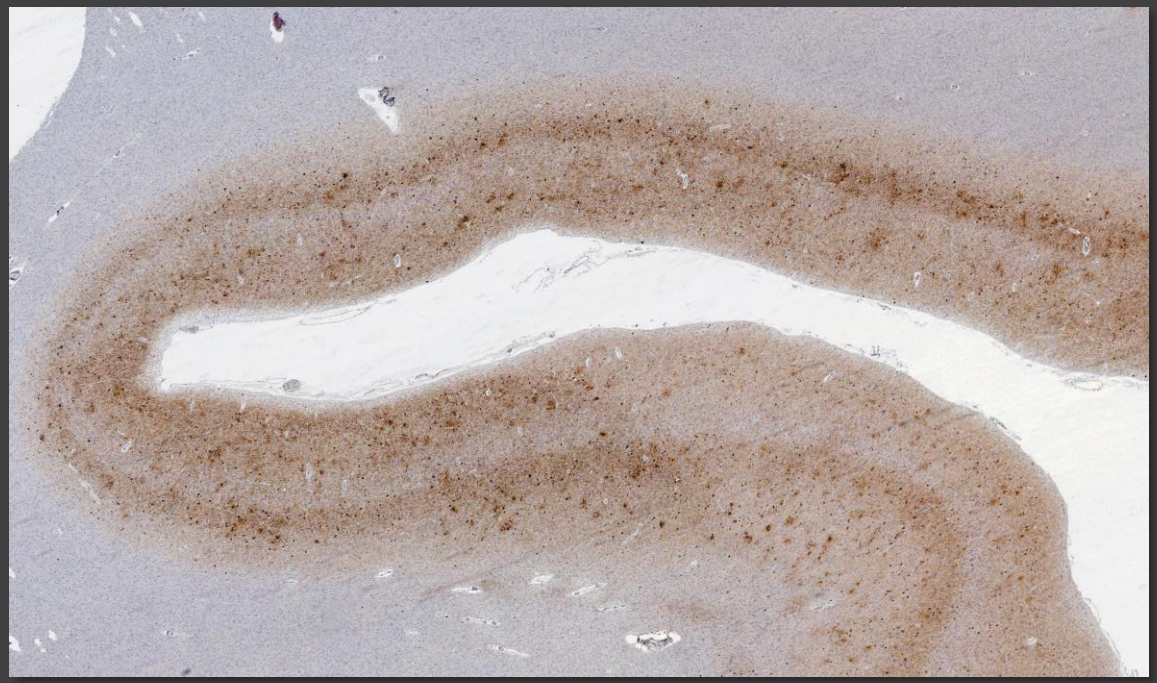
La patología de tipo Alzheimer



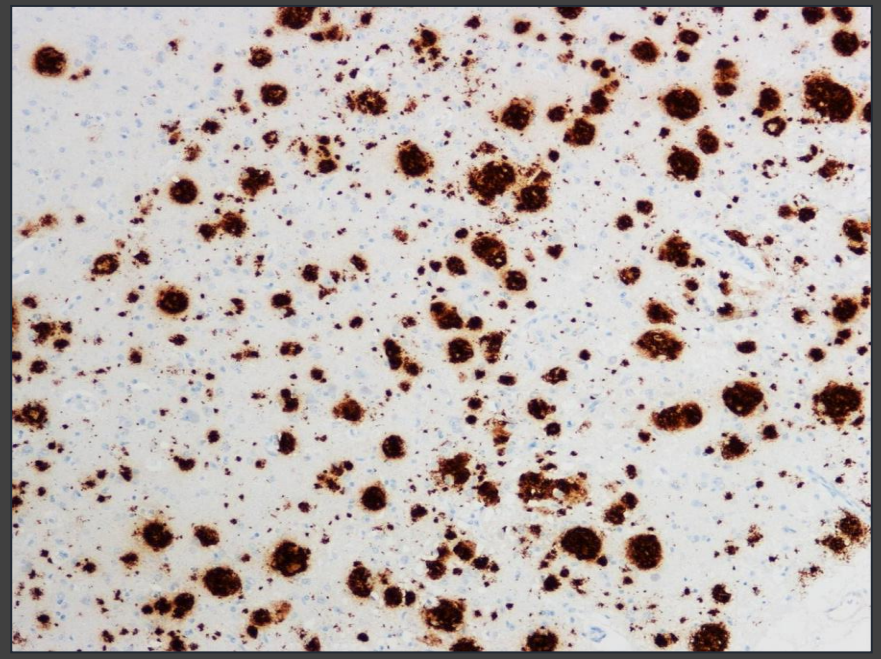
Fase celular de la enfermedad de Alzheimer

Scheltens P *et al*., 2021

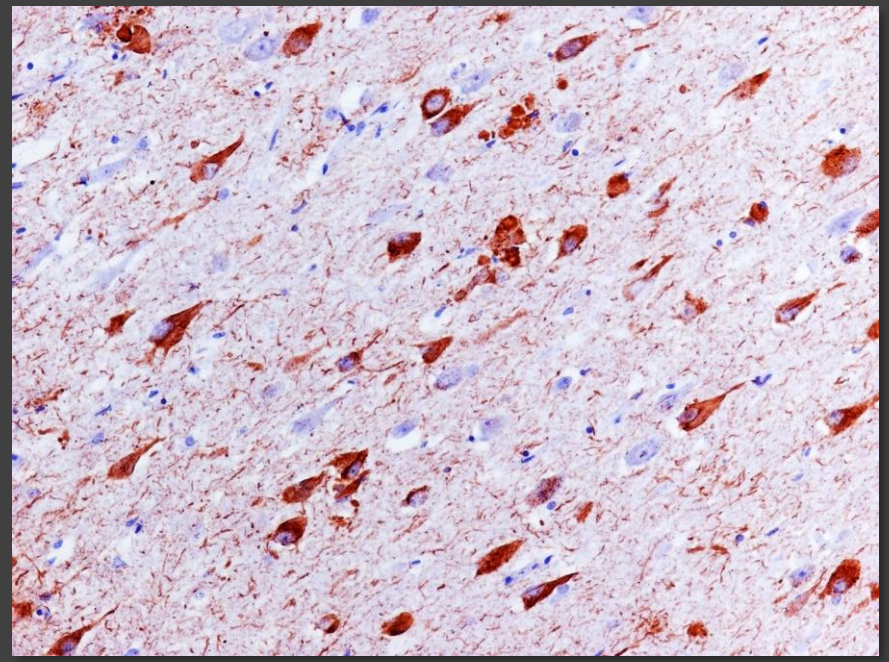




Aβ



Tau



2012

Acta Neuropathol (2012) 123:1–11
DOI 10.1007/s00401-011-0910-3

CONSENSUS PAPER

National Institute on Aging–Alzheimer's Association guidelines for the neuropathologic assessment of Alzheimer's disease: a practical approach

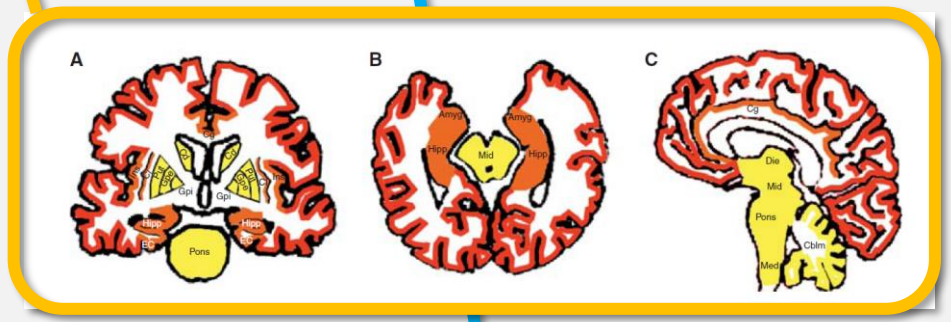
Thomas J. Montine · Creighton H. Phelps · Thomas G. Beach · Eileen H. Bigio · Nigel J. Cairns · Dennis W. Dickson · Charles Duyckaerts · Matthew P. Frosch · Eliezer Masliah · Suzanne S. Mirra · Peter T. Nelson · Julie A. Schneider · Dietmar Rudolf Thal · John Q. Trojanowski · Harry V. Vinters · Bradley T. Hyman

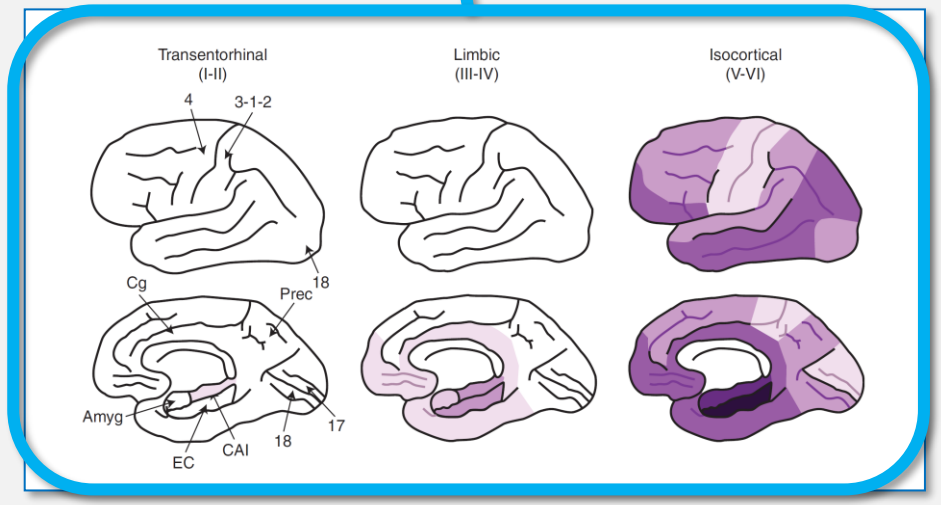
Table 2 "ABC" score for AD neuropathologic change

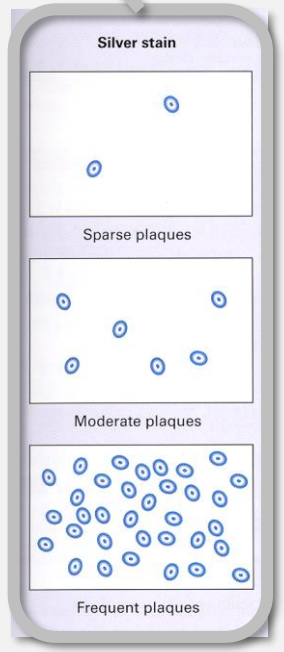
"A"	Thal Phase for Aβ plaques [57]	"B"	Braak and Braak NFT stage [14,15]	"C"	CERAD neuritic plaque score [41]
0	0	0	None	0	None
1	1 or 2	1	I or II	1	Sparse
2	3	2	III or IV	2	Moderate
3	4 or 5	3	V or VI	3	Frequent

Table 3 "ABC" score for level of AD neuropathologic change

AD neuropathologic change		B[a]		
A[b]	C[c]	0 or 1	2	3
0	0	Not[d]	Not[d]	Not[d]
1	0 or 1	Low	Low	Low[e]
	2 or 3[f]	Low	Intermediate	Intermediate[e]
2	Any C	Low[g]	Intermediate	Intermediate[e]
3	0 or 1	Low[g]	Intermediate	Intermediate[e]
	2 or 3	Low[g]	Intermediate	High

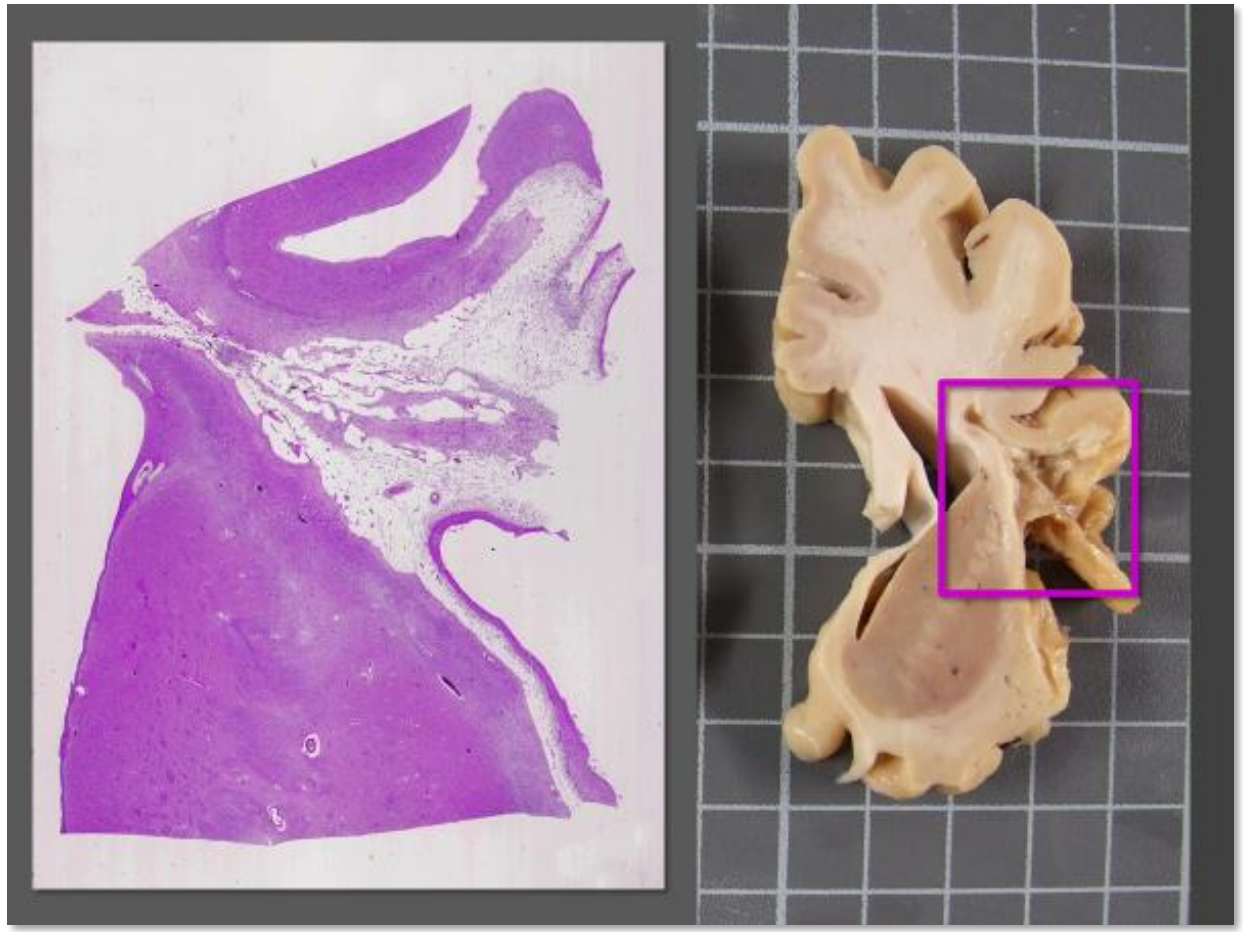






Alzheimer's disease neuropathological change: A1 B2 C3

La patología vascular cerebral



Staging and natural history of cerebrovascular pathology in dementia

2012

Neurology® 2012;78:1-1

V. Deramecourt, MD, PhD
J.Y. Slade, BSc
A.E. Oakley, MBiol
R.H. Perry, FRCPath
P.G. Ince, FRCPath
C.-A. Maurage, MD, PhD
R.N. Kalaria, FRCPath

Frontal lobe (0-6)

Temporal lobe (0-6)

Hippocampus (0-4)

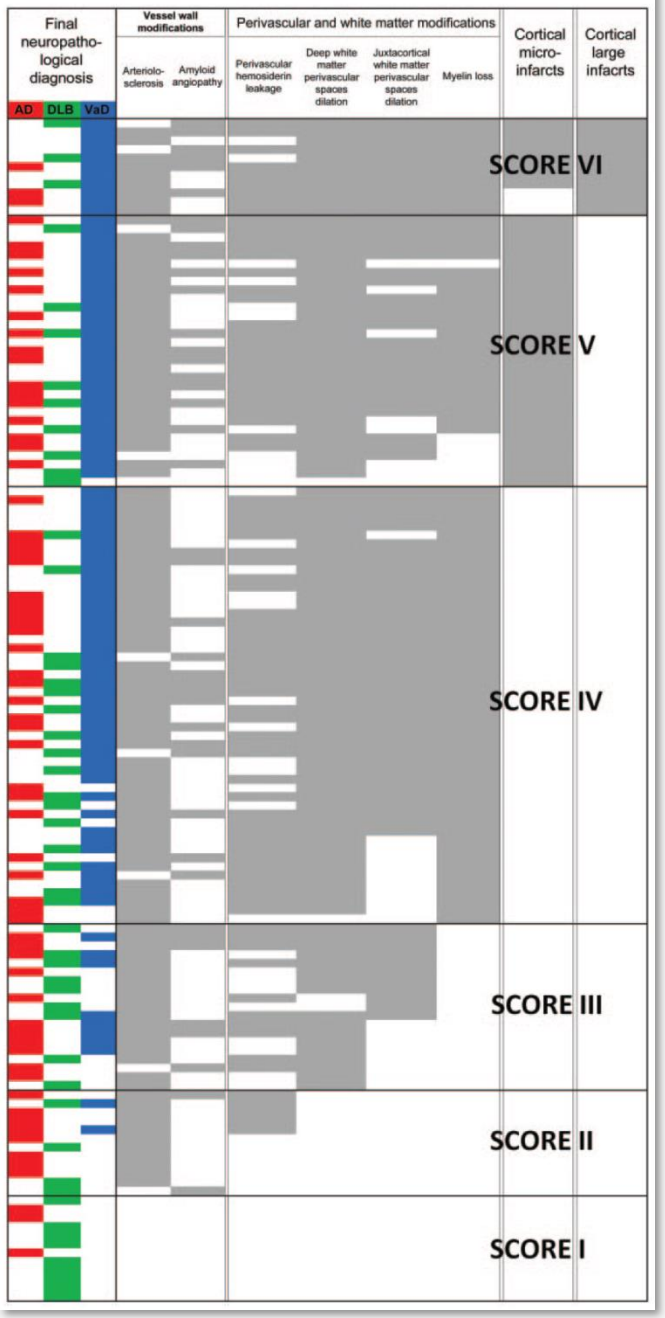
Basal ganglia (0-4)

