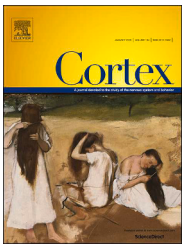
CORTEX 194 (2026) 121–142

Available online at www.sciencedirect.com

ScienceDirect

Journal homepage: www.elsevier.com/locate/cortex



Research Report

Cognitive-linguistic skills in production of expository discourse: Insights from longitudinal changes and neural correlates in primary progressive aphasia

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ARTICLE INFO

Article history:
Received 3 May 2025
Revised 18 November 2025
Accepted 20 November 2025
Published online 1 December 2025

Keywords:
Discourse
Connected speech
Connected language
Neuroscience
Cognition
Primary progressive aphasia

ABSTRACT

Background: Engaging in conversational and story-telling discourse involves an interplay of language and cognitive skills, including working memory, attention, and inference-making. Primary progressive aphasia (PPA) provides a model for exploring discourse, as both language and cognitive abilities change over time with changes in cortical atrophy. Here, associations between morphosyntactic discourse skills and patterns of cortical atrophy are measured over time in nonfluent (nfv), logopenic (lv) and semantic (sv) variants of PPA.

Method: Participants were 27 individuals with nfvPPA (M = 66.6 years ± 8.3), 30 lvPPA (M = 66.7 ± 7.3), 33 svPPA (M = 64.8 ± 6.7), and 36 healthy controls (HC; M = 65.5 ± 6.8). Picture descriptions were analysed for word density and diversity, sentence complexity, well-formedness, and fluency annually for up to three timepoints. Associations between language measures and cortical thickness on structural MRI scans were analysed.

Results: At timepoint 1, nfvPPA performed below other groups on most measures; lvPPA were differentiated from svPPA on fluency measures only. Longitudinally, utterance length declined in all variants. For nfvPPA, this was linked with reduced sentence complexity and cortical atrophy in regions engaged by higher attentional demand. For lvPPA, it was linked with increasing grammatical errors and atrophy extending into perisylvian language network. No associations were identified for svPPA.

Conclusions: Findings provide insight into how discourse production is underpinned by a network that extends beyond classic language regions, with morphosyntactic elements of discourse associated in part with regions involved in domain-general cognitive skills such as error-monitoring and elaborative encoding. Findings can also inform assessment, prognosis, and intervention for communication through the PPA disease course.

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https://doi.org/10.1016/j.cortex.2025.11.011

1. Introduction

Discourse is conceptualised as connected language that extends beyond individual words and single sentences, enabling individuals to express coherent thoughts and ideas across diverse communicative contexts (Kong, 2023). It affords analysis of both microstructural linguistic (i.e., vocabulary, morphology, syntax) and macrostructural discourse organisation skills (e.g., cohesion and coherence across sentences). Discourse-level tasks (e.g., describing scenes, telling stories, or expressing opinions) are complex in their integration of multiple cognitive processes (i.e., working memory, attention, and inference-making; Federmeier et al., 2020). They have been used as sensitive markers of change in the earliest stages of neurodegeneration affecting core regions of the brain involved in language as well as domain general cognitive processes of attention, memory and executive function (Themistocleous, 2023; Mueller, 2023). Not surprisingly, discourse tasks have been used frequently to detect and explore progressive changes in microstructural skills in individuals with Primary Progressive Aphasia (PPA), who experience progressive linguistic deterioration over time due to frontotemporal or atypical Alzheimer's dementia (e.g., Leyton et al., 2011). However, while PPA initially presents with symptoms disproportionately affecting the language system, and pathology in the classic left perysylvian language regions, changes in cognitive abilities emerge over time and progress alongside the language symptoms. This population, therefore, provides a unique opportunity to study temporal associations between microstructural linguistic skills, and cognitive skills as pathology extends beyond the classic left perisylvian language network. Examining the PPA profiles of change in cortical atrophy and microstructural behaviour over time has potential to reveal whether and how these core linguistic skills may be influenced by cortical regions involved in more domain-general cognitive processes.

PPA has three language variants typically caused by different neurodegenerative pathology and tending to emerge in different regions within the left hemisphere (Gorno-Tempini et al., 2011). The nonfluent "agrammatic" variant (nfvPPA) has been characterised by word-finding difficulty and errors or simplification of morphology and syntax with early atrophy observed in the left posterior inferior frontal region. It frequently co-occurs with the motor speech disorder of apraxia of speech, which causes distortions of speech sounds and the intonational contour of speech (Duffy et al., 2014, 2021). The logopenic variant (lvPPA) shows word-finding difficulty accompanied by phonological processing and working memory challenges. Memory span for sentence repetition is reduced, and words often contain phonemic errors. It has often been associated with pathology in and around the left temporoparietal junction. The semantic variant (svPPA) affects semantically-based word retrieval and comprehension, with early atrophy focused in the left anterior temporal lobe. While agrammatism (i.e., impairment in production of grammatical structures, including morphology and/or syntax) is listed as a key diagnostic feature of nfvPPA (Gorno-Tempini et al., 2011; Leyton et al., 2011), simplification or errors of morphology and syntax have been reported in all three variants (Mack et al., 2021; Meteyard & Patterson, 2009; Sajjadi et al., 2012; Thompson et al., 2012; Wilson, Henry, et al., 2010). Here, we explore longitudinal associations between microstructural discourse components and distribution of cortical atrophy, to gain insight into whether and how this level of discourse production is underpinned by a network that extends beyond classic language regions.

Selecting a discourse genre (e.g., expository, procedural or narrative discourse) is essential in informing the selection of measures, as these can help capture fine-grained linguistic (i.e., microstructural) and/or broader discourse (i.e., macrostructural) elements. According to Kintz (2023), monologic, stimulus-based discourse can be characterised as narrative (e.g., retelling the Cinderella story) or expository (e.g., describing a complex scene). Narrative discourse tasks are defined by a temporal structure that contains a beginning, middle and end, while engaging macro-level components, cohesion and coherence across story elements. While expository monologues lack this temporal structure, they yield rich microstructural metrics (Greenslade et al., 2024) which can quantify linguistic changes across a range of complexity for individuals with PPA. This makes expository monologues more sensitive detectors of language decline, especially for individuals whose cognitive profiles impair episodic memory and sequencing skills (Kong et al., 2023). Therefore, such a focused approach can help signal broader communicative decline at the discourse level.

Previous studies of discourse production in PPA, in both narrative and expository tasks, have focused on measurement at the microstructural level, examining (i) lexical factors (e.g., diversity of words, density of content, ratios of nouns to verbs or open class to closed class words), (ii) morphosyntactic factors (e.g., inflectional morphology, verb argument structure, number of dependent clauses per sentence as an index of complexity, and presence of grammatical errors and (iii) fluency (e.g., words per minute) (Marshall, 2011; Thompson & Mack, 2014). An overview of common measures and associated findings is shown in Table 1. While these variables have differentiated the three PPA variants in some studies, there have been conflicting findings. Individuals with nfvPPA have shown *elevated* noun:verb and/or open:closed class word ratios, related to inefficient retrieval of verbs and grammatical morphology (e.g., verb inflections, determiners, pronouns) that defines agrammatism (Ash et al., 2013; Fraser et al., 2014; Themistocleous et al., 2021; Thompson & Mack, 2014).

Table 1 – Microstructural discourse measures (i.e., lexical, morphosyntactic, and fluency) reported in the literature to differentiate the PPA variants and used in the current study.

Measure	Formulas[a]	Supporting studies
Lexical measures		
Type:Token ratio	Number of unique words/total number of words (lexical diversity)	Correlates with grammatical deficits; can be a consequence of relying on limited morphosyntactic structures (Fraser et al., 2014).
Propositional idea density	Number of non-noun entities (verbs, adjectives, adverbs, prepositions, conjunctions)/total number of words	Lower density in general cognitive decline (Engelman et al., 2010; Harrag et al., 2024); may differentiate nfvPPA[b] from other variants (Themistocleous et al., 2021).
Noun:Verb ratio	Number of nouns/number of verbs	Higher ratio (verb retrieval impairment) in nfvPPA; lower ratio (noun retrieval impairment) in svPPA[b] and lvPPA[b] (Ash et al., 2013; Faroqi-Shah et al., 2020; Thompson et al., 2012; Wilson et al., 2010).
Open:Closed class ratio	Number of open class words/number of closed class words	Higher ratio (fewer closed class words) in nfvPPA; lower ratio (fewer open class words) in svPPA and lvPPA (Ash et al., 2013; Faroqi-Shah et al., 2020; Fraser et al., 2014).
Morphosyntactic measures		
Mean length of utterance in morphemes (MLU)	Total number of morphemes/total number of utterances	Lower in nfvPPA; may be reduced in all variants relative to healthy controls (Ash et al., 2006, 2013; Graham et al., 2016; Thompson et al., 2012; Wilson et al., 2010).
Sentences with flawed syntax (%)	Number of sentences with grammatical error/ number of sentences	Higher proportion of ungrammatical sentences in nfvPPA and lvPPA than svPPA or controls (Lavoie et al., 2021; Mack et al., 2021).
Embedded Clauses:Sentences Ratio	Number of embedded clauses/number of sentences (sentence complexity)	Declines with healthy aging and typical dementia (Kemper et al., 2001); reduced in nfvPPA relative to other PPA variants (Ash et al., 2013; Fraser et al., 2014; Sajjadi et al., 2012, Wilson et al., 2010).
Fluency measures		
Words/Minute	Total number of words/total length of audio in minutes	Correlated in nfvPPA with grammaticality/ sentence complexity (Ash et al., 2019; Gunawardena et al., 2010) and presence of motor speech disorder (i.e., apraxia, dysarthria; Duffy, 2014).
Retracings	Number of self-corrections or changes (e.g., "the boy … girl is reaching up")	Correlated with syntactic errors in lvPPA (Wilson et al., 2012).
Repetitions	Number of sequentially repeated words/ phrases (e.g., "boy … boy")	Differentiates PPA from healthy controls and those with mild cognitive impairment (Faroqi-Shah et al., 2020).

[a] MacWhinney, 2000.
[b] nfvPPA = nonfluent variant of primary progressive aphasia, lvPPA = logopenic variant, svPPA = semantic variant.

Consistent with degradation of the semantic system, cases with svPPA typically have shown a *reduced* noun:verb ratio against other variants and healthy controls (Themistocleous et al., 2021; Thompson et al., 2012; Thompson & Mack, 2014). Open:closed class word ratio has been elevated in both nfvPPA and lvPPA relative to svPPA in some studies (Thompson et al., 2012). Individuals with nfvPPA tend to show reduced length of sentences, fewer dependent clauses per sentence and, in some studies, more sentences containing grammatical errors (Ash et al., 2013, 2019; Faroqi-Shah et al., 2020; Fraser et al., 2014; Lorca-Puls et al., 2023; Thompson & Mack, 2014; Wilson, Dronkers, et al., 2010). While individuals with lvPPA and svPPA are often reported to have performance similar to healthy controls (Ash et al., 2013, 2019; Meteyard & Patterson, 2009; Sajjadi et al., 2012; Thompson et al., 2012; Thompson & Mack, 2014; Wilson, Dronkers, et al., 2010), lvPPA and nfvPPA are not always well differentiated on these measures. Fluency is typically lowest in nfvPPA (Ash et al., 2013; Faroqi-Shah et al., 2020; Thompson et al., 2012; Thompson & Mack, 2014) but also reduced in lvPPA and svPPA compared with healthy controls (Ash et al., 2013; Sajjadi et al., 2012; Thompson et al., 2012; Wilson, Henry, et al., 2010), likely related to inefficient word retrieval. Equivocal findings across studies, with at times limited differentiation of the PPA variants, could have several sources. Small sample sizes, initial diagnostic uncertainty of participants, and small samples of language are common

problems in studies of PPA, which is a rare condition requiring sample collection over many years and, therefore, often retrospective study designs.

