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REVIEW

The Cerebellum in Frontotemporal Dementia: a Meta-Analysis of Neuroimaging Studies

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Abstract

Frontotemporal dementia (FTD) is a neurodegenerative brain disorder primarily affecting the frontal and/or temporal lobes. Three main subtypes have been recognized: behavioural-variant FTD (bvFTD), semantic dementia (SD), and progressive nonfluent aphasia (PNFA), each of which has a distinct clinical and cognitive profile. Although the role of the cerebellum in cognition is increasingly accepted, knowledge of cerebellar changes across neuroimaging modalities and their contribution to behavioural and cognitive changes in FTD syndromes is currently scant. We conducted an anatomical/activation likelihood estimation (ALE) meta-analysis in 53 neuroimaging studies (structural MRI: 42; positron emission tomography: 6; functional MRI: 4; single-photon emission computed tomography: 1) to identify the patterns of cerebellar changes and their relations to profiles of behavioural and cognitive deficits in FTD syndromes. Overall, widespread bilateral cerebellar changes were found in FTD and notably the patterns were subtype specific. In bvFTD, ALE peaks were identified in the bilateral Crus, left lobule VI, right lobules VIIb and VIIIb. In SD, focal cerebellar changes were located in the left Crus I and lobule VI. A separate ALE meta-analysis on PNFA studies was not performed due to the limited number of studies available. In addition, the ALE analysis indicated that bilateral Crus I and Crus II were associated with behavioural disruption and cognitive dysfunction. This ALE meta-analysis provides the quantification of the location and extent of cerebellar changes across the main FTD syndromes, which in turn provides evidence of cerebellar contributions to behavioural and cognitive changes in FTD. These results bring new insights into the mechanisms mediating FTD symptomatology.

Keywords Frontotemporal dementia · Cerebellum · Cognition · Meta-analysis · Structural imaging · Functional imaging

Introduction

Frontotemporal dementia (FTD) is a neurodegenerative brain disorder affecting primarily the frontal and temporal lobes (Rascovsky et al., 2011; Gorno-Tempini et al., 2011). It is a common cause of dementia in individuals aged 45–64 years with a prevalence roughly equal to that of younger-onset Alzheimer's disease (10–15 per 100,000) (Ratnavalli, Brayne, Dawson, & Hodges, 2002; Coyle-Gilchrist et al., 2016). Patients with FTD present with personality and behavioural changes, impairment of social cognition, or language disturbances (Rascovsky et al., 2011; Gorno-Tempini et al., 2011). Based on distinct clinical and cognitive profiles, three subtypes of FTD have been recognised: behavioural-variant FTD (bvFTD), semantic dementia (SD), and progressive nonfluent aphasia (PNFA) (Rascovsky et al., 2011; Gorno-Tempini et al., 2011).

It is now accepted that, beyond motor function, the cerebellum plays a role in cognition with contributions to executive function, visuospatial processing, language, and behaviour regulation. Human and animal research has shown that the cerebellum receives projections from the cerebral cortex (Ramnani, 2006; Schmahmann & Pandya, 1997a; Schmahmann & Pandya, 1995; Strick, Dum, & Fiez, 2009; Ramnani, 2012; Ramnani et al., 2006; Krienen & Buckner, 2009; Jissendi, Baudry, & Baleriaux, 2008; Schmahmann & Sherman, 1998). Cerebellar Crus I and Crus II are subregions

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that connect to the prefrontal cortex (Kelly & Strick, 2003). In healthy populations, these subregions have been associated with cognitive abilities, including attention, visuospatial ability, memory, language, executive function, emotion processing, and social cognition (Van Overwalle, D'Aes, & Marien, 2015; Stoodley & Schmahmann, 2009). Changes in these subregions have also been reported in clinical populations, such as developmental dyslexia (Gross-Tsur, Ben-Bashat, Shalev, Levav, & Sira, 2006), autism spectrum disorder (Laidi et al., 2017), alcohol-related brain damage (Cheng et al., 2017), movement disorders (Dogan et al., 2016; Gao et al., 2017; de Azevedo et al., 2017), dementia (Colloby, O'Brien, & Taylor, 2014; Synn et al., 2018; Tan et al., 2015), and psychiatric disorders (Andreasen & Pierson, 2008; Shinn et al., 2017). Of note, many of the clinical features observed in FTD syndromes overlap with cognitive processes that are in part supported by the cerebellum.

In FTD, circumscribed atrophy (Tan et al., 2014; Guo et al., 2016; Tan et al., 2015; Chen et al., 2018), disrupted functional networks (Guo et al., 2016), as well as abnormal gene expression (Belzil et al., 2013) and pathological changes (Ferrer et al., 2015) of the cerebellum have been reported. Focus on the cerebellum has especially increased since recognition of the *C9orf72* gene expansion as the most common genetic cause for FTD and amyotrophic lateral sclerosis (ALS) (Belzil et al., 2013; Suarez-Calvet et al., 2015; Whitwell et al., 2012). Specifically, some studies have suggested that cerebellar abnormalities may be more common in individuals with the *C9orf72* gene expansion than in sporadic, non-genetic, cases of FTD (Whitwell et al., 2012; Devenney et al., 2014; Sha et al., 2012). Despite this, research on the clinical phenomenology of FTD has generally been limited to cerebral regions with scant attention to cerebellar contribution. Only two studies to date have investigated cerebellar involvement in cognition in FTD (Chen et al., 2018; Tan et al., 2015), both of which found cerebellar grey matter atrophy and demonstrated its relations with cognitive dysfunction.

Anatomical/Activation Likelihood Estimation (ALE) is a widely used and accepted coordinate-based meta-analysis technique which enables the synthesis of imaging findings across studies. ALE aims to estimate the likelihood (but not the effect size) that a peak change lies in any given voxel. As such, the algorithm treats reported foci as spatial probability distributions centred at the given coordinates (Eickhoff, Bzdok, Laird, Kurth, & Fox, 2012; Eickhoff et al., 2009). Applied to the cerebellum, this technique has demonstrated cerebellar involvement in a range of cognitive domains in healthy participants (Van Overwalle et al., 2015; Stoodley & Schmahmann, 2009). Only one ALE analysis has investigated cerebellar grey matter atrophy in FTD to date, and this study was limited to bvFTD and structural neuroimaging studies (Gellersen et al., 2017). Knowledge of affected cerebellar subregions across neuroimaging modalities in FTD syndromes is currently absent as is knowledge about how these cerebellar changes relate to behavioural and cognitive dysfunction. To address these questions, we conducted a meta-analysis of published neuroimaging studies using an inclusive search strategy to provide an overview of the current literature on cerebellar changes across FTD syndromes. Furthermore, we investigated the association between cerebellar changes and behaviour and cognitive function in FTD syndromes. Based on previous findings in both healthy adults and patients with FTD (Chen et al., 2018; Stoodley & Schmahmann, 2009), we hypothesised that cerebellar changes in both posterior and anterior lobules would be found in FTD syndromes and would be subtype-specific. In addition, we hypothesised that distinct cerebellar changes would relate to specific behavioural and cognitive changes (e.g., attention, visuospatial, memory, language, executive function, emotion processing) in FTD syndromes.

Methods

Systematic Literature Search

Neuroimaging studies (structural MRI, positron emission tomography (PET), functional MRI (fMRI) and single-photon emission computed tomography (SPECT)) were identified using PubMed, EMBASE and Web of Science databases. The systematic literature search identified all relevant studies published until 31st January 2019 according to the recommendations for neuroimaging meta-analysis (Muller et al., 2018). Given the potential under-reporting of cerebellar findings, the search did not include the term 'cerebellum' but referred to FTD and imaging, to ensure all potential eligible studies were identified (Table 1). *Reference Lists of Eligible Articles and Relevant Review Articles Were Also Screened for Potential Additional Publications. The studies' corresponding authors were contacted for missing/uncertain information if the necessary data were not available or unclear in the published material. Studies were included based on the following criteria:

i. Written in English;
ii. Studies compared FTD with a healthy control group;
iii. Correlations between neuropsychological or clinical performance and cerebellar changes in the FTD patient group or FTD and healthy controls combined were included;
iv. FTD patients (i.e., bvFTD, FTD-ALS, SD, PNFA, unclassified FTD) were diagnosed according to internationally recognised clinical diagnostic criteria (Rascovsky et al., 2011; Neary et al., 1998; Rascovsky et al., 2007; Strong et al., 2009; Gorno-Tempini et al., 2011);

Table 1 Search terms for the systematic literature search

Database	Search terms	Limits
PubMed	All Fields ("frontotemporal lobar degeneration" OR "Pick disease of the brain" OR "frontotemporal dementia" OR "semantic dementia" OR "primary progressive nonfluent aphasia" OR "tau proteins" OR "TDP-43 proteinopathies" OR "C9orf72" OR "GRN" OR "progranulin" OR "MAPT" OR "microtubule-associated protein tau") AND ("imaging" OR "neuroimaging" OR "functional imaging" OR "magnetic resonance imaging" OR "positron-emission tomography" OR "single photon emission computed tomography")	Clinical Trial & Case Report & Journal Article
EMBASE	All Fields ("frontotemporal lobar degeneration" OR "frontotemporal dementia" OR "Pick presenile dementia" OR "semantic dementia" OR "progressive nonfluent aphasia" OR "tau protein" OR "TDP 43 proteinopathy" OR "C9orf72" OR "GRN" OR "progranulin" OR "MAPT" OR "microtubule-associated protein tau") AND ("imaging" OR "neuroimaging" OR "functional imaging" OR "magnetic resonance imaging" OR "positron emission tomography" OR "single photon emission computed tomography")	–
Web of Science	Same as PUBMED	Article

v. The results reported cerebellar involvement (i.e., changes in grey matter intensity, functional activation or metabolism) and provided coordinates for the location of cerebellar involvement in standard brain spaces (either Montreal Neurological Institute (MNI) or Talairach & Tournoux).