Total score (∑) (0-20)

Table 2 Staging of the cerebrovascular lesions

Score	Staging
Frontal and temporal lobes	
0	Normal appearance of brain, vessels, white matter, and cortex
I	Mild modification of vessel walls, perivascular spaces, or white matter
II	Moderate to severe but isolated modification of the vessel walls (arteriolosclerosis or amyloid angiopathy), usually associated with hemosiderin deposits in the perivascular spaces
III	Moderate to severe perivascular space dilatations either in the deep or the juxtacortical white matter
IV	Moderate to severe myelin loss
V	Presence of cortical microinfarcts
VI	Presence of large infarcts
Hippocampus	
0	Normal appearance
I	Mild modification of vessel walls or perivascular spaces
II	Moderate to severe perivascular space dilatations
III	Presence of microinfarcts (usually in Ammon horn or the subiculum)
IV	Presence of large infarcts
Basal ganglia	
0	Normal appearance
I	Mild modification of vessel walls or perivascular spaces
II	Moderate to severe perivascular space dilatations
III	Presence of microinfarcts
IV	Presence of large infarcts
Total vascular score	
Frontal lobe + Temporal lobe + Hippocampus + Basal ganglia (/20)	

Figure 3 Distribution of the cerebrovascular lesions, example of the frontal lobe



2016

doi:10.1093/brain/aww214 BRAIN 2016: Page 1 of 13 | 1

BRAIN
A JOURNAL OF NEUROLOGY

Vascular cognitive impairment neuropathology guidelines (VCING): the contribution of cerebrovascular pathology to cognitive impairment

Olivia A. Skrobot,[1] Johannes Attems,[2] Margaret Esiri,[3] Tibor Hortobágyi,[4,5] James W. Ironside,[6] Rajesh N. Kalaria,[2] Andrew King,[7] George A. Lammie,[8] David Mann,[9] James Neal,[10] Yoav Ben-Shlomo,[11] Patrick G. Kehoe[1] and Seth Love[1]

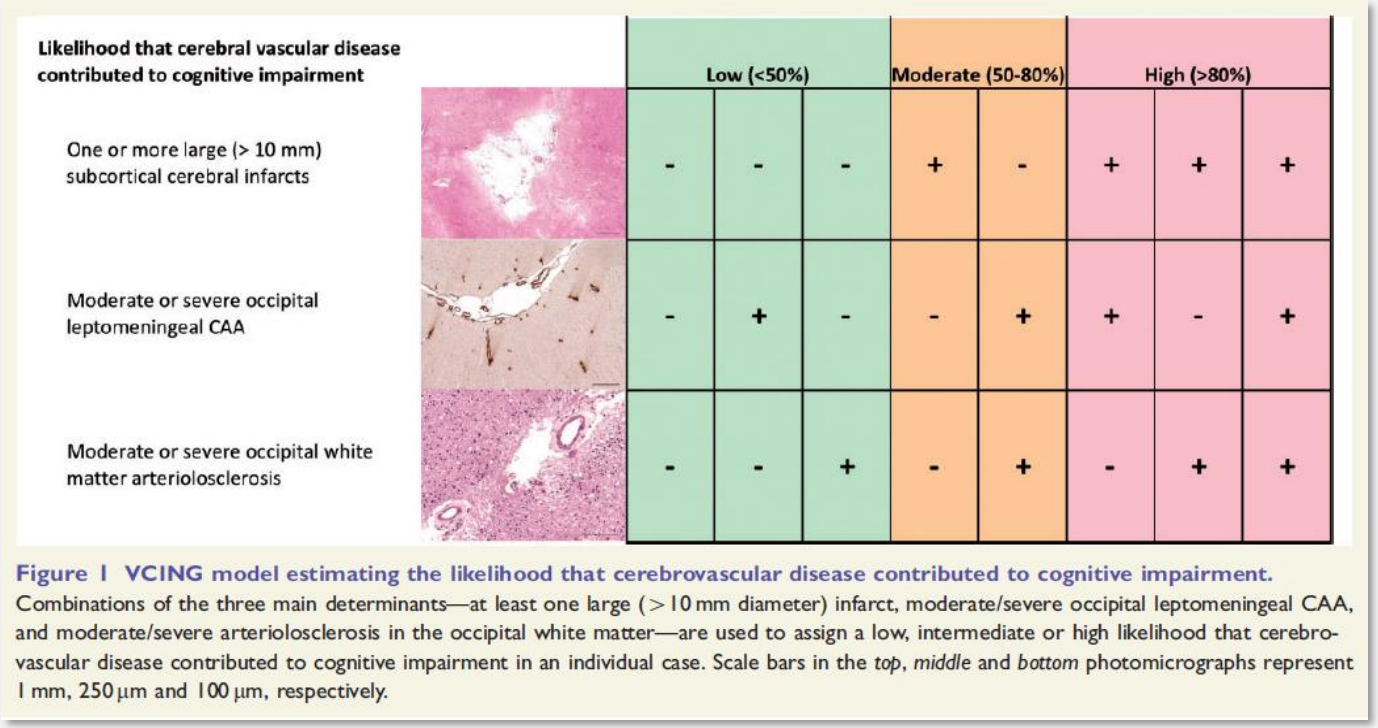


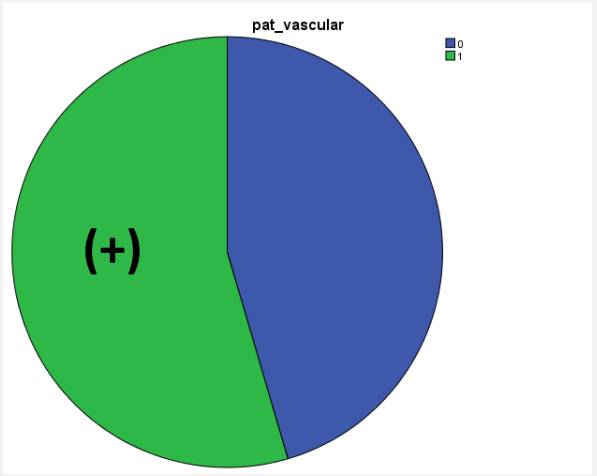
Figure 1 VCING model estimating the likelihood that cerebrovascular disease contributed to cognitive impairment. Combinations of the three main determinants—at least one large (>10 mm diameter) infarct, moderate/severe occipital leptomeningeal CAA, and moderate/severe arteriolosclerosis in the occipital white matter—are used to assign a low, intermediate or high likelihood that cerebrovascular disease contributed to cognitive impairment in an individual case. Scale bars in the *top*, *middle* and *bottom* photomicrographs represent 1 mm, 250 μm and 100 μm, respectively.

Sperman's correlation test

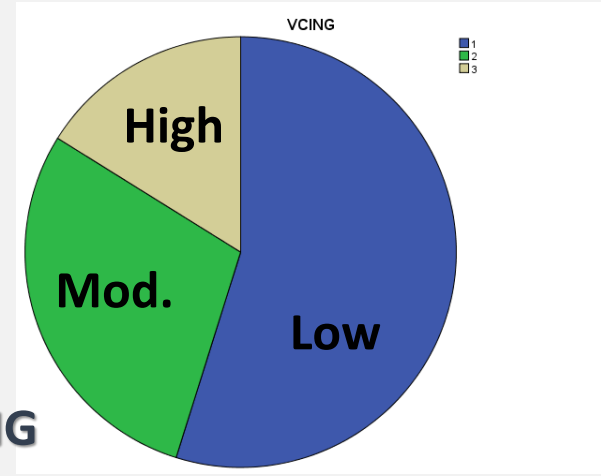
Vascular score vs. VCING

Correlation coeff. = 0.233

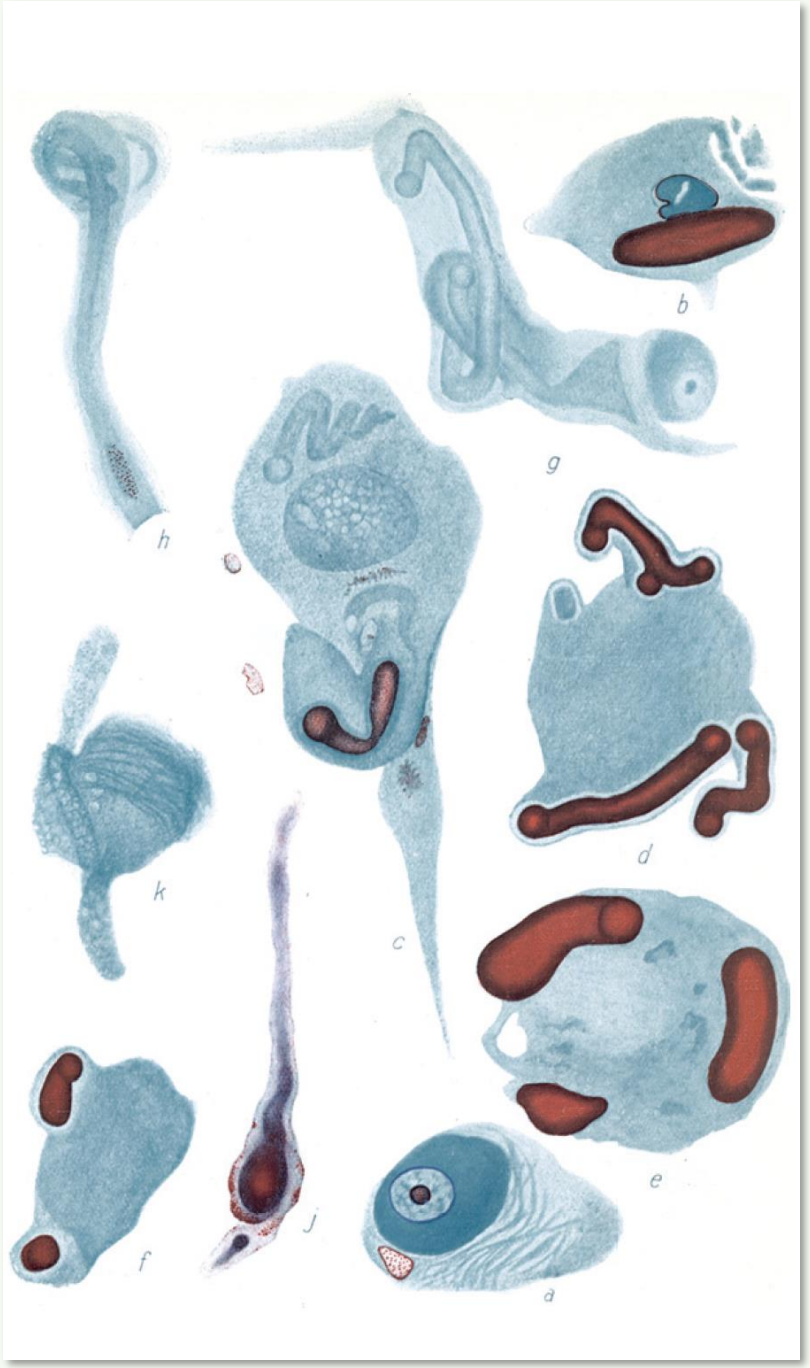
p-value = 0.074



Vascular score



VCING



Lewy FH. Paralysis agitans. 1. Pathologische Anatomie. In: Lewandowsky M, editor. Handbuch der Neurologie, Dritter Band, Spezielle Neurologie I. Berlin: Julius Springer; 1912. p. 920-33

Lafora GR. Contribución a la histopatologia de la parálisis agitante. Trab Lab Invest Biol Univers Madrid. 1913;11:43-54

E. de Parkinson, n. motor dorsal del n. vago. F.H. Lewy, 1923

Contribución á la histopatología de la parálisis agitante

POR

GONZALO R. LAFORA

A las primeras hipótesis de Parkinson y otros, según los cuales las lesiones causales de la parálisis agitante yacerían probablemente en el

Lafora GR. Contribución a la histopatologia de la parálisis agitante.
Trab Lab Invest Biol Univers Madrid. 1913;11:43-54

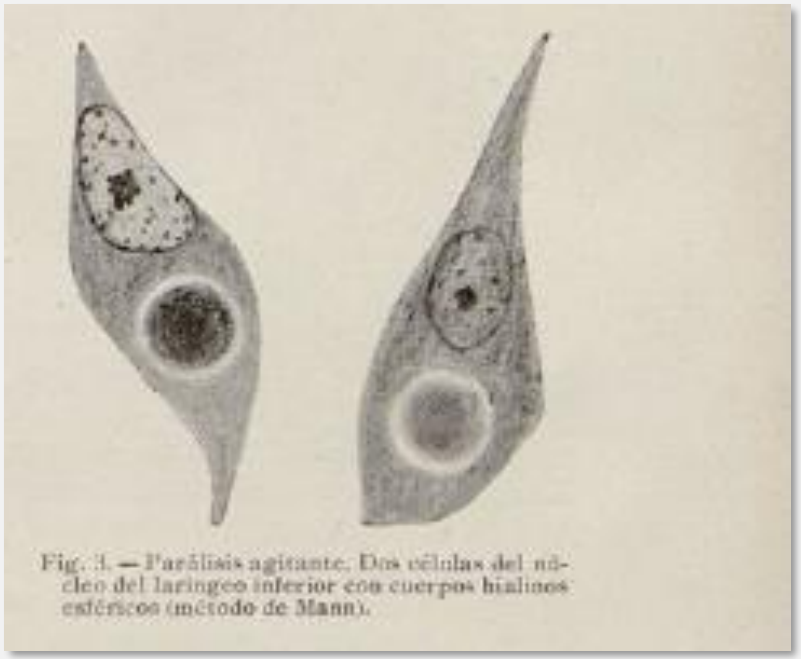


Fig. 3. — Parálisis agitante. Dos células del núcleo del laríngeo inferior con cuerpos hialinos esféricos (método de Mann).

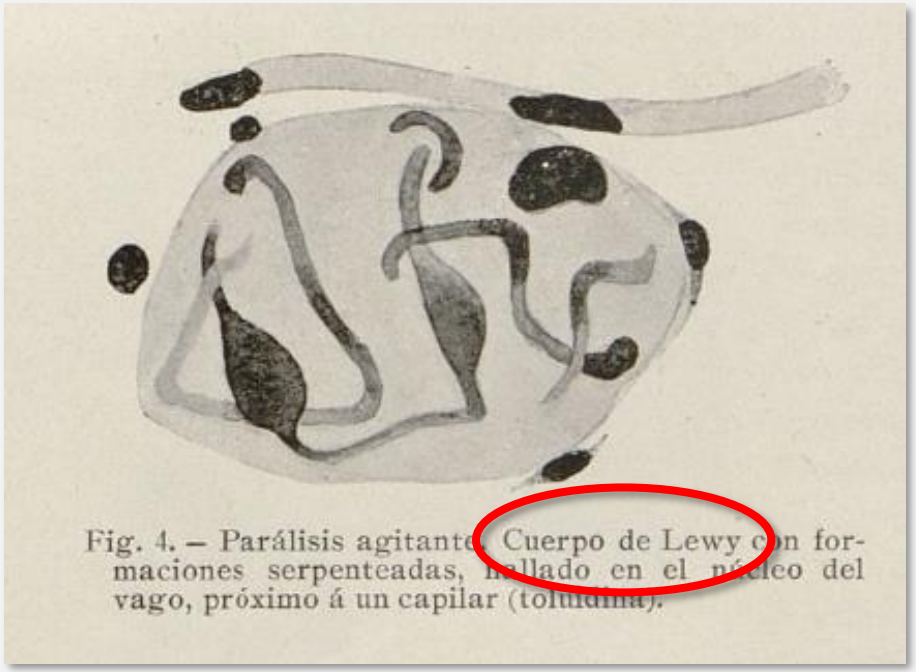
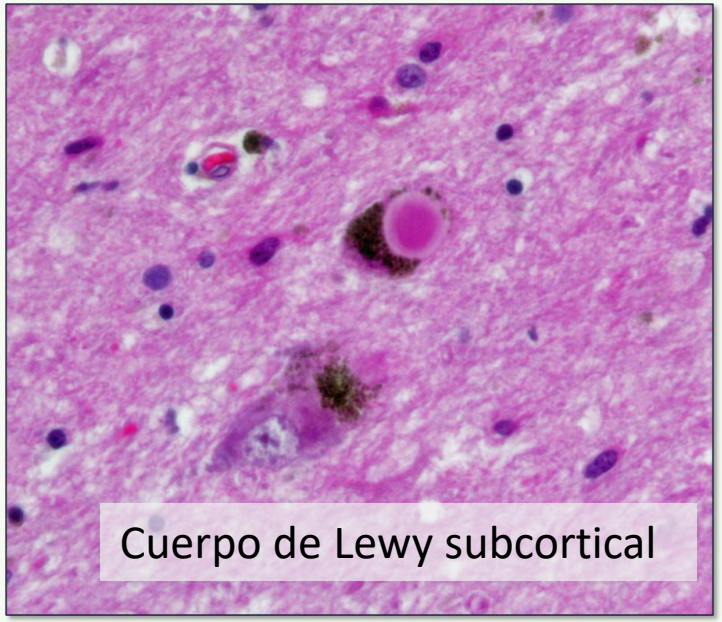
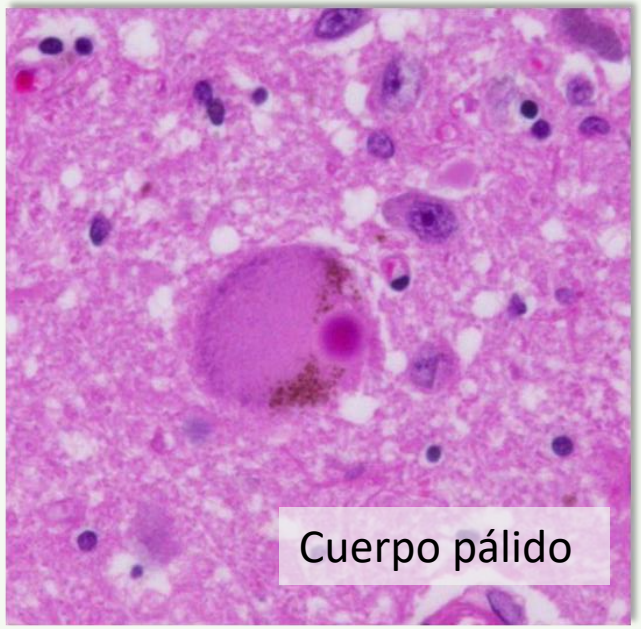
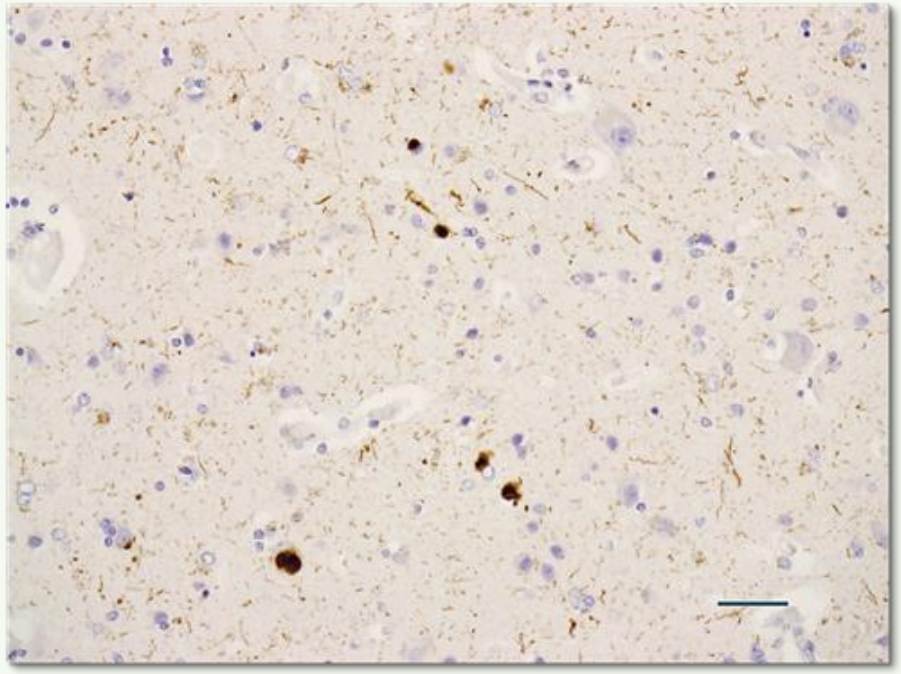


