Supplementary Material 1

Supplementary Table 1. Search terms for the systematic literature search in PubMed. The common terms are listed in the last row and were the same for all disease groups.

Supplementary Table 2. Characteristics of studies included in the coordinate-based meta-analysis.

Supplementary Table 3. Summary of studies that included the cerebellum in analyses assessing associations between regional grey matter decrease and clinical or behavioral data.

Supplementary Table 1: Search terms for the systematic literature search in pubmed. The common terms are listed in the last row and were the same for all disease groups.

Disease	Search terms	Publication dates
AD	Title/Abstract ("AD" OR "Alzheimer") AND common terms	Any date – July 14th 2016
ALS	Title/Abstract ("ALS" OR "amyotrophic lateral sclerosis" OR "motor neuron disease" OR "MND" OR "Lou Gehrig" OR "Charcot") AND common terms	
FTD	Title/Abstract ("frontotemporal dementia" OR "FTD" OR "frontotemporal lobar degeneration" OR "FTLD") AND common terms	
HD	Title/Abstract ("Huntington" OR "HD") AND common terms	
MSA	Title/Abstract ("MSA" OR "multiple system atrophy") AND common terms	
PD	Title/Abstract ("PD" OR "Parkinson") AND common terms	
PSP	Title/Abstract ("PSP" OR "progressive supranuclear palsy") AND common terms	
Common terms: ("VBM" OR "voxel-based morphometry" OR "structural MRI") Filter: humans		

Abbreviations. AD: Alzheimer's disease; ALS: amyotrophic lateral sclerosis; FTD: frontotemporal dementia; FTLD: frontotemporal lobar degeneration; HD: Huntington's disease; MND: motor neuron disease; MSA: multiple system atrophy; PD: Parkinson's disease; PSP: progressive supranuclear palsy.

Supplementary Table 2. Study characteristics of records included in the coordinate-based meta-analysis.

Authors	Diagnosis	*N* patients (% female)	*N* controls (% female)	Age patients ± *SD*	Age controls ± *SD*	*p*-value age difference	MMSE patients ± *SD*	Disease Duration (years ± *SD*)	Coordinates (MNI)
Guo et al (2016)[1]	AD	34 (44)	34 (53)	62±6	64±5	NS	NA	3±3	-32 -72 -29 -31 -60 -19 27 -71 -28 27 -76 -26
Ossenkoppele et al (2015)[2]	Typical AD	58 (39)	61 (38)	64±9	64±8	NS	23±4	NA	-39 -82 -33 46 -73 -36
Colloby et al (2014)[3]	AD	47	39	79±9	77±6	NS	21±4	NA	-33 -43 -24 42 -43 -26 21 -85 -35 39 -79 -44 -41 -48 -32
Serra et al (2014)[4]	AD	48 (35)	20 (65)	71±6	70±6	NS	19±3	4±3	-12 -86 -24
Möller et al (2013)[5]	Late onset AD	120 (46)	71 (50)	72±5	71±4	NS	21±5	NA	33 -60 -27 30 -69 -38 12 -61 -23 26 -49 -47 -26 -48 -45 -34 -48 -45 -30 -42 -42 10 -67 -36
Canu et al (2011)[6]	AD	17 (82)	13 (46)	77±6	73±7	NS	21±5	NA	32 -64 -36 42 -59 -25 -29 -70 -39 -36 -67 -32
Lehmann et al (2011)[7]	Typical AD	30 (53)	50 (66)	69±9	63±10	<.005	19±5	5	8 -49 -30
Mazere et al (2008)[8]	AD	8 (63)	8 (75)	80±7	74±3	NS	24±2	NA	-44 -65 -42 27 -66 -11
Farrow et al (2007)[9]	Early stage AD	7	11	78±7	71±4	.014	25±4	4	25 -40 -29 -24 -36 -29