Despite some studies reporting limited utility of microstructural measures in differentiating between PPA subtypes, the existing literature reveals correlations between specific language patterns and regions of cortical involvement across PPA variants and healthy controls. The inferior frontal gyrus (i.e., BA44/45 or Broca's area) is a key node within the distributed language network (Hickok & Poeppel, 2004; Kemmerer, 2022) that is strongly associated with production and processing of morphology and syntax, particularly activation of verbs and their arguments for sentence generation (Botha & Josephs, 2019). Consistent with this, verb-related deficits (e.g., verb inflection errors, shorter utterances, and paucity of embedded clause structures) in nfvPPA have been associated with atrophy in left posterior inferior frontal cortex (Ash et al., 2013, 2019; Deleon et al., 2012; Lorca-Puls et al., 2023; Thompson et al., 2013). However, Wilson, Dronkers, et al. (2010) reported, for nfvPPA, that superior and middle temporal lobe also play roles in verb and noun retrieval for sentence construction. Also, reduced utterance length and sentence well-formedness have been observed in lvPPA and svPPA and associated with atrophy in different cortical regions: left inferior parietal and superior temporal areas in lvPPA and left temporal, orbital frontal, and insula regions in svPPA (Ash et al., 2013). For these two variants, word retrieval difficulties disproportionately affect nouns, rather than verbs, and are related to activation of phonological or semantic representations, respectively. For individuals with lvPPA, impaired phonological short-term memory has been implicated as the cause of grammatical errors (Mack et al., 2021; Mesulam, Coventry, Rader, et al., 2021). As noted, the microstructural linguistic characteristics and changes in the three PPA variants are likely arising for different reasons and confounding interpretation. Studies of the microstructural change in discourse across the PPA, variants have almost exclusively, used cross-sectional designs. In the few longitudinal studies reporting microstructural measures and cortical integrity, nfvPPA and lvPPA have not been well-differentiated over time (Ash et al., 2019; Rogalski et al., 2011; Rohrer et al., 2009). This may be due to blurring of the boundaries between the variants as pathology extends throughout the language network or beyond canonical language regions. Ash et al. (2019) tested participants twice over an average 26 months and noted that percent of sentences with grammatical errors rose significantly and words produced per minute fell over time for all PPA variants. Both nfvPPA and svPPA groups used fewer complex sentences over time, while the lvPPA group remained stable on this measure. Ash et al. (2019) found an association between increased grammatical errors in sentences and increased cortical thinning in multiple regions (i.e., left frontal operculum/anterior insula, left middle/posterior insula, left inferior temporal gyrus, bilateral superior and middle temporal cortex; right posterior temporal and opercular cortex). No relationships were detected for the number of sentences with embedded clauses and words per minute. Participants were pooled, across variant, for brain-behaviour analyses due to small sample sizes. Further, it has been reported that pathology in the PPA variants invariably extends over time into right homologues (e.g., Kumfor et al., 2016; Landin-Romero et al., 2021; Leyton et al., 2019) and there is little known about the impact of this contralateral damage on grammatical profiles in the three PPA variants. In summary, the microstructural language measures used to date may not be sufficiently sensitive or specific to isolate morphosyntactic impairment from lexical or other cognitive changes in PPA. Also, small sample sizes (i.e., fewer than 15 per variant) have prevented analysis of variant-specific brain-behaviour relationships over time.

1.1. *Purpose*

Examining associations between these linguistic behaviours and regions of cortical atrophy, and how these might change longitudinally with the differing spread of atrophy across PPA variants, could shed light on the contribution of broader domain-general cognitive processes in generating the microstructural elements of discourse. The current study aimed to explore longitudinal changes in discourse-level lexical, morphosyntactic, and fluency measures, and associated changes in cortical integrity, in a large sample of individuals spanning the three clinical variants of PPA. We hypothesised that, at the first timepoint, (a) the nfvPPA group would produce fewer words per minute, have shorter utterance length, higher noun:verb and open:closed class word ratios and lower grammatical accuracy and complexity than the lvPPA and svPPA and healthy control groups, and (b) the lvPPA and svPPA groups would produce fewer words per minute, have shorter utterance length, lower noun:verb ratio and lower grammatical accuracy than healthy controls, and (c) measures of language impairment would associate with atrophy distributed in the canonical regions for the three variants. Further, we hypothesised that, longitudinally, (d) the nfvPPA group would deteriorate over time on the measures of verb retrieval and sentence complexity associated with increased atrophy in the left posterior inferior frontal region and extension to the right homologue, (e) the lvPPA and svPPA groups would show greater decrement on lexical and semantic measures associated with increased atrophy in regions along the ventral pathway (Hickok & Poeppel, 2004) and right homologues, and (f) as individuals with lvPPA can show spread of atrophy along the dorsal pathway, it is also hypothesised that associations between new regions of frontal atrophy and measures of verb retrieval and sentence complexity would emerge for this group (Mandelli et al., 2023). Overall, these longitudinal behavioural and brain imaging analyses have potential to reveal associations between domain-general cognitive processes and the ability to produce microstructural linguistic components within this discourse-level production task.

2. Methods

This study was approved by the Human Research Ethics Committee of the South Eastern Sydney Local Area Health District (HREC 10/126) and the Human Research Ethics Committee of the University of Sydney (HREC 2020/224 and 2020/408). All participants or their person responsible provided written informed consent in accordance with the Declaration of Helsinki. Participants volunteered their time and were reimbursed for travel costs.

Table 2 – Baseline demographics and diagnostic assessments for participants with nonfluent (nfv), logopenic (lv) and semantic (sv) variants of primary progressive aphasia (PPA) and healthy controls (HC).

Variable	nfvPPA (n = 27)	lvPPA (n = 30)	svPPA (n = 33)	HC (n = 36)	*Test*	*p*	Post-hoc
Sex (Male: Female)	9:18	16:14	17:16	20:16	3.58[a]	.31	N/A
Age at visit (year)	66.1 (12.9)	67.5 (8.0)	63.3 (10.6)	66.4 (5.3)	2.21[b]	.53	N/A
Education (year)	11.0 (4.0)	12.0 (4.8)	12.0 (4.0)	14.0 (2.6)	10.14[b]	.02	nfvPPA < HC
First language English	26/27	30/30	29/33	36/36	8.48[a]	.04	svPPA < HC
Disease duration (year)	3.2 (2.5)	2.4 (2.5)	3.5 (2.5)	N/A	4.77[b]	.09	N/A
Addenbrooke's cognitive examination – III[c]							
Total (/100)	81.2 (7.4)	68.6 (12.5)	59.0 (18.1)	94.5 (4.3)	85.36[b]	<.001	svPPA < nfvPPA < HC lvPPA < HC
Attention (/18)	16.0 (2.0)	14.0 (2.4)	15.4 (2.6)	18.0 (1.0)	52.17[b]	<.001	lvPPA < nfvPPA < HC svPPA < HC
Memory (/26)	23.0 (4.5)	15.5 (6.8)	12.0 (9.0)	25.0 (3.0)	73.00[b]	<.001	lvPPA, svPPA < HC, nfvPPA
Fluency (/14)	6.0 (3.5)	5.0 (4.0)	4.0 (4.0)	12.0 (2.0)	73.56[b]	<.001	nfvPPA, lvPPA, svPPA < HC
Language (/26)	22.0 (3.4)	20.1 (3.5)	13.0 (6.7)	26.0 (1.0)	80.61[b]	<.001	svPPA < nfvPPA, lvPPA < HC
Visuospatial (/16)	15.0 (3.0)	14.0 (3.0)	16.0 (2.0)	16.0 (1.0)	22.89[b]	<.001	lvPPA < HC, svPPA
Clinical dementia rating sum of boxes[d]							
	1.3 (2.0)	1.8 (2.4)	3.0 (3.8)	.0 (.5)	42.18[b]	<.001	HC < nfvPPA < svPPA HC < lvPPA
Sydney language battery[e]							
Naming (/30)	23.0 (5.5)	18.5 (7.8)	5.0 (4.5)	27.0 (2.0)	92.49[b]	<.001	svPPA < lvPPA, nfvPPA < HC
Semantic association (/30)	28.0 (4.0)	26.5 (2.0)	20.0 (10.3)	29.0 (1.0)	58.00[b]	<.001	svPPA < lvPPA < HC svPPA < nfvPPA
Comprehension (/30)	29.0 (2.0)	27.0 (3.8)	20.0 (13.5)	30.0 (1.0)	57.48[b]	<.001	svPPA < lvPPA < HC svPPA < nfvPPA
Repetition (/30)	25.0 (7.0)	28.0 (3.0)	29.0 (2.0)	30.0 (.0)	57.50[b]	<.001	lvPPA, svPPA < HC nfvPPA < HC, svPPA

Note. Medians (IQR); N/A = not applicable.
[a] Chi-square test.
[b] Kruskal Wallis test, posthoc Dunn's Test with Bonferroni correction.
[c] Total score <84/100 indicates impairment, score ≥88/100 indicates normal range (So et al., 2018).
[d] Sum of Boxes score for non-language sections of the test.
[e] Savage et al. (2013).

2.1. *Participants*

Data from 90 participants with PPA, assessed by FRONTIER, the Frontotemporal Dementia Research Group in Sydney Australia between 2008 and 2023, were included in the study. All participants received an expert consensus diagnosis of PPA according to current consensus criteria (Gorno-Tempini et al., 2011) based on neurological and neuropsychological clinical examination. Of the included participants, 27 were diagnosed with nfvPPA, 33 with left hemisphere svPPA, and 30 with lvPPA. Participants with PPA met inclusion criteria if they had: 1) received a clinical diagnosis of PPA, 2) had self/family-report of premorbid fluency in English, 3) completed the 'Cookie Theft' spoken picture description task at least once, but possibly more than once during annual follow-up assessments, each with an audio-recording, and 4) undergone a T1-weighted MRI scan within 3 months of each picture description sample. Participants were excluded based on: 1) evidence of severely reduced verbal output or mutism, 2) past history of stroke, epilepsy, alcoholism, and significant traumatic brain imagery, 3) MRI scans affected by movement or other abnormality, 4) a moderate to severe Clinical Dementia Rating (CDR) score (Hughes et al., 1982), 5) unintelligible speech, such that a discourse sample could not be reliably transcribed, 6) presence of other neurological disorders, such as supranuclear palsy or corticobasal syndrome, 7) audio-recording of the 'Cookie Theft' picture description language sample missing or corrupted, 8) record of developmental language or learning disorder, and (9) <10 years of formal education. An additional 36 age-matched healthy controls (HC) were recruited from the FRONTIER healthy control registry. Healthy controls were subject to the same inclusion and exclusion criteria, with the exception that they must not have a diagnosis of PPA and must have achieved a total score within the normal range of ≥88/100 on the Addenbrooke's Cognitive Examination (ACE) (Mioshi et al., 2006; So et al., 2018).

All participants had undergone comprehensive clinical, neurological and neuropsychological assessment, along with structural brain MRI, at the first timepoint when a Cookie Theft picture description sample was available (see Table 2; hereto referred to as baseline) and at each subsequent timepoint. General cognitive functioning (i.e., attention, memory, verbal fluency, language, and visuospatial skills) was assessed using either the ACE-R (Mioshi et al., 2006) or ACE-III (Hsieh et al., 2013). ACE-R scores were converted to ACE-III scores prior to analysis (So et al., 2018). The CDR (Hughes et al., 1982) measured functional capacity in daily life. To support differential diagnosis of PPA variants, the Sydney Language Battery (SYDBAT) (Savage et al., 2013) was used to assess single-word naming, comprehension, repetition and semantic associations. Note that diagnosis is, at times, uncertain at first

assessment but confirmed at later timepoints. The final consensus diagnosis was used to categorise participants. While some participants contributed only one timepoint for a discourse sample, they attended FRONTIER more than once, so this method of diagnosis still applied.

Apraxia of speech is commonly reported in individuals with nfvPPA (Duffy et al., 2021). Its presence was objectively measured at baseline using the pairwise variability index (PVI) and polysyllabic word duration (Ballard et al., 2014; Cordella et al., 2017; Landin-Romero et al., 2021; Vergis et al., 2014), to determine the need to factor AOS presence into analyses of language metrics. PVI quantifies the relative duration of vowels in the first two syllables of 3-syllables words with weak-strong stress (e.g., banana, tomato), with a median PVI value < 100 being associated with presence of apraxia of speech. As average word duration (i.e., articulation rate) for these polysyllabic words has also been explored as an index of progressive apraxia of speech (Cordella et al., 2017), it is reported for transparency.