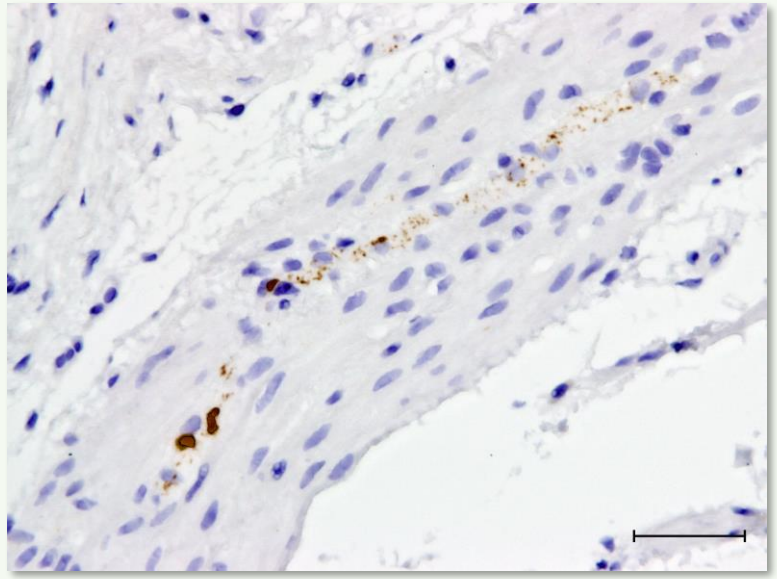
Fig. 4. — Parálisis agitante. Cuerpo de Lewy con formaciones serpenteadas, hallado en el núcleo del vago, próximo á un capilar (toluidina).







Patología de Lewy cortical, α-sinucleína total

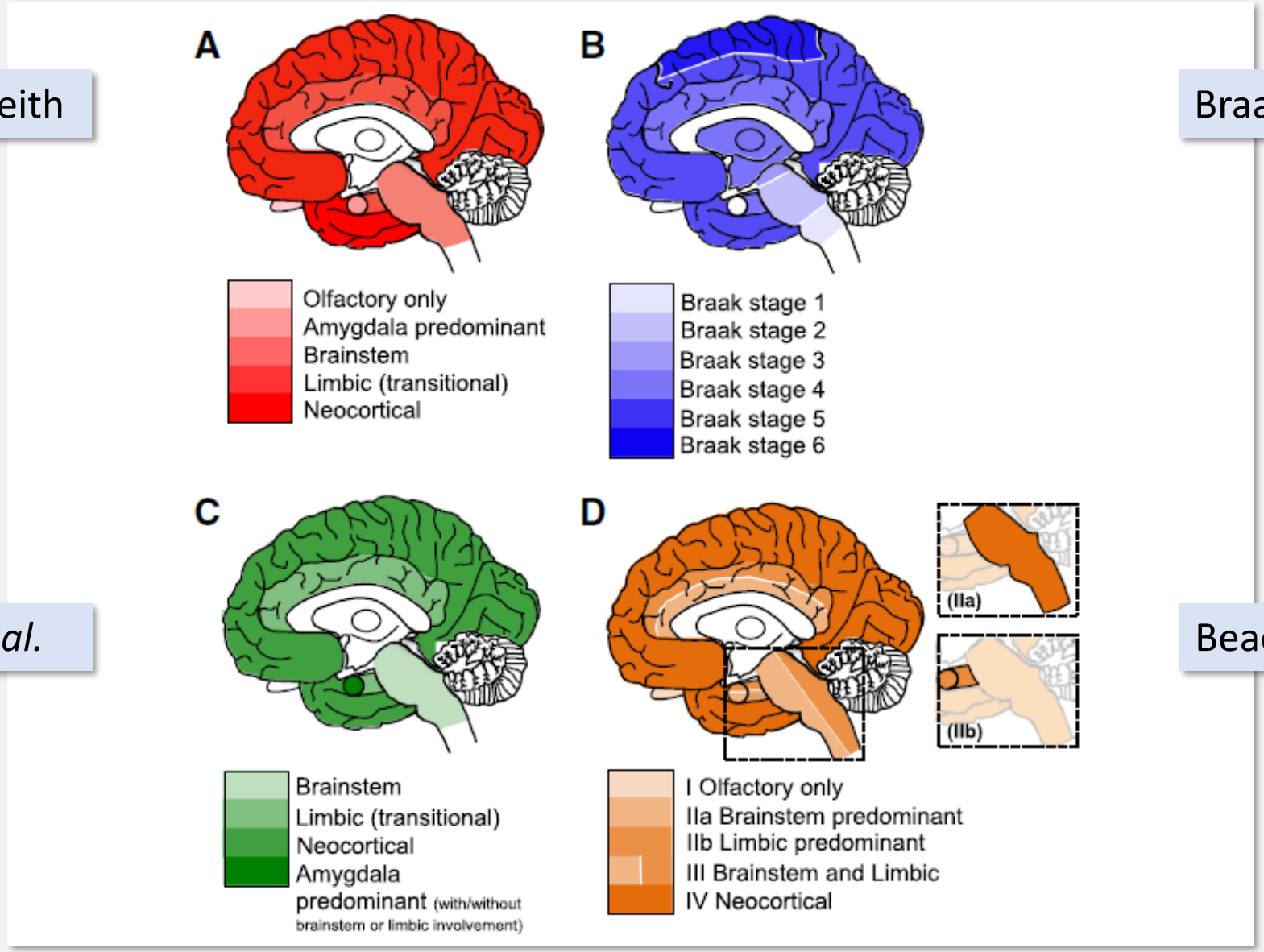


Fibras + en la mucosa gástrica, α-sinucleína fosforilada

Sánchez-Ferro Á, Rábano A *et al*., Mov Disord, 2015

Newcastle-McKeith

Braak



Leverenz *et al.*

Beach *et al.*

Clasificación LPC

2021

Acta Neuropathologica (2021) 141:159–172
https://doi.org/10.1007/s00401-020-02255-2

ORIGINAL PAPER

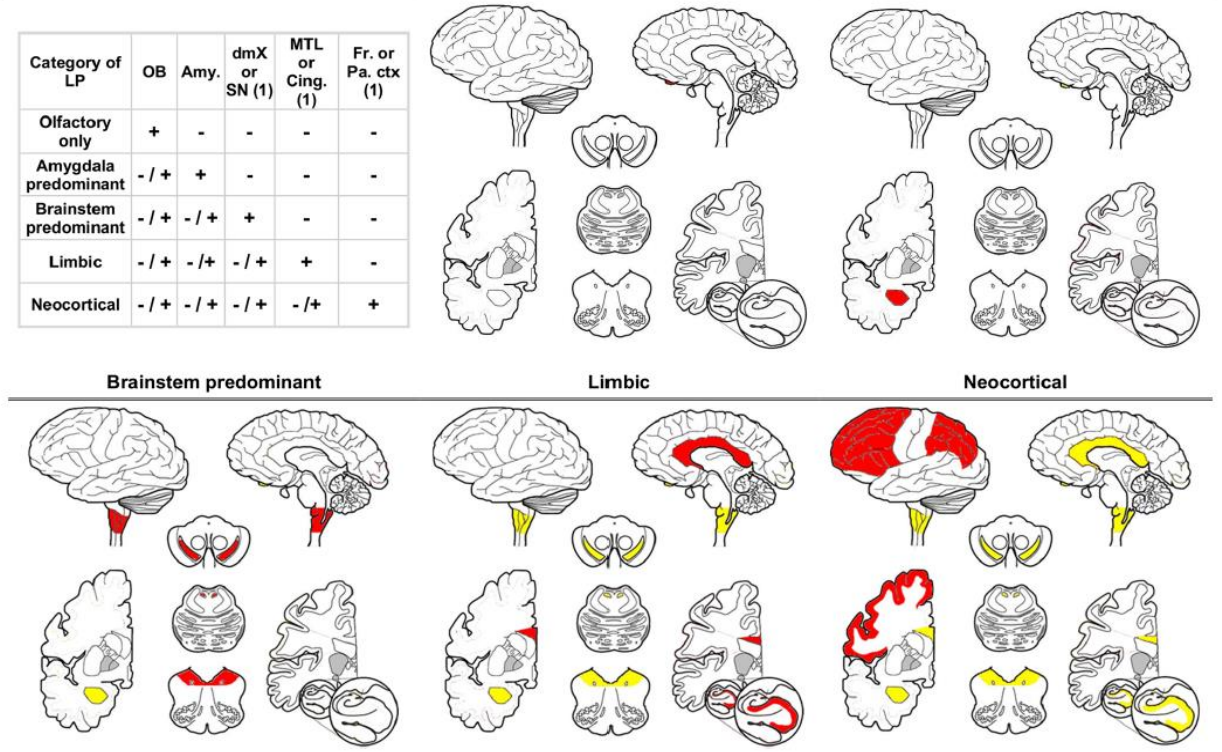
Check for updates

Neuropathological consensus criteria for the evaluation of Lewy pathology in post-mortem brains: a multi-centre study

Johannes Attems[1] · Jon B. Toledo[2,3] · Lauren Walker[1] · Ellen Gelpi[4,5] · Steve Gentleman[6] · Glenda Halliday[7] · Tibor Hortobagyi[8,9,10,11] · Kurt Jellinger[12] · Gabor G. Kovacs[13,14] · Edward B. Lee[3] · Seth Love[15] · Kirsty E. McAleese[1] · Peter T. Nelson[16] · Manuela Neumann[17,18] · Laura Parkkinen[19,20] · Tuomo Polvikoski[1] · Beata Sikorska[21] · Colin Smith[22] · Lea Tenenholz Grinberg[23,24] · Dietmar R. Thal[25] · John Q. Trojanowski[3] · Ian G. McKeith[1]

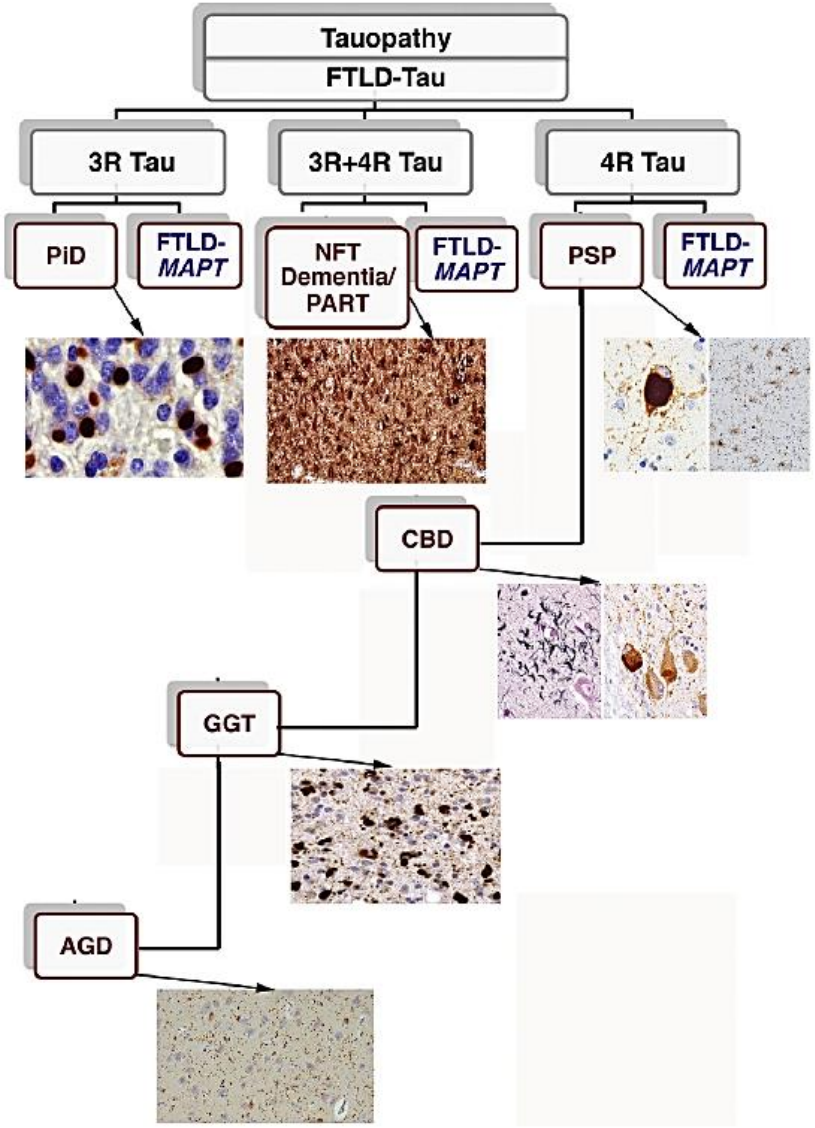
Category of LP	OB	Amy.	dmX or SN (1)	MTL or Cing. (1)	Fr. or Pa. ctx (1)
Olfactory only	+	-	-	-	-
Amygdala predominant	- / +	+	-	-	-
Brainstem predominant	- / +	- / +	+	-	-
Limbic	- / +	- /+	- / +	+	-
Neocortical	- / +	- / +	- / +	- /+	+

Lewy Pathology Consensus Criteria (LPC)



- Evaluación dicotómica (presencia/ausencia) de CL o NL.
- Región (+) si al menos score 1 en el sistema de McKeith.