The literature search identified 17,819 publications. The studies were narrowed down in a four-step assessment including literature identification, literature screening, eligibility assessment, and study inclusion (Fig. 1; See Supplementary Fig. 1 for details). After initial exclusion (i.e., duplicates, papers published prior to the Neary FTD diagnostic criteria (Neary et al., 1998) and non-English publications) 12,037 publications remained. Based on article categories, 3875 publications of non-original studies were excluded (i.e., reviews, practice guidelines, editorials/commentaries etc.). Individual single case studies were also excluded. Finally, assessment of the methods and results was conducted on the remaining 8162 publications. Ninety-eight studies reported significant clusters in the cerebellum. Of these, 45 could not be included because the necessary information was not available, even after contacting the study authors. In some instances, the corresponding authors could no longer provide the requested data ($n = 9$). In many instances, however, the authors failed to reply ($n = 36$). Two studies used the same participant cohort (Tan et al., 2015; Tan et al., 2014). Only the study with the larger sample size was included in the ALE analysis.

The primary outcome was the peak coordinates of cerebellar changes in any modalities in the FTD group. In addition to anatomical data, demographic characteristics (i.e., age, disease duration, clinical diagnosis) and imaging processing information (i.e., analysis type (whole-brain or region of interest analysis)) were obtained from the publications or from the authors directly (see Supplementary Table 1).

Anatomical/Activation Likelihood Estimation Meta-Analysis

Nine separate ALE analyses were conducted. First, an overall ALE analysis with all available studies ($n = 53$; 156 foci) was carried out. Six ALE analyses were then performed on specific sub-datasets. Three analyses focused on methods of brain imaging acquisition: voxel-based morphometry (VBM) studies (structural MRI studies) ($n = 42$; 135 foci), FDG-PET studies ($n = 6$; 11 foci), fMRI studies (n = 4; 9 foci); two analyses focused on clinical groups: bvFTD studies ($n = 41$; 91 foci),

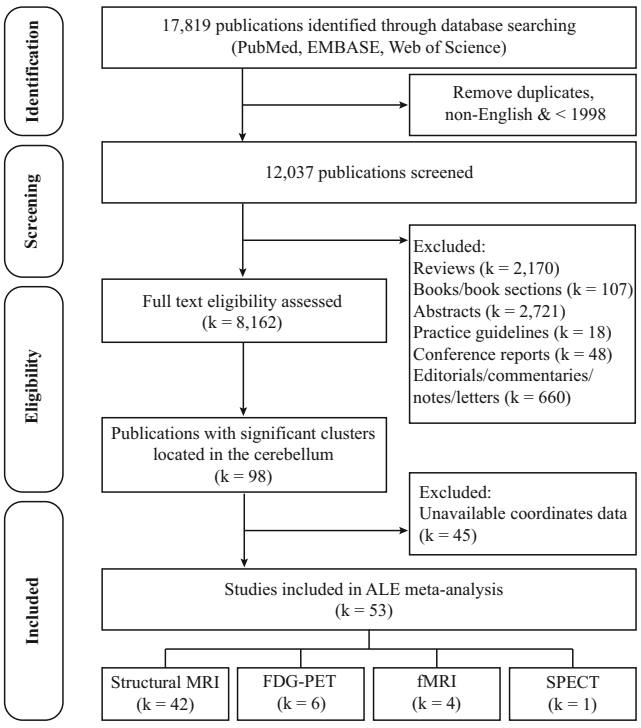

Fig. 1 Flowchart of literature search and selection strategy. ALE = Anatomical/Activation Likelihood Estimation; FDG-PET = fludeoxyglucose-positron emission tomography; SPECT = single-photon emission computed tomography

SD studies ($n = 9$; 17 foci); and one analysis focused on task-related correlation analyses regardless of methods of acquisition ($n = 20$; 43 foci). Finally, separate ALE analyses were also conducted on VBM studies that included direct contrasts between FTD and healthy controls ($n = 29$; 99 foci) and on those that included task-related correlation analyses ($n = 17$; 36 foci). Separate ALE analyses on the SPECT (n = 1; 1 focus) and PNFA ($n = 3$; 8 foci) studies were not performed due to the limited number of studies available.

Three region-of-interest (ROI) VBM studies were included in this investigation. As such, we also repeated the ALE analyses after including only the whole-brain studies in order to determine the contribution of the ROI studies to the results.

ALE analysis was performed in MNI space using GingerALE software V2.3.6 (http://www.brainmap.org/ale/) (Turkeltaub et al., 2012; Eickhoff et al., 2012; Eickhoff et al., 2009). Prior to the main analysis, any coordinates (foci) that were reported in Talairach & Tournoux space were converted to MNI standard space using the GingerALE convert tool icbm2tal transformation (Lancaster et al., 2007). Then, MNI coordinates and sample size for each study were entered into GingerALE via text files. The full-width half-maximum value of 12 mm was used and the Gaussian function was calculated for each focus derived from the subject sample size, with larger sample sizes allocated tighter and taller Gaussian functions. Gaussian probabilities of each set of foci were given to each voxel yielding a modelled activation map. The activation maps were subsequently combined into one ALE-map by taking the union of the probabilities. ALE values represent the differential likelihood of brain changes in grey matter or brain activations at each voxel. ALE values were then compared to a non-linear histogram integration based on the frequency of distinct modelled activation values (Eickhoff et al., 2012). An algorithm was used to model the probability distribution of each focus using an estimation of both between-subject and between-experiment variability. This algorithm limited the meta-analysis to an anatomically constrained space specified by a grey matter mask and calculated the above-chance clustering between experiments (i.e., random-effect analysis) rather than between foci (i.e., fixed-effects analysis) (Eickhoff et al., 2012, Eickhoff et al., 2009). Then, a cluster analysis was performed to localize peak voxels of significant clusters.

For all analyses, as recommended by the GingerALE manual, the cluster-forming threshold was set at $p < .001$ uncorrected, and the cluster-level inference threshold was set at .05 (Eickhoff et al., 2012; Gellersen et al., 2017). Imaging results were displayed on surface-based cerebellar flatmaps using the SUIT toolbox (Diedrichsen & Zotow, 2015) from MATLAB (https://au.mathworks.com/products/matlab.html) and SPM12 (http://www.fil.ion.ucl.ac.uk/spm).

Results

Systematic Search

Fifty-three publications met criteria for inclusion in the ALE meta-analysis: 42 structural MRI studies, 6 fludeoxyglucose-PET (FDG-PET) studies, 4 functional MRI (fMRI) studies, and 1 single-photon emission computed tomography (SPECT) study (Fig. 1) (see Online Resource for the full list of publications). These encompassed a total of 77 analysis results which included 1261 FTD patients (bvFTD = 918; SD = 184; PNFA = 94, FTD-ALS = 36; unclassified FTD = 29), and 974 healthy controls. Study information including sample size, demographic characteristics of participants (i.e., age, disease duration if applicable), main instrument, assessments of cognitive function/clinical performance, analysis type (i.e., whole-brain or ROI analysis), and number of foci are reported in the Online Resource (Supplementary Table 1).

ALE Meta-Analysis

(i) All Available Studies (156 Foci)

Widespread cerebellar changes were identified in the cerebellar hemispheres bilaterally and parts of the vermis. Peak clusters were found in the bilateral posterior, and the right anterior cerebellum (bilateral Crus and lobules VIIb; right lobules I-V, and VIIIb; left lobules VI and X). The peak coordinate of the most significant cluster was centered in the right Crus (Fig. 2, Table 2).

(ii) VBM Studies (135 Foci)

Regions identified from the analysis of VBM studies were comparable with the analysis of all available studies. Cerebellar grey matter changes were primarily found in the posterior regions (bilateral Crus, lobules VI, VIIb and X; right lobule VIIIb) with additional involvement of the right anterior cerebellum (lobules I-V). The peak coordinate of the most significant cluster was located in the left Crus I (Fig. 2, Table 3).

(iii) FDG-PET Studies (11 Foci)

No convergent cerebellar subregions were identified across FDG-PET studies from this ALE analysis.

(iv) Functional MRI Studies (9 Foci)

ALE analyses in fMRI studies revealed one focal cluster in the left lobule VI and Crus I with the peak voxels located in the left lobule VI (Fig. 2, Table 4).