Authors	**Diagnosis**	***N* patients (% female)**	***N* controls (% female)**	**Age patients ± *SD***	**Age controls ± *SD***	***p*-value age difference**	**ALSFRS-R score ± *SD***	**Disease Duration (years ± *SD*)**	**Coordinates (MNI)**
Tan et al (2014)[10]	ALS	23 (39)	16 (50)	61±12	64±5	NS	37±8	4±5	1 -70 -56 -30 -76 -48 -5 -69 -27
Mioshi et al (2013)[11]	ALS	22 (27)	18 (50)	60±12	64±5	NS	35±11	3±3	16 -62 -58
Thivard et al (2007)[12]	Sporadic ALS	15 (40)	25 (44)	52±9	45±12	NS	30±6	3±1	10 -56 -14
Authors	**Diagnosis**	***N* patients (% female)**	***N* controls (% female)**	**Age patients ± *SD***	**Age controls ± *SD***	***p*-value age difference**	**Disease Duration (years ± *SD*)**		**Coordinates (MNI)**
Ahmed et al (2016)[13]	bvFTD	19 (47)	25 (48)	62±8	66±8	NS	6±4		40 -58 -42
Guo et al (2016)[1]	bvFTD	33 (42)	34 (53)	61±7	64±5	NS	3±3		-27 -63 -25 -24 -73 -28 -26 -49 -21 -32 -67 -40
Tan et al (2014)[10]	bvFTD	23 (35)	16 (50)	62±10	64±5	NS	4±2		-15 -54 -35 -22 -76 -35
Irish et al (2014)[14]	bvFTD	19 (42)	19 (61)	64±8	68±5	NS	4±2		-50 -70 -38 34 -78 -54 24 -40 -30
Premi et al (2014)[15]	FTD (*n*=18, 54% bvFTD)	33	12	66±7	NA	NS	3±2		-40 -36 -36 -55 -77 -36 -53 -76 -48 46 -40 -37 53 -67 -26
Irish et al (2013)[16]	bvFTD	15 (40)	15 (33)	64±9	65±4	NS	3±2		-50 -50 -52 -38 -58 -42
Lillo et al (2012)[17]	bvFTD	15 (27)	18 (50)	62±7	65±5	NS	3		-40 -68 -42 42 -60 -46
Whitwell et al (2012)[18]	FTD-C9orf72 with behavioral variant	19 (53)	40 (50)	55	58	NA[a]	6		-45 -75 -41 -6 -79 -27 -39 -40 -28

Authors	**Diagnosis**	***N* patients (% female)**	***N* controls (% female)**	**Age patients ± *SD***	**Age controls ± *SD***	***p*-value age difference**	**Disease Duration (years ± *SD*)**		**Coordinates (MNI)**
Lee et al (2011)[19]	bvFTD-CBD	3 (40)	44 (50)	66	69±5	NA[a]	8		29 -86 -41 35 -82 -46
Knutson et al (2008)[20]	Frontal variant [i.e. bv]FTD	25 (48)	14 (50)	60±8	61±6	NS	NA		26 -86 -38 50 -68 -34 -24 -86 -40 -16 -88 -36
Seeley et al (2008)[21]	bvFTD	15 (53)	45 (49)	62±10	68±8	NS	6±3		48 -67 -48
Grossman et (2004)[22]	Non-aphasic [i.e. bv] FTD	14	25	63±12	69±9	NS	4±3		11 -85 -52
Authors	**Diagnosis**	***N* patients (% female)**	***N* controls (% female)**	**Age patients ± *SD***	**Age controls ± *SD***	***p*-value age difference**	**UHDRS-III**	**Disease Duration (years ± *SD*)**	**Coordinates (MNI)**
Wolf et al (2015)[23]	Manifest HD	20 (43)	20 (35)	49±9	47±9	.56	25±12	3±2	44 -62 -26
Scharmüller et al (2013)[24]	Symptomatic HD	18	18	45±3	49±10	NA[a]	31±18	4± 3	-6 -36 -18 8 -36 -18 -21 -52 -14 22 -49 -15 -21 -55 -14 20 -66 -14 45 -60 -59 -21 -57 -45 22 -60 -47 -21 -52 -47 3 -54 -39 0 -51 -35 3 -36 -20 6 -36 -17 3 -60 -35 2 -54 -36 -21 -52 -14 22 -49 -15

									3 -36 -20
Authors	**Diagnosis**	***N* patients (% female)**	***N* controls (% female)**	**Age patients ± *SD***	**Age controls ± *SD***	***p*-value age difference**	**UHDRS-III**	**Disease Duration (years ± *SD*)**	**Coordinates (MNI)**
Gomez-Anson et al (2009)[25]	Preclinical HD	20	21	33±9	33±9	NS	3±2	NA	-19 -57 -61 22 -55 -62 21 -55 -63 -23 -57 -64 -31 -74 -44
Tabrizi et al (2009)[26]	HD stage 2	46	123	51±9	46±10	NA[a]	NA	NA	-27 -61 -25 27 -69 -35
Authors	**Diagnosis**	***N* patients (% female)**	***N* controls (% female)**	**Age patients ± *SD***	**Age controls ± *SD***	***p*-value age difference**	**Disease Duration (years ± *SD*)**		**Coordinates (MNI)**
Planetta et al (2015)[27]	MSA-P	14 (43)	14 (36)	65±9	62±8	NS	7±3		-2 -67 -27
Shigemoto et al (2013)[28]	MSA-P	20 (65)	30 (67)	63±8	63±8	NS	4±2		44 -51 -41 40 -73 -15 27 -49 -15 -42 -59 -24 -21 -80 -19
Minnerop et al (2010)[29]	MSA-P (*n*=4); MSA-C (*n*=10)	14 (50)	14 (50)	61±3	59±5	NA[a]	3±2		2 -36 -18 -20 -58 -56 28 -64 -54
Tzarouchi et al (2010)[30]	MSA-P	11 (18)	11 (27)	62±12	65±10	NA[a]	5±3		5 -59 -12 -3 -66 -14 3 -29 -4 -8 -45 -22 7 -51 -38
Chang et al (2009)[31]	MSA-C (*n*=10); MSA-P (*n*=13)	26 (46)	37 (39)	59±9	56±9	NS	NA		-41 -50 -54 17 -40 -16 25 -36 -47
Minnerop et al (2007)[32]	MSA-P (*n*=16); MSA-C (*n*=32)	48 (44)	46 (52)	62±6	59±6	NA[a]	5±2		-19 -55 -10 10 -57 -44 -9 -68 -8 25 -75 -40 -7 -48 -4