La patología de granos argirófilos

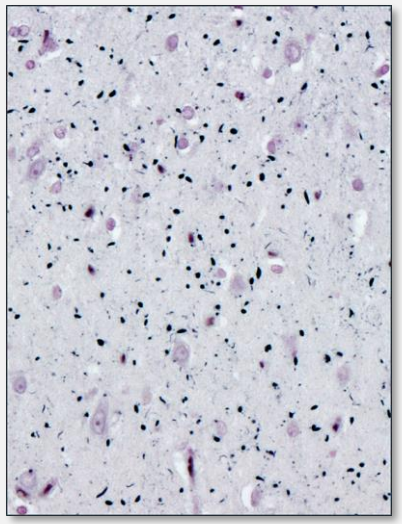


Clasificación molecular de las taupatías primarias

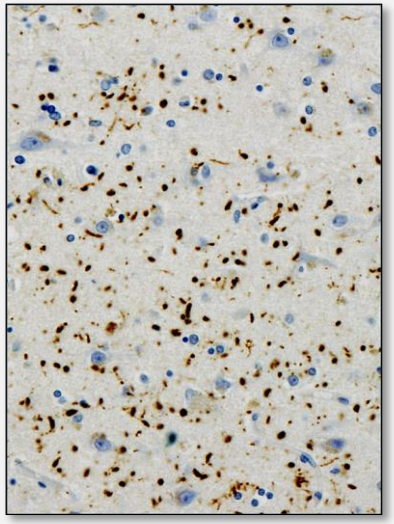
Höglinger et al., 2018

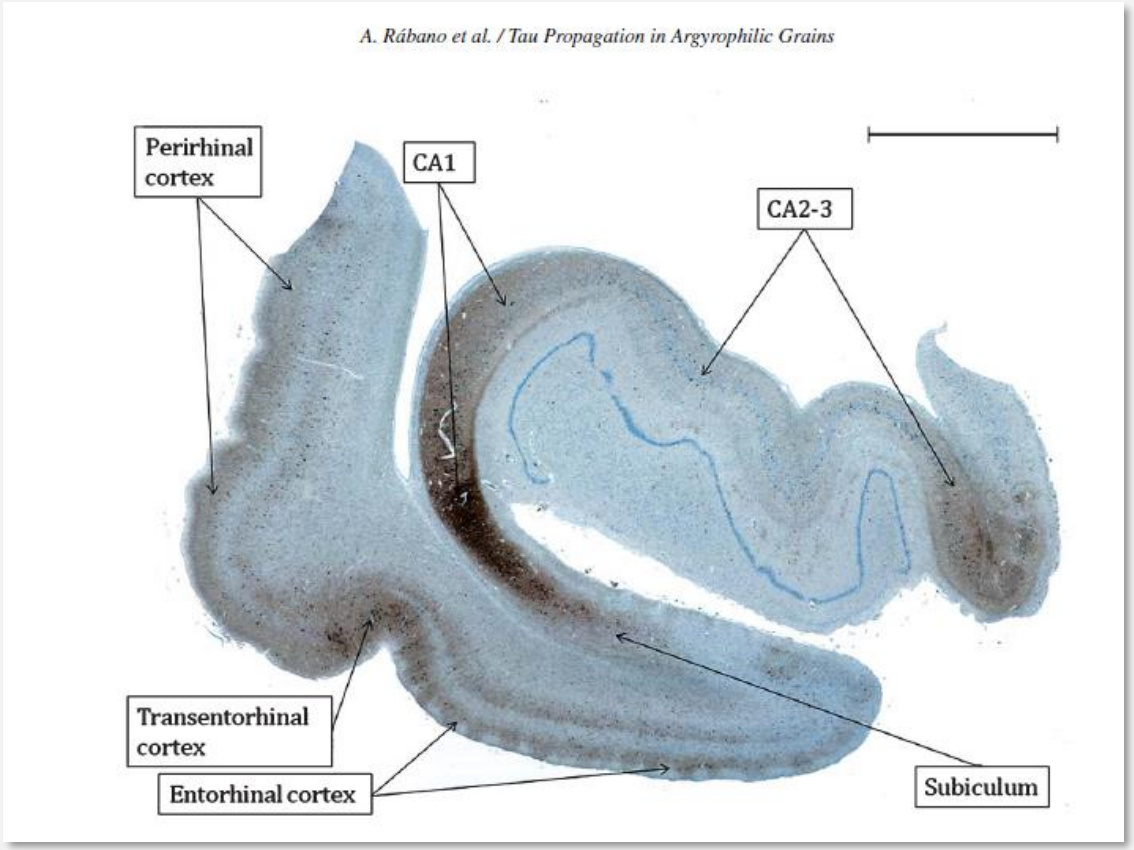
Argyrophilic grain disease

Gallyas

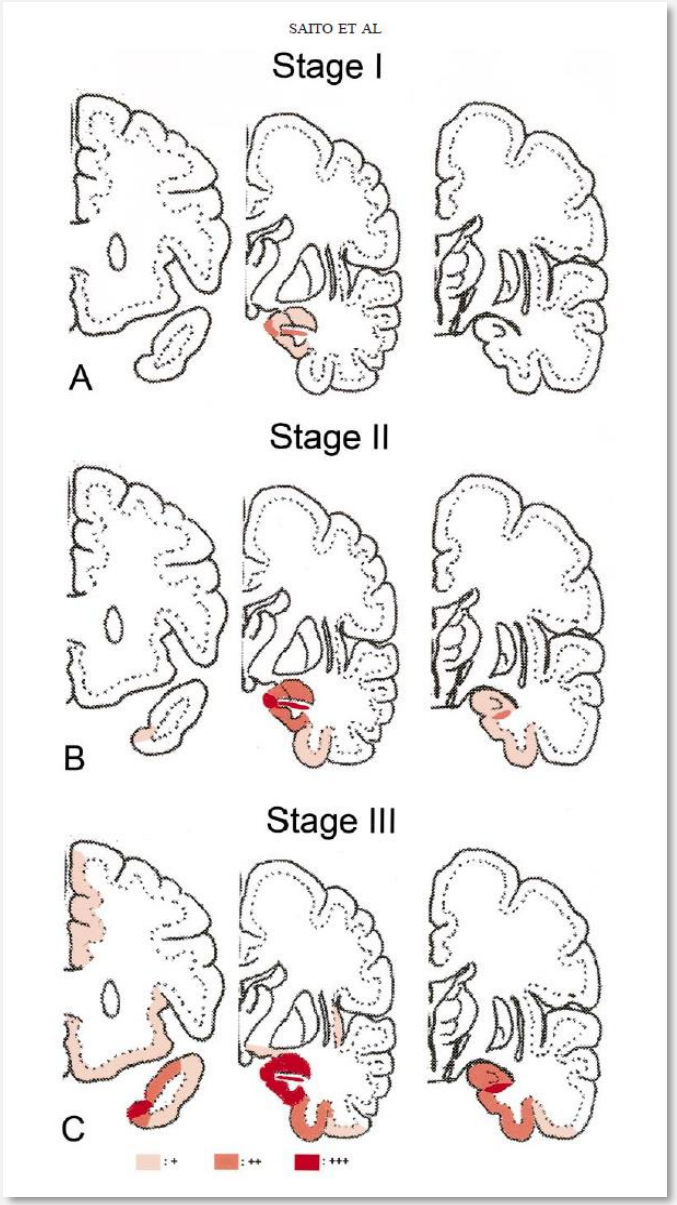


Tau AT8



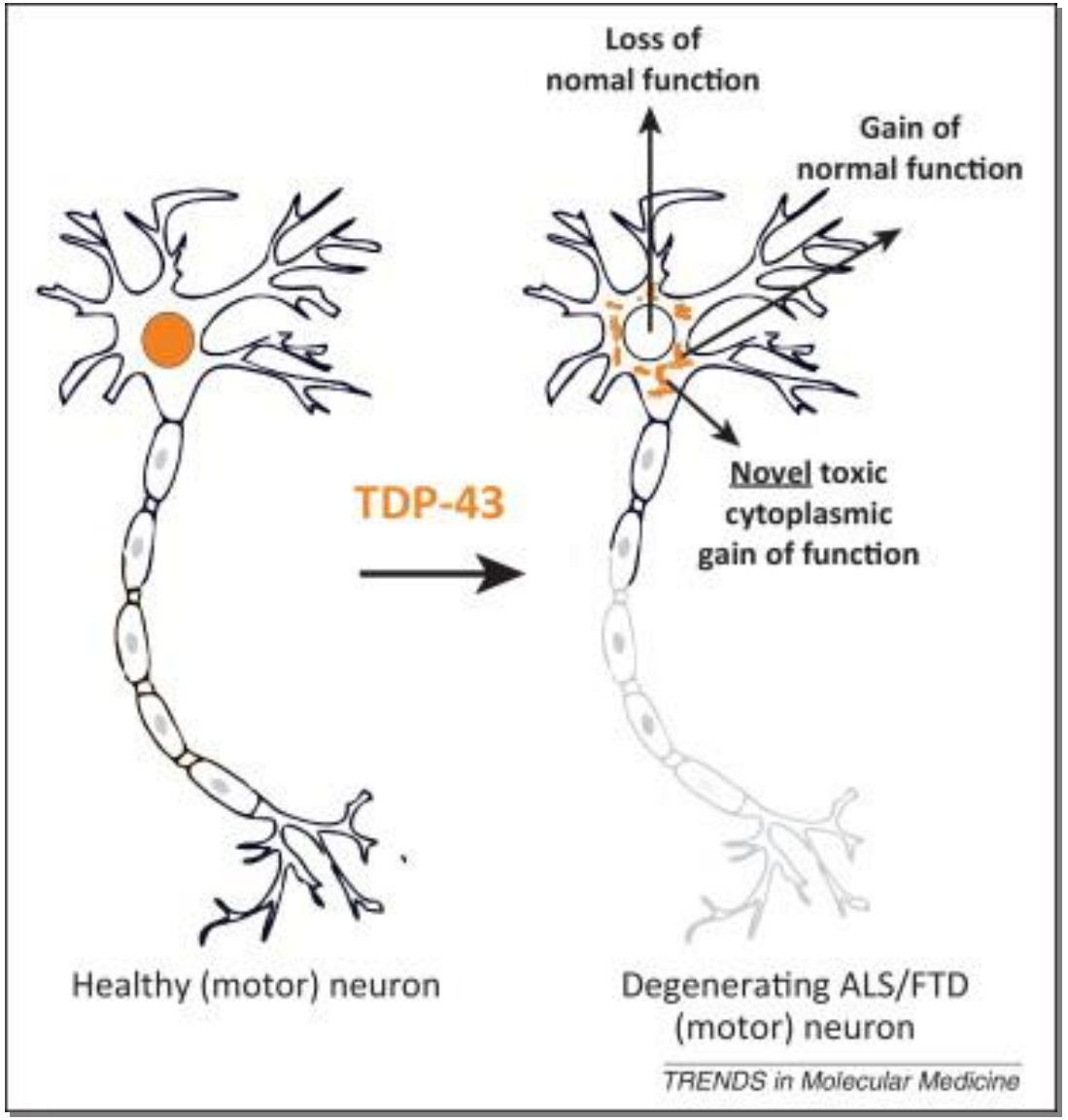


Rábano *et al*., 2014



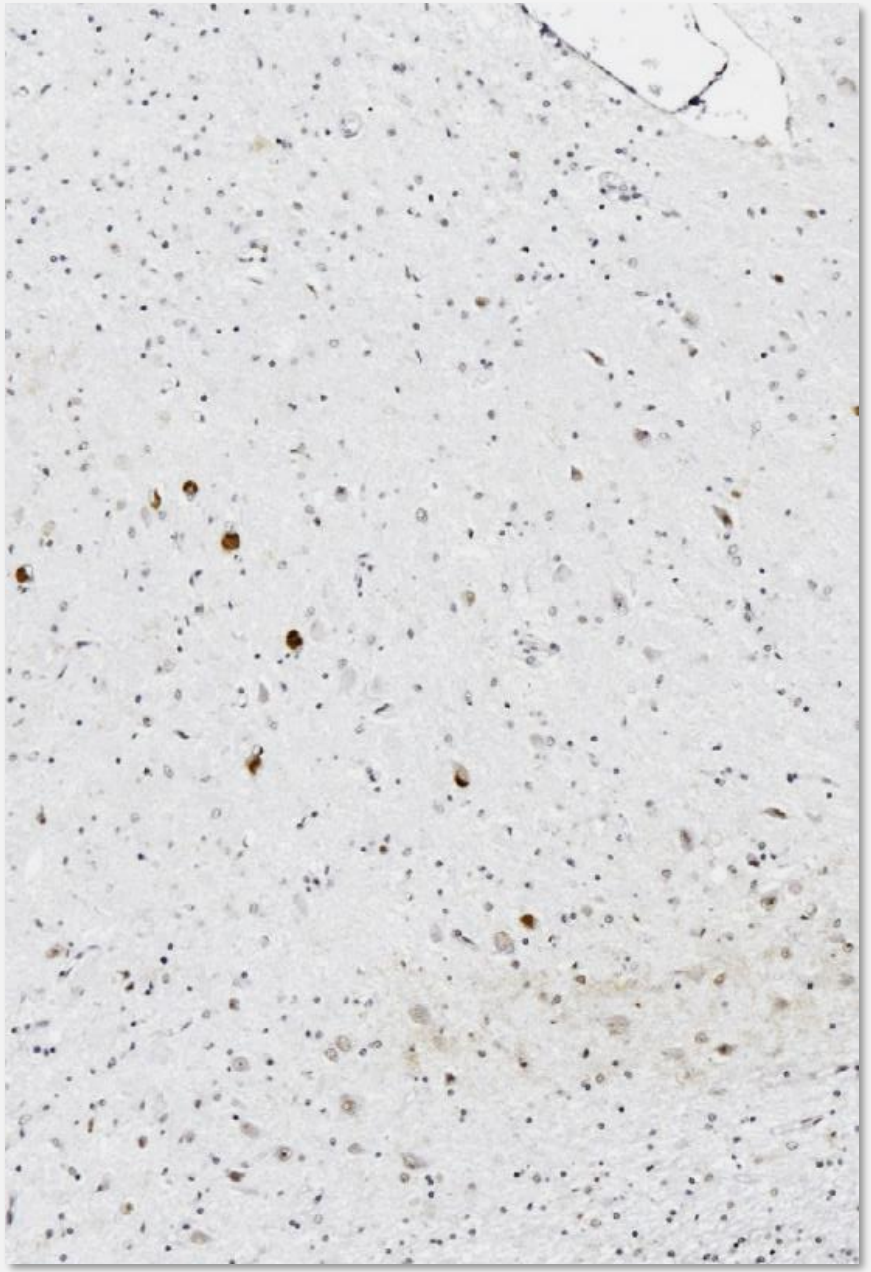
Saito *et al*., 2004

La patología TDP-43 asociada a la edad avanzada (LATE)

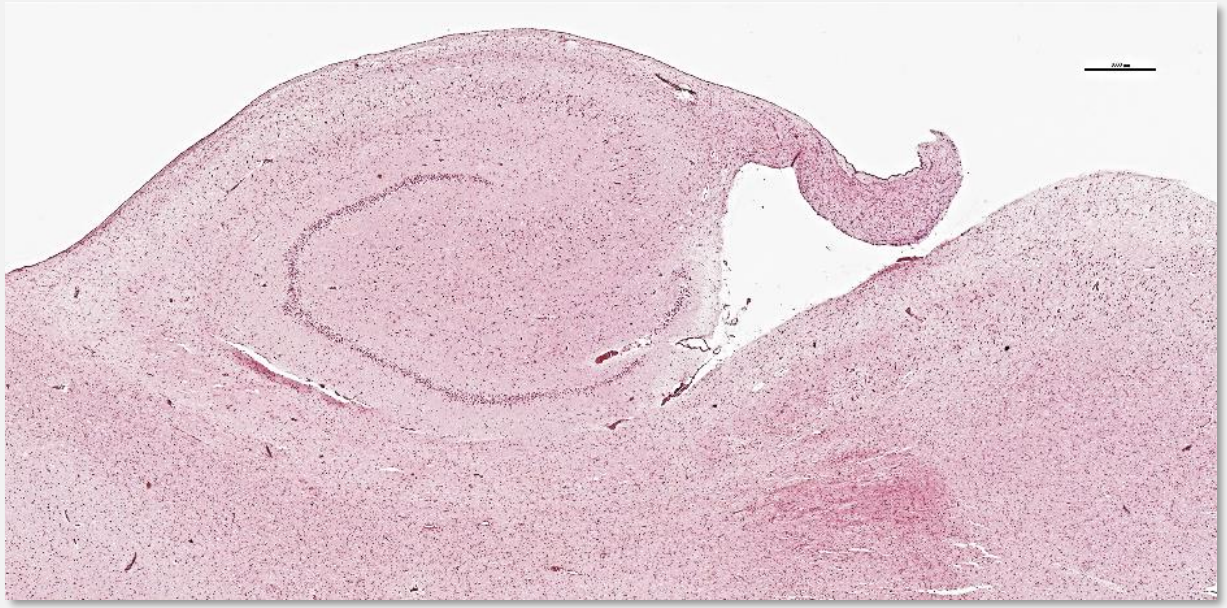


Hippocampal sclerosis

TDP-43



H / E



doi:10.1093/brain/awz099 BRAIN 2019: Page 1 of 25 | 1

BRAIN
A JOURNAL OF NEUROLOGY

REVIEW

Limbic-predominant age-related TDP-43 encephalopathy (LATE): consensus working group report

Peter T. Nelson,[1] Dennis W. Dickson,[2] John Q. Trojanowski,[3] Clifford R. Jack Jr.,[4] Patricia A. Boyle,[5] Konstantinos Arfanakis,[5,6] Rosa Rademakers,[2] Irina Alafuzoff,[7] Johannes Attems,[8] Carol Brayne,[9] Ian T.S. Coyle-Gilchrist,[9] Helena C. Chui,[10] David W. Fardo,[1] Margaret E. Flanagan,[11] Glenda Halliday,[12] Suvi R.K. Hokkanen,[9] Sally Hunter,[9] Gregory A. Jicha,[1] Yuriko Katsumata,[1] Claudia H. Kawas,[13] C. Dirk Keene,[14] Gabor G. Kovacs,[15] Walter A. Kukull,[14] Allan I. Levey,[16] Nazanin Makkinejad,[6] Thomas J. Montine,[17] Shigeo Murayama,[18] Melissa E. Murray,[2] Sukriti Nag,[5] Robert A. Rissman,[19] William W. Seeley,[20] Reisa A. Sperling,[21] Charles L. White III,[22] Lei Yu[5] and Julie A. Schneider[5]

2019

LATE-NC
Stages 0 → 3

B LATE-NC related stages based on anatomic distribution of TDP-43 pathology

Simplified staging of TDP-43 proteinopathy* for routine LATE-NC diagnosis (consensus recommendation)		Josephs TDP-43 proteinopathy staging (KA Josephs et al, 2013)		Rush University TDP-43 proteinopathy staging (S Nag et al, 2017)	
0	None	0	None	0	None
1	Amygdala	1	Amygdala	1	Amygdala
2	Hippocampus	2	Entorhinal cortex, subiculum	2	Entorhinal cortex, CA1
		3	Dentate, Occipitotemporal cortex	3	Anterior temporal cortex
		4	Insula, Inf temporal cortex	4	Midtemporal and orbitofrontal cortex
		5	Inf olive, midbrain		
3	Middle frontal gyrus (MFG)	6	Basal ganglia, MFG	5	MFG