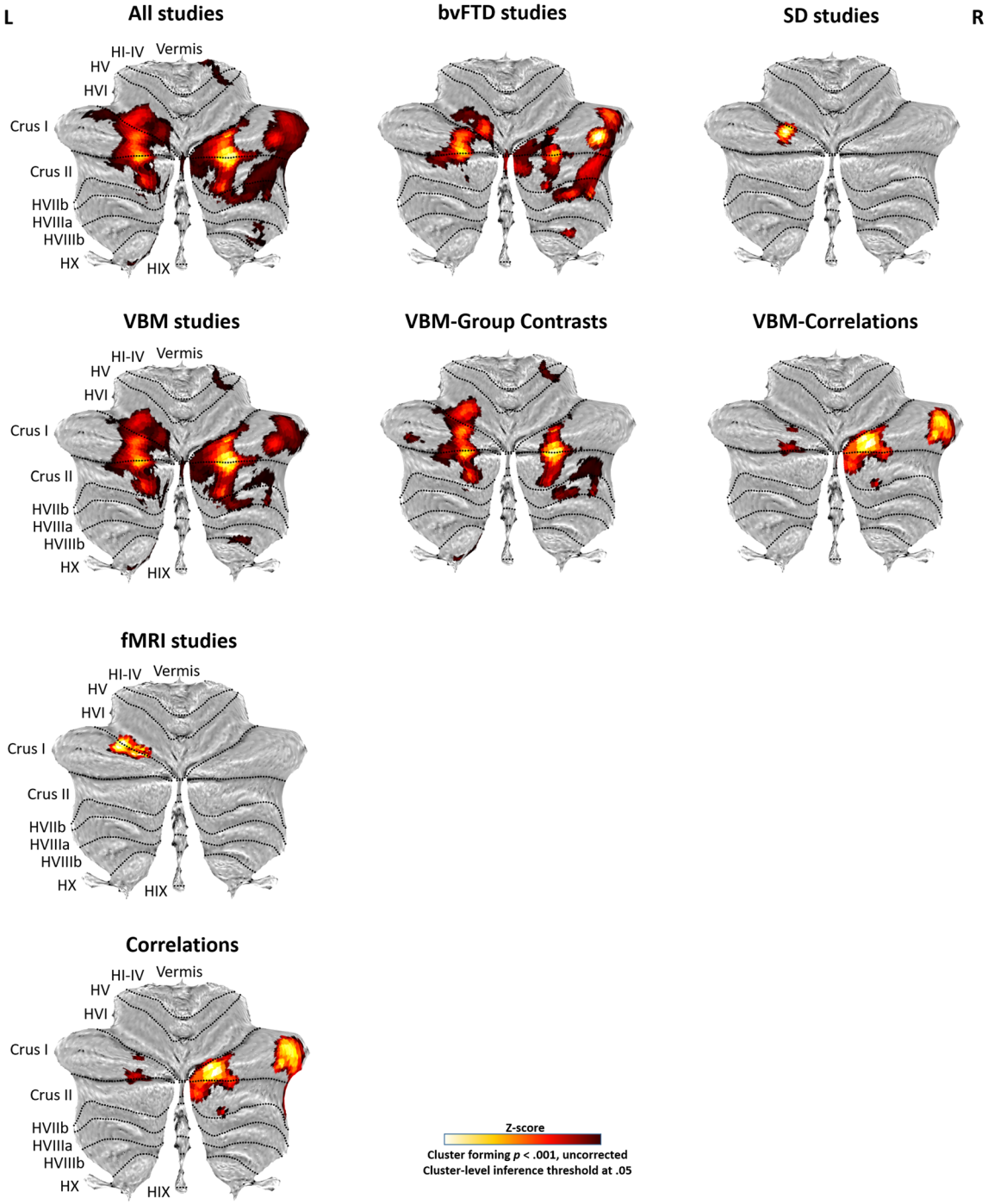

Fig. 2 Cerebellar involvement in FTD (combining both whole-brain and region-of-interest studies). Cerebellar flatmaps of significant clusters are colour coded (cluster forming threshold $p < .001$ uncorrected, cluster-level inference threshold at 0.05). VBM = voxel-based morphometry; fMRI = functional magnetic resonance imaging; bvFTD = behavioural-variant frontotemporal dementia; SD = semantic dementia

(v) bvFTD Studies (91 Foci)

Cerebellar changes spanned the bilateral cerebellar hemispheres (bilateral Crus; left lobule VI; right lobules VIIb and VIIIb) and parts of the vermis. Only the posterior cerebellum was involved with the peak coordinates of the most significant cluster located in the right lobule VIIb and Crus II (Fig. 2, Table 5).

(vi) SD Studies (17 Foci)

The pattern of cerebellar changes in SD was quite distinct from that found in bvFTD. Marked hemispheric asymmetry with left predominance was observed. Only one significant cluster was found in the left lobule VI and Crus I with the peak coordinates located in the left Crus I (Fig. 2, Table 6).

Table 2 Peak ALE coordinates of all available studies

Location	Local extrema (x, y, z)			Volume (mm^3)	Extrema ALE value
From (0,-88,-62) to (58,-38,-20) centered at (32.6,-65.9,-39.3)					
Right Crus I, Crus II	20	−72	−34	11,120	0.046042826
Right Crus I	52	−66	−36		0.024166528
Right Crus I	48	−52	−34		0.020772701
Right Crus I	28	−62	−38		0.020680897
Right lobule VIIb	44	−56	−58		0.019933205
Right lobule VIIb	36	−68	−58		0.017921649
Right Crus II, lobule VIIb	38	−64	−48		0.014643208
Right Crus II	42	−60	−46		0.014459331
Right Crus II	6	−78	−30		0.013974056
Right Crus I	52	−66	−24		0.013835791
From (−40,-80,-42) to (−6,-50,-18) centered at (−22.1,-67.4,-31.8)					
Left Crus II, Crus I	−20	−72	−36	8768	0.035806876
Left Crus I, lobule VI	−24	−72	−28		0.030611152
Left lobule VI	−26	−62	−28		0.02420393
Left lobule VI	−22	−60	−28		0.024028912
Left lobule VI	−10	−66	−26		0.01702343
Left Crus I, lobule VI	−34	−56	−32		0.013376212
From (8,-52,−48) to (28,-32,-20) centered at (21.1,-42,-33.4)					
Right lobule VIIIa	22	-48	−44	1568	0.016044289
Right lobules I-IV	10	−36	−22		0.014632501
Right lobules V, I-IV	22	−36	−24		0.012550315
Right lobule V	24	−40	−30		0.012421303

Clusters are thresholded at cluster forming threshold $p < .001$ uncorrected, cluster-level inference threshold at 0.05. Coordinates are given in MNI152 space. "Local extrema" refers to the coordinates of the location of the maximum ALE value; "Extrema ALE value" refers to the maximum ALE value observed in the cluster. Locations are reported based on the Cerebellar Atlas in MNI152 space

ALE Anatomical/Activation Likelihood Estimation

(vii) Task-Related Correlation Studies (43 Foci)

Cerebellar changes were associated with several cognitive domains including attention (Chen et al., 2018), memory (working memory, episodic memory, emotional memory) (Irish et al., 2013; Kumfor, Irish, Hodges, & Piguet, 2013b; Seelaar et al., 2011; Henley et al., 2014; Bertoux et al., 2018; Chen et al., 2018), visuospatial function (Chen et al., 2018), language (Grossman et al., 2004; Chen et al., 2018), executive function (Kloeters, Bertoux, O'Callaghan, Hodges, & Hornberger, 2013; Strenziok et al., 2011; Laisney et al., 2009), emotion processing (Kumfor, Irish, Hodges, & Piguet, 2013a; Hutchings, Bruggemann, Hodges, Piguet, & Kumfor, 2018; Kumfor et al., 2018a), face recognition (De Winter et al., 2016; Hutchings et al., 2018), and social cognition (Kumfor et al., 2017; Synn et al., 2017; Wong et al., 2017). In addition, cerebellar changes were also associated with psychosis (Devenney et al., 2017) and behavioural changes such as abnormal eating habit (Ahmed et al., 2016), anosognosia (Amanzio et al., 2016), and loss of empathy (Dermody et al., 2016). Regions identified from ALE analysis of these studies spanned the bilateral Crus, with the peak voxels located in the right Crus I (Fig. 2, Table 7).

(viii) VBM Studies – Direct Contrasts between FTD and Healthy Controls (99 Foci)

The pattern of cerebellar grey matter changes from this subgroup analysis was comparable with the analysis of all VBM studies. Cerebellar changes spanned bilateral Crus, lobules VI, VIIb, right lobules I-V, and left lobule X. The peak coordinate of the most significant cluster was located in left Crus I (Fig. 2, Table 8).

(ix) VBM Studies – Task-Related Correlation Studies (36 Foci)

In keeping with sub-dataset (vii), ALE analyses of only VBM studies found correlations between cerebellar grey matter changes and FTD symptomatology. Regions identified from this analysis were remarkably consistent with findings from

Table 3 Peak ALE coordinates of voxel-based morphometry studies only

Location	Local extrema (x, y, z)			Volume (mm^3)	Extrema ALE value
From (−40,-80,-42) to (−8,-50,-24) centered at (−21,-68.4,-32.8)					
Left Crus II, Crus I	−20	−72	−36	7000	0.03579308
Left Crus I, lobule VI	−24	−72	−28		0.029645205
Left lobule VI	−20	−60	−28		0.022865655
Left lobule VI	−12	−66	−26		0.01666895
From (0,-88,-60) to (50,-52,-26) centered at (23,-70,-37.5)					
Right Crus I, Crus II	20	−72	−34	6192	0.04604283
Right Crus I	28	−62	−38		0.020678893
Right lobule VIIb	44	−56	−58		0.019774511
Right Crus II	6	−78	−30		0.013970773
Right Crus II	36	−66	−46		0.011775645
Right Crus II	48	−66	−48		0.011291356
Right Crus II	42	−60	−46		0.011038466
Right Crus II	18	−84	−46		0.010431599
From (44,-72,-42) to (58,-40,-28) centered at (50.5,-59.9,-34.8)					
Right Crus I	52	−66	−36	2296	0.024146823
Right Crus I	48	−52	−34		0.019825626
From (26,-78,-62) to (40,-64,-54) centered at (33.4,-70.6,-58.2)					
Right lobule VIIb	36	−68	−60	968	0.016979381
From (18,-50,-44) to (28,-32,-20) centered at (24.7,-41.4,-31.7)					
Right Crus I	26	−46	−38	912	0.014701418
Right lobules V, I-IV	24	−38	−24		0.012517499
Right lobule V	24	−40	−30		0.012412731
From (10,-58,-68) to (20,-44,-58) centered at (15.4,-50.4,-62.8)					
Right lobule VIIIb	14	−52	−66	848	0.017011557
From (−36,-86,-54) to (−22,-78,-44) centered at (−29.1,-81.5,-49.3)					
Left Crus II	−28	−82	−50	832	0.027974322

Clusters are thresholded at cluster forming threshold $p < .001$ uncorrected, cluster-level inference threshold at 0.05. Coordinates are given in MNI152 space. "Local extrema" refers to the coordinates of the location of the maximum ALE value; "Extrema ALE value" refers to the maximum ALE value observed in the cluster. Locations are reported based on the Cerebellar Atlas in MNI152 space

ALE Anatomical/Activation Likelihood Estimation

sub-dataset (vii), as only two studies were excluded (1 SPECT study, 1 PET study). In brief, significant clusters were found bilaterally in Crus I and Crus II with the peak voxels located in the right Crus I (Fig. 2, Table 9).

(x) Whole-Brain Meta-Analysis

Separate analyses of only whole-brain studies were conducted to assess the potential impact of the ROI approach on the meta-analysis results (Supplementary Fig. 2). ROI-based studies ($n = 3$) were excluded from these analyses. From these analyses, significant clusters were identified which were broadly consistent with those combining whole-brain and ROI-based studies. Notable differences from the combined dataset analyses were the absence of right cerebellar cluster in task-related correlation studies. In addition, no significant clusters were observed in SD studies.