Authors	**Diagnosis**	***N* patients (% female)**	***N* controls (% female)**	**Age patients ± *SD***	**Age controls ± *SD***	***p*-value age difference**	**Disease Duration (years ± *SD*)**	**Coordinates (MNI)**
Brenneis et al (2006)[33]	MSA-C	13 (38)	13	61±6	61±4	NS	4±1	-40 -49 -29 50 -61 -40 -6 -44 -24 7 -44 -24
Specht et al (2005)[34]	MSA-C	14 (64)	13 (62)	59±7	55±7	NS	4±1	9 -36 -20

Authors	**Diagnosis**	***N* patients (% female)**	***N* controls (% female)**	**Age patients ± *SD***	**Age controls ± *SD***	***p*-value age difference**	**UPDRS-III score**	**HY score**	**Disease Duration (years ± *SD*)**	**Coordinates (MNI)**
Chen et al (2016)[36]	Idiopathic PD	23 (35)	15 (67)	61±7	56±9	.21	29±14	2±1	4±5	36 -55 -44 -45 -46 -38
O'Callaghan et al (2016)[36]	PD	78 (32)	51 (73)	67±8	66±8	NS	32±15	2±1	6±4	-24 -64 -63
Zeng et al (2016)[37]	Probable PD	45 (49)	40 (55)	62±11	60±9	.44	28±12	NA	5±2	39 -65 -36 -33 -69 -35 3 -47 -11
Gerrits et al (2014)[38]	PD	93 (34)	46 (39)	63±10	61±8	NS	25±10	2	NA	26 -87 -39
Lee et al (2014)[39]	PD-MCI	15 (33)	25 (48)	73±6	70±3	NS	17±8	2	2±2	-28 -40 -19
Rektorova et al (2014)[40]	PD	126 (40)	25 (48)	67	58	.001	NA	3	6	-32 -82 -25
Xia et al (2013)[41]	PD	32 (47)	25 (44)	70±9	67±8	NS	NA	2±1	NA	27 -70 -71
Hong et al (2012)[42]	PD (subgroup without subjective memory complaints)	15 (53)	25	65±8	66	NS	18±8	NA	2±2	1 -66 -1

Authors	**Diagnosis**	***N* patients (% female)**	***N* controls (% female)**	**Age patients ± *SD***	**Age controls ± *SD***	***p*-value age difference**	**UPDRS-III score**	**HY score**	**Disease Duration (years ± *SD*)**	**Coordinates (MNI)**
Nishio et al (2010)[43]	PD with cognitive impairment	13 (7)	13 (46)	68±6	63±5	NS	22±6	3±0	6±6	-22 -48 -32
Lehericy et al (2010)[44]	Guadeloupean PD	9 (33)	9 (44)	68±10	67±5	NS	41	NA	7±4	-8 -48 -10 16 -46 -12 32 -44 -32
Camicioli et al (2009)[45]	PD	43 (44)	43 (44)	71±4	71±5	NS	14±7 (dopamine responsive) 3±3 (dopamine non-responsive)	2±1	8±5	-16 -53 -21 20 -59 -20 7 -61 -38
Pereira et al (2009)[46]	PD	36 (61)	20 (50)	73±6	73±7	NS	27±13	3±1	12±5	-34 -44 -42 -14 -72 -32 10 -88 -33 -26 -74 -54
Authors	**Diagnosis**	***N* patients (% female)**	***N* controls (% female)**	**Age patients ± *SD***	**Age controls ± *SD***	***p*-value age difference**	**UPDRS-III score**	**HY score**	**Disease Duration (years ± *SD*)**	**Coordinates (MNI)**
Piattella et al (2015)[47]	PSP	16	16	68±6	NA	NS	27±17	3±1	3	10 -64 -24
Wang et al (2015)[48]	PSP	24 (25)	23 (39)	64±7	61±6	.07	NA	3	4±3	-6 -36 -16
Sandhya et al (2014)[49]	PSP	10 (10)	8 (38)	NA	NA	NA	NA	NA	NA	-16 -29 -17 -38 -39 -31 -44 -47 -43 23 -90 -20 -4 -80 -17 9 -83 -17 -50 -68 -31