*-*Any* TDP-43 proteinopathy is seen in that anatomic region

Received: 6 October 2022 | Revised: 5 December 2022 | Accepted: 21 December 2022

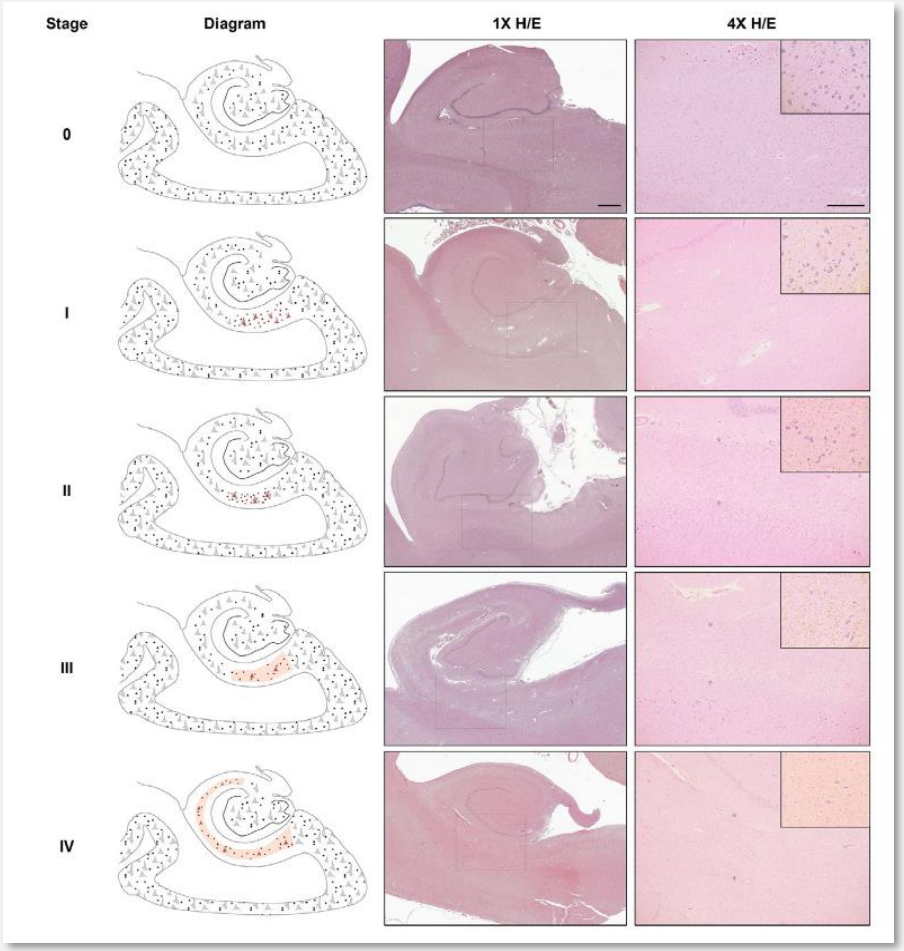
DOI: 10.1002/alz.12942

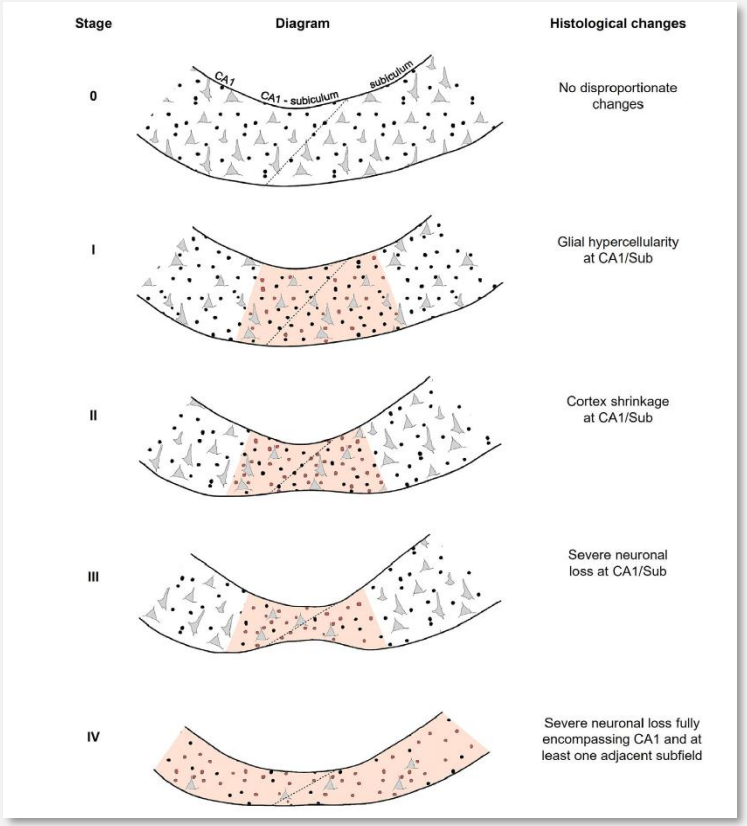
Alzheimer's & Dementia®
THE JOURNAL OF THE ALZHEIMER'S ASSOCIATION

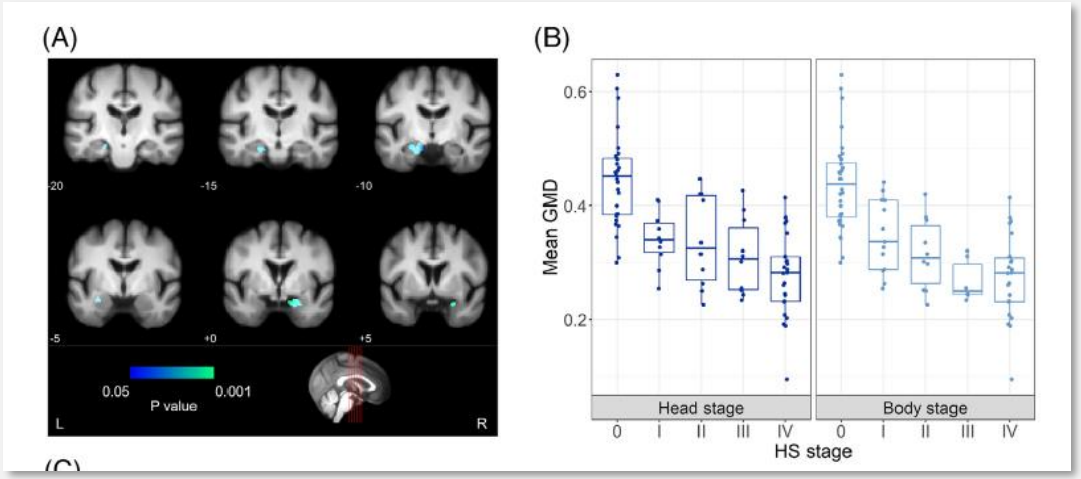
RESEARCH ARTICLE

A novel histological staging of hippocampal sclerosis that is evident in gray matter loss in vivo

Diana Ortega-Cruz[1,2] | Alicia Uceda-Heras[2,3] | Juan Eugenio Iglesias[4,5] | María Ascensión Zea-Sevilla[2] | Bryan Strange[1,2] | Alberto Rabano[2]





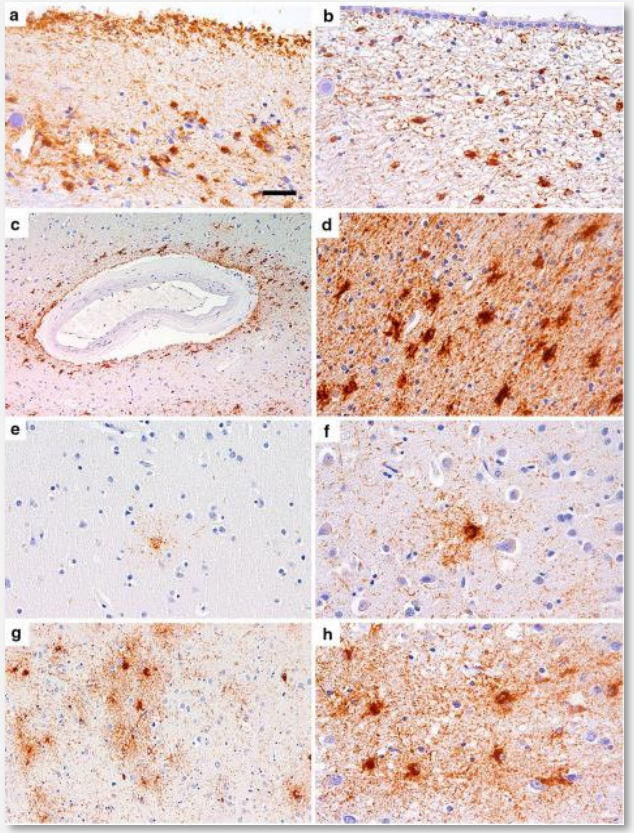


Aging-related tau astrogliopathy (ARTAG)

Acta Neuropathol. 2016 January ; 131(1): 87–102. doi:10.1007/s00401-015-1509-x.

Aging-related tau astrogliopathy (ARTAG): harmonized evaluation strategy

A full list of authors and affiliations appears at the end of the article.



Kovacs *et al. Acta Neuropathologica Communications* (2018) 6:50
https://doi.org/10.1186/s40478-018-0552-y

Acta Neuropathologica Communications

RESEARCH Open Access

Sequential stages and distribution patterns of aging-related tau astrogliopathy (ARTAG) in the human brain

Gabor G. Kovacs[1,2*], Sharon X. Xie[3], John L. Robinson[2], Edward B. Lee[2], Douglas H. Smith[4], Theresa Schuck[2], Virginia M.-Y. Lee[2] and John Q. Trojanowski[2*]

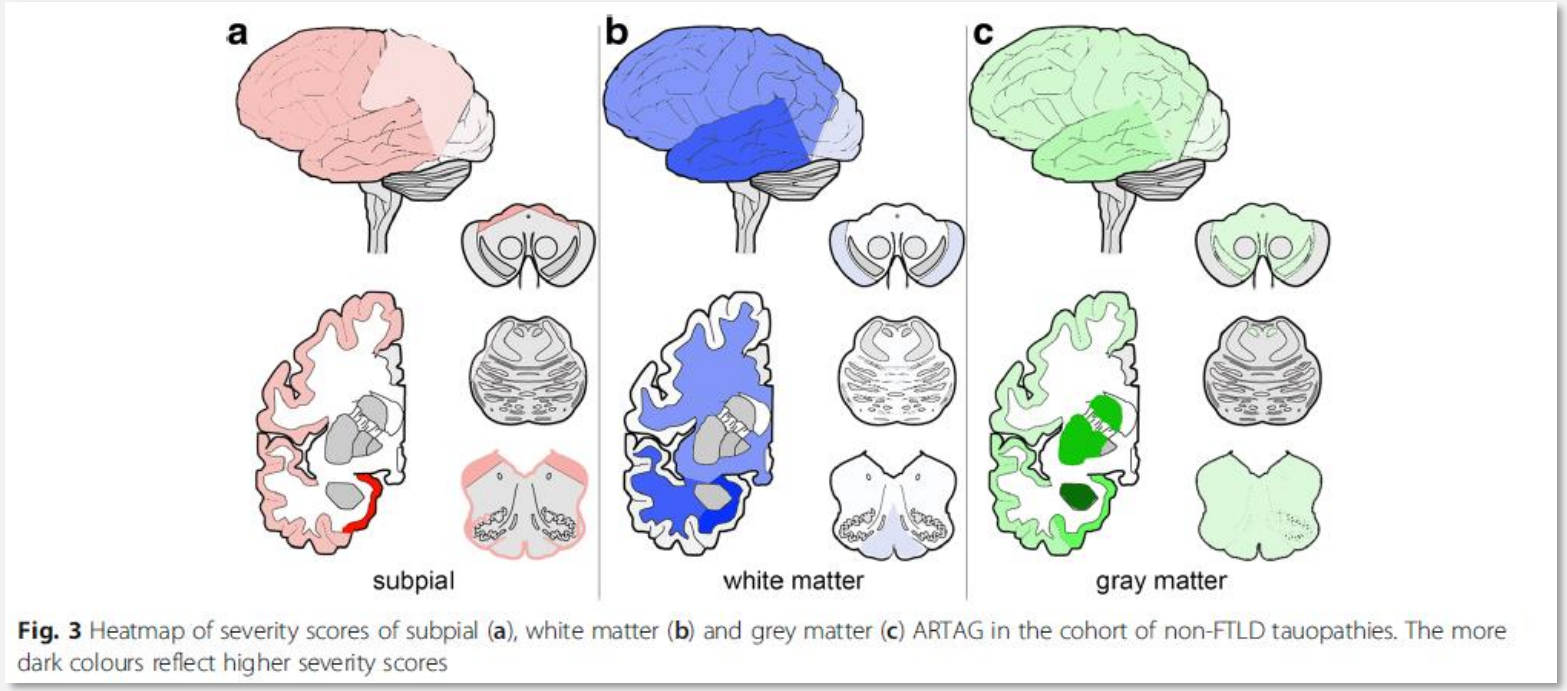


Fig. 3 Heatmap of severity scores of subpial (**a**), white matter (**b**) and grey matter (**c**) ARTAG in the cohort of non-FTLD tauopathies. The more dark colours reflect higher severity scores

ELSEVIER
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Alzheimer's & Dementia: Translational Research & Clinical Interventions 3 (2017) 83-91
Alzheimer's & Dementia

Perspective

Multiple comorbid neuropathologies in the setting of Alzheimer's disease neuropathology and implications for drug development

Gil D. Rabinovici[a], Maria C. Carrillo[b], Mark Forman[c], Susan DeSanti[d], David S. Miller[e], Nicholas Kozauer[f], Ronald C. Petersen[g], Christopher Randolph[h,i], David S. Knopman[g], Eric E. Smith[j], Maria Isaac[k], Niklas Mattsson[l,m], Lisa J. Bain[n], James A. Hendrix[b,*], John R. Sims[o]

Alzheimers Dement. 2017 June ; 13(6): 654–662. doi:10.1016/j.jalz.2016.09.015.

Mixed neuropathologies and estimated rates of clinical progression in a large autopsy sample

Willa D. Brenowitz[1], **Rebecca A. Hubbard**[2], **C. Dirk Keene**[3], **Stephen E. Hawes**[4], **W.T. Longstreth Jr**[1,5], **Randy L. Woltjer**[6], and **Walter A. Kukull**[1]

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Acta Neuropathol. 2018 September ; 136(3): 377–388. doi:10.1007/s00401-018-1872-5.

Non-Alzheimer's contributions to dementia and cognitive resilience in The 90+ Study

John L. Robinson[1], **Maria M. Corrada**[2], **Gabor G. Kovacs**[1,3], **Myrna Dominique**[1], **Carrie Caswell**[4], **Sharon X. Xie**[4], **Virginia M.-Y. Lee**[1], **Claudia H. Kawas**[5], and **John Q. Trojanowski**[1]

doi:10.1093/brain/awab099 BRAIN 2021: 144; 2186–2198 | 2186
BRAIN
ORIGINAL ARTICLE

Comorbid neuropathological diagnoses in early versus late-onset Alzheimer's disease

Salvatore Spina,[1,†] Renaud La Joie,[1,†] Cathrine Petersen,[1] Amber L. Nolan,[1] Deion Cuevas,[1] Celica Cosme,[1] Mackenzie Hepker,[1] Ji-Hye Hwang,[1] Zachary A. Miller,[1] Eric J. Huang,[2] Anna M. Karydas,[1] Harli Grant,[1] Adam L. Boxer,[1] Maria Luisa Gorno-Tempini,[1] Howard J. Rosen,[1] Joel H. Kramer,[1] Bruce L. Miller,[1] William W. Seeley,[1,2] Gil D. Rabinovici[1,3] and Lea T. Grinberg[1,2]

doi:10.1093/brain/awy146 BRAIN 2018: 141; 2181–2193 | 2181
BRAIN
A JOURNAL OF NEUROLOGY

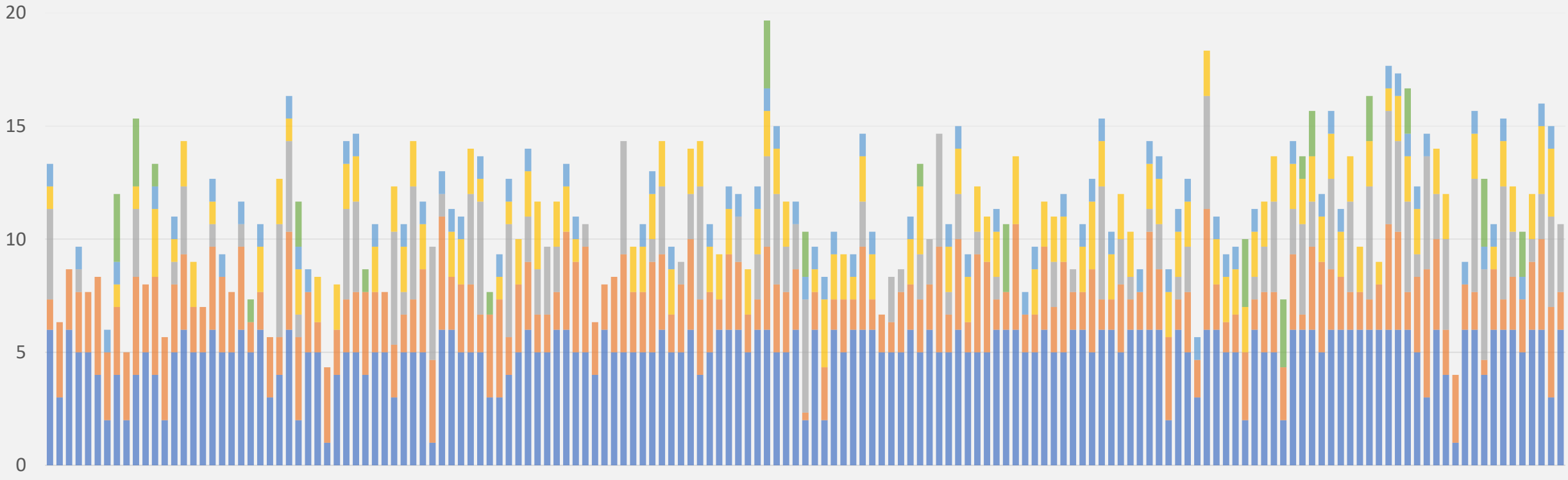
Neurodegenerative disease concomitant proteinopathies are prevalent, age-related and APOE4-associated