Discussion

This ALE meta-analysis provides the first quantification of the location and extent of cerebellar changes across FTD syndromes using different imaging modalities. Consistent clusters

Table 4 Peak ALE coordinates of functional MRI studies only

Location	Local extrema (x, y, z)			Volume (mm^3)	Extrema ALE value
From (−36,-82,-32) to (−20,-60,-14) centered at (−29.3,-70.6,-22.4)					
Left Crus I	−26	−76	−18	1712	0.011013831
Left Crus I, lobule VI	−32	−66	−26		0.010917673

Clusters are thresholded at cluster forming threshold $p < .001$ uncorrected, cluster-level inference threshold at 0.05. Coordinates are given in MNI152 space. "Local extrema" refers to the coordinates of the location of the maximum ALE value; "Extrema ALE value" refers to the maximum ALE value observed in the cluster. Locations are reported based on the Cerebellar Atlas in MNI152 space

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Table 5 Peak ALE coordinates of behavioural-variant frontotemporal dementia studies only

Location	Local extrema (x, y, z)			Volume (mm^3)	Extrema ALE value
From (26,-78,-62) to (52,-44,-28) centered at (41.1,-61.7,-50.5)					
Right lobule VIIb	44	−56	−58	3936	0.019933078
Right lobule VIIb	36	−68	−58		0.01787766
Right Crus I	48	−52	−32		0.016442165
Right Crus I, Crus II	44	−58	−44		0.014148136
From (−2,-88,-46) to (30,-62,-26) centered at (17.3,-74.3,-35.2)					
Right Crus II	20	−70	−36	2640	0.01850615
Right Crus II	6	−78	−30		0.013881652
Right Crus II	28	−64	−42		0.012281639
Right Crus II	26	−80	−40		0.01189486
Right Crus II	26	−84	−38		0.011452802
Right Crus I, lobule VI	12	−72	−28		0.011392617
From (−42,-78,-44) to (−16,-64,-24) centered at (−24.1,-71.9,-34.1)					
Left Crus I, Crus II	−22	−72	−36	2256	0.019778134
Left Crus II, Crus I	−32	−68	−40		0.012550566
Left Crus I, Crus II	−38	−68	−42		0.010715586
From (48,-72,-42) to (58,-60,-30) centered at (52.9,−66.2,−36.1)					
Right Crus I	52	-66	-36	1144	0.023216639
From (−28,-70,-30) to (−8,-58,-22) centered at (−14.8,-64.5,-26.3)					
Left lobule VI	−12	−66	−26	800	0.016135342
Left lobule VI	−20	−60	−26		0.011170035
Left lobule VI	−26	−62	−26		0.010596783
From (10,-62,-68) to (20,-44,-58) centered at (16.2,-50,-63.3)					
Right lobule VIIIb	14	−52	−66	792	0.01642279
Right lobule VIIIb	18	−48	−62		0.015001545

Clusters are thresholded at cluster forming threshold $p < .001$ uncorrected, cluster-level inference threshold at 0.05. Coordinates are given in MNI152 space. "Local extrema" refers to the coordinates of the location of the maximum ALE value; "Extrema ALE value" refers to the maximum ALE value observed in the cluster. Locations are reported based on the Cerebellar Atlas in MNI152 space

ALE Anatomical/Activation Likelihood Estimation

of cerebellar atrophy, activation and metabolic changes were identified. Our results revealed distinct patterns of cerebellar changes in the two most common FTD subtypes (bvFTD vs. SD) reflecting syndrome-specific cerebellar involvement. In addition, we also uncovered associations between these cerebellar abnormalities and changes in behaviour and cognition. Although previously found in other neurological disorders, such associations have not been systematically reported in FTD. We discuss these findings in detail below.

Our meta-analysis revealed widespread cerebellar changes in FTD bilaterally involving most lobules with additional changes in parts of the vermis. Importantly, regions identified in the analysis restricted to the VBM studies were comparable with the all-studies analysis. This finding indicates that the results of all-studies analysis were primarily driven by the VBM studies, which is not unexpected given the limited number of studies using FDG-PET, fMRI and SPECT. The small number of studies prevented separate analyses for the functional imaging techniques using SPECT ($n = 1$). Surprisingly, despite relatively limited number of studies available, focal clusters were found in fMRI studies ($n = 4$, 9 foci) primarily located in the left lobule VI and Crus I, suggesting convergent cerebellar findings across fMRI studies. This difference in the topography of changes between VBM and fMRI is likely explained by the different methodologies (structural vs. functional imaging) and the limited number of studies of fMRI studies. In bvFTD, cerebellar changes were pronounced, spanning bilateral posterior and the right inferior cerebellum. Focal changes were also present in SD localized in the left lobule VI and Crus I. The patterns of cerebellar changes in bvFTD and SD are consistent with a recent ROI-based structural neuroimaging study (Chen et al., 2018). These findings demonstrate the heterogeneous patterns of cerebellar changes across FTD subtypes that may be specific to the underlying pathological processes and reflect the specific clinical profiles manifested by different phenotypes. Cerebellar atrophy was also observed in PNFA, localized bilaterally in the lobules VI, Crus and VIIb (Sturm et al., 2015; Guo et al., 2016; Chen et al., 2018). Due to

Table 6 Peak ALE coordinates of semantic dementia studies only

Location	Local extrema (x, y, z)			Volume (mm^3)	Extrema ALE value
From (−30,-76,-32) to (−22,-68,-24) centered at (−25.6,-72.1,-27.5)					
Left Crus I	−26	−72	−28	632	0.01562541

Clusters are thresholded at cluster forming threshold $p < .001$ uncorrected, cluster-level inference threshold at 0.05. Coordinates are given in MNI152 space. "Local extrema" refers to the coordinates of the location of the maximum ALE value; "Extrema ALE value" refers to the maximum ALE value observed in the cluster. Locations are reported based on the Cerebellar Atlas in MNI152 space

ALE Anatomical/Activation Likelihood Estimation

the small number of available studies ($n = 3$, 8 foci), however, separate ALE analyses could not be conducted for this FTD subtype.

Left-lateralised asymmetry was observed in SD. Based on the cross-hemispheric cerebro-cerebellar connections (Schmahmann & Pandya, 1997b), this asymmetry likely reflects right cortical changes in SD. Inspection of the publications involving SD cases, however, indicated that mixed samples containing both right- and left- predominant SD were included in these studies. Indeed, left and right SD represent distinct clinical entities at baseline but evolve into a similar clinical profile with disease progression despite showing divergent patterns of cortical thinning progression (Kumfor et al., 2016). Clearly, the patterns of cerebellar changes in these phenotypes warrant further investigations.

The *C9orf72* gene expansion (Renton et al., 2011; DeJesus-Hernandez et al., 2011) is now considered the most common genetic cause of FTD and accounts for a high proportion of familial FTD cases (Boeve et al., 2012). Structural and functional cerebellar abnormalities in individuals harbouring the *C9orf72* gene expansion have been reported (Whitwell et al., 2012; Savica et al., 2012; Boeve et al., 2012; Suarez-Calvet et al., 2015). Importantly, however, the existing literature has also identified cerebellar changes in sporadic cases that are even greater than in *C9orf72* expansion carriers, even in the presence of a shorter disease duration, (Lee et al., 2014; Devenney et al., 2014; Irish et al., 2013). Here, we were unable to compare *C9orf72* carriers vs. sporadic cases because this information is not routinely reported in clinical studies. How *C9orf72* interacts with cerebellar changes in FTD remains a challenge for future research.

Resting-state functional MRI studies have identified abnormal network connectivity between the cerebellum and cortical regions in FTD (Farb et al., 2013; Meijboom et al., 2016). These studies suggested that the observed cerebellar changes may be mediated by structural cerebral damage interfering with cortico-cerebellar loops, and reflecting functional reorganisation in FTD. The findings from the VBM studies, however, indicate the presence of local structural changes as well. How disruption of the cerebellar networks impacts cognitive function, independently from those arising from the cerebral functional network disturbances in FTD, still needs to be established.

Similar cerebellar regions implicated in cognition and behavioural changes arising from the analyses on task-related correlation studies and VBM task-related correlation studies were identified. Significant clusters were found in the bilateral Crus. These regions have been considered as the "cognitive cerebellum" (Van Overwalle et al., 2015; Stoodley & Schmahmann, 2009; Petacchi, Laird, Fox, & Bower, 2005)

Table 7 Peak ALE coordinates of correlation analyses

Location	Local extrema (x, y, z)			Volume (mm^3)	Extrema ALE value
From (44,-68,-46) to (58,-42,-30) centered at (50.3,-54.8,-36.5)					
Right Crus I	54	−64	−38	2064	0.01621537
Right Crus I	48	−50	−36		0.015940774
From (4,-84,-42) to (26,-66,-26) centered at (14.3,-73.6,-32.1)					
Right Crus I, lobule VI	14	−72	−30	1824	0.01748178
Right Crus II	8	−80	−32		0.012947307
From (−30,-74,-38) to (−14,-66,-32) centered at (−21.4,-70.4,-34.7)					
Left Crus I, lobule VI	−20	−68	−34	512	0.01087771
Left Crus I, Crus II	−28	−72	−36		0.010341066

Clusters are thresholded at cluster forming threshold $p < .001$ uncorrected, cluster-level inference threshold at 0.05. Coordinates are given in MNI152 space. "Local extrema" refers to the coordinates of the location of the maximum ALE value; "Extrema ALE value" refers to the maximum ALE value observed in the cluster. Locations are reported based on the Cerebellar Atlas in MNI152 space

ALE Anatomical/Activation Likelihood Estimation

Table 8 Peak ALE coordinates of VBM studies of direct contrasts between patients with frontotemporal dementia and healthy controls