Authors	Diagnosis	*N* patients (% female)	*N* controls (% female)	Age patients ± *SD*	Age controls ± *SD*	*p*-value age difference	UPDRS-III score	HY score	Disease Duration (years ± *SD*)	Coordinates (MNI)
										-41 -77 -22 -44 -69 -22 41 -49 -54 47 -48 -44 48 -56 -49 15 -33-19 1 -38 -11 33 -80 -48 40 -79 -41
Giordano et al (2013)[50]	PSP	15 (47)	15 (47)	69±1	66±6	NS	38±4	4±1	3±1	-21 -56 -32 9 -52 -44
Lagarde et al (2013)[51]	PSP	21 (62)	18 (65)	66±7	68±5	NS	NA	NA	4±2	21 -84 -27 -46 -55 -47
Ghosh et al (2012)[52]	PSP	23	22	71±9	71±8	NS	34±16	NA	3	-34 -88 -44 46 -70 -56 -46 -44 -48 -48 -60 -58 -8 -40 -18
Lee et al (2011)[19]	PSP-CBS	5 (40)	44 (50)	69	69±5	NS	NA	NA	8	10 -41 -25 14 -55 -35
Agosta et al (2010)[53]	PSP parkinsonism (*n*=10) PSP Richardson Syndrome (*n*=10)	20 (70)	24 (46)	65	64	NS	33	3.0	4	32 -49 -18
Cordato et al (2005)[54]	PSP	21 (33)	25 (36)	70±6	72±7	NA[a]	23±10	4±1	4±3	6 -48 -35

Abbreviations. AD: Alzheimer's disease; ALS: amyotrophic lateral sclerosis; ALSFRS-R: ALS Functional Rating Scale – Revised; (bv)FTD: (behavioural variant)

frontotemporal dementia; CBD: corticobasal degeneration; HD: Huntington's disease; HY: Hoehn and Yahn; M: motor score; MCI: mild cognitive impairment; MMSE: Mini Mental State Exam; MNI: Montreal Neurological Institute; MSA: multiple system atrophy; MSA-C: MSA cerebellar subtype; MSA-P: MSA Parkinsonian subtype; NA: not available; NS: not significant; PD: Parkinson's disease; PSP: progressive supranuclear palsy; SD: standard deviation; UPDRS-III: Unified Parkinson's Disease Rating Scale, motor subscore; UHDRS-III: Unified Huntington's Disease Rating Scalem motor subscore. [a]Author report controls and patients as age-matched but do not report whether a statistical test confirmed this for the patient group included in this study.

Supplementary Table 3. Summary of studies that included the cerebellum in analyses assessing associations between regional grey matter decrease and clinical or behavioral data.

Study	Analysis	Result	Disease Duration
Alzheimer's Disease			
Colloby et al (2014)[3]	Correlation of cognitive and clinical measures (CAMCOG, MMSE, NPI, UPDRS III, CAF scores) with volume loss	No significant findings in any brain region	NA
Möller et al (2013)[5]	Correlation between regional GM reductions and dementia severity measured using MMSE	No significant findings in cerebellum	NA
Farrow et al (2007)[9]	Partial correlations (controlling for global grey matter volume and age) between GM volume and ADAS-TES	No significant findings in cerebellum	25±4
*Amyotrophic Lateral Sclerosis and Frontotemporal Dementia**			
Tan et al (2014)[10] *(both patient groups were included in the analysis)	Correlation of GM volume loss with measures of cognitive, neuropsychiatric, and motor function as measured with ACE-R, CBI-R, and ALSFRS-R (motor analysis included only ALS and ALS-bvFTD patients; bvFTD patients were excluded)	ACE-R scores correlated with grey matter volumes of the cerebellum in bilateral lobules I-IV, V, VI, VII (Crus I), VII (Crus II), VIIb, and right VIIIa, VIIIb, IX CBI-R measures were associated with grey matter volumes in right lobule V, and bilateral lobule VI and VII (Crus I) ALSFRS-R scores correlated with grey matter volumes in right lobule V, VIIIa, VIIIb, and IX, in bilateral lobule VI and VIIb, and left lobule VII	4±5 (ALS) 4±2 (FTD)
Frontotemporal Dementia			
Irish et al (2013)[16]	Correlation of GM intensity decrease and episodic memory recall performance	No significant findings in cerebellum for episodic memory dysfunction in C9orf72 FTD patients In sporadic FTD, memory performance correlated with GM intensity decrease in bilateral cerebellum	3±2