John L. Robinson,[1,2,3,4] Edward B. Lee,[1,2,3,4] Sharon X. Xie,[1,2,3,4,5] Lior Rennert,[1,2,3,4,5] EunRan Suh,[1,2,3,4] Colin Bredenberg,[1,2,3,4] Carrie Caswell,[1,2,3,4,5] Vivianna M. Van Deerlin,[1,2,3,4] Ning Yan,[1,2,3,4,6] Ahmed Yousef,[1,2,3,4] Howard I. Hurtig,[1,2,3,7] Andrew Siderowf,[1,2,3,7] Murray Grossman,[1,2,3,7,8] Corey T. McMillan,[7,8] Bruce Miller,[9] John E. Duda,[3,10] David J. Irwin,[1,2,3,7,8] David Wolk,[1,2,3,7,8,11] Lauren Elman,[3,7] Leo McCluskey,[3,7] Alice Chen-Plotkin,[1,2,3,7] Daniel Weintraub,[2,3,12] Steven E. Arnold,[13] Johannes Brettschneider,[14] Virginia M.-Y. Lee[1,2,3,4,7] and John Q. Trojanowski[1,2,3,4,7]

The problem of pathological heterogeneity and comorbity in dementia

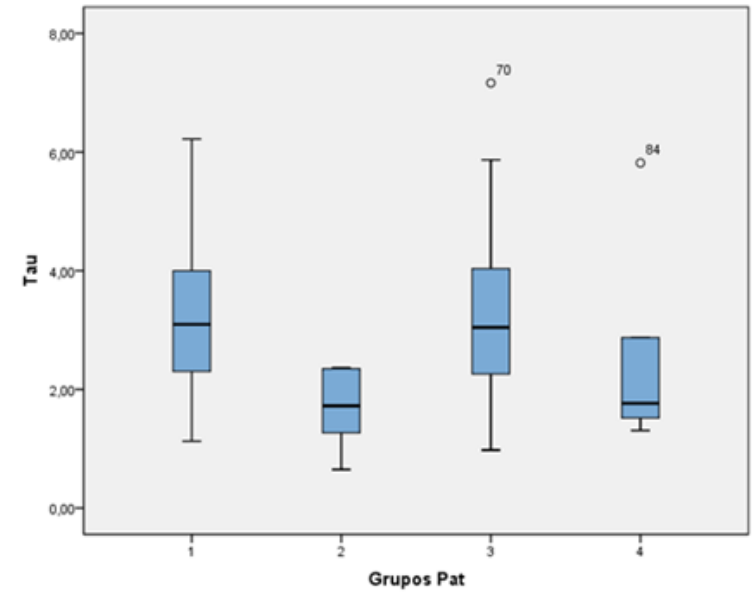
- Alzheimer's disease neuropathology change
- Cerebrovascular pathology
- Lewy type pathology
- Limbic-predominant age-related TDP-43 encephalopathy (LATE)
- Aging-related tau astrogliopathy (ARTAG)
- Argyrophilic grain disease
- Other pathologies

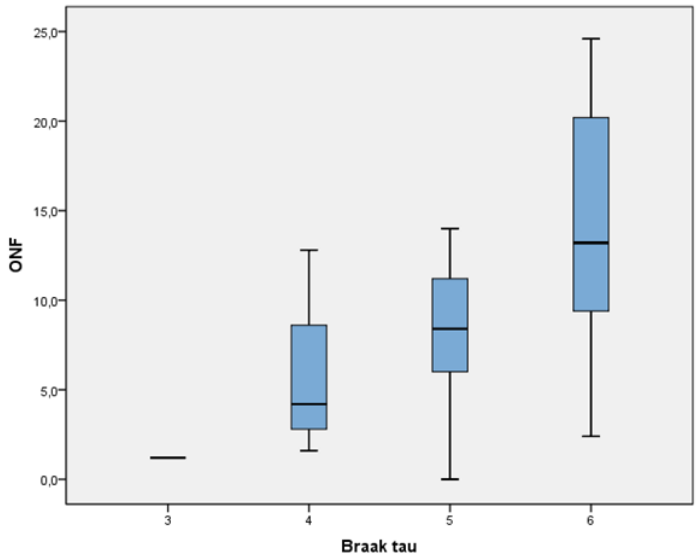
Vallecas Alzheimer's Study

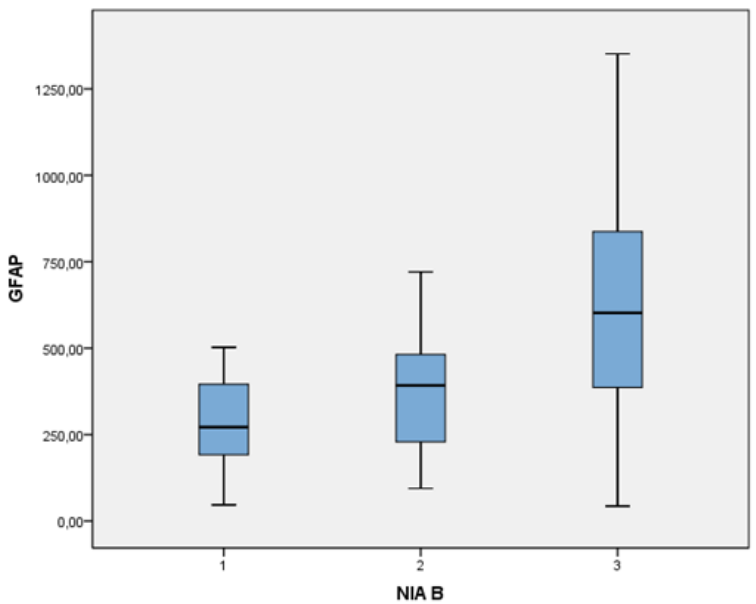


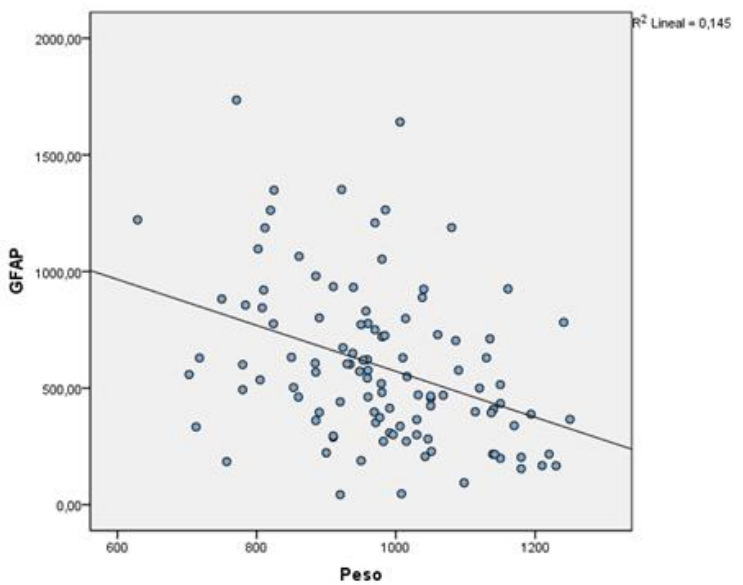
- Alzheimer' pathology (Braak stage 0 – 6)
- Cerebrovascular pathology (0 – 5)
- Lewy type pathology (0 – 6)
- TDP-43 pathology (LATE) (0 – 3)
- ARTAG (0 – 1)
- Argyrophilic grain disease (0 – 3)

Biomarcadores en plasma (SIMOA)









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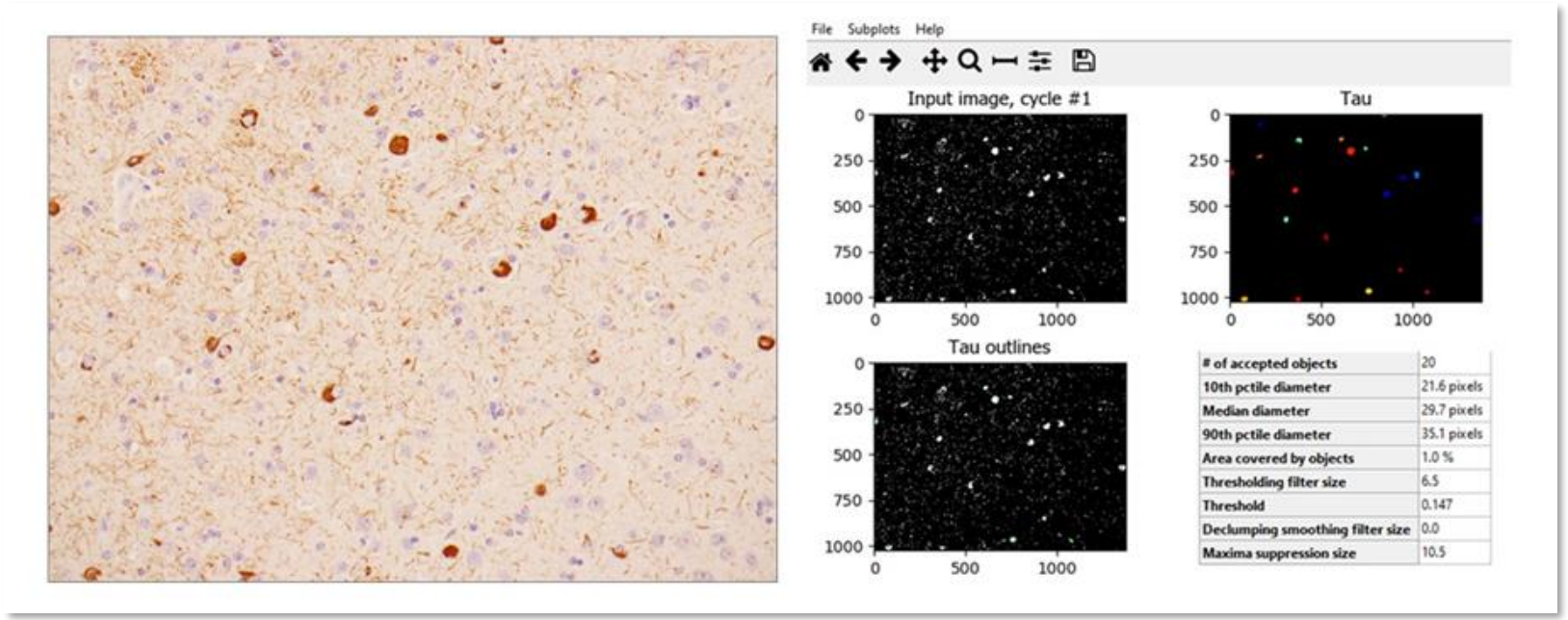
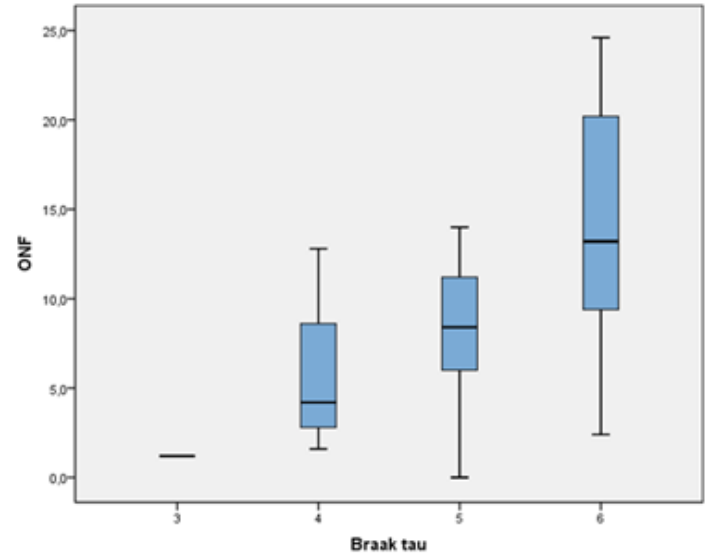
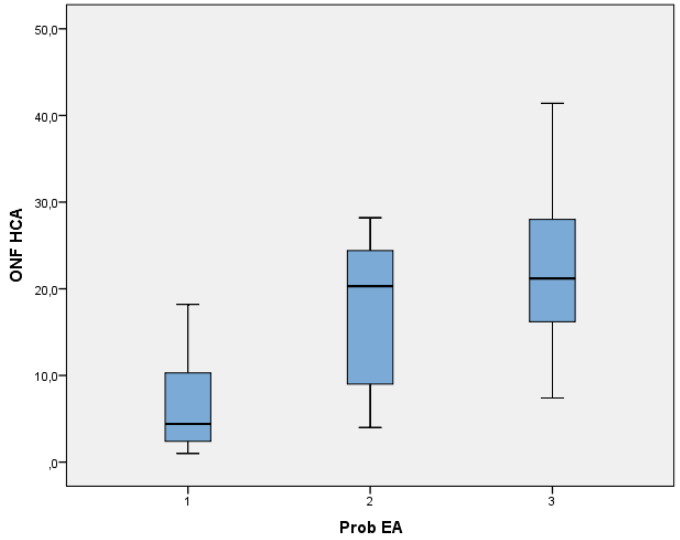
cell image analysis software



Córtex temporal lateral



Córtex hipocampo



Perosa *et al. acta neuropathol commun* (2021) 9:141
https://doi.org/10.1186/s40478-021-01235-1

Acta Neuropathologica Communications

METHODOLOGY ARTICLE Open Access

Deep learning assisted quantitative assessment of histopathological markers of Alzheimer's disease and cerebral amyloid angiopathy

Valentina Perosa[1,2*†], Ashley A. Scherlek[3,4†], Mariel G. Kozberg[4], Lindsey Smith[5], Thomas Westerling-Bui[5], Corinne A. Auger[4], Serge Vasylechko[6], Steven M. Greenberg[1] and Susanne J. van Veluw[1,4*]

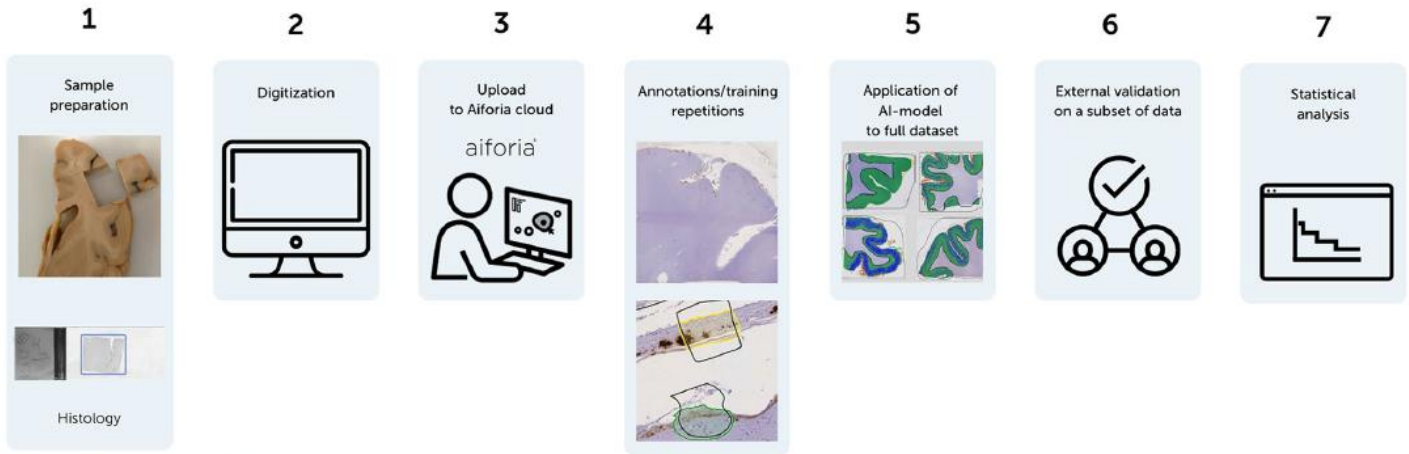
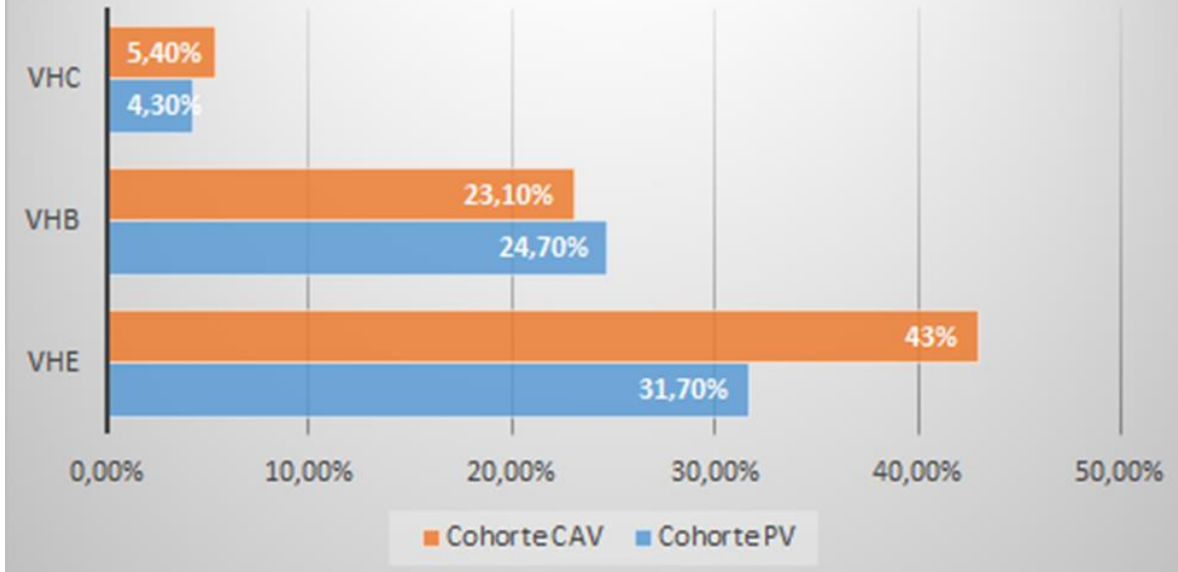
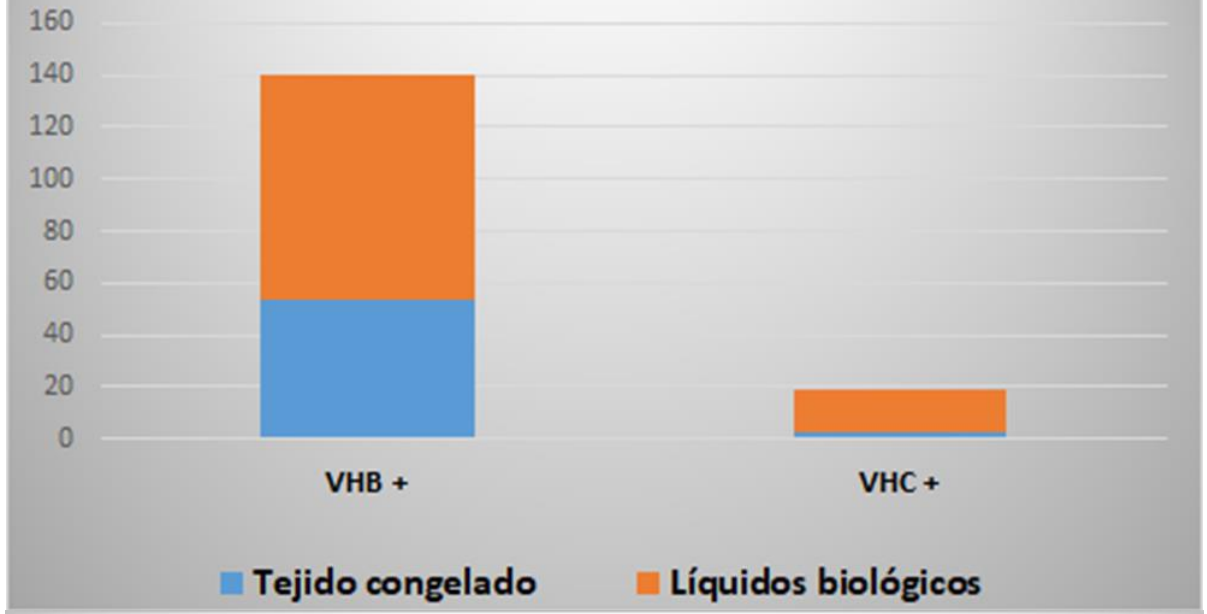