2.2. Assessment of discourse

The 'Cookie Theft' picture description task was used to elicit samples of monologue discourse (Goodglass et al., 2001). Participants were instructed to describe a black and white line drawing of a domestic scene. Only general prompting was provided, such as "What else?" and "Anything else you can describe?". No duration restrictions were enforced, ending only when the participant confirmed that they had nothing else to describe.

Samples were audio-recorded using a Marantz PMD660 or Zoom HN8 digital recorder in.wav format at 44 kHz sampling rate, the microphone placed within 30 cm of the mouth. Samples were transcribed by qualified speech pathologists experienced in Computerised Language ANalysis (CLAN) conventions and blinded to diagnosis.[1] The CHAT software program was used for transcription, and the CLAN program was used to analyse the transcriptions (MacWhinney, 2000). CLAN's linguistic coding system was used to mark specific word- and sentence-level grammatical errors (e.g., omission of '-ed' on past tense verbs). The MOR command was used to automatically tag and categorise all morphemes (e.g., nouns, verbs, pronouns, plural "-s", past tense "-ed"). Two CLAN automated evaluation routines were then run on all transcribed and coded samples: EVAL and the Northwestern Narrative Language Analysis (C-NNLA). Ten measures, previously reported to be sensitive to discourse changes in PPA, were extracted (see in Table 1).

Between 10% and 20% of samples per group were randomly selected and transcribed by a second rater, blind to the first rater's transcription, to establish reliability on language measures. High inter-rater reliability was achieved with intraclass correlation values ranging from .80 for ratio of embedded clauses:sentences (95% CI .64 to .89) to .96 for words per minute (95% CI .92 to .98). Discrepancies were discussed and resolved through consensus.

[1] Transcripts can be accessed for research purposes by contacting FRONTIER through the website www.frontier.org and upon provision of ethical approvals from the researcher's academic institution.

2.3. Brain imaging acquisition and preprocessing

Whole-brain MRI scans were acquired from participants annually at either one, two or three timepoints, using a Philips Achieva (125/188 scans, 66.5%) or GE Discovery 3.0T MRI scanner (63/188 scans, 33.5%) with standard 8-channel head coil. Two whole-brain 3D T1-weighted structural MRI sequences were acquired during each visit with the following parameters: 200 slices, slice thickness 1 mm, in-plane resolution 1×1 mm, in-plane matrix 256×256, flip angle $\alpha = 8°$, and echo time/repetition time of 2.6/5.8 msec on the Philips and 2.4/6.6 on the GE; equivalent acquisition parameters after comprehensive harmonisation of sequences for both machines. After visual comparison for quality and motion artifact, the sequence with the best quality selected for further processing.

Scans were processed using the FreeSurfer v6.0 imaging analysis package (http://surfer.nmr.mgh.harvard.edu/). The "recon-all" pipeline in FreeSurfer was utilized for cortical reconstruction and volumetric segmentation of the T1-weighted images (Fischl & Dale, 2000). For each participant in the study, a longitudinal, unbiased within-subject template was created using robust, reverse consistent registration between time points (Reuter et al., 2010). This template was used to enhance reliability (Reuter & Fischl, 2011), allowing for the parcellation of gyral and sulcal units (Desikan et al., 2006; Fischl et al., 2004) to generate maps of curvature and sulcal depth. Cortical thickness was measured as the shortest distance between the grey/CSF and grey/white boundaries at each vertex on the brain's surface (Fischl & Dale, 2000). To improve the signal-to-noise ratio and reduce the impact of imperfect cortical alignment, cortical thickness measurements were smoothed using a 20 mm full-width at half-height Gaussian kernel in all analyses (Lerch & Evans, 2005). All scans were visually inspected for segmentation and parcellation errors; none required manual correction or exclusion.

2.4. Statistical analyses

Language data were analysed using SPSS Statistics, version 28.0 (IBM). Initially, variables were checked for normality, and either parametric or nonparametric analysis of variance was employed to identify baseline group differences for each dependent variable. Posthoc pairwise comparisons were conducted using Sidak or Independent samples tests, respectively. Next, we selected only those language variables for which the nfvPPA group's baseline performance was significantly different to the other PPA groups. These variables were then analysed with Linear Mixed Effects (LME) modelling to explore longitudinal effects (see Supplementary Materials for syntax). LME modelling accommodates missing data, variations in event timing across participants, and single time points, ensuring optimal use of all available data. The model included fixed effects for diagnostic group, time, and their interaction term. The random effect of participants accounted for baseline variability, assuming independence among participants and applying a random intercept model. Residual errors and random intercepts for each participant at baseline were assumed to be normally distributed. For each language measure showing a significant effect, posthoc pairwise comparisons were performed. As healthy controls volunteering at

FRONTIER are not routinely tested over multiple years, this group was not included in the longitudinal modelling. Variables of interest from these analyses were then selected to explore associations with cortical atrophy at baseline and over time.

Whole-brain differences in cortical thickness for each patient group (nfvPPA, lvPPA, and svPPA) at baseline were initially examined using vertex-wise general linear models (GLM). These models included cortical thickness as the dependent variable and group (patient or healthy control) as the independent variable. Analyses were run using standard preprocessing in FreeSurfer, which accounts for brain volume differences when comparing cortical thickness between groups. Vertex-wise GLMs were also employed to investigate brain-behaviour associations at baseline within each patient group, with cortical thickness as the dependent variable and each language measure as a covariate in separate models. Healthy control data were incorporated into each baseline association model to increase the number and distribution of data points, thereby enhancing the power to detect associations.

Annualized rates of change in cortical thickness within each patient group were examined using vertex-wise comparisons with the Spatiotemporal LME Matlab tools (Bernal-Rusiel, Greve, et al., 2013; Bernal-Rusiel, Reuter, et al., 2013). Spatiotemporal models of cortical atrophy were fitted with the following fixed effects: (i) time from baseline MRI acquisition, (ii) language measure, and (iii) the interaction between time and language measure. The intercept was included as a random effect. Null hypotheses of no effect of time on cortical thickness and no time by language measure interaction (i.e., no change in brain-behaviour association over time) were tested.

The statistical threshold for all neuroimaging analyses was set at $p < .005$ uncorrected, with a conservative cluster extent threshold of $k > 100$ mm^2 to balance the risks of Type I and Type II errors. This approach circumvented software limitations that necessitate a false discovery rate correction for multiple comparisons per hemisphere. Such corrections can complicate the interpretation of whole-brain cortical changes across different group comparisons, due to differing interhemispheric thresholds (Landin-Romero & Piguet, 2017).

3. Results

As shown in Table 2, the PPA groups were matched at baseline for sex ratio, age at first visit, years of education, and patient/family-reported disease duration. The groups differed in proportion reporting a non-English first language, due to 5/33 participants in the svPPA group but one or none in the other groups. Scores on the ACE, SYDBAT, and CDR tests showed group differences consistent with PPA diagnostic profiles (see Table 1). Mean PVI value and word duration for the nfvPPA group were in the normal range (PVI: M = 109.34, SD = 26.82; word duration: M = 718.03 msec [4.17 syllables/sec], SD = 186.82); with 7/27 of nfvPPA participants having a PVI value < 100 indicating concomitant apraxia of speech.

Of the 90 cases with PPA, 51/90 (56.7%) were available for testing at time point 2 and 25/51 (49.0%; 27.8% of baseline sample) at time point 3 (see Table 3). The 36 control participants were tested at baseline only. Thus, a total of 202 discourse language assessments were analysed and 188 MRI scans were available for analysis (93.1% of assessment sessions). Demographics, neuropsychological test data, and Cookie Theft language data for individual participants across all timepoints are provided in Supplementary Materials).

Table 3 – Number of participants with discourse language assessments (MRI scans) for the nonfluent (nfvPPA), logopenic (lvPPA), and semantic (svPPA) variants of primary progressive aphasia and healthy controls (HC) across the three timepoints.

Timepoint	nfvPPA	lvPPA	svPPA	HC	Total
1 - baseline	27 (25)	30 (28)	33 (30)	36 (36)	126 (119)
2	12 (10)	20 (18)	19 (18)	Nil	51 (46)
3	5 (3)	10 (10)	10 (10)	Nil	25 (22)
Total	44 (38)	60 (56)	62 (58)	36 (36)	202 (188)

3.1. *Baseline analyses*

3.1.1. *Discourse measures*

Baseline differences between PPA groups and HC for each of the 10 language measures are summarised in Table 4. The nfvPPA group were differentiated from the other three groups, with large effect sizes, on seven of the 10 measures (see Fig. 1, time point 0 for comparison across PPA groups). Type:token ratio, retracing, and repetition were non-discriminatory. The nfvPPA group were characterized by the lowest idea density, highest proportion of nouns to verbs and open class to closed class words; lowest mean length of utterance, highest percent of sentences with flawed syntax, fewest embedded clauses; and fewest words produced per minute. For the nfvPPA group, word duration and PVI were explored to determine whether presence of apraxia of speech may be associated with language performance. While these two measures were significantly correlated with each other ($r = -.60$, $p = .001$), they were not correlated with any language or fluency variable (all p values > .1) and, therefore, were not explored further (see also Wilson, Henry, et al., 2010). The lvPPA group was distinguished from the svPPA group and HC by two fluency measures: they produced fewer words per minute and had more word/phrase repetitions. As a group, they also exhibited shorter utterance lengths compared to the HC group. However, they did not significantly differ from the svPPA group on any lexical measures used in this discourse-level analysis, despite the SYDBAT revealing clear differences in single-word naming, comprehension, and semantic association.

3.1.2. *Neuroimaging measures*

Baseline neuroimaging analyses showed the expected distributions of reduced cortical thickness consistent with PPA diagnosis (Gorno-Tempini et al., 2011) (Fig. 2, left panel). Significant clusters in nfvPPA were in left pars opercularis (BA44), caudal middle frontal, and medial superior frontal regions. In lvPPA, clusters were confined to the left hemisphere, focused on the ventral pathway from the temporoparietal junction through to anterior temporal pole. Additional small clusters were noted in the vicinity of left BA44/left pars triangularis (BA45), medial superior frontal, and posterior cingulate cortex. In svPPA, clusters were found in the left superior, middle and inferior temporal cortices, left insula cortex, and right anterior temporal pole.

Table 4 – Baseline differences between groups for each language measure.

Measures	nfvPPA n = 27	lvPPA n = 30	svPPA n = 33	HC n = 36	η2[a]	F statistic	*p*-value	Sidak post-hoc test
Word density/diversity								
Propositional idea density	.35 (.05)	.43 (.05)	.42 (.05)	.42 (.04)	.279	15.71	**<.001**	nfvPPA < lvPPA, svPPA, HC
Type:Token ratio	.65 (.12)	.60 (.10)	.60 (.12)	.58 (.10)	.047	1.99	.118	–
Noun:Verb ratio	1.65 (.43)	1.22 (.54)	1.16 (.46)	1.40 (.41)	.140	6.61	**<.001**	nfvPPA > lvPPA, svPPA
Open:Closed class ratio	1.18 (.47)	.89 (.15)	.91 (.22)	.97 (.20)	.138	6.47	**<.001**	nfvPPA > lvPPA, svPPA, HC
Sentence structure								
Mean length of utterance in morphemes	8.11 (3.19)	11.04 (4.53)	11.54 (4.65)	13.96 (4.49)	.191	9.59	**<.001**	nfvPPA < svPPA, HC lvPPA < HC
% sentences with flawed syntax	35.31 (28.20)	19.99 (14.07)	14.20 (15.76)	8.30 (9.25)	.220	11.39	**<.001**	nfvPPA > lvPPA, svPPA, HC
Embedded clauses: Sentence	.29 (.35)	.61 (.44)	.672 (.38)	.84 (.40)	.199	10.03	**<.001**	nfvPPA < lvPPA, svPPA, HC
Fluency								
Words per minute	49.46 (22.93)	86.97 (31.68)	113.00 (37.60)	130.53 (28.68)	.495	39.86	**<.001**	nfvPPA < lvPPA < svPPA, HC
Retracing	2.11 (2.03)	2.47 (2.64)	1.82 (2.08)	1.47 (1.92)	.029	1.23	.300	–
Repetition	1.78 (2.21)	3.03 (3.43)	1.36 (1.88)	.67 (1.10)	.133	6.22	**<.001**	lvPPA > svPPA, HC

Note. Data shown are mean and standard deviation.
[a] Cohen's effect size; values above .01 are small, above .06 medium, and above .14 large (Cohen, 1988).