Study	Analysis	Result	Disease Duration
Knutson et al (2008)[20]	Correlation of caregiver burden and NPI scores with GM atrophy	No significant findings in cerebellum	NA
Grossman et al (2004)[22]	Correlations of GM atrophy with confrontation naming	Correlations between GM loss in cerebellum and confrontation naming performance only in patient subgroups of corticobasal degeneration with FTD and non-aphasic FTD	4±3
Huntington's Disease			
Wolf et al (2015)[23]	Correlation of GM volume decrease with UHDRS score	No significant findings in cerebellum	3±2
Scharmüller et al (2013)[24]	Correlations of GM volume with affect recognition intensity, symptom severity as measured with UHDRS, and disease duration	Lower anger ratings were correlated with reduced GM volume in vermal and lateral cerebellar areas Degree of anger misclassification was associated with reduced GM volume of vermal lobule III and hemispheric lobule III Positive correlation between volume of vermal lobule VI and UHDRS independence score, indicating that patients with more GM volume have smaller impairment Symptom duration in months showed negative correlation with GM volume of hemispheric lobule X	4± 3
Gomez-Anson et al (2009)[25]	Correlations of GM volume with visuomotor performance and CAG number	Negative correlations between focal volume loss on VBM and visuomotor performance (the 15-Objects test, time to achieve the task) in right cerebellum (corrected $p<.05$) No significant findings in cerebellum for CAG number	NA
Multiple System Atrophy			
Shigemoto et al (2013)[28]	Correlation of GM loss and disease duration and severity	No brain regions showed significant correlations	4±2

Study	Analysis	Result	Disease Duration
Chang et al (2009)[31]	Correlation of CVLT-MS memory scores with GM atrophy	No significant findings in cerebellum	NA
Minnerop et al (2007)[32]	Correlations of GM loss and disease duration	In both MSA-C and MSA-P patients, GM loss was correlated with disease duration in cerebellar vermis and adjacent parts of cerebellar hemispheres	5±2
Brenneis et al (2006)[33]	Correlation of GM densities with cerebellar ataxia score	Negative correlation between GM density and cerebellar ataxia score in cerebellar hemispheres	4±1
Parkinson's Disease			
Chen et al (2016)[35]	Partial correlation using demographic data, cardiovascular data, and circulatory epithelial progenitor cell levels (controlled for age and sex)	Left lobule VIIa GM volume correlated positively with baroreflex sensitivity and negatively with numbers of epithelial progenitor cells	4±5
O'Callaghan et al (2016)[36]	Correlated average cerebellar atrophy score against average resting state connectivity separately between each cerebellar module (motor and cognitive) and resting state networks (default mode, frontoparietal, ventral attention, the dorsal attention and sensorimotor network)	Correlation between GM atrophy and UPDRS-III Correlation of extent of cerebellar atrophy with relative loss of connectivity between the motor cerebellum and default mode, sensorimotor, and dorsal attention network Correlation of cerebellar atrophy with increase in connectivity between motor cerebellum and frontoparietal network Correlation of atrophy in cognitive cerebellum with loss of connectivity with sensorimotor network	6±4
Zeng et al (2016)[37]	Partial correlation between GM densities and UPDRS score, controlling for age	No significant findings in cerebellum	5±2
Gerrits et al (2014)[38]	Correlations between GM volume and visuospatial learning and memory score, and executive functioning	No significant findings in cerebellum	NA

Study	Analysis	Result	Disease Duration
Camicioli et al (2009)[45]	Correlations between CVLT-II long delay free recall z-scores and executive functions with GM volume	No significant findings in cerebellum for CVLT-II long delay free recall scores Correlation between GM volume and executive function in left cerebellum	8±5
Pereira et al (2009)[46]	Correlation between performance on facial recognition test, VFDT, and recognition memory test	No significant findings in cerebellum	12±5
Progressive Supranuclear Palsy			
Giordano et al (2013)[50]	Correlations of FAB score, disease duration, phonological verbal fluency, PIGDs, UPDRS-III, and TPTC with GM volume	No significant findings in cerebellum for UPDRS-III performance Higher FAB score correlated positively with larger GM volume in cerebellum Disease duration was positively associated with GM loss in bilateral cerebellum PIGDs was negatively correlated with right cerebellum volume Phonological verbal fluency was positively correlated right cerebellum volumes	3±1
Lagarde et al (2013)[51]	Correlations of GM density and environmental dependency	No significant findings in cerebellum	4±2
Ghosh et al (2012)[52]	Correlations of GM atrophy and voice emotion recognition performance and theory of mind task	GM atrophy correlated with performance in voice emotion recognition in cerebellum Theory of mind task performance correlated negatively with grey matter atrophy in cerebellum	3