Location	Local extrema (x, y, z)			Volume (mm^3)	Extrema ALE value
From (−40,-80,-42) to (−8,-50,-24) centered at (−22.2,-67.1,-32.9)					
Left Crus II	−20	−72	−38	5232	0.029366927
Left Crus I, lobule VI	−24	−72	−28		0.025781462
Left lobule VI	−20	−60	−28		0.022449885
Left Crus I, Crus II	−30	−66	−38		0.014550786
Left Crus II, Crus I	−38	−68	−42		0.010431402
From (14,-86,-48) to (32,-58,-28) centered at (22.3,-71.8,-36.9)					
Right Crus I, Crus II	20	−72	−34	2992	0.035623137
Right Crus II	26	−64	−42		0.015227625
Right Crus II	18	−84	−46		0.010273452
From (34,-68,-62) to (50,-52,-44) centered at (43.8,-59.3,-52.2)					
Right lobule VIIb	44	−56	−58	1208	0.019774511
Right Crus II	36	−66	−46		0.011566442
Right Crus II	48	−66	−48		0.011196542
Right Crus II	42	−60	−46		0.011022932
From (26,-78,-62) to (40,-64,-52) centered at (33.3,-70.6,-58)					
Right lobule VIIb	36	−68	−60	1136	0.016979381
From (20,-50,-40) to (30,-32,-20) centered at (24.6,-40.8,-30.4)					
Right lobule VI	26	−44	−36	912	0.013966357
Right lobules V, I-IV	24	−38	−24		0.01249635
Right lobule V	24	−40	−30		0.012409609
From (−56,-76,-42) to (−48,-56,-34) centered at (−52.8,-64.1,-38.4)					
Left Crus I	−54	−62	−38	712	0.017438238

Clusters are thresholded at cluster forming threshold $p < .001$ uncorrected, cluster-level inference threshold at 0.05. Coordinates are given in MNI152 space. "Local extrema" refers to the coordinates of the location of the maximum ALE value; "Extrema ALE value" refers to the maximum ALE value observed in the cluster. Locations are reported based on the Cerebellar Atlas in MNI152 space

ALE Anatomical/Activation Likelihood Estimation

and their contribution to cognitive processes have been previously demonstrated in FTD (Chen et al., 2018). The inclusion of studies examining multiple cognitive domains using different experimental paradigms in the present ALE analysis indicates a fundamental role of the bilateral Crus in cognition, irrespective of the processes.

Associations between cognition and both anterior and posterior cerebellar regions have been reported in healthy participants (Stoodley & Schmahmann, 2009; Van Overwalle, Baetens, Marien, & Vandekerckhove, 2014). While typically considered as a motor centre, in FTD the anterior cerebellum has been associated with eating behaviours (Ahmed et al., 2016), instrumental activities of daily living (Amanzio et al., 2016), decision making (Strenziok et al., 2011), and theory of mind (Synn et al., 2018). Task-based fMRI studies in mild cognitive impairment and Alzheimer's disease have also found reduced activation in the anterior cerebellar regions as disease progresses suggesting a posterior-to-anterior involvement with disease progression (for review see Jacobs et al., 2017). Cerebellar changes are probably related to disease severity in FTD (Seeley et al., 2008), but whether the anterior cerebellar involvement reflects increasing disease severity is unknown. No studies to date have examined changes in cerebellar integrity and their relations to cognition with disease progression in FTD. Longitudinal studies are needed to understand the timeframe for the progressive cerebellar pathological expansion in FTD syndromes and how such changes relate to cognitive dysfunction.

In addition, separate analyses of whole-brain studies were performed to assess the specific effect of ROI methodology on resultant ALE findings. In brief, the patterns of results were mostly similar to those arising from the analyses combining all available studies (i.e., where both whole-brain and ROI-based studies were included), but notable differences were found in the analyses of task-related correlation studies. Significant clusters were found bilaterally spanning cerebellar hemispheres when all available studies were included. In contrast, significant clusters were found only in the left hemisphere when only whole-brain studies were included. Similarly, smaller clusters in the right cerebellar hemisphere were also observed in the analysis of VBM task-related correlation studies when only whole-brain studies were included.

Table 9 Peak ALE coordinates of VBM studies of task-related correlation analyses

Location	Local extrema (x, y, z)			Volume (mm^3)	Extrema ALE value
From (4,-84,-42) to (26,-66,-26) centered at (14.3,-73.6,-32.1)					
Right Crus I, lobule VI	14	−72	−30	1842	0.01748178
Right Crus II	8	−80	−32		0.012947002
From (44,-68,-42) to (58,-46,-30) centered at (50.9,-57.3,-36.2)					
Right Crus I	54	−64	−38	1560	0.016215298
Right Crus I	48	−52	−34		0.015313339
From (−30,-74,-38) to (−14,-66,-32) centered at (−21.4,-70.4,-34.7)					
Left Crus I, lobule VI	−20	−68	−34	512	0.010877576
Left Crus I, Crus II	−28	−72	−36		0.010341067

Clusters are thresholded at cluster forming threshold $p < .001$ uncorrected, cluster-level inference threshold at 0.05. Coordinates are given in MNI152 space. "Local extrema" refers to the coordinates of the location of the maximum ALE value; "Extrema ALE value" refers to the maximum ALE value observed in the cluster. Locations are reported based on the Cerebellar Atlas in MNI152 space

ALE Anatomical/Activation Likelihood Estimation

These findings, however, should be interpreted with caution as the number of studies is indeed reduced in the whole-brain analysis leading to reduced power to detect significant voxels. Future ALE analyses may need to consider including both ROI and whole-brain studies, and conduct separate analyses.

Our results likely underestimate the extent and severity of cerebellar changes in FTD. For instance, the cerebellum is still considered the brain region least affected in FTD (Dukart et al., 2010; Dukart et al., 2011) and is often used as a reference region for PET and SPECT measurements. In addition, the cerebellum is often not included in standard scanning or analysis protocols (e.g., Vemuri et al., 2011; Jiskoot et al., 2019). Studies also occasionally report large clusters that encompass both cerebral and cerebellar regions where the peak change is located in the cerebral cortex and not in the cerebellum (e.g., Bertoux et al., 2018). These studies were excluded, increasing the risk of under-estimation of cerebellar changes in FTD. Importantly, the relations between cerebellar changes and clinical features in FTD have received little attention to date despite being present in many studies (e.g., Ahmed et al., 2016; Kumfor, Zhen, Hodges, Piguet, & Irish, 2018b), possibly because they have been previously thought to be artefacts. Notably, our literature search uncovered cerebellar changes in the figures of numerous studies. These changes were, however, not routinely reported in tables or discussed, demonstrating how the involvement of the cerebellum has been downplayed (e.g., Rosen et al., 2002; Whitwell et al., 2010). Expanding the search by removing the 'cerebellum/cerebellar' limit search terms and visually inspecting all relevant papers for incidental cerebellar findings identified approximately three times as many publications. Unfortunately, coordinates of incidental, but not discussed, cerebellar changes were unavailable for 45 studies, either because the original data were no longer available or because the authors did not reply to our requests. These limitations suggest that cerebellar changes in FTD may be more common than previously reported and more extensive than our results indicate. The results of this meta-analysis provide a strong argument that the cerebellum should be routinely included in neuroimaging data acquisition protocols and systematically reported in future studies investigating FTD in order to gain a full picture of the role of the cerebellum.

Although our meta-analysis findings have provided novel insights into the cerebellar involvement in FTD syndromes, several caveats should be considered. First, ALE analysis has the limitation of representing activation/anatomical peaks weighted only by sample size. As such, other measurements such as the cluster size (i.e., the spread of changes) and the statistical thresholds set for group-level comparisons cannot be adequately examined. This limitation, however, is addressed by the ALE algorithm by treating each focus as the centre of probability distributions and using an algorithm that takes into account both between-subject and between-experiment variability (Eickhoff et al., 2009). This approach achieves a good balance between potential Type I and Type II errors (Bossier et al., 2017). In this way, it is able to deal with the inter-study differences in scanning parameters and imaging analysis methods (Bossier et al., 2017; Turkeltaub, Eden, Jones, & Zeffiro, 2002). It should be noted that it is not technically possible to include studies without any peak coordinates in the ALE algorithm (Acar, Seurinck, Eickhoff, & Moerkerke, 2018). Thus, we restricted our investigations to studies reporting cerebellum findings only, in line with previous ALE meta-analyses (Gellersen et al., 2017; Bernard & Mittal, 2015; Stoodley & Schmahmann, 2009). As such, this meta-analysis did not include negative findings. These negative findings may have arisen for a variety of reasons such as limited statistical power or group differences (e.g., disease duration). Future original research (including both structural and functional imaging studies) with larger sample sizes will be needed to confirm the robustness of our findings. A second

limitation is that we included studies with participants at different disease stages, which may have influenced results. Some studies included individuals at a relatively early stage of around 1 year after disease onset (e.g., Cistaro et al., 2014) while the longest disease duration was approximately 8 years (e.g., Lee et al., 2014). In some instances, because of technical difficulties or medical contraindications, imaging analyses were conducted in a subset of the study sample only, of which the demographics were not reported. In addition, our meta-analysis may be limited by the small number of available studies, although the number of studies included is acceptable in the context of a relatively rare disorder. Indeed, the number of studies in our meta-analysis is comparable to most prior cerebellar meta-analyses (Addis, Moloney, Tippett, & Hach, 2016; Riedel et al., 2015; Lange et al., 2015; Bernard & Mittal, 2015; Stoodley & Schmahmann, 2009; Petacchi et al., 2005; Gellersen et al., 2017) and larger than a recent meta-analysis in bvFTD (Gellersen et al., 2017). Third, only studies with explicit diagnostic criteria were included. According to current FTD diagnostic criteria, three levels of diagnostic certainty are proposed: possible (based on clinical evidence only), probable (with additional presence of change on neuroimaging), or definite (with pathological confirmation). Studies considered in this meta-analysis included participants meeting "probable" disease certainty; as such, in the absence of pathological confirmation, the diagnosis may be incorrect in some cases. This risk is mitigated by the fact that most studies were conducted by research groups with extensive experience in FTD investigations. Future neuroimaging studies in pathologically confirmed FTD are however needed. Finally, the present findings were derived from studies of cognition in FTD that employed many different tasks. Future studies using common experimental paradigms/cognitive tests will be helpful to determine the regions supporting specific cognitive processes that are affected in FTD.

In conclusion, the comprehensive literature search for ALE meta-analysis summarizes extensive evidence of structural and functional cerebellar changes in FTD syndromes and associations with behavioural and cognitive dysfunctions. Our findings confirm that the cerebellum is affected in FTD with patterns of cerebellar changes that are phenotype-specific and that relate to behavioural and cognitive changes. We hope our findings can draw attention to cerebellar contributions in future investigations in FTD. More research is needed to understand how these changes progress over time, how the cerebellum contributes to cognition, and how the cerebellum interacts with other brain regions in this disorder and related conditions.