3.1.3. *Relationships between discourse and neuroimaging measures*

Associations between regions of cortical thinning and the four language measures of interest were explored within each PPA group, at baseline and longitudinally. Associations for idea density are shown in Fig. 3, mean length of utterance in Fig. 4, embedded clauses:sentences in Fig. 5, and percentage of flawed sentences in Fig. 6.

At baseline, for the nfvPPA group, reduced cortical thickness in the left medial superior frontal cortex was associated with reduced idea density, utterance length, ratio of embedded clauses: sentences, and with higher percentage of flawed sentences. Higher percentage of flawed sentences was also associated with reduced cortical thickness in right medial superior frontal cortex and posterior cingulate cortex.

In lvPPA, reduced utterance length was associated with clusters of reduced cortical thickness in left temporoparietal junction, inferior frontal (BA44/45) cortex, and left medial posterior cingulate, as well as a small cluster on the back of the right superior temporal cortex on the lateral surface. Higher percentage of flawed sentences was also linked to reduced cortical thickness in left temporoparietal junction, as well as damage in middle-inferior temporal cortices, medially in anterior and posterior cingulate cortices, and in superior frontal gyrus. Ratio of idea density and embedded clauses: sentence were not associated with any regions of atrophy in this group and no substantive associations were found with right hemisphere regions.

In svPPA, reduced utterance length showed associations with reduced cortical thickness in the left hemisphere only with scattered clusters in inferior and middle temporal cortices, anterior inferior frontal cortex, and medially in the middle/posterior cingulate cortex. No other measures showed associations with regions of atrophy for this group.

3.2. *Longitudinal analyses*

3.2.1. *Discourse measures*

In longitudinal analyses comparing the three PPA groups, all variables showed a significant main effect of group. Mean length of utterance and words per minute exhibited a main effect of time (Table 5, Fig. 1) and only mean length of utterance demonstrated a significant group-by-time interaction ($F = 3.89$, $p = .024$) (Fig. 1d). Utterance length remained stable over time in svPPA ($p = .813$) but declined in nfvPPA and lvPPA ($p = .009$ and $p = .003$, respectively). NfvPPA showed significantly lower utterance length at baseline and the second time point compared to lvPPA and svPPA. LvPPA was no longer differentiated from nfvPPA at the second follow up, and both groups had significantly lower utterance lengths compared to svPPA. To understand the factors influencing utterance length, we examined the effect of time within each PPA group including a broader set of language variables (Table 6, Fig. 1). In nfvPPA, declining utterance length was accompanied by declining sentence complexity (i.e., fewer embedded clauses in sentences; $p = .026$). In lvPPA, it was associated with an increasing percentage of flawed sentences ($p = .045$). Words uttered per minute also deteriorated over time across all three PPA groups. The only other significant change over time was observed in svPPA, which unexpectedly showed increasing idea density. Based on these findings, we selected idea density, mean length of utterance, embedded clauses per sentence, and percentage of flawed sentences for further analysis exploring brain-behaviour associations for each of the three variants.

3.2.2. *Neuroimaging measures*

Longitudinal changes are shown in Fig. 2 (right panel) (see Supplementary Materials for the extracted cortical thickness data used in analyses). The nfvPPA group showed

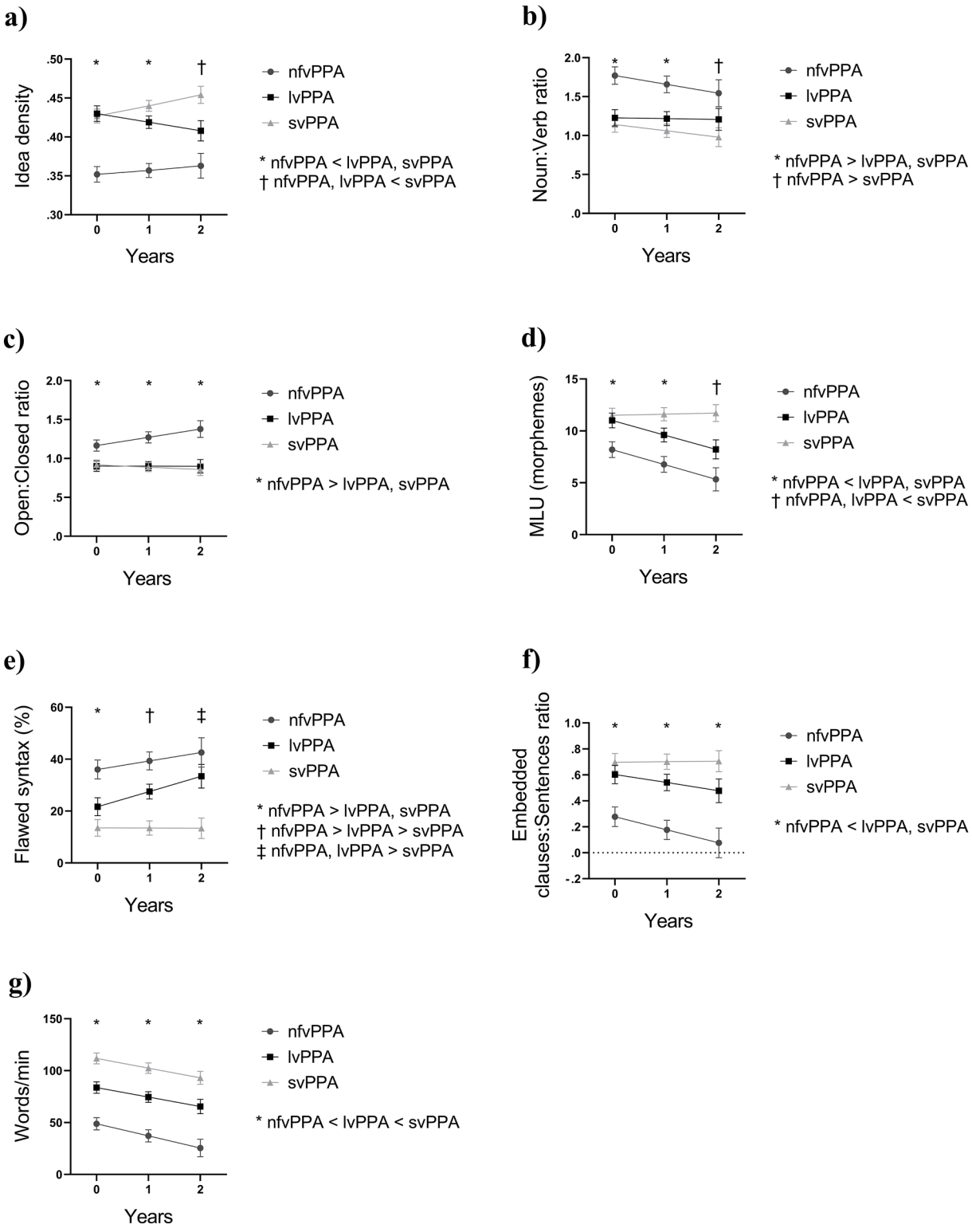

Fig. 1 – Measures differentiating the nonfluent variant of primary progressive aphasia (nfvPPA) from the logopenic (lvPPA) and semantic variants (svPPA) at baseline, modeling the trajectory over the three timepoints. Measures shown are a) propositional idea density, b) noun:verb ratio, c) open:closed class word ratio, d) mean length of utterance (MLU) in morphemes, e) percentage of sentences with flawed syntax, f) embedded clauses: sentences ratio, and g) fluency, in words per minute. The key to significant effects is shown on each graph.

further decline in brain regions identified at baseline, including left posterior inferior frontal (BA44) and caudal middle frontal and peri-insular regions. Over time, cortical thinning emerged in left middle inferior temporal and

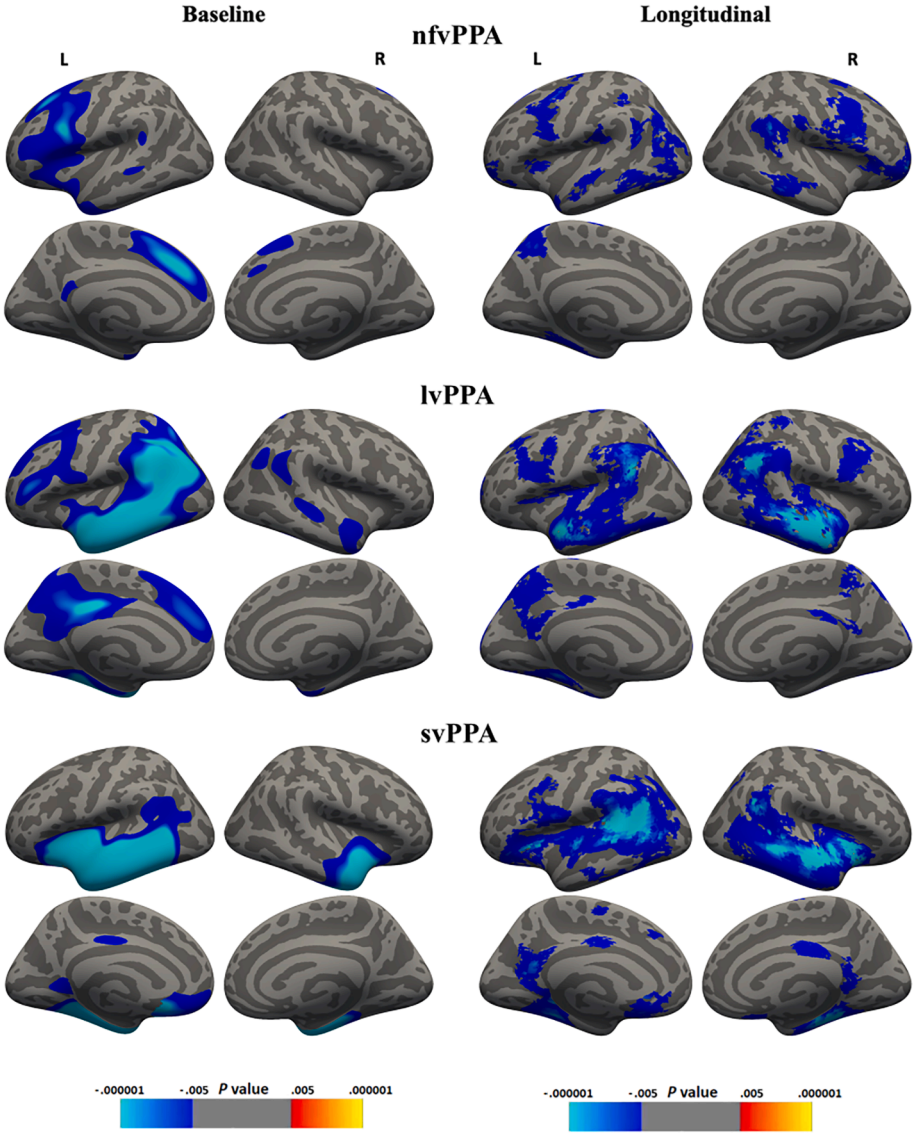

Fig. 2 – Baseline (left) and longitudinal (right) patterns of cortical thinning in nfvPPA (top), lvPPA (middle) and svPPA (bottom) relative to healthy controls (HC). Left hemisphere on the left and medial surface on bottom for each PPA variant, smoothed at 20 FWHM, showing clusters with $p < .005$ (uncorrected for multiple comparisons) and size >100 mm^2.

posterior parietal regions, as well as mirroring of left hemisphere patterns of atrophy on the right, affecting motor cortices, temporoparietal junction, and middle-inferior temporal areas. In contrast, medial cortices appeared relatively stable. The lvPPA group showed ongoing decline affecting anterior left temporal lobe, temporoparietal junction and, to a lesser degree, decline in left middle frontal and pars opercularis areas. Motor regions remained relatively stable. Medially, ongoing atrophy was seen in the precuneus and left posterior cingulate cortex with new extension into the right hemisphere that mirrored baseline left-sided atrophy. The svPPA group showed decline along an anterior-posterior gradient from left anterior and middle temporal regions toward temporoparietal junction, and superiorly into insular and inferior frontal cortices. Medially on the left, orbitofrontal atrophy was evident and progression posteriorly toward fusiform gyrus and precuneus. There was mirroring of baseline atrophy into the right hemisphere with decline in the entire temporal region, including medially in entorhinal and posterior cingulate cortices.