Study	Analysis	Result	Disease Duration
Agosta et al. (2010)[53]	Correlation of GM volume and BNT, Letter Fluency, and Category Fluency	No significant findings in cerebellum for BNT and Category Fluency Letter Fluency performance was associated with GM loss in left cerebellum	4
Cordato et al (2005)[54]	Correlations of UPDRS-motor subscore, frontal behavioral disturbance, disease duration, and MMSE with GM loss	No significant findings in cerebellum for frontal behavioral disturbance and motor scores No significant findings for MMSE and disease duration in any brain region	4±3

Abbreviations. ACE-R: Addenbrooke's Cognitive Examination Revised; ADAS-Cog: Alzheimer's Disease Assessment Scale-Cognitive; ADAS-TES: Alzheimer's Disease Assessment Scale – Total Error Score; ALS: amyotrophic lateral sclerosis; ALSFRS-R: Amyotrophic Lateral Sclerosis Functional Rating Score-Revised; BNT: Boston Naming Test; (bv)FTD: (behavioral variant) frontotemporal dementia; CAF: Clinical Assessment of Fluctuation; CAG: cytosine-adenin-guanine; CAMCOG: Cambridge Cognitive Examination; CBI-R: Cambridge Behavioural Inventory-Revised; CVLT(-MS): California Verbal Learning Test(-Mental Status); FAB: Frontal Assessment Battery; FEW: Family-wise error; GM: Gray matter; MMSE: Mini Mental State Exam; MSA-C: multiple system atrophy-cerebellar type; MSA-P: multiple system atrophy-parkinsonian type; NA: not available; NPI: Neuropsychiatric Inventory; PIGDs: Postural Instability Gait Disturbance sub-score; TPTC: Ten Point Clock Test; UHDRS: Unified Huntington's Disease Rating Scale; UPDRS: Unified Parkinson's Disease Rating Scale; VFDT: Visual Form Discrimination Test.

REFERENCES

1 Guo CC, Tan R, Hodges JR, *et al*. Network-selective vulnerability of the human cerebellum to Alzheimer's disease and frontotemporal dementia. *Brain* 2016;**139**:1527-38.

2 Ossenkoppele R, Pijnenburg YA, Perry DC, *et al*. The behavioural/dysexecutive variant of Alzheimer's disease: clinical, neuroimaging and pathological features. *Brain* 2015;**138**:2732-49.

3 Colloby SJ, O'Brien JT, Taylor J-P. Patterns of cerebellar volume loss in dementia with Lewy bodies and Alzheimer′s disease: A VBM-DARTEL study. *Psychiat Res-Neuroim* 2014;**223**:187-91.

4 Serra L, Fadda L, Perri R, *et al*. Constructional apraxia as a distinctive cognitive and structural brain feature of pre-senile Alzheimer's disease. *J Alzheimers Dis* 2014;**38**:391-402.

5 Möller C, Vrenken H, Jiskoot L, *et al*. Different patterns of gray matter atrophy in early- and late-onset Alzheimer's disease. *Neurobiol Aging* 2013;**34**:2014-22.

6 Canu E, McLaren DG, Fitzgerald ME, *et al*. Mapping the structural brain changes in Alzheimer's disease: the independent contribution of two imaging modalities. *J Alzheimers Dis* 2011;**26**(Suppl 3):263-74.

7 Lehmann M, Crutch SJ, Ridgway GR, *et al*. Cortical thickness and voxel-based morphometry in posterior cortical atrophy and typical Alzheimer's disease. *Neurobiol Aging* 2011;**32**:1466-76.

8 Mazere J, Prunier C, Barret O, *et al*. In vivo SPECT imaging of vesicular acetylcholine transporter using [(123)I]-IBVM in early Alzheimer's disease. *Neuroimage* 2008;**40**:280-8.

9 Farrow TF, Thiyagesh SN, Wilkinson ID, *et al*. Frontotemporal-lobe atrophy in early-stage Alzheimer's disease identified using an improved detection methodology. *Psychiatry Res* 2007;**155**:11-9.

10 Tan RH, Devenney E, Dobson-Stone C, *et al*. Cerebellar Integrity in the Amyotrophic Lateral Sclerosis - Frontotemporal Dementia Continuum. *PLoS One* 2014;**9**:1-10.

11 Mioshi E, Lillo P, Yew B, *et al*. Cortical atrophy in ALS is critically associated with neuropsychiatric and cognitive changes. *Neurology* 2013;**80**:1117-23.

12 Thivard L, Pradat PF, Lehericy S, *et al*. Diffusion tensor imaging and voxel based morphometry study in amyotrophic lateral sclerosis: relationships with motor disability. *J Neurol Neurosurg Psychiatry* 2007;**78**:889-92.