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References

References Marked with an Asterisk Indicate Studies Included in the Meta-Analysis

Acar, F., Seurinck, R., Eickhoff, S. B., & Moerkerke, B. (2018). Assessing robustness against potential publication bias in activation likelihood estimation (ALE) meta-analyses for fMRI. *PLoS One, 13*(11), e0208177. https://doi.org/10.1371/journal.pone.0208177

Addis, D. R., Moloney, E. E., Tippett, L. J., & Hach, S. (2016). Characterizing cerebellar activity during autobiographical memory retrieval: ALE and functional connectivity investigations. *Neuropsychologia, 90*, 80–93. https://doi.org/10.1016/j.neuropsychologia.2016.05.025

*Ahmed, R. M., Irish, M., Henning, E., Dermody, N., Bartley, L., Kiernan, M. C., Piguet O., Farooqi S., Hodges J. R. (2016). Assessment of eating behavior disturbance and associated neural networks in frontotemporal dementia. JAMA Neurology, 73(3), 282–290, doi:https://doi.org/10.1001/jamaneurol.2015.4478.

*Amanzio, M., D'Agata, F., Palermo, S., Rubino, E., Zucca, M., Galati, A., et al. (2016). Neural correlates of reduced awareness in instrumental activities of daily living in frontotemporal dementia. Experimental Gerontology, 83, 158–164, doi:https://doi.org/10.1016/j.exger.2016.08.008.

Andreasen, N. C., & Pierson, R. (2008). The role of the cerebellum in schizophrenia. *Biological Psychiatry, 64*(2), 81–88. https://doi.org/10.1016/j.biopsych.2008.01.003

Belzil, V. V., Bauer, P. O., Prudencio, M., Gendron, T. F., Stetler, C. T., Yan, I. K., … Petrucelli, L. (2013). Reduced C9orf72 gene expression in c9FTD/ALS is caused by histone trimethylation, an epigenetic event detectable in blood. *Acta Neuropathologica, 126*(6), 895–905. https://doi.org/10.1007/s00401-013-1199-1

Bernard, J. A., & Mittal, V. A. (2015). Dysfunctional activation of the cerebellum in schizophrenia: A functional neuroimaging meta-analysis. *Clinical Psychological Science: A Journal of the Association for Psychological Science, 3*(4), 545–566. https://doi.org/10.1177/2167702614542463

*Bertoux, M. F.;Hobbs, M.;Ruiz-Tagle, A.;Delgado, C.;Miranda, M.;Ibanez, A.;Slachevsky, A.;Hornberger, M. (2018). Structural anatomical investigation of long-term memory deficit in behavioral frontotemporal dementia. Journal of Alzheimer's Disease, 62(4), 1887–1900, doi:https://doi.org/10.3233/jad-170771.

Boeve, B. F., Boylan, K. B., Graff-Radford, N. R., DeJesus-Hernandez, M., Knopman, D. S., Pedraza, O., … Rademakers, R. (2012). Characterization of frontotemporal dementia and/or amyotrophic lateral sclerosis associated with the GGGGCC repeat expansion in

C9ORF72. *Brain, 135*(Pt 3), 765–783. https://doi.org/10.1093/brain/aws004

Bossier, H., Seurinck, R., Kuhn, S., Banaschewski, T., Barker, G. J., Bokde, A. L. W., et al. (2017). The influence of study-level inference models and study set size on coordinate-based fMRI meta-analyses. *Frontiers in Neuroscience, 11*, 745. https://doi.org/10.3389/fnins.2017.00745

*Chen, Y., Kumfor, F., Landin-Romero, R., Irish, M., Hodges, J. R., & Piguet, O. (2018). Cerebellar atrophy and its contribution to cognition in frontotemporal dementias. Annals of Neurology, 84(1), 98–109, doi:https://doi.org/10.1002/ana.25271.

Cheng, D. T., Meintjes, E. M., Stanton, M. E., Dodge, N. C., Pienaar, M., Warton, C. M. R., … Jacobson, S. W. (2017). Functional MRI of human Eyeblink classical conditioning in children with fetal alcohol Spectrum disorders. *Cerebral Cortex, 27*(7), 3752–3767. https://doi.org/10.1093/cercor/bhw273

*Cistaro, A., Pagani, M., Montuschi, A., Calvo, A., Moglia, C., Canosa, A., Restagno G., Brunetti M., Traynor B. J., Nobili F., Carrara G., Fania P., Lopiano L., Valentini M. C., Chiò A. (2014). The metabolic signature of C9ORF72-related ALS: FDG PET comparison with nonmutated patients. European Journal of Nuclear Medicine and Molecular Imaging, 41(5), 844–852, doi:https://doi.org/10.1007/s00259-013-2667-5.

Colloby, S. J., O'Brien, J. T., & Taylor, J. P. (2014). Patterns of cerebellar volume loss in dementia with Lewy bodies and Alzheimers disease: A VBM-DARTEL study. *Psychiatry Research, 223*(3), 187–191. https://doi.org/10.1016/j.pscychresns.2014.06.006

Coyle-Gilchrist, I. T., Dick, K. M., Patterson, K., Vazquez Rodriquez, P., Wehmann, E., Wilcox, A., et al. (2016). Prevalence, characteristics, and survival of frontotemporal lobar degeneration syndromes. *Neurology, 86*(18), 1736–1743. https://doi.org/10.1212/WNL.0000000000002638

de Azevedo, P. C., Guimaraes, R. P., Piccinin, C. C., Piovesana, L. G., Campos, L. S., Zuiani, J. R., et al. (2017). Cerebellar Gray Matter Alterations in Huntington Disease: A Voxel-Based Morphometry Study. In *Cerebellar gray matter alterations in Huntington*. Disease: A Voxel-Based Morphometry Study. Cerebellum. https://doi.org/10.1007/s12311-017-0865-6

*De Winter, F. L., Timmers, D., de Gelder, B., Van Orshoven, M., Vieren, M., Bouckaert, M., et al. (2016). Face shape and face identity processing in behavioral variant fronto-temporal dementia: A specific deficit for familiarity and name recognition of famous faces. NeuroImage: Clinical, 11, 368–377.

DeJesus-Hernandez, M., Mackenzie, I. R., Boeve, B. F., Boxer, A. L., Baker, M., Rutherford, N. J., … Rademakers, R. (2011). Expanded GGGGCC hexanucleotide repeat in noncoding region of C9ORF72 causes chromosome 9p-linked FTD and ALS. *Neuron, 72*(2), 245–256. https://doi.org/10.1016/j.neuron.2011.09.011

*Dermody, N., Wong, S., Ahmed, R., Piguet, O., Hodges, J. R., & Irish, M. (2016). Uncovering the neural bases of cognitive and affective empathy deficits in Alzheimer's disease and the behavioral-variant of frontotemporal dementia. Journal of Alzheimer's Disease, 53(3), 801–816, doi:https://doi.org/10.3233/jad-160175.

Devenney, E., Hornberger, M., Irish, M., Mioshi, E., Burrell, J., Tan, R., … Hodges, J. R. (2014). Frontotemporal dementia associated with the C9ORF72 mutation: A unique clinical profile. *JAMA Neurology, 71*(3), 331–339. https://doi.org/10.1001/jamaneurol.2013.6002

*Devenney, E., Landin-Romero, R., Irish, M., Hornberger, M., Mioshi, E., Halliday, G. M., et al. (2017). The neural correlates and clinical characteristics of psychosis in the frontotemporal dementia continuum and the C9orf72 expansion. Neuroimage Clin, 13, 439–445, doi:https://doi.org/10.1016/j.nicl.2016.11.028.

Dogan, I., Tinnemann, E., Romanzetti, S., Mirzazade, S., Costa, A. S., Werner, C. J., … Reetz, K. (2016). Cognition in Friedreich's ataxia: A behavioral and multimodal imaging study. *Annals of Clinical Translational Neurology, 3*(8), 572–587. https://doi.org/10.1002/acn3.315

Dukart, J., Mueller, K., Horstmann, A., Barthel, H., Moller, H. E., Villringer, A., et al. (2011). Combined evaluation of FDG-PET and MRI improves detection and differentiation of dementia. *PLoS One, 6*(3), e18111. https://doi.org/10.1371/journal.pone.0018111

Dukart, J., Mueller, K., Horstmann, A., Vogt, B., Frisch, S., Barthel, H., … Schroeter, M. L. (2010). Differential effects of global and cerebellar normalization on detection and differentiation of dementia in FDG-PET studies. *Neuroimage, 49*(2), 1490–1495. https://doi.org/10.1016/j.neuroimage.2009.09.017

Eickhoff, S. B., Bzdok, D., Laird, A. R., Kurth, F., & Fox, P. T. (2012). Activation likelihood estimation meta-analysis revisited. *Neuroimage, 59*(3), 2349–2361. https://doi.org/10.1016/j.neuroimage.2011.09.017

Eickhoff, S. B., Laird, A. R., Grefkes, C., Wang, L. E., Zilles, K., & Fox, P. T. (2009). Coordinate-based activation likelihood estimation meta-analysis of neuroimaging data: A random-effects approach based on empirical estimates of spatial uncertainty. *Human Brain Mapping, 30*(9), 2907–2926. https://doi.org/10.1002/hbm.20718

*Farb, N. A., Grady, C. L., Strother, S., Tang-Wai, D. F., Masellis, M., Black, S., et al. (2013). Abnormal network connectivity in frontotemporal dementia: Evidence for prefrontal isolation. Cortex, 49(7), 1856–1873, doi:https://doi.org/10.1016/j.cortex.2012.09.008.

Ferrer, I., Legati, A., Garcia-Monco, J. C., Gomez-Beldarrain, M., Carmona, M., Blanco, R., et al. (2015). Familial behavioral variant frontotemporal dementia associated with astrocyte-predominant tauopathy. *Journal of Neuropathology and Experimental Neurology, 74*(4), 370–379.