3.2.3. *Relationships between discourse and neuroimaging measures*

Longitudinally, associations between declining language skills and regions of cortical thinning varied across PPA variants. In nfvPPA, declining utterance length was not associated with a specific neurological region. However, a decreasing ratio of embedded clauses:sentences was correlated with deterioration in the left caudal middle frontal cortex. The lvPPA group showed a more complex picture. Declining utterance length was associated with deterioration in the right inferior-anterior temporal cortex, while increased percentage of sentences with flawed syntax was associated with atrophy in the left supramarginal gyrus, a small cluster in the middle temporal gyrus, and right anterior temporal and posterior middle temporal cortices. For the svPPA group, idea density was the only

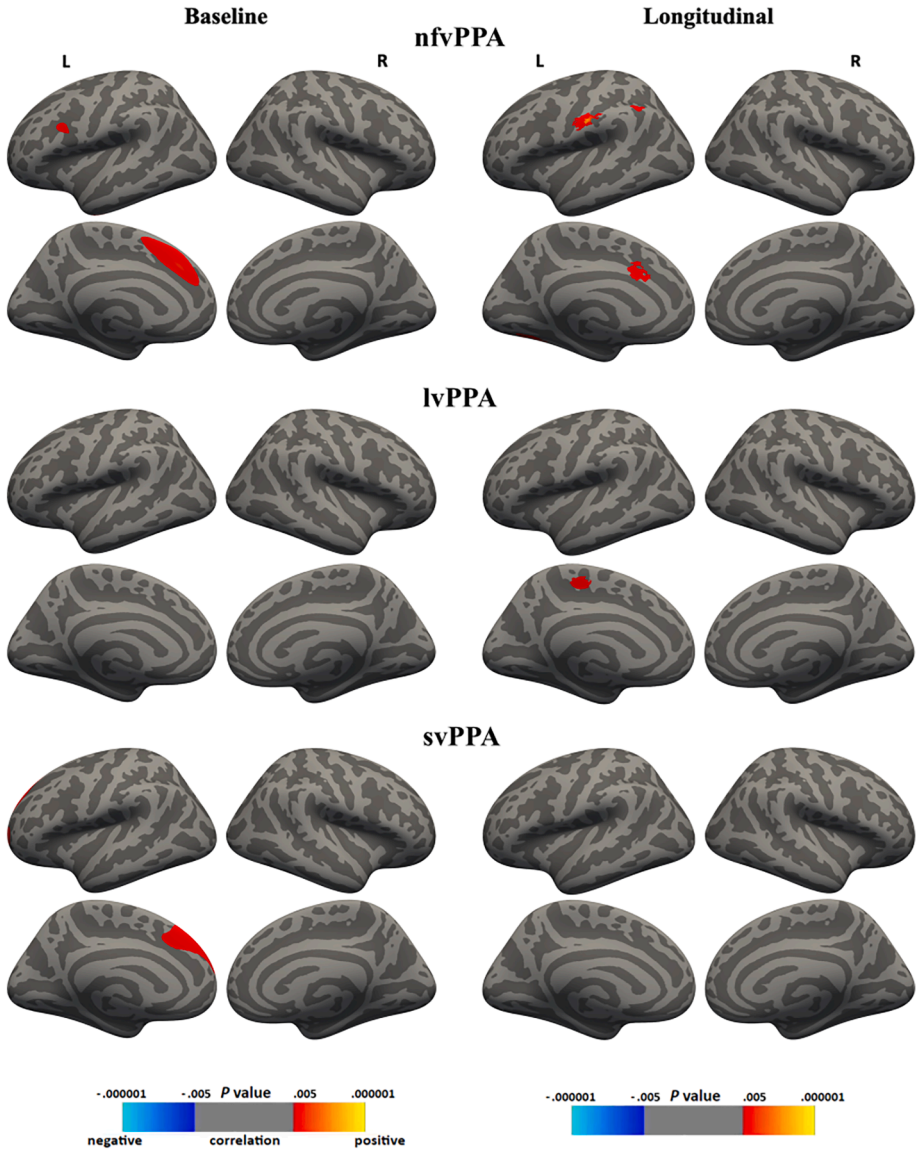

Fig. 3 – Baseline (left) and longitudinal (right) associations between idea density and cortical thinning in nfvPPA (top), lvPPA (middle) and svPPA (bottom). Left hemisphere on the left and medial surface on bottom for each PPA variant, smoothed at 20 FWHM, showing clusters with $p < .005$ (uncorrected for multiple comparisons) and size >100 mm^2.

language measure declining over time, yet no reliable associations with regions of cortical thinning over time were detected.

4. Discussion

Primary progressive aphasia (PPA) begins as a predominantly language-based impairment but, over time, other cognitive changes develop across its three variants, making it a powerful model for exploring the interactions between cognitive and linguistic processes employed in discourse. This study is the largest to date to explore longitudinal changes in discourse-level lexical, morphosyntactic, and fluency measures, and associated changes in cortical thickness, across the three variants of primary progressive aphasia (PPA). These selected metrics offer sensitivity to cognitive-linguistic decline even when surface-level discourse overall appears relatively preserved. Robust group differences in language skills were observed at baseline and over time, along with different patterns of association between language changes and grey-matter degeneration. All groups showed shortening utterance lengths over time, consistent with increasing difficulty generating spoken language. However, in nfvPPA, this was alongside declining sentence complexity that was associated with reduced cortical thickness outside the traditional perisylvian language network, in frontal regions engaged by tasks of higher cognitive attention and error monitoring. In lvPPA, it was linked with increasing grammatical errors in sentences and atrophy was observed to extend along both ventral and dorsal pathways. This implied worsening of phonological processing and working memory skills and emergence of grammatical and semantic processing deficits. In both nfvPPA and lvPPA, cortical regions involved in

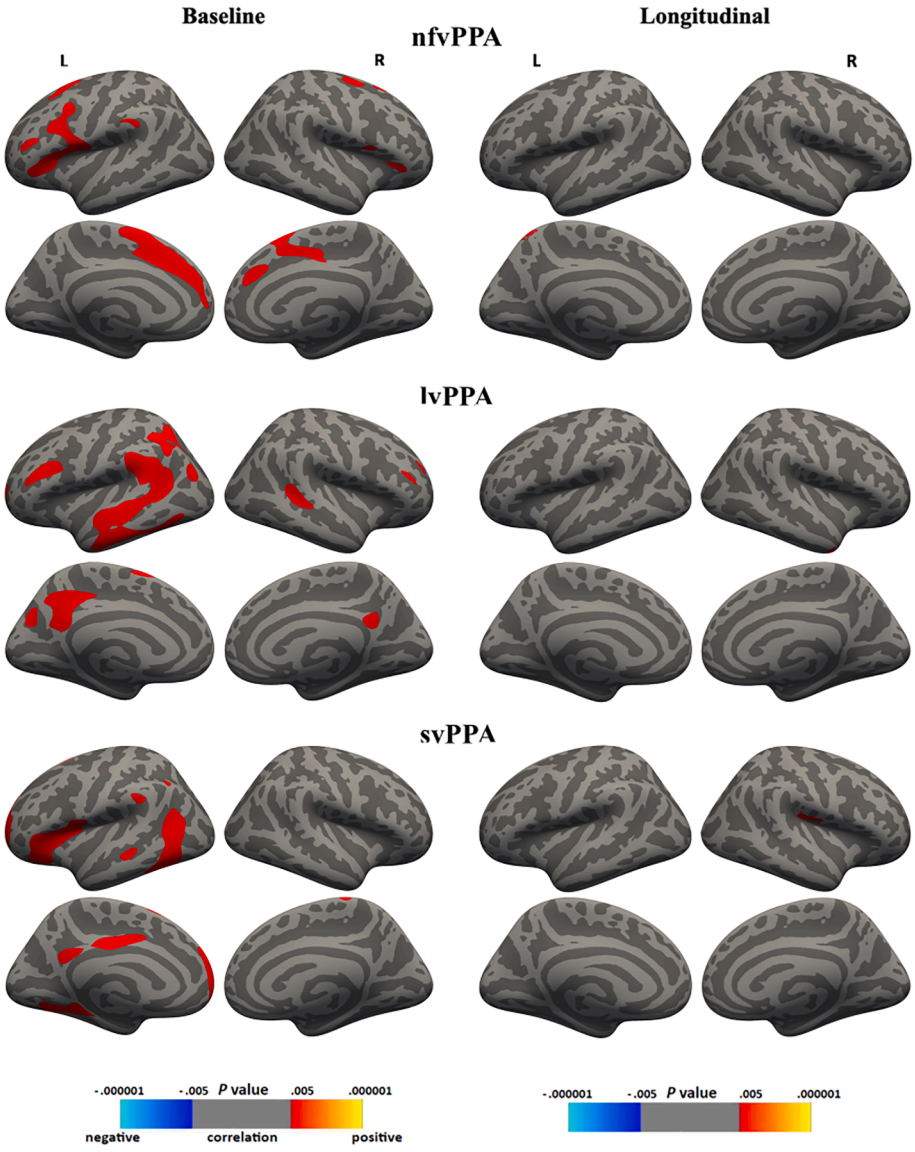

Fig. 4 – Baseline (left) and longitudinal (right) associations between mean length of utterance in morphemes (MLU) and cortical thinning in nfvPPA (top), lvPPA (middle) and svPPA (bottom). Left hemisphere on the left and medial surface on bottom for each PPA variant, smoothed at 20 FWHM, showing clusters with $p < .005$ (uncorrected for multiple comparisons) and size >100 mm^2.

cognitive memory, attentional, and/or executive functioning systems were associated with ability to produce microstructural behaviours at the discourse level. No associations were found for the svPPA group between the microstructural language measures used here and regions of cortical thinning over time, with further research needed to understand the mechanism underlying the discourse changes in this group.

In the following sections, we will discuss the findings by PPA variant. First, we will interpret the findings relative to previous studies of PPA to explore consistencies and departures from these studies given the larger sample size studied here and the contribution of longitudinal data. Second, we will consider how these findings can inform our understanding of production of microstructural language in discourse more generally.

4.1. *Discourse simplification and economy of effort in nfvPPA*

4.1.1. *Baseline discourse and neuroimaging*

As predicted, individuals with nfvPPA exhibited higher noun:verb and open:closed class word ratios, and lower grammatical accuracy and complexity relative to those with lvPPA, svPPA and healthy control groups at baseline. Low proportional idea density supported an overall reduction in content words, that some have attributed to production of fewer verbs and reduced utterance length in nfvPPA (Themistocleous et al., 2021; Faroqi-Shah et al., 2020; Fraser et al., 2014). In the present study, the nfvPPA group showed the most severely reduced utterance length and sentence complexity, and the highest rate of grammatical errors. Unsurprisingly, their fluency was impacted, with