13 Ahmed RM, Irish M, Henning E, *et al*. Assessment of Eating Behavior Disturbance and Associated Neural Networks in Frontotemporal Dementia. *JAMA Neurol* 2016;73:282-290.

14 Irish M, Piguet O, Hodges JR, *et al*. Common and unique gray matter correlates of episodic memory dysfunction in frontotemporal dementia and Alzheimer's disease. *Hum Brain Mapp* 2014;**35**:1422-35.

15 Premi E, Garibotto V, Gazzina S, *et al*. Subcortical and Deep Cortical Atrophy in Frontotemporal Dementia due to Granulin Mutations. *Dement Geriatr Cogn Dis Extra* 2014;**4**:95-102.

16 Irish M, Devenney E, Wong S, *et al*. Neural substrates of episodic memory dysfunction in behavioural variant frontotemporal dementia with and without C9ORF72 expansions. *Neuroimage Clin* 2013;**2**:836-43.

17 Lillo P, Mioshi E, Burrell JR, *et al*. Grey and white matter changes across the amyotrophic lateral sclerosis-frontotemporal dementia continuum. *PLoS One* 2012;**7**:e43993. doi:http://dx.doi.org/10.1371/journal.pone.0043993

18 Whitwell JL, Weigand SD, Boeve BF, *et al*. Neuroimaging signatures of frontotemporal dementia genetics: C9ORF72, tau, progranulin and sporadics.

Brain 2012;**135**:794-806.
19 Lee SE, Rabinovici GD, Mayo MC, *et al*. Clinicopathological correlations in corticobasal degeneration. *Ann Neurol* 2011;**70**:327-40.
20 Knutson KM, Zamboni G, Tierney MC, *et al*. Neural correlates of caregiver burden in cortical basal syndrome and frontotemporal dementia. *Dement Geriatr Cogn Disord* 2008;**26**:467-74.
21 Seeley WW, Crawford R, Rascovsky K, *et al*. Frontal paralimbic network atrophy in very mild behavioral variant frontotemporal dementia. *Arch Neurol* 2008;**65**:249-55.
22 Grossman M, McMillan C, Moore P, *et al*. What's in a name: voxel-based morphometric analyses of MRI and naming difficulty in Alzheimer's disease, frontotemporal dementia and corticobasal degeneration. *Brain* 2004;**127**:628-49.
23 Wolf RC, Thomann PA, Sambataro F, *et al*. Abnormal cerebellar volume and corticocerebellar dysfunction in early manifest Huntington's disease. *J Neurol* 2015;**262**:859-69.
24 Scharmüller W, Ille R, Schienle A. Cerebellar contribution to anger recognition deficits in Huntington's disease. *Cerebellum* 2013;**12**:819-25.
25 Gomez-Anson B, Alegret M, Munoz E, *et al*. Prefrontal cortex volume reduction on MRI in preclinical Huntington's disease relates to visuomotor performance and CAG number. *Parkinsonism Relat Disord* 2009;**15**:213-9.
26 Tabrizi SJ, Langbehn DR, Leavitt BR, *et al*. Biological and clinical manifestations of Huntington's disease in the longitudinal TRACK-HD study: cross-sectional analysis of baseline data. *Lancet Neurol* 2009;**8**:791-801.
27 Planetta PJ, Kurani AS, Shukla P, *et al*. Distinct functional and macrostructural brain changes in Parkinson's disease and multiple system atrophy. *Hum Brain Mapp* 2015;**36**:1165-79.
28 Shigemoto Y, Matsuda H, Kamiya K, *et al*. In vivo evaluation of gray and white matter volume loss in the parkinsonian variant of multiple system atrophy using SPM8 plus DARTEL for VBM. *Neuroimage Clin* 2013;**2**:491-6.
29 Minnerop M, Luders E, Specht K, *et al*. Callosal tissue loss in multiple system atrophy--a one-year follow-up study. *Mov Disord* 2010;**25**:2613-20.
30 Tzarouchi LC, Astrakas LG, Konitsiotis S, *et al*. Voxel-based morphometry and Voxel-based relaxometry in parkinsonian variant of multiple system atrophy. *J Neuroimaging* 2010;**20**:260-6.
31 Chang CC, Chang YY, Chang WN, *et al.* Cognitive deficits in multiple system atrophy correlate with frontal atrophy and disease duration. *Eur J Neurol* 2009;**16**:1144-50.
32 Minnerop M, Specht K, Ruhlmann J, *et al.* Voxel-based morphometry and voxel-based relaxometry in multiple system atrophy-a comparison between clinical subtypes and correlations with clinical parameters. *Neuroimage* 2007;**36**:1086-95.
33 Brenneis C, Boesch SM, Egger KE, *et al.* Cortical atrophy in the cerebellar variant of multiple system atrophy: a voxel-based morphometry study. *Mov Disord* 2006;**21**:159-65.
34 Specht K, Minnerop M, Muller-Hubenthal J, *et a*l. Voxel-based analysis of multiple-system atrophy of cerebellar type: complementary results by combining voxel-based morphometry and voxel-based relaxometry. *Neuroimage* 2005;**25**:287- 93.
35 Chen MH, Lu CH, Chen PC, *et al.* Association between autonomic impairment and structural deficit in Parkinson disease. *Medicine* 2016;**95**:e3086. doi:10.1097/MD.0000000000003086
36 O'Callaghan C, Hornberger M, Balsters JH, *et al*. Cerebellar atrophy in Parkinson's disease and its implication for network connectivity. *Brain* 2016;**139**:845-55.
37 Zeng LL, Xie L, Shen H, *et al*. Differentiating Patients with Parkinson's Disease