Fig. 1 Workflow for development of histopathological deep learning-based models. (1) Preparation and staining of histopathological tissue sections; (2) Digitization of high resolution whole slide images; (3) Uploading on the Aiforia ® cloud based platform; (4) Annotations on a representative subset (approximately 10%) of the whole dataset and repetitive training of the separate convolutional neural networks (CNNs), that constitute the deep learning-based model (AI-model); (5) Application of the model on the whole dataset; (6) Validation of each CNN; (7) Statistical analysis

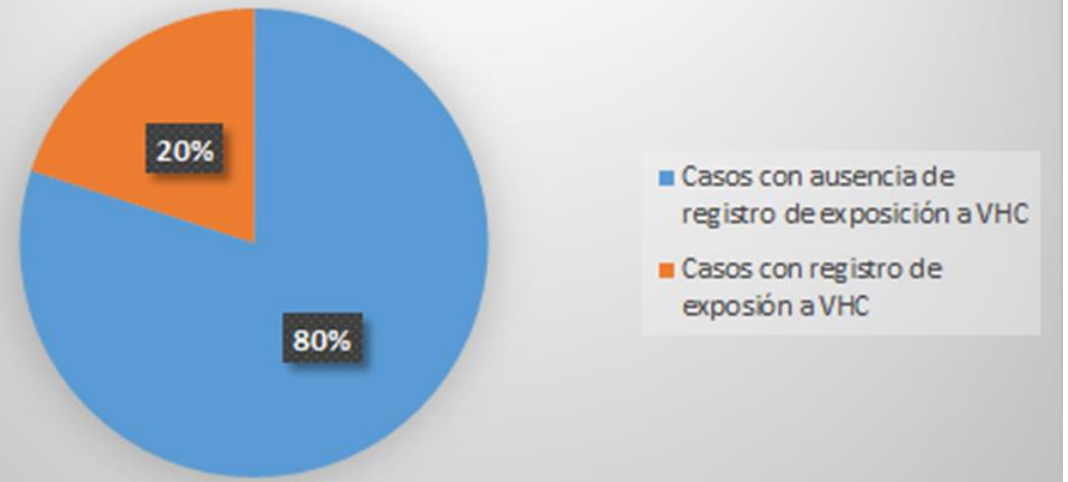
Seroprevalencia



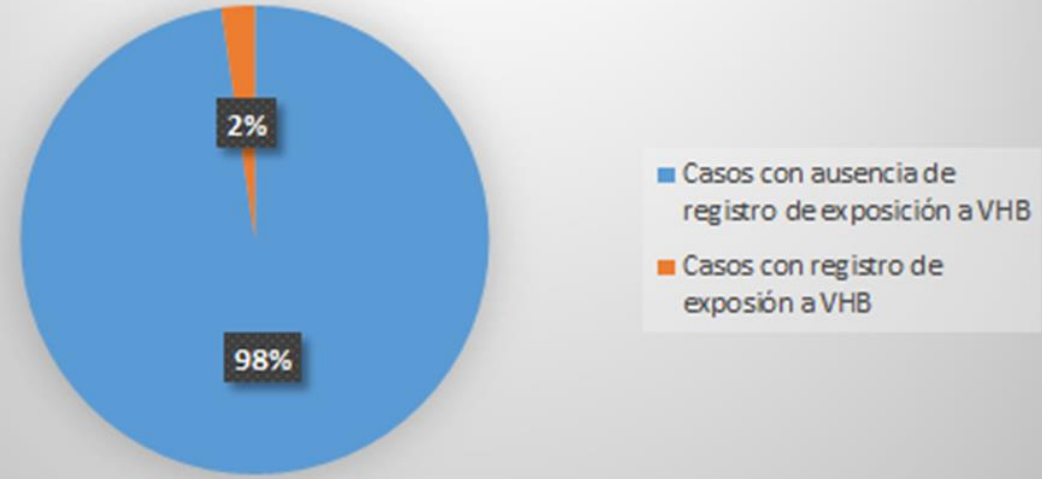
Nº de muestras cedidas



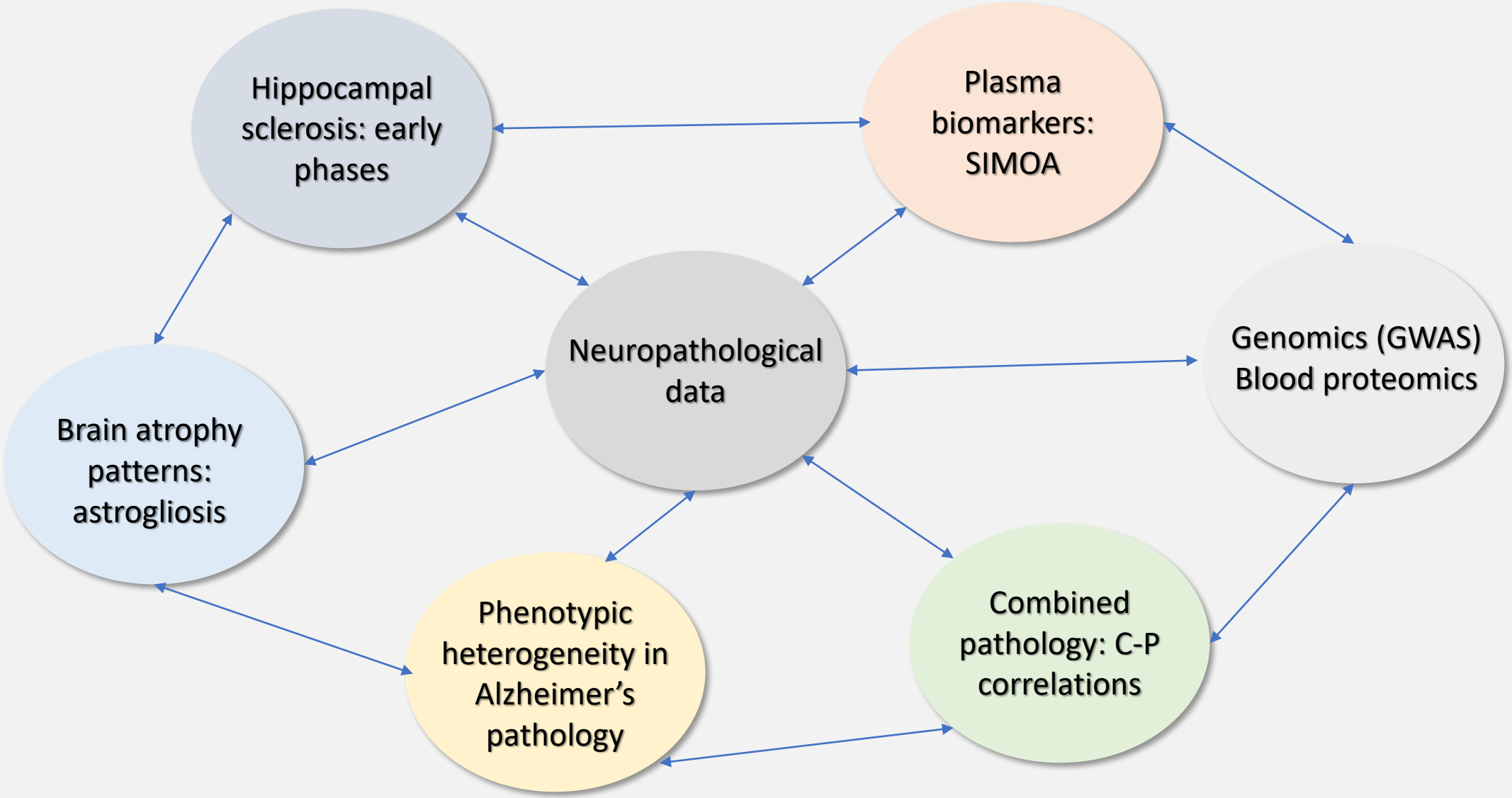
Conocimiento previo de exposición a VHC



Conocimiento previo de exposición a VHB



Neuropathological data of the Vallecas Alzheimer's Study: research lines at the CIEN Foundation




Toma de muestras Transferencia
Solicitud investigador
Evaluación técnica y disponibilidad
Aprobación Comité Científico
Aprobación Comité Ético

Centros receptores de muestras del BT-CIEN

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• Instituto de Neurociencias Ramón y Cajal, CSIC.
• Centro Nacional de Biotecnología, CSIC.
• Instituto de Neurociencias de Alicante, CSIC, Universidad Miguel Hernández.
• Instituto de Investigaciones Biomédicas “Alberto Sols”, CSIC.
• Instituto de Química-Física “Rocasolano”, CSIC.
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• Universidad de Valencia.
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• Biocross, S.L., Madrid.
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•Instituto de Investigación Sanitaria I + 12, Madrid.
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•Royal College of Surgeons, Irlanda.
•Grenoble Institut des Neurosciences, Grenoble, Francia.
•Karolinska Institutet, Estocolmo, Suecia

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Redox Biol. 2022
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Acta Neuropathol Commun. 2023
Alzheimers Res Ther. 2023
Mol Neurobiol. 2023
Neurobiol Dis. 2023
Commun Biol. 2023

Descripción de nuevas entidades

JMG Online First, published on April 6, 2013 as 10.1136/jmedgenet-2013-101525

ORIGINAL ARTICLE

A new seipin-associated neurodegenerative syndrome

Encarna Guillén-Navarro,[1] Sofía Sánchez-Iglesias,[2] Rosario Domingo-Jiménez,[3] Berta Victoria,[2] Alejandro Ruiz-Riquelme,[2] Alberto Rábano,[4] Lourdes Loidi,[5] Andrés Beiras,[6] Blanca González-Méndez,[2] Adriana Ramos,[2] Vanesa López-González,[1] María Juliana Ballesta-Martínez,[1] Miguel Garrido-Pumar,[7] Pablo Aguiar,[7] Alvaro Ruibal,[7] Jesús R Requena,[2] David Araújo-Vilar[2]

doi:10.1093/brain/awh501 *Brain* (2005), **128**, 1707–1715

A multigenerational pedigree of late-onset Alzheimer's disease implies new genetic causes

Adriano Jimenez-Escrig,[1] Estrella Gomez-Tortosa,[2] Manuel Baron,[3] Alberto Rabano,[3] Mauricio Arcos-Burgos,[8] Luis Guillermo Palacios,[8] Antonio Yusta,[6] Pilar Anta,[1] Immaculada Perez,[7] Margarita Hierro,[7] David G. Munoz[4] and Sagrario Barquero[5]

[1]Hospital Ramon y Cajal, Universidad de Alcala, [2]Fundacion Jimenez Diaz, [3]Fundacion Hospital Alcorcon, [4]Banco de Tejidos para la Investigacion Neurologica and [5]Hospital Clinico de San Carlos, Madrid, [6]Hospital General Universitario and [7]C.A.P. Peñalver, Guadalajara, Spain and [8]Universidad de Antioquia, Grupo de Genetica de Poblaciones, Mutacarcinogenesis y Epidemiologia Genetica, Antioquia, Colombia

nature COMMUNICATIONS

ARTICLE

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CCNF mutations in amyotrophic lateral sclerosis and frontotemporal dementia

Kelly L. Williams[1,2,3], Simon Topp[4], Shu Yang[1,2], Bradley Smith[4], Jennifer A. Fifita[1,2], Sadaf T. Warraich[1], Katharine Y. Zhang[1], Natalie Farrawell[5], Caroline Vance[4], Xun Hu[4], Alessandra Chesi[6], Claire S. Leblond[7,8], Albert Lee[1,9], Stephanie L. Rayner[1], Vinod Sundaramoorthy[1,10], Carol Dobson-Stone[11,12], Mark P. Molloy[1,9], Marka van Blitterswijk[13], Dennis W. Dickson[13], Ronald C. Petersen[14], Neill R. Graff-Radford[15], Bradley F. Boeve[14], Melissa E. Murray[13], Cyril Pottier[13], Emily Don[1], Claire Winnick[1], Emily P. McCann[1], Alison Hogan[1], Hussein Daoud[7,8], Annie Levert[7,8], Patrick A. Dion[7,8], Jun Mitsui[16], Hiroyuki Ishiura[16], Yuji Takahashi[16], Jun Goto[16], Jason Kost[17,18], Cinzia Gellera[19], Athina Soragia Gkazi[4], Jack Miller[4], Joanne Stockton[20], William S. Brooks[11], Karyn Boundy[21], Meraida Polak[22], José Luis Muñoz-Blanco[23], Jesús Esteban-Pérez[24,25], Alberto Rábano[26], Orla Hardiman[27], Karen E. Morrison[20,28,29], Nicola Ticozzi[30,31], Vincenzo Silani[30,31], Jacqueline de Belleroche[32], Jonathan D. Glass[22], John B.J. Kwok[11,12], Gilles J. Guillemin[1], Roger S. Chung[1], Shoji Tsuji[16,33], Robert H. Brown Jr[18], Alberto García-Redondo[24,25], Rosa Rademakers[13], John E. Landers[18], Aaron D. Gitler[6], Guy A. Rouleau[7,8], Nicholas J. Cole[1,3], Justin J. Yerbury[5], Julie D. Atkin[1,10], Christopher E. Shaw[4], Garth A. Nicholson[1,2,3,34] & Ian P. Blair[1,2]

doi:10.1093/brain/awy137 BRAIN 2018: 0; 1–17 | 1

BRAIN
A JOURNAL OF NEUROLOGY

Clinical, genetic and neuropathological characterization of spinocerebellar ataxia type 37

Marc Corral-Juan,[1] Carmen Serrano-Munuera,[2] Alberto Rábano,[3] Daniel Cota-González,[1] Anna Segarra-Roca,[1] Lourdes Ispierto,[4] Antonio Tomás Cano-Orgaz,[5] Astrid D. Adarmes,[6] Carlota Méndez-del-Barrio,[6] Silvia Jesús,[6] Pablo Mir,[6,7] Victor Volpini,[8] Ramiro Alvarez-Ramo,[4] Ivelisse Sánchez[1] and Antoni Matilla-Dueñas[1]

Neurociencia básica / investigación patogénica

doi:10.1093/brain/awt088 Brain 2013: Page 1 of 16 | 1

BRAIN
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The influence of phospho-tau on dendritic spines of cortical pyramidal neurons in patients with Alzheimer's disease

Paula Merino-Serrais,[1,2,3] Ruth Benavides-Piccione,[1,2,3] Lidia Blazquez-Llorca,[1,2,3] Asta Kastanauskaite,[1,2,3] Alberto Rábano,[4] Jesús Avila[3,5] and Javier DeFelipe[1,2,3]

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SCIENTIFIC REPORTS

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Different Brain Regions are Infected with Fungi in Alzheimer's Disease

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Diana Pisa[1], Ruth Alonso[1], Alberto Rábano[2], Izaskun Rodal[2] & Luis Carrasco[1]

ANN NEUROL 2019;85:691–703

RESEARCH ARTICLE

Seeding Variability of Different Alpha Synuclein Strains in Synucleinopathies

Niccolò Candelise,[1*] Matthias Schmitz,[1*] Franc Llorens,[2] Anna Villar-Piqué,[1] Maria Cramm,[1] Tobias Thom,[1] Susana Margarida da Silva Correia,[1] José Eriton Gomes da Cunha,[3] Wiebke Möbius,[4,6] Tiago F. Outeiro,[5,6,7] Valentina González Álvarez,[8] Martina Banchelli,[9] Cristiano D'Andrea,[9] Marella de Angelis,[9] Saima Zafar,[1] Alberto Rabano,[8] Paolo Matteini,[9] and Inga Zerr[1]