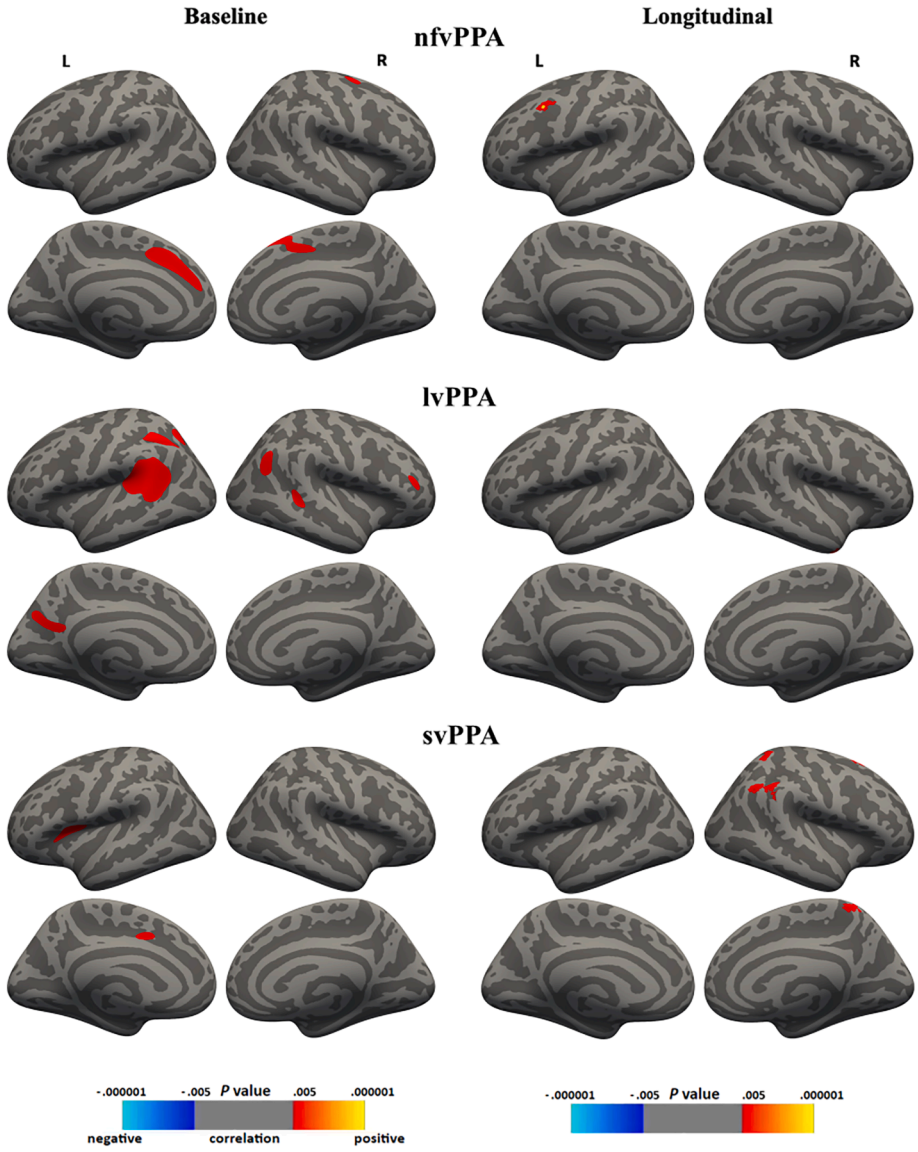

Fig. 5 – Baseline (left) and longitudinal (right) associations between embedded clauses:sentences (i.e., sentence complexity) and cortical thinning in nfvPPA (top), lvPPA (middle) and svPPA (bottom). Left hemisphere on the left and medial surface on bottom for each PPA variant, smoothed at 20 FWHM, showing clusters with $p < .005$ (uncorrected for multiple comparisons) and size >100 mm^2.

a speaking rate about one third that of healthy controls. Together, these findings suggest a breakdown in verb retrieval, in turn reducing utterance length and complexity when attempting to produce coherent connected speech. Ultimately, this cluster of symptoms at baseline, supports use of the term "agrammatism", which traditionally has been associated with damage to the left inferior posterior frontal lobe (BA44/45). Since this term can oversimplify the complex nature of language impairment in PPA and present itself as a single deficit, we refer to it here as a cluster of symptoms such as reduced verb retrieval and sentence complexity that impact discourse. Further, there are conflicting reports that other word and sentence metrics such as noun:verb ratio and sentence well-formedness or complexity may not be discriminatory (Ash et al., 2013, 2019; Wilson, Henry, et al., 2010). These, however, may relate more to sample size differences across studies; with similar findings to ours being reported by Faroqi-Shah et al. (2020).

Lower idea density, utterance length, number of embedded clauses, and higher rate of flawed sentences in the nfvPPA group were associated with decreased cortical thickness in the left frontal lobe at baseline. However, the specific region implicated was medial superior frontal cortex rather than the classic inferior posterior frontal regions linked to agrammatism. The right medial superior frontal cortex and posterior cingulate cortex were also associated with higher rates of flawed sentences. The medial superior frontal cortex includes the supplementary motor and pre-supplementary motor areas, which are linked with movement skills as well as cognitive control and decision making, performance monitoring, and syntactic processing (Europa

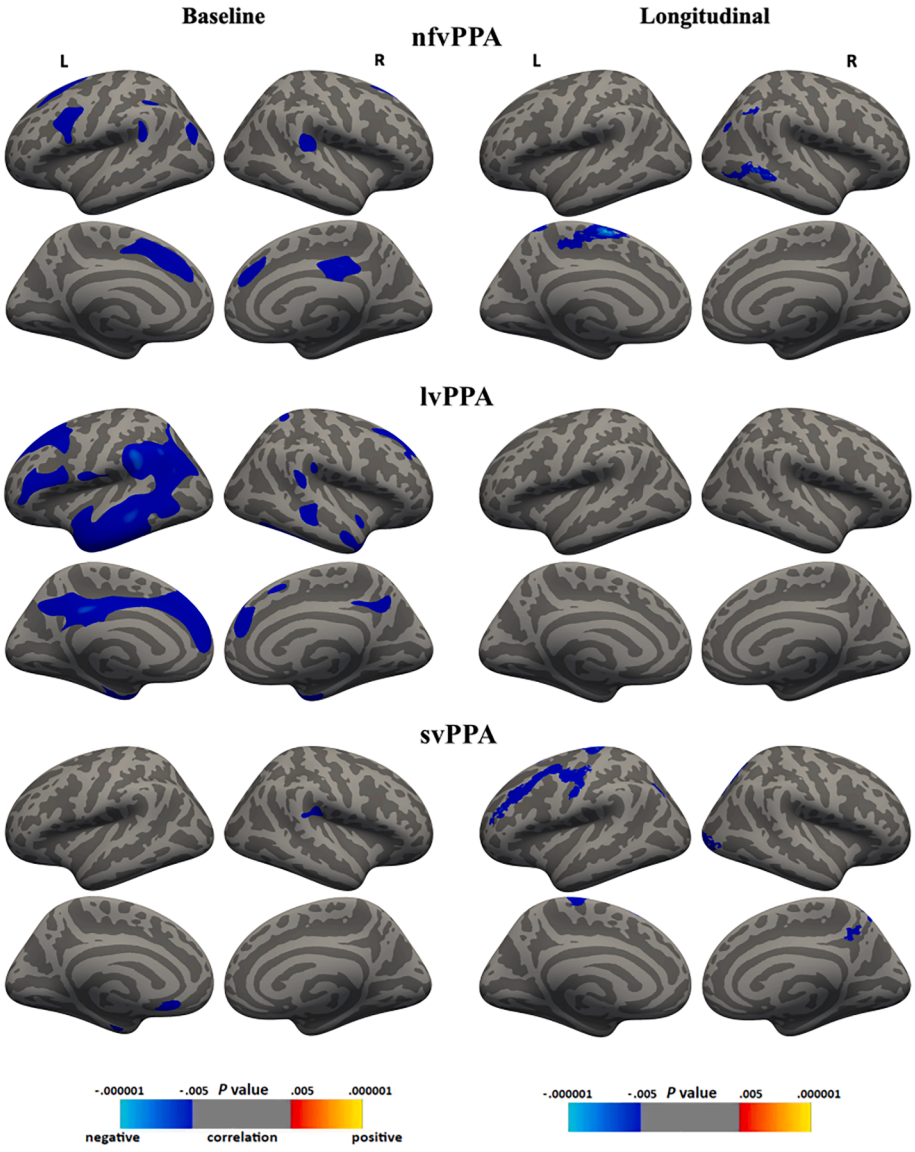

Fig. 6 – Baseline (left) and longitudinal (right) associations between percentage of flawed sentences and cortical thinning in nfvPPA (top), lvPPA (middle) and svPPA (bottom). Left hemisphere on the left and medial surface on bottom for each PPA variant, smoothed at 20 FWHM, showing clusters with $p < .005$ (uncorrected for multiple comparisons) and size >100 mm^2.

Table 5 – Longitudinal LME models comparing language measures between groups across timepoints, for measures that differentiated the nonfluent variant of primary progressive aphasia (nfvPPA) from other PPA variants at baseline.

Variable	Group Main Effect		Time Main Effect		Group × Time Interaction	
	F	*p*-value	F	*p*-value	F	*p*-value
Lexical						
Propositional idea density	19.82	**<.001**	.31	.579	2.67	.073
Noun:Verb ratio	9.87	**<.001**	1.85	.177	.35	.709
Open: Closed class word ratio	4.52	**.013**	.81	.371	1.91	.153
Morphosyntactic						
Mean length of utterance in morphemes	5.78	**.004**	10.23	**.002**	3.89	**.024**
% sentences with flawed syntax	10.70	**<.001**	3.38	.069	1.34	.265
Embedded clauses: Sentence	9.27	**<.001**	2.88	.093	1.08	.344
Fluency						
Words per minute	32.06	**<.001**	23.48	**<.001**	.12	.886

Note. Values in bold font are statistically significant.

Table 6 – Longitudinal LME models showing the effect of time for each language measure within nonfluent (nfvPPA), logopenic (lvPPA), and semantic (svPPA) variants of primary progressive aphasia.

Measure	nfvPPA			lvPPA			svPPA		
	t	*p*-value	Difference in EMM[a]	t	*p*-value	Difference in EMM[a]	t	*p*-value	Difference in EMM[a]
Lexical									
Propositional idea density	.54	.591	.005	−1.41	.165	−.011	2.26	.029	.013
Noun: Verb ratio	−.88	.387	−.114	.09	.928	−.01	−1.49	.144	−.081
Open: Closed class word ratio	1.10	.281	.106	−.07	.281	.00	−1.36	.182	−.03
Morphosyntactic									
Mean length of utterance in morphemes	−2.83	.009	−1.437	−3.23	.003	−1.393	.24	.813	.111
% sentences with flawed syntax	.70	.492	3.288	2.05	.045	5.867	−.05	.957	−.086
Embedded clauses: Sentence	−2.38	.026	−.101	−.95	.350	−.062	−.02	.983	.004
Fluency									
Words per minute	−4.99	<.001	−11.638	−2.56	.015	−9.155	−2.70	.010	−9.401

Note. Highlighted values represent statistical significance at $p < .05$.
[a] EMM = Estimated Marginal Means, difference in EMM is the amount that the variable increases/decreases for each PPA group per year per the model.

et al., 2019; Newman et al., 2001; Ni et al., 2000; Ridderinkhof et al., 2004; Zhang et al., 2011). Interestingly, this region has been implicated in progressive grammatical impairment as reported by multiple studies (Europa et al., 2019; Newman et al., 2001; Shetreet & Friedmann, 2014; Wilson, Dronkers, et al., 2010). For example, Wilson, Dronkers, et al. (2010) reported that well-formed sentences and use of embedded clauses in nfvPPA were associated with the more traditional left posterior inferior frontal regions but identified additional involvement of supplementary motor area. Similarly, Europa et al. (2019) and Newman et al. (2001) reported activation of the medial superior frontal gyrus involving syntactic complexity and violations, supporting a role in procedural monitoring and discourse-level integration. Further, the posterior cingulate cortex also plays a role in higher-order cognitive functions such as error monitoring and executive control (Raichle et al., 2001). These studies indicate a central role for these regions in tasks, across multiple modalities, that engage higher cognitive attention and effort. Critically, they support a role of domain-general cognitive processes in generating the semantic, morphological and syntactic components for expository discourse. This is significant given that expository discourse is perhaps one of the least complex discourse tasks, that does not invoke the higher cognitive demand of other discourse forms that require temporal sequencing, coherence and cohesion across each story element. These baseline findings, therefore, predict a longitudinal course for nfvPPA patients that will diverge from the traditional non-progressive form of agrammatism, revealing a progressive disruption of cognitive-linguistic networks that will continually degrade discourse integrity and its microstructural components in ways not observable in stroke-related conditions.

4.1.2. *Longitudinal discourse and neuroimaging*

We predicted that the nfvPPA group would deteriorate over time on metrics associated with sentence structure such as ability to retrieve verbs (i.e., noun:verb ratio) and sentence complexity (i.e., embedded clauses:sentences). We also predicted that these language changes would be associated with increasing atrophy in the left posterior inferior frontal region and emerging in the right homologue. These hypotheses were partially supported. Over time, utterance length in nfvPPA declined at a faster rate compared with the lvPPA and svPPA groups and this was associated with reduced sentence complexity for this group. This finding of declining sentence complexity has been previously reported (Thompson et al., 1997; Thompson & Mack, 2014; Wilson, Dronkers, et al., 2010) and attributed, in part, to verb retrieval deficits (Montembeault et al., 2018; Thompson & Mack, 2014), given that complex sentences must contain more than one verb phrase. Our results suggest that declining sentence complexity may be linked to overall word retrieval deficits, given that verb retrieval did not decline more rapidly than noun retrieval in our study.