from Normal Controls Using Gray Matter in the Cerebellum. *Cerebellum* 2017; **16**:151-7.
38 Gerrits NJ, van der Werf YD, Hofman M, *et al*. Gray matter differences contribute to variation in cognitive performance in Parkinson's disease. *Eur J Neurol* 2014;**21**:245-52.
39 Lee JE, Cho KH, Song SK, *et al*. Exploratory analysis of neuropsychological and neuroanatomical correlates of progressive mild cognitive impairment in Parkinson's disease. *J Neurol Neurosurg Psychiatry* 2014;**85**:7-16.
40 Rektorova I, Biundo R, Marecek R, *et al*. Grey Matter Changes in Cognitively Impaired Parkinson's Disease Patients. *PLoS One* 2014;**9**:1-8.
41 Xia J, Wang J, Tian W, *et al*. Magnetic resonance morphometry of the loss of gray matter volume in Parkinson's disease patients. *Neural Regen Res* 2013;**8**:2557-65.
42 Hong JY, Lee JE, Sohn YH, *et al*. Neurocognitive and atrophic patterns in Parkinson's disease based on subjective memory complaints. *J Neurol* 2012;**259**:1706-12.
43 Nishio Y, Hirayama K, Takeda A, *et al*. Corticolimbic gray matter loss in Parkinson's disease without dementia. *Eur J Neurosci* 2010;**17**:1090-7.
44 Lehericy S, Hartmann A, Lannuzel A, *et al*. Magnetic resonance imaging lesion pattern in Guadeloupean parkinsonism is distinct from progressive supranuclear palsy. *Brain* 2010;**133**:2410-25.
45 Camicioli R, Gee M, Bouchard TP, *et al*. Voxel-based morphometry reveals extra-nigral atrophy patterns associated with dopamine refractory cognitive and motor impairment in parkinsonism. *Parkinsonism Relat Disord* 2009;**15**:187-95.
46 Pereira JB, Junqué C, Martí M-J, *et al*. Neuroanatomical substrate of visuospatial and visuoperceptual impairment in Parkinson's disease. *Mov Disord* 2009;**24**:1193-9.
47 Piattella MC, Upadhyay N, Bologna M, *et al*. Neuroimaging evidence of gray and white matter damage and clinical correlates in progressive supranuclear palsy. *J Neurol* 2015;**262**:1850-8.
48 Wang G, Wang J, Zhan J, *et al*. Quantitative assessment of cerebral gray matter density change in progressive supranuclear palsy using voxel based morphometry analysis and cerebral MR T1-weighted FLAIR imaging. *J Neurological Sci* 2015;**359**:367-72.
49 Sandhya M, Saini J, Pasha S, *et al*. A voxel based comparative analysis using magnetization transfer imaging and T1-weighted magnetic resonance imaging in progressive supranuclear palsy. *Ann Indian Acad Neurol* 2014;**17**:193-8.
50 Giordano A, Tessitore A, Corbo D, *et al*. Clinical and cognitive correlations of regional gray matter atrophy in progressive supranuclear palsy. *Parkinsonism Relat Disord* 2013;**19**:590-4.
51 Lagarde J, Valabregue R, Corvol JC, *et al*. The clinical and anatomical heterogeneity of environmental dependency phenomena. *J Neurol* 2013;**260**:2262-70.
52 Ghosh BC, Calder AJ, Peers PV, *et al*. Social cognitive deficits and their neural correlates in progressive supranuclear palsy. *Brain* 2012;**135**:2089-102.
53 Agosta F, Kostic VS, Galantucci S, *et al*. The in vivo distribution of brain tissue loss in Richardson's syndrome and PSP-parkinsonism: a VBM-DARTEL study. *Eur J Neurosci* 2010;**32**:640-7.
54 Cordato NJ, Duggins AJ, Halliday GM, *et al*. Clinical deficits correlate with regional cerebral atrophy in progressive supranuclear palsy. *Brain* 2005;**128**:1259-66.