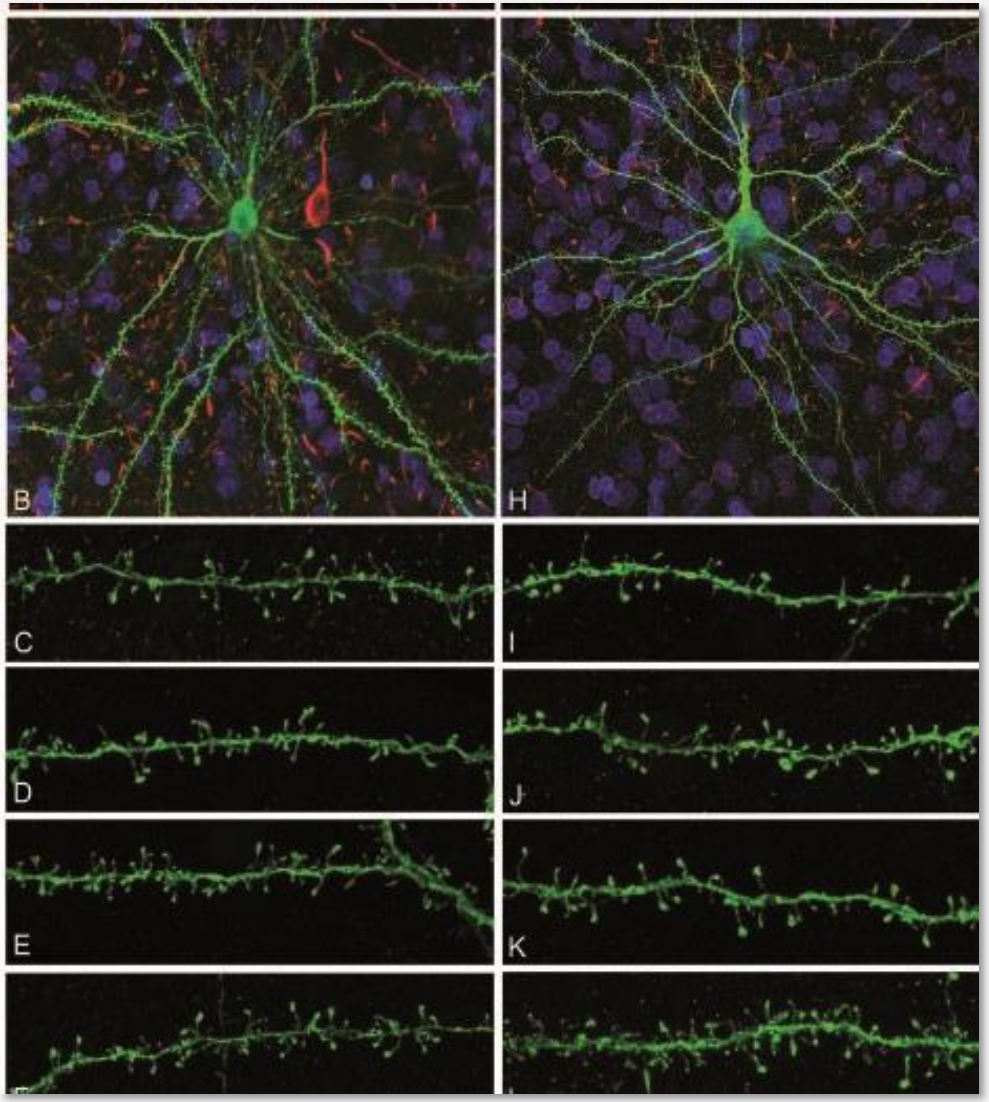
nature medicine

NATURE MEDICINE VOLUME 20 | NUMBER 8 | AUGUST 2014

Huntington's disease is a four-repeat tauopathy with tau nuclear rods

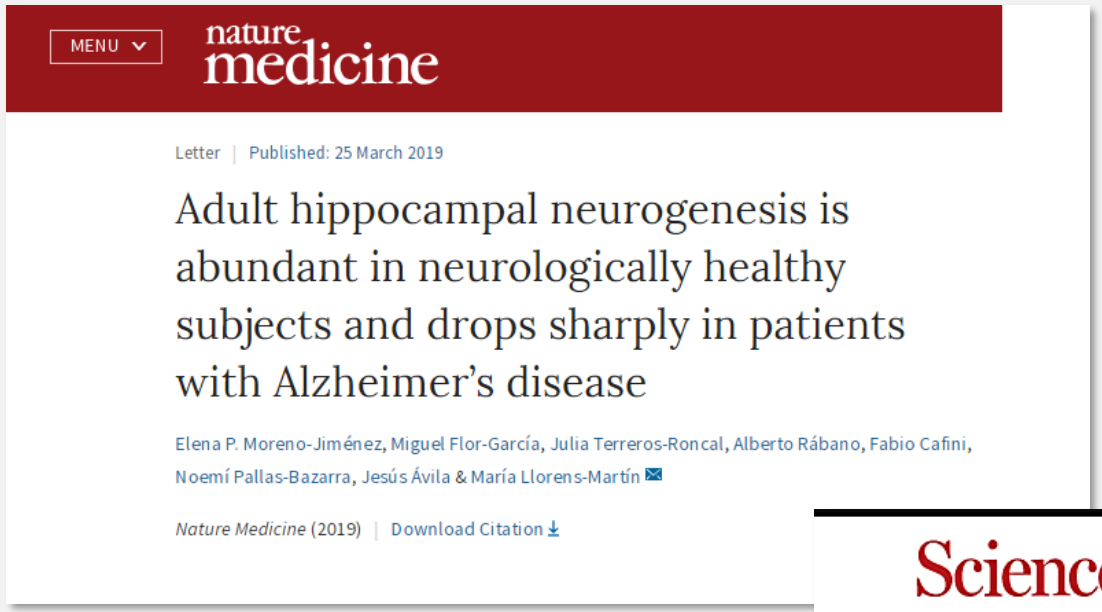
Marta Fernández-Nogales[1,2], Jorge R Cabrera[1,2,5], María Santos-Galindo[1,2,5], Jeroen J M Hoozemans[3], Isidro Ferrer[2,4], Annemieke J M Rozemuller[3], Félix Hernández[1,2], Jesús Avila[1,2] & José J Lucas[1,2]

La importancia del intervalo post mortem (IPM)



Javier de Felipe (CTB – UPM)

Cuando la clave está en el procesamiento



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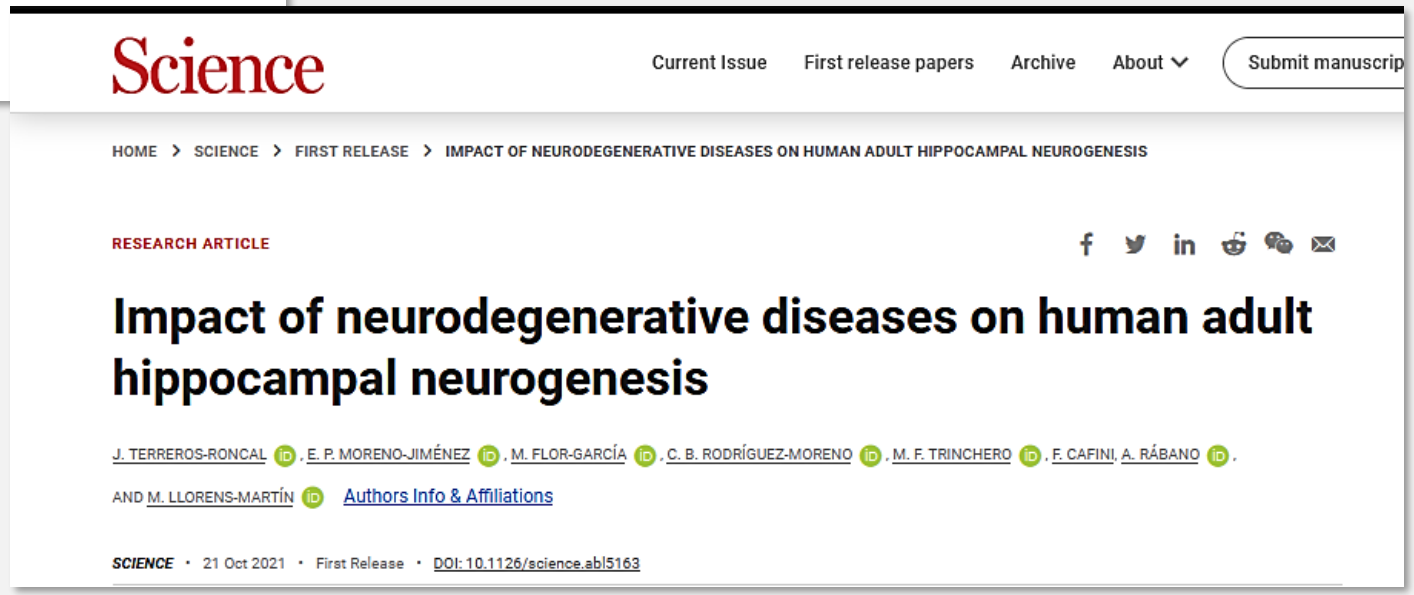
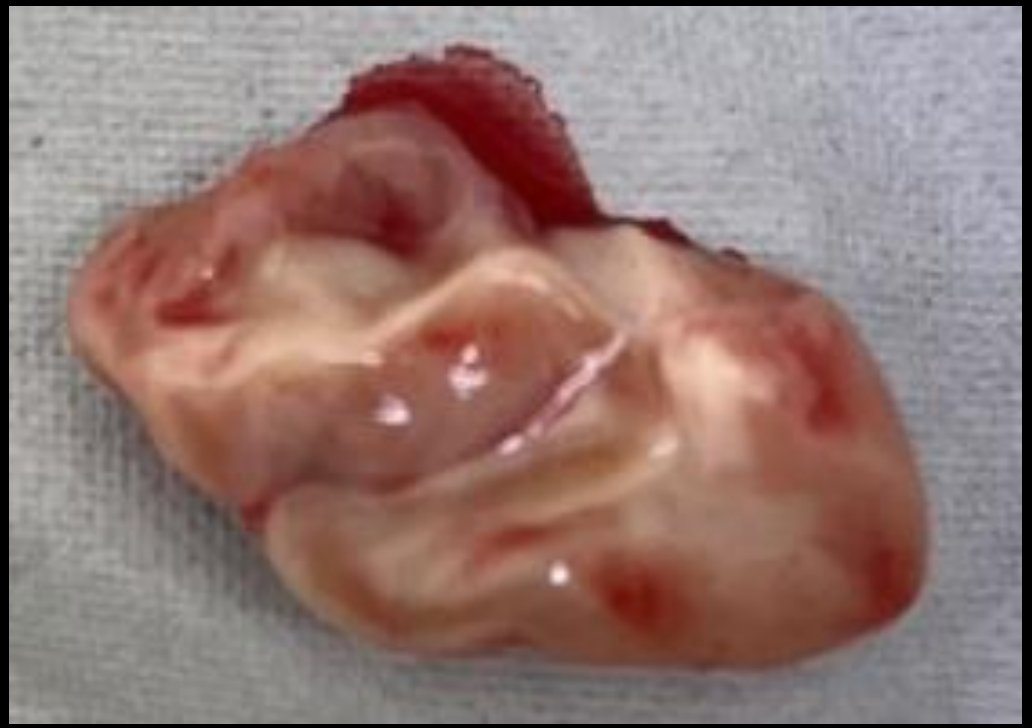
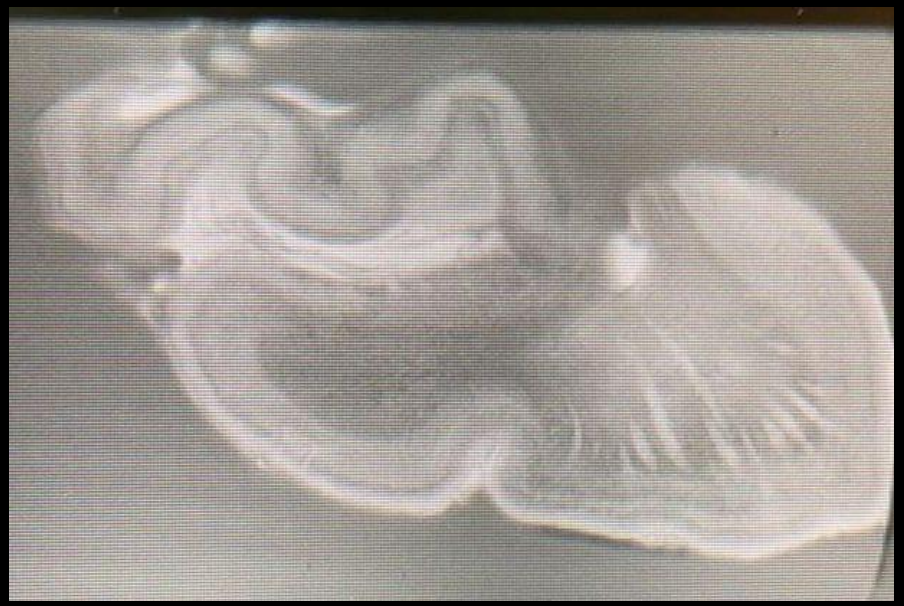
María Llorens Martín
(CBM – UAM – CSIC)



2021





Cuando lo más importante es la “n”

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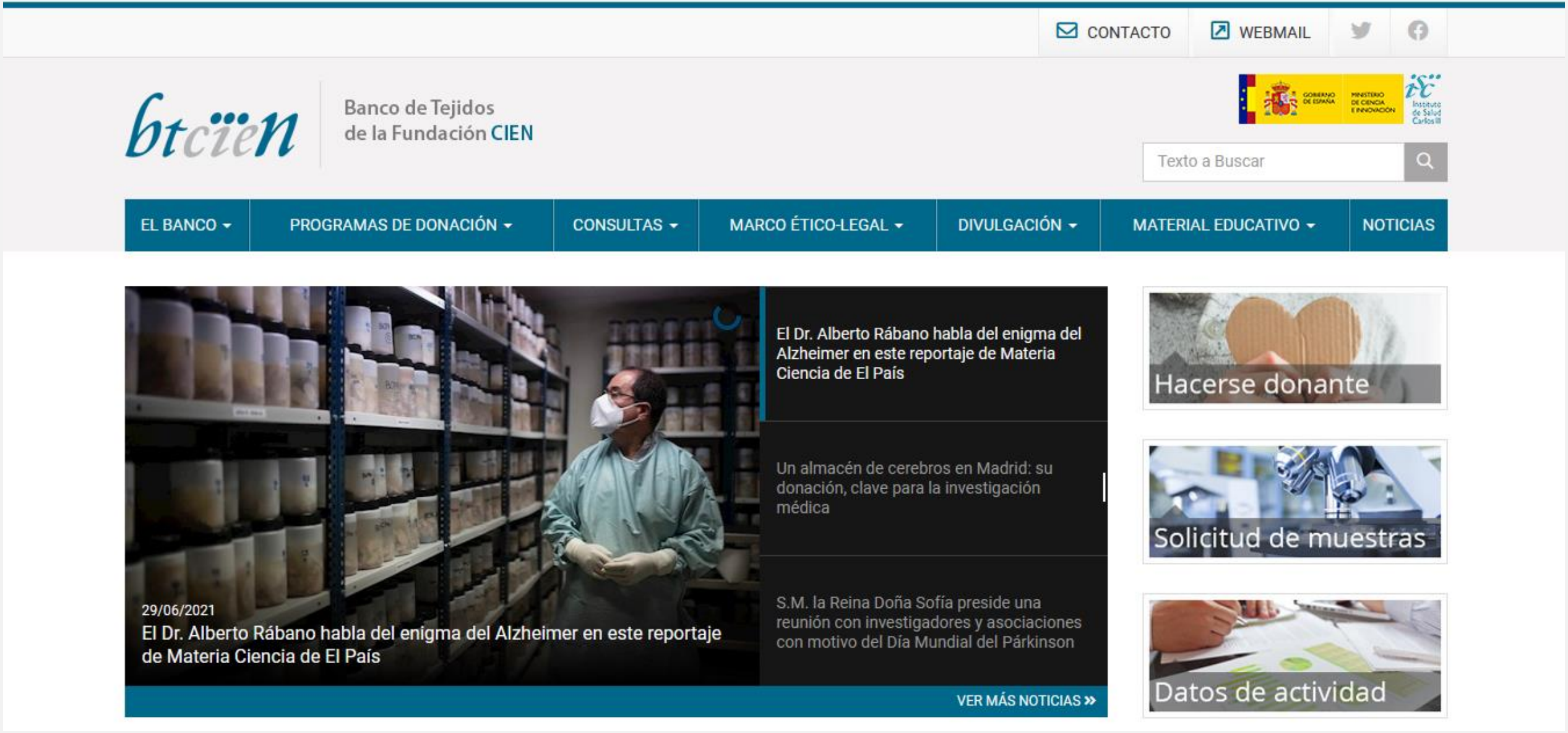
New insights into the genetic etiology of Alzheimer’s disease and related dementias

Characterization of the genetic landscape of Alzheimer’s disease (AD) and related dementias (ADD) provides a unique opportunity for a better understanding of the associated pathophysiological processes. We performed a two-stage genome-wide association study totaling 111,326 clinically diagnosed/‘proxy’ AD cases and 677,663 controls. We found 75 risk loci, of which 42 were new at the time of analysis. Pathway enrichment analyses confirmed the involvement of amyloid/tau pathways and highlighted microglia implication. Gene prioritization in the new loci identified 31 genes that were suggestive of new genetically associated processes, including the tumor necrosis factor alpha pathway through the linear ubiquitin chain assembly complex. We also built a new genetic risk score associated with the risk of future AD/dementia or progression from mild cognitive impairment to AD/dementia. The improvement in prediction led to a 1.6- to 1.9-fold increase in AD risk from the lowest to the highest decile, in addition to effects of age and the *APOE* ε4 allele.

https://bt.fundacioncien.es/



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