Given that declining utterance length in nfvPPA was not consistently correlated with cortical changes over time, and utterance length declined in all variants, it is a less specific and sensitive measure likely influenced by several linguistic and non-linguistic factors. In nfvPPA specifically, decreasing utterance length was correlated with decreasing sentence complexity, and the latter was associated with decline in the left caudal middle frontal region (Husa et al., 2017). This region has previously been implicated in nfvPPA for maintaining syntactic rules essential for constructing complex sentences (Kielar et al., 2011; Tetzloff et al., 2018). In healthy adults, greater grey matter volume in the left caudal middle frontal region has also been linked to the use of self-initiated elaborative encoding strategies. These are general high-effort cognitive processes recruited in generating structured discourse and personal narratives. Conversely, reduced grey matter volume in the middle frontal gyrus is, in part, correlated with apathy in the frontotemporal dementias that underlie nfvPPA (Eslinger et al., 2012; Le Heron et al., 2018; Rosen et al., 2005; Zamboni et al., 2008). These findings again suggest that simplification of language in nfvPPA may not only reflect syntactic degradation but also dampening of lexical activation; potentially associated with emerging apathy and reduced engagement of high-effort cognitive strategies that support elaboration of detail and structure needed to construct multi-sentence discourse.

Based on our findings, we suggest the term "agrammatism" from the field of stroke-related aphasia has caused confusion in the effort to understand the contributing factors in the devolution of language in the PPAs. However, these differences across populations provide the opportunity for discovery. The emergence of pathology in our nfvPPA participants is in regions of domain-specific language networks, rather than in regions following the vasculature. This results in potential differences in PPA symptoms from stroke-related agrammatism from the outset. Second, the progressive spreading nature of the disease results in different distributions of neural damage to those observed after stroke, with pathology travelling between regions of high connectivity that implicate language and non-language functions. This can complicate the symptom profile with time, as observed here for the nfvPPA group, but enable discovery of dependencies across inter-connected systems. Third, the gradual (mal)adaptation made by each individual to the changes in their language skill, over years, interact with and potentially obscure some aphasia symptoms (see Mesulam, Coventry, Bigio, et al., 2021). This adaptation must result in quite different manifestations of deficits and compensations in comparison to the adaptation/compensation that emerges from an instantaneous, catastrophic change in language ability seen post-stroke. As such, the findings presented here suggest that individuals with nfvPPA demonstrate some symptoms of classical agrammatism but, with progression, show a complex interplay between damage to key language regions and regions that support high-effort cognitive strategies necessary for discourse-level integration. Therefore, the term "agrammatism", referring to loss of grammatical knowledge or ability, is misaligned with our current understanding of the network-level nature of microstructural language decline in PPA.

4.2. *Influence of dorsal stream degeneration and working memory on discourse in lvPPA*

4.2.1. *Baseline discourse and neuroimaging*

Our second hypothesis proposed that the lvPPA group would demonstrate poorer performance on lexical-semantic measures of word density and diversity, relative to healthy controls, rather than morphosyntactic changes. At baseline, the lvPPA group showed shorter utterance length than controls. They produced fewer words per minute and higher number of word/phrase repetitions than both svPPA and controls, but sentence complexity and grammaticality did not differentiate these three groups, supporting a predominantly lexical deficit in lvPPA as well as in svPPA. The higher number of word/phrase repetitions can also be interpreted in support of this finding. We suggest that spontaneous repetitions in discourse may be potential markers of instability within the phonological loop, supported by the imaging findings below (also see Santi et al., 2024).

Baseline neuroimaging analysis of the lvPPA group showed atrophy already extending beyond the left temporoparietal junction toward regions affected in the other two variants. Clusters were identified along the ventral pathway from temporoparietal junction to anterior temporal pole, but also along the dorsal pathway into BA44/45. Despite this, the baseline language profile was still distinct from the other variants. The reduction in utterance length in the lvPPA group was significantly associated with clusters of atrophy distributed across multiple cortical regions, including left temporoparietal junction, inferior frontal (BA44/45) cortex, and left medial posterior cingulate, as well as a small cluster on the lateral surface of the right superior posterior temporal gyrus, but not the emerging atrophy in the anterior temporal pole. This implies that utterance length in this variant was affected by lexical retrieval and phonological working memory (Dial et al., 2021; Mesulam, 2013; Rohrer et al., 2010; Stark et al., 2019; Whitwell et al., 2015) as well as sentence formulation and coherence (Leech & Sharp, 2014), but not with deterioration in the semantic network. While this group were not yet differentiated from controls and the svPPA group on percentage of grammatically flawed sentences, this behaviour was associated with atrophy distributed across left temporoparietal junction, middle-inferior temporal cortices, medial anterior and posterior cingulate cortices, and superior frontal gyrus. These baseline associations imply that strain to multiple processes can manifest as sentence formulation difficulties in discourse. Presumably, disruptions in phonological working memory and inefficient phonological retrieval causing word/phrase repetitions interfere with sentence formulation and possibly error monitoring/detection.

4.2.2. *Longitudinal discourse and neuroimaging*

It was predicted that the lvPPA group would show greater decrement over time on word-level measures of lexical and semantic processing, with atrophy extending along the ventral pathway and into right temporo-parietal and temporal homologues, respectively. It was also predicted that atrophy would spread along the dorsal pathway revealing new associations between frontal atrophy and measures of verb retrieval and sentence complexity. Consistent with this, the lvPPA group's decline in utterance length was associated with increased percentage of grammatically flawed sentences and the lvPPA and svPPA groups gradually separated on the sentence-level measures of utterance length and flawed syntax. However, neither of these behaviours was associated with worsening atrophy in BA44/45, suggesting fundamental morphosyntactic skills were relatively spared. Declining utterance length was associated with deterioration in the right inferior-anterior temporal cortex, suggesting a worsening of semantic processing ability (Conca et al., 2022; Leyton et al., 2015; Rice et al., 2015). On the other hand, increasingly flawed syntax was associated with atrophy in the left supramarginal gyrus, a small cluster in the middle temporal gyrus, and right anterior temporal and posterior middle temporal cortices. As in baseline, the mechanism appears more related to declining verbal working memory ability (Deschamps et al., 2014; Oberhuber et al., 2016; Whitwell et al., 2015) with additional emerging impairment in semantic processing (Davey et al., 2016). While the cortical atrophy and brain-behaviour associations in the lvPPA group were focused largely within the language network, findings again support the notion that formulation of grammatical structures in sentences and discourse is subserved by a distributed network, with breakdowns arising at multiple potential sites.

4.3. *Compensatory discourse mechanisms in svPPA*

4.3.1. *Baseline discourse and neuroimaging*

The svPPA group did not significantly differ from controls on any of our language measures, which did not support our second hypothesis of lower performance on semantic measures. This was despite participants with svPPA possessing the lowest scores on the ACE language subtest and the SYDBAT picture naming, word comprehension and semantic association subtests (see Table 2) indicative of semantic impairment. This demonstrates the importance of assessing spoken language skills across multiple contexts, as structured test batteries reveal deficits when stimulus-response requirements are tightly constrained. Discourse tasks, such as the Cookie Theft, remove many of these constraints and evaluate how flexible an individual can be in drawing upon intact and residual skills to express thoughts and ideas (see Kong, 2023). However, this study's focus was on microstructural discourse measures to capture parallel linguistic and cognitive changes in all variants. As such, we did not use macrostructural discourse-level measures such as main concept analysis (Dalton & Richardson, 2019) and productivity, completeness and elaboration of sequential story elements. It is expected that these measures would also show variant-specific profiles and provide greater insight into the quality of the story-telling in each variant (Coelho, 2002; Greenslade et al., 2024).

Although average utterance length for the svPPA group was not significantly lower than controls, it was associated with atrophy in left hemisphere temporal regions, anterior inferior frontal cortex, and medial middle-posterior cingulate cortex. These findings reveal the influence of impaired semantic processing on discourse production, even when surface level discourse structure and fluency may appear relatively preserved. This can be attributed to the nature of discourse tasks as they permit elaboration and the opportunity to explore semantic error patterns in tasks with different language and processing demands. Additionally, looking across the three PPA variants, it is clear that utterance length is affected by a variety of factors. It is associated with the inferior frontal language area in nfvPPA (i.e., sentence formulation), temporo-parietal language area in lvPPA (i.e., lexical processing), and the temporal regions in svPPA (i.e., semantic processing). As such, utterance length is a marker of discourse integrity but, by itself, is not practically or theoretically informative in differentiating PPA variants. On the other hand, these findings highlight the diverse and distributed suite of skills that must be coordinated in the process of generating the microstructural components of discourse.

4.3.2. *Longitudinal discourse and neuroimaging*

It was predicted that the svPPA group would show deterioration on semantic measures associated with increased atrophy along the ventral pathway (Hickok & Poeppel, 2004) and right homologues. Contrary to expectation, the svPPA group experienced an increase in propositional idea density over time. In the context of their relatively stable noun:verb and open:-closed class word ratios, this could be associated with increased circumlocution, wherein individuals provide more descriptive content when specific words cannot be retrieved (Mesulam, Coventry, Bigio, et al., 2021). Consistent with an explanation of a more functional or compensatory response to the semantic word retrieval deficit in svPPA, the change in idea density observed in our sample was not associated with atrophic changes. Gallant et al. (2019), in a study comparing individuals with Alzheimer's dementia or mild cognitive impairment, noted that moderate semantic impairment tends to enable contentful (i.e., "precise") circumlocutions while more severe impairment leads to "vague" circumlocutions. A detailed analysis of word retrieval error types and behaviours was beyond the scope of this study. However, this finding highlights the importance of evaluating linguistic skills across multiple contexts to reveal core deficits (e.g., naming tasks) as well as their functional consequences for daily communication (e.g., discourse tasks). While our findings are amplified in our svPPA group due to underlying pathology, they shed light on universal compensatory mechanisms used in discourse by healthy ageing individuals. Hardy et al. (2020) found that older adults were more vulnerable to errors during word retrieval, although the integrity of syntactic frames and scaffolds remained relatively preserved. Additional evidence has supported a shift towards top-down processing in healthy older individuals that further promotes dependence on circumlocutory language (Payne & Silcox, 2019).

4.4. *Limitations and future directions*

While PPA is a valuable model for examining relationships between cognition and language in discourse production, there are limitations. PPA is a relatively rare condition and, as such, most studies are retrospective in nature. The discourse language analysis here was limited to the Cookie Theft picture description. While this task elicits several story elements, it typically elicits a shorter language sample with limited story grammar in comparison to other discourse tasks such as telling the stories of Cinderella (Thompson et al., 1997) or 'Frog, Where Are You' (Mayer, 1969). Therefore, prospective longitudinal studies should collect samples using a range of discourse genres (Kintz, 2023), considering both microstructural and macrostructural analyses for a more comprehensive picture of cognitive-linguistic interactions over time. These limitations may have impacted our ability to detect between-group differences in language performance. While beyond the scope of the current study, complementing micro- and macro-structural analyses should provide insight into the quality of the story-telling and ability to participate in the broad range of discourse contexts people encounter daily (Coelho, 2002; Greenslade et al., 2024).

While our language analyses used standard CLAN protocols, these include a subset of all possible microstructural discourse measures. It was beyond the scope of the current study to quantify additional behaviours such as abandoned versus completed utterances, morpheme substitution versus omission, verb argument structure, and type of paraphasias. As such, our analysis does not inform differentiation of agrammatism from other disorders such as paragrammatism (Wilson, Henry, et al., 2010), and mechanisms underlying

these different behaviours. These metrics could be included in future longitudinal discourse analysis studies. Despite this, the measures of spoken language structure and complexity used here (e.g., mean length of utterance, embedded clauses:sentences, and percent of flawed sentences) were shown to be highly correlated across these two discourse task types of (Ash et al., 2013). Furthermore, comparing discourse level language with constrained tasks that elicit specific morphosyntactic structures (Billette et al., 2015; Cupit et al., 2017; Deleon et al., 2012) under varying cognitive load may provide new perspectives on cognitive-linguistic interactions that are occurring in discourse. While these tasks sacrifice ecological validity, potentially under-estimating functional capacity, they provide detail across the spectrum of language function and can inform theories of grammatical processing and use.