Gao, L., Zhang, J., Hou, Y., Hallett, M., Chan, P., & Wu, T. (2017). The cerebellum in dual-task performance in Parkinson's disease. *Scientific Reports, 7*(1), 45662. https://doi.org/10.1038/srep45662

Gellersen, H. M., Guo, C. C., O'Callaghan, C., Tan, R. H., Sami, S., & Hornberger, M. (2017). Cerebellar atrophy in neurodegeneration-a meta-analysis. *Journal of Neurology, Neurosurgery, and Psychiatry, 88*(9), 780–788. https://doi.org/10.1136/jnnp-2017-315607

Gorno-Tempini, M. L., Hillis, A. E., Weintraub, S., Kertesz, A., Mendez, M., Cappa, S. F., … Grossman, M. (2011). Classification of primary progressive aphasia and its variants. *Neurology, 76*(11), 1006–1014. https://doi.org/10.1212/WNL.0b013e31821103e6

Gross-Tsur, V., Ben-Bashat, D., Shalev, R. S., Levav, M., & Sira, L. B. (2006). Evidence of a developmental cerebello-cerebral disorder. *Neuropsychologia, 44*(12), 2569–2572. https://doi.org/10.1016/j.neuropsychologia.2006.04.028

*Grossman, M., McMillan, C., Moore, P., Ding, L. J., Glosser, G., Work, M., et al. (2004). What's in a name: Voxel-based morphometric analyses of MRI and naming difficulty in Alzheimer's disease, frontotemporal dementia and corticobasal degeneration. Brain, 127, 628–649, doi:https://doi.org/10.1093/brain/awh075, 3.

*Guo, C. C., Tan, R., Hodges, J. R., Hu, X., Sami, S., & Hornberger, M. (2016). Network-selective vulnerability of the human cerebellum to Alzheimer's disease and frontotemporal dementia. Brain, 139(Pt 5), 1527–1538, doi:https://doi.org/10.1093/brain/aww003.

*Henley, S. M., Downey, L. E., Nicholas, J. M., Kinnunen, K. M., Golden, H. L., Buckley, A., et al. (2014). Degradation of cognitive timing mechanisms in behavioural variant frontotemporal dementia. Neuropsychologia, 65, 88–101, doi:https://doi.org/10.1016/j.neuropsychologia.2014.10.009.

*Hutchings, R. P.;Bruggemann, J.;Hodges, J. R.;Piguet, O.;Kumfor, F. (2018). Looking but not seeing: Increased eye fixations in behavioural-variant frontotemporal dementia. *Cortex, 103*, 71–81, doi:https://doi.org/10.1016/j.cortex.2018.02.011.

*Irish, M., Devenney, E., Wong, S., Dobson-Stone, C., Kwok, J. B., Piguet, O., Hodges J. R., Hornberger M. (2013). Neural substrates of episodic memory dysfunction in behavioural variant

frontotemporal dementia with and without C9ORF72 expansions. Neuroimage Clin, 2, 836–843, doi:https://doi.org/10.1016/j.nicl.2013.06.005.

Jacobs, H. I. L., Hopkins, D. A., Mayrhofer, H. C., Bruner, E., van Leeuwen, F. W., Raaijmakers, W., & Schmahmann, J. D. (2017). The cerebellum in Alzheimer's disease: Evaluating its role in cognitive decline. *Brain, 141*(1), 37–47. https://doi.org/10.1093/brain/awx194

Jiskoot, L. C. P., Meeter, L. H., Dopper, E. G. P., Donker Kaat, L., Franzen, S., van der Ende, E. L., … van Swieten, J. C. (2019). Longitudinal multimodal MRI as prognostic and diagnostic biomarker in presymptomatic familial frontotemporal dementia. *Brain, 142*(1), 193–208. https://doi.org/10.1093/brain/awy288

Jissendi, P., Baudry, S., & Baleriaux, D. (2008). Diffusion tensor imaging (DTI) and tractography of the cerebellar projections to prefrontal and posterior parietal cortices: A study at 3T. *Journal of Neuroradiology, 35*(1), 42–50. https://doi.org/10.1016/j.neurad.2007.11.001

Kelly, R. M., & Strick, P. L. (2003). Cerebellar loops with motor cortex and prefrontal cortex of a nonhuman primate. *The Journal of Neuroscience, 23*(23), 8432–8444.

*Kloeters, S., Bertoux, M., O'Callaghan, C., Hodges, J. R., & Hornberger, M. (2013). Money for nothing - atrophy correlates of gambling decision making in behavioural variant frontotemporal dementia and Alzheimer's disease. Neuroimage Clin, 2, 263–272, doi:https://doi.org/10.1016/j.nicl.2013.01.011.

Krienen, F. M., & Buckner, R. L. (2009). Segregated fronto-cerebellar circuits revealed by intrinsic functional connectivity. *Cerebral Cortex, 19*(10), 2485–2497. https://doi.org/10.1093/cercor/bhp135

*Kumfor, F., Honan, C., McDonald, S., Hazelton, J. L., Hodges, J. R., & Piguet, O. (2017). Assessing the "social brain" in dementia: Applying TASIT-S. Cortex, 93, 166–177, doi:https://doi.org/10.1016/j.cortex.2017.05.022.

Kumfor, F., Ibanez, A., Hutchings, R., Hazelton, J. L., Hodges, J. R., & Piguet, O. (2018a). Beyond the face: How context modulates emotion processing in frontotemporal dementia subtypes. *Brain, 141*(4), 1172–1185. https://doi.org/10.1093/brain/awy002

*Kumfor, F., Irish, M., Hodges, J. R., & Piguet, O. (2013a). Discrete neural correlates for the recognition of negative emotions: Insights from frontotemporal dementia. PLoS One, 8(6), e67457, doi:https://doi.org/10.1371/journal.pone.0067457.

*Kumfor, F., Irish, M., Hodges, J. R., & Piguet, O. (2013b). The orbitofrontal cortex is involved in emotional enhancement of memory: Evidence from the dementias. Brain, 136(Pt 10), 2992–3003, doi:https://doi.org/10.1093/brain/awt185.

Kumfor, F., Landin-Romero, R., Devenney, E., Hutchings, R., Grasso, R., Hodges, J. R., & Piguet, O. (2016). On the right side? A longitudinal study of left- versus right-lateralized semantic dementia. *Brain, 139*(Pt 3), 986–998. https://doi.org/10.1093/brain/awv387

*Kumfor, F., Zhen, A., Hodges, J. R., Piguet, O., & Irish, M. (2018b). Apathy in Alzheimer's disease and frontotemporal dementia: Distinct clinical profiles and neural correlates. Cortex, 103, 350–359, doi:https://doi.org/10.1016/j.cortex.2018.03.019.

Laidi, C., Boisgontier, J., Chakravarty, M. M., Hotier, S., d'Albis, M. A., Mangin, J. O., et al. (2017). Cerebellar anatomical alterations and attention to eyes in autism. *Scientific Reports, 7*(1), 12008. https://doi.org/10.1038/s41598-017-11883-w

*Laisney, M., Matuszewski, V., Mezenge, F., Belliard, S., de la Sayette, V., Eustache, F., et al. (2009). The underlying mechanisms of verbal fluency deficit in frontotemporal dementia and semantic dementia. Journal of Neurology, 256(7), 1083–1094, doi:https://doi.org/10.1007/s00415-009-5073-y.

Lancaster, J. L., Tordesillas-Gutierrez, D., Martinez, M., Salinas, F., Evans, A., Zilles, K., et al. (2007). Bias between MNI and Talairach coordinates analyzed using the ICBM-152 brain template. *Human Brain Mapping, 28*(11), 1194–1205. https://doi.org/10.1002/hbm.20345

Lange, I., Kasanova, Z., Goossens, L., Leibold, N., De Zeeuw, C. I., van Amelsvoort, T., et al. (2015). The anatomy of fear learning in the cerebellum: A systematic meta-analysis. *Neuroscience and Biobehavioral Reviews, 59*, 83–91. https://doi.org/10.1016/j.neubiorev.2015.09.019

Lee, S. E., Khazenzon, A. M., Trujillo, A. J., Guo, C. C., Yokoyama, J. S., Sha, S. J., … Seeley, W. W. (2014). Altered network connectivity in frontotemporal dementia with C9orf72 hexanucleotide repeat expansion. *Brain, 137*(Pt 11), 3047–3060. https://doi.org/10.1093/brain/awu248

*Meijboom, R., Steketee, R. M., de Koning, I., Osse, R. J., Jiskoot, L. C., de Jong, F. J., et al. (2016). Functional connectivity and microstructural white matter changes in phenocopy frontotemporal dementia. European Radiology, doi:https://doi.org/10.1007/s00330-016-4490-4, 27, 4, 1352, 1360.

Muller, V. I., Cieslik, E. C., Laird, A. R., Fox, P. T., Radua, J., Mataix-Cols, D., et al. (2018). Ten simple rules for neuroimaging meta-analysis. *Neuroscience and Biobehavioral Reviews, 84*, 151–161. https://doi.org/10.1016/j.neubiorev.2017.11.012

Neary, D., Snowden, J. S., Gustafson, L., Passant, U., Stuss, D., Black, S., … Benson, D. F. (1998). Frontotemporal lobar degeneration: a consensus on clinical diagnostic criteria. *Neurology, 51*(6), 1546–1554.

Petacchi, A., Laird, A. R., Fox, P. T., & Bower, J. M. (2005). Cerebellum and auditory function: An ALE meta-analysis of functional neuroimaging studies. *Human Brain Mapping, 25*(1), 118–128. https://doi.org/10.1002/hbm.20137

Ramnani, N. (2006). The primate cortico-cerebellar system: Anatomy and function. *Nature Reviews. Neuroscience, 7*(7), 511–522. https://doi.org/10.1038/nrn1953

Ramnani, N. (2012). Frontal lobe and posterior parietal contributions to the cortico-cerebellar system. *Cerebellum, 11*(2), 366–383. https://doi.org/10.1007/s12311-011-0272-3

Ramnani, N., Behrens, T. E., Johansen-Berg, H., Richter, M. C., Pinsk, M. A., Andersson, J. L., et al. (2006). The evolution of prefrontal inputs to the cortico-pontine system: Diffusion imaging evidence from macaque monkeys and humans. *Cerebral Cortex, 16*(6), 811–818. https://doi.org/10.1093/cercor/bhj024

Rascovsky, K., Hodges, J. R., Kipps, C. M., Johnson, J. K., Seeley, W. W., Mendez, M. F., … Miller, B. M. (2007). Diagnostic criteria for the behavioral variant of frontotemporal dementia (bvFTD): Current limitations and future directions. *Alzheimer Disease and Associated Disorders, 21*(4), S14–S18. https://doi.org/10.1097/WAD.0b013e31815c3445

Rascovsky, K., Hodges, J. R., Knopman, D., Mendez, M. F., Kramer, J. H., Neuhaus, J., … Miller, B. L. (2011). Sensitivity of revised diagnostic criteria for the behavioural variant of frontotemporal dementia. *Brain, 134*(Pt 9), 2456–2477. https://doi.org/10.1093/brain/awr179

Ratnavalli, E., Brayne, C., Dawson, K., & Hodges, J. R. (2002). The prevalence of frontotemporal dementia. *Neurology, 58*(11), 1615–1621.

Renton, A. E., Majounie, E., Waite, A., Simon-Sanchez, J., Rollinson, S., Gibbs, J. R., et al. (2011). A hexanucleotide repeat expansion in C9ORF72 is the cause of chromosome 9p21-linked ALS-FTD. *Neuron, 72*(2), 257–268. https://doi.org/10.1016/j.neuron.2011.09.010

Riedel, M. C., Ray, K. L., Dick, A. S., Sutherland, M. T., Hernandez, Z., Fox, P. M., … Laird, A. R. (2015). Meta-analytic connectivity and behavioral parcellation of the human cerebellum. *Neuroimage, 117*, 327–342. https://doi.org/10.1016/j.neuroimage.2015.05.008

Rosen, H. J., Gorno-Tempini, M. L., Goldman, W. P., Perry, R. J., Schuff, N., Weiner, M., et al. (2002). Patterns of brain atrophy in frontotemporal dementia and semantic dementia. *Neurology, 58*(2), 198–208.

Savica, R., Adeli, A., Vemuri, P., Knopman, D. S., Dejesus-Hernandez, M., Rademakers, R., et al. (2012). Characterization of a family with c9FTD/ALS associated with the GGGGCC repeat expansion in C9ORF72. *Archives of Neurology, 69*(9), 1164–1169. https://doi.org/10.1001/archneurol.2012.772

Schmahmann, J. D., & Pandya, D. N. (1995). Prefrontal cortex projections to the basilar pons in rhesus monkey: Implications for the cerebellar contribution to higher function. *Neuroscience Letters, 199*(3), 175–178.

Schmahmann, J. D., & Pandya, D. N. (1997a). Anatomic organization of the basilar pontine projections from prefrontal cortices in rhesus monkey. *The Journal of Neuroscience, 17*(1), 438–458.

Schmahmann, J. D., & Pandya, D. N. (1997b). The cerebrocerebellar system. *International Review of Neurobiology, 41*, 31–60.

Schmahmann, J. D., & Sherman, J. C. (1998). The cerebellar cognitive affective syndrome. *Brain, 121*(4), 561–579.

*Seelaar, H., Papma, J. M., Garraux, G., de Koning, I., Reijs, A. E., Valkema, R., Rozemuller A. J. M., Salmon E., van Swieten J. C. (2011). Brain perfusion patterns in familial frontotemporal lobar degeneration. Neurology, 77(4), 384–392, doi:https://doi.org/10.1212/WNL.0b013e3182270456.

Seeley, W. W., Crawford, R., Rascovsky, K., Kramer, J. H., Weiner, M., Miller, B. L., & Gorno-Tempini, M. L. (2008). Frontal paralimbic network atrophy in very mild behavioral variant frontotemporal dementia. *Archives of Neurology, 65*(2), 249–255. https://doi.org/10.1001/archneurol.2007.38

Sha, S. J., Takada, L. T., Rankin, K. P., Yokoyama, J. S., Rutherford, N. J., Fong, J. C., … Boxer, A. L. (2012). Frontotemporal dementia due to C9ORF72 mutations: Clinical and imaging features. *Neurology, 79*(10), 1002–1011. https://doi.org/10.1212/WNL.0b013e318268452e

Shinn, A. K., Roh, Y. S., Ravichandran, C. T., Baker, J. T., Ongur, D., & Cohen, B. M. (2017). Aberrant cerebellar connectivity in bipolar disorder with psychosis. *Biol Psychiatry Cogn Neurosci Neuroimaging, 2*(5), 438–448. https://doi.org/10.1016/j.bpsc.2016.07.002

Stoodley, C. J., & Schmahmann, J. D. (2009). Functional topography in the human cerebellum: A meta-analysis of neuroimaging studies. *Neuroimage, 44*(2), 489–501. https://doi.org/10.1016/j.neuroimage.2008.08.039

Strenziok, M., Pulaski, S., Krueger, F., Zamboni, G., Clawson, D., & Grafman, J. (2011). Regional brain atrophy and impaired decision making on the balloon analog risk task in behavioral variant frontotemporal dementia. *Cognitive and Behavioral Neurology, 24*(2), 59–67. https://doi.org/10.1097/WNN.0b013e3182255a7c

Strick, P. L., Dum, R. P., & Fiez, J. A. (2009). Cerebellum and nonmotor function. *Annual Review of Neuroscience, 32*(1), 413–434. https://doi.org/10.1146/annurev.neuro.31.060407.125606

Strong, M. J., Grace, G. M., Freedman, M., Lomen-Hoerth, C., Woolley, S., Goldstein, L. H., … Figlewicz, D. (2009). Consensus criteria for the diagnosis of frontotemporal cognitive and behavioural syndromes in amyotrophic lateral sclerosis. *Amyotrophic Lateral Sclerosis, 10*(3), 131–146.

*Sturm, V. E., Yokoyama, J. S., Eckart, J. A., Zakrzewski, J., Rosen, H. J., Miller, B. L., Seeley W. W., Levenson R. W. (2015). Damage to left frontal regulatory circuits produces greater positive emotional reactivity in frontotemporal dementia. Cortex, 64, 55–67, doi:https://doi.org/10.1016/j.cortex.2014.10.002.

Suarez-Calvet, M., Camacho, V., Gomez-Anson, B., Anton, S., Vives-Gilabert, Y., Dols-Icardo, O., et al. (2015). Early cerebellar Hypometabolism in patients with frontotemporal dementia carrying the C9orf72 expansion. *Alzheimer Disease and Associated Disorders, 29*(4), 353–356. https://doi.org/10.1097/WAD.0000000000000056

*Synn, A., Mothakunnel, A., Kumfor, F., Chen, Y., Piguet, O., Hodges, J. R., et al. (2017). Mental states in moving shapes: Distinct cortical and subcortical contributions to theory of mind impairments in dementia. *Journal of Alzheimer's Disease, 61*(2), 521-535, doi: https://doi.org/10.3233/JAD-170809.

Synn, A., Mothakunnel, A., Kumfor, F., Chen, Y., Piguet, O., Hodges, J. R., & Irish, M. (2018). Mental states in moving shapes: Distinct cortical and subcortical contributions to theory of mind impairments in dementia. *Journal of Alzheimer's Disease, 61*(2), 521–535. https://doi.org/10.3233/JAD-170809

Tan, R. H., Devenney, E., Dobson-Stone, C., Kwok, J. B., Hodges, J. R., Kiernan, M. C., … Hornberger, M. (2014). Cerebellar integrity in the amyotrophic lateral sclerosis-frontotemporal dementia continuum. *PLoS One, 9*(8), e105632. https://doi.org/10.1371/journal.pone.0105632

*Tan, R. H., Devenney, E., Kiernan, M. C., Halliday, G. M., Hodges, J. R., & Hornberger, M. (2015). Terra incognita-cerebellar contributions to neuropsychiatric and cognitive dysfunction in behavioral variant frontotemporal dementia. Frontiers in Aging Neuroscience, 7, 121, doi:https://doi.org/10.3389/fnagi.2015.00121.

Turkeltaub, P. E., Eden, G. F., Jones, K. M., & Zeffiro, T. A. (2002). Meta-analysis of the functional neuroanatomy of single-word reading: Method and validation. *Neuroimage, 16*(3 Pt 1), 765–780.

Turkeltaub, P. E., Eickhoff, S. B., Laird, A. R., Fox, M., Wiener, M., & Fox, P. (2012). Minimizing within-experiment and within-group effects in activation likelihood estimation meta-analyses. *Human Brain Mapping, 33*(1), 1–13. https://doi.org/10.1002/hbm.21186

Van Overwalle, F., Baetens, K., Marien, P., & Vandekerckhove, M. (2014). Social cognition and the cerebellum: A meta-analysis of over 350 fMRI studies. *Neuroimage, 86*, 554–572. https://doi.org/10.1016/j.neuroimage.2013.09.033

Van Overwalle, F., D'Aes, T., & Marien, P. (2015). Social cognition and the cerebellum: A meta-analytic connectivity analysis. *Human Brain Mapping, 36*(12), 5137–5154. https://doi.org/10.1002/hbm.23002

Vemuri, P., Simon, G., Kantarci, K., Whitwell, J. L., Senjem, M. L., Przybelski, S. A., … Jack, C. R., Jr. (2011). Antemortem differential diagnosis of dementia pathology using structural MRI: Differential-STAND. *Neuroimage, 55*(2), 522–531. https://doi.org/10.1016/j.neuroimage.2010.12.073

Whitwell, J. L., Avula, R., Senjem, M. L., Kantarci, K., Weigand, S. D., Samikoglu, A., … Jack, C. R. (2010). Gray and white matter water diffusion in the syndromic variants of frontotemporal dementia. *Neurology, 74*(16), 1279–1287. https://doi.org/10.1212/WNL.0b013e3181d9edde

Whitwell, J. L., Weigand, S. D., Boeve, B. F., Senjem, M. L., Gunter, J. L., DeJesus-Hernandez, M., … Josephs, K. A. (2012). Neuroimaging signatures of frontotemporal dementia genetics: C9ORF72, tau, progranulin and sporadics. *Brain, 135*(Pt 3), 794–806. https://doi.org/10.1093/brain/aws001

*Wong, S., Irish, M., O'Callaghan, C., Kumfor, F., Savage, G., Hodges, J. R., et al. (2017). Should I trust you? Learning and memory of social interactions in dementia. Neuropsychologia, 104, 157–167, doi: https://doi.org/10.1016/j.neuropsychologia.2017.08.016.