4.5. *Conclusions*

This study is the largest to date to explore longitudinal changes in discourse and associated changes in cortical thickness, across the three PPA variants. Findings show that longitudinal changes in sentence complexity and grammatical errors in expository discourse production PPA are not associated with traditional regions underpinning morphosyntactic processing. Rather, they are associated with broader cognitive processes and with related lexical and semantic processing. For nfvPPA, this evokes the earliest accounts of agrammatism, which proposed that symptoms were a consequence of an "economy of effort" strategy (e.g., Fedorenko et al., 2023). However, in individuals with stroke, this strategy is proposed to compensate for a core morphosyntactic impairment, which contrasts with nfvPPA where it may be due to a secondary deficit linked to processes requiring higher cognitive effort. In nfvPPA and lvPPA, grammatical changes over time reflected extension of pathology both beyond and within the left perisylvian language network, respectively, providing insight into how discourse production is underpinned by a network that extends beyond classic language regions and involves domain-general cognitive processes such as error-monitoring and elaborative encoding. From a clinical standpoint, while assessing discourse may provide an estimate of functional language capacity for daily communication needs in PPA, the measures we are currently using are not specific to the grammatical changes. Using more targeted and systematically controlled elicitation tasks and measures, perhaps drawing from the long history in stroke-related agrammatism research (Feng et al., 2024), may enable us to better define grammatical impairments across variants and more accurately assign (or define) the label of agrammatism. From a clinical standpoint, we have interventions that have positive effect for prolonging spoken communication capacity in nfvPPA (Wauters et al., 2024). With more in-depth knowledge of the grammatical and contributing cognitive deficits in PPA, we may be able to refine these interventions to improve communication success.

CRediT authorship contribution statement

Marina A. Anwia: Writing – original draft, Methodology, Investigation, Formal analysis. **Mara Steinberg Lowe:** Writing – review & editing, Validation, Methodology, Investigation, Formal analysis, Conceptualization. **Sophie Matis:** Writing – review & editing, Visualization, Methodology, Investigation, Formal analysis, Data curation. **James Carrick:** Writing – review & editing, Validation, Methodology, Data curation. **Olivier Piguet:** Writing – review & editing, Resources, Funding acquisition, Conceptualization. **Ramon Landin-Romero:** Writing – review & editing, Visualization, Supervision, Methodology, Investigation, Funding acquisition, Formal analysis, Conceptualization. **Kirrie J. Ballard:** Writing – review & editing, Validation, Supervision, Project administration, Methodology, Investigation, Data curation, Conceptualization.

Funding

This work was supported in part by funding to ForeFront, a collaborative research group dedicated to the study of FTD and motor neuron disease, from the National Health and Medical Research Council (NHMRC: GNT1037746) and the Australian Research Council Centre of Excellence in Cognition and its Disorders Memory Program (ARC: CE11000102). JC is supported by an Australian Government Research Training Program Scholarship. OP and RLR are supported by NHMRC Investigator grants (GNT2008020 to OP; GNT2010064 to RLR).

Declaration of competing interest

The authors report no conflicts of interest.

Acknowledgements

We thank all the participants and their carers for their time and contribution to this study. Penelope Monroe and Jessica Feng assisted with language sample transcription. We gratefully acknowledge the infrastructure and subsidised access provided by Sydney Imaging and the Sydney Informatics Hub core facilities at the University of Sydney.

Scientific transparency statement

DATA: Some raw and processed data supporting this research are publicly available, while some are subject to restrictions: Data contained in the manuscript or supplemental files.

CODE: All analysis code supporting this research is publicly available: http://surfer.nmr.mgh.harvard.edu/Code contained in the manuscript or supplemental files.

MATERIALS: This research did not make use of any materials to generate or acquire data.

DESIGN: This article reports, for all studies, how the author(s) determined all sample sizes, all data exclusions, all

data inclusion and exclusion criteria, and whether inclusion and exclusion criteria were established prior to data analysis.

PRE-REGISTRATION: No part of the study procedures was pre-registered in a time-stamped, institutional registry prior to the research being conducted. No part of the analysis plans was pre-registered in a time-stamped, institutional registry prior to the research being conducted.

For full details, see the *Scientific Transparency Report* in the supplementary data to the online version of this article.

Supplementary data

Supplementary data to this article can be found online at https://doi.org/10.1016/j.cortex.2025.11.011.

REFERENCES

Ash, S., Evans, E., O'Shea, J., Powers, J., Boller, A., Weinberg, D., Haley, J., McMillan, C., Irwin, D. J., Rascovsky, K., & Grossman, M. (2013). Differentiating primary progressive aphasias in a brief sample of connected speech. *Neurology, 81*(4), 329–336. https://doi.org/10.1212/wnl.0b013e31829c5d0e

Ash, S., Moore, P., Antani, S., McCawley, G., Work, M., & Grossman, M. (2006). Trying to tell a tale: Discourse impairments in progressive aphasia and frontotemporal dementia. *Neurology, 66*(9), 1405–1413. https://doi.org/10.1212/01.wnl.0000210435.72614.3

Ash, S., Nevler, N., Phillips, J., Irwin, D. J., McMillan, C. T., Rascovsky, K., & Grossman, M. (2019). A longitudinal study of speech production in primary progressive aphasia and behavioral variant frontotemporal dementia. *Brain and Language, 194*, 46–57. https://doi.org/10.1016/j.bandl.2019.04.006

Ballard, K. J., Savage, S., Leyton, C. E., Vogel, A. P., Hornberger, M., & Hodges, J. R. (2014). Logopenic and nonfluent variants of primary progressive aphasia are differentiated by acoustic measures of speech production. *PLos One, 9*(2), Article e89864. https://doi.org/10.1371/journal.pone.0089864

Bernal-Rusiel, J. L., Greve, D. N., Reuter, M., Fischl, B., & Sabuncu, M. R. (2013). Statistical analysis of longitudinal neuroimage data with linear mixed effects models. *Neuroimage, 66*, 249–260. https://doi.org/10.1016/j.neuroimage.2012.10.065

Bernal-Rusiel, J. L., Reuter, M., Greve, D. N., Fischl, B., & Sabuncu, M. R. (2013). Spatiotemporal linear mixed effects modeling for the mass-univariate analysis of longitudinal neuroimage data. *Neuroimage, 81*, 358–370. https://doi.org/10.1016/j.neuroimage.2013.05.049

Billette, O. V., A, S. S., Karalyn, P., & Nestor, P. J. (2015). SECT and MAST: New tests to assess grammatical abilities in primary progressive aphasia. *Aphasiology, 29*(10), 1135–1151. https://doi.org/10.1080/02687038.2015.1037822

Botha, H., & Josephs, K. A. (2019). Primary progressive aphasias and apraxia of speech. *Continuum: Lifelong Learning in Neurology, 25*(1), 101–127. https://doi.org/10.1212/con.0000000000000699

Coelho, C. A. (2002). Story narratives of adults with closed head injury and non-brain-injured adults: Influence of socioeconomic status, elicitation task, and executive functioning. *Journal of Speech, Language, and Hearing Research: JSLHR, 45*(6), 1232–1248. https://doi.org/10.1044/1092-4388(2002/099

Cohen, J. (1988). In *Statistical power analysis for the behavioral sciences* (2nd ed.). Hillsdale, NJ: Erlbaum.

Conca, F., Esposito, V., Giusto, G., Cappa, S. F., & Catricalà, E. (2022). Characterization of the logopenic variant of primary progressive aphasia: A systematic review and meta-analysis. *Ageing Research Reviews, 82*, Article 101760. https://doi.org/10.1016/j.arr.2022.101760

Cordella, C., Dickerson, B. C., Quimby, M., Yunusova, Y., & Green, J. R. (2017). Slowed articulation rate is a sensitive diagnostic marker for identifying non-fluent primary progressive aphasia. *Aphasiology, 31*(2), 241–260. https://doi.org/10.1080/02687038.2016.1191054

Cupit, J., Carol, L. L. G. N., Bruna, S. L., David, T. W. E. B. S., & Rochon, E. (2017). Analysing syntactic productions in semantic variant PPA and non-fluent variant PPA: How different are they? *Aphasiology, 31*(3), 282–307. https://doi.org/10.1080/02687038.2016.1180661

Dalton, S. G. H., & Richardson, J. D. (2019). A large-scale comparison of main concept production between persons with aphasia and persons without brain injury. *The American Journal of Surgical Pathology, 28*(1S), 293–320. https://doi.org/10.1044/2018_ajslp-17-0166

Davey, J., Thompson, H. E., Hallam, G., Karapanagiotidis, T., Murphy, C., De Caso, I., Krieger-Redwood, K., Bernhardt, B. C., Smallwood, J., & Jefferies, E. (2016). Exploring the role of the posterior middle temporal gyrus in semantic cognition: Integration of anterior temporal lobe with executive processes. *Neuroimage, 137*, 165–177. https://doi.org/10.1016/j.neuroimage.2016.05.051

Deleon, J., Gesierich, B., Besbris, M., Ogar, J., Henry, M. L., Miller, B. L., Gorno-Tempini, M. L., & Wilson, S. M. (2012). Elicitation of specific syntactic structures in primary progressive aphasia. *Brain and Language, 123*(3), 183–190. https://doi.org/10.1016/j.bandl.2012.09.004

Deschamps, I., Baum, S. R., & Gracco, V. L. (2014). On the role of the supramarginal gyrus in phonological processing and verbal working memory: Evidence from rTMS studies. *Neuropsychologia, 53*, 39–46. https://doi.org/10.1016/j.neuropsychologia.2013.10.015

Desikan, R. S., Ségonne, F., Fischl, B., Quinn, B. T., Dickerson, B. C., Blacker, D., Buckner, R. L., Dale, A. M., Maguire, R. P., Hyman, B. T., Albert, M. S., & Killiany, R. J. (2006). An automated labeling system for subdividing the human cerebral cortex on MRI scans into gyral based regions of interest. *Neuroimage, 31*(3), 968–980. https://doi.org/10.1016/j.neuroimage.2006.01.021

Dial, H. R., Gnanateja, G. N., Tessmer, R. S., Gorno-Tempini, M. L., Chandrasekaran, B., & Henry, M. L. (2021). Cortical tracking of the speech envelope in logopenic variant primary progressive aphasia. *Frontiers in Human Neuroscience, 14*. https://doi.org/10.3389/fnhum.2020.597694

Duffy, J. R., Strand, E. A., & Josephs, K. A. (2014). Motor speech disorders associated with primary progressive aphasia. *Aphasiology, 28*(8–9), 1004–1017. https://doi.org/10.1080/02687038.2013.869307

Duffy, J. R., Utianski, R. L., & Josephs, K. A. (2021). Primary progressive apraxia of speech: From recognition to diagnosis and care. *Aphasiology, 35*(4), 560–591. https://doi.org/10.1080/02687038.2020.1787732

Engelman, M., Agree, E. M., Meoni, L. A., & Klag, M. J. (2010). Propositional density and cognitive function in later life: Findings from the precursors study. *The Journal of Gerontology Series B, Psychological Sciences and Social Sciences, 65*(6), 706–711. https://doi.org/10.1093/geronb/gbq064

Eslinger, P. J., Moore, P., Antani, S., Anderson, C., & Grossman, M. (2012). Apathy in frontotemporal dementia: Behavioral and neuroimaging correlates. *Behavioural Neurology, 25*(2), 127–136. https://doi.org/10.3233/ben-2011-0351

Europa, E., Gitelman, D. R., Kiran, S., & Thompson, C. K. (2019). Neural connectivity in syntactic movement processing. *Frontiers in Human Neuroscience, 13*. https://doi.org/10.3389/fnhum.2019.00027

Faroqi-Shah, Y., Treanor, A., Ratner, N. B., Ficek, B., Webster, K., & Tsapkini, K. (2020). Using narratives in differential diagnosis of neurodegenerative syndromes. *Journal of communication disorders, 85*, 105994. https://doi.org/10.1016/j.jcomdis.2020.105994, 105994.

Federmeier, K. D., Jongman, S. R., & Szewczyk, J. M. (2020). Examining the role of general cognitive skills in language processing: A window into complex cognition. *Current Directions in Psychological Sciences, 29*(6), 575–582. https://doi.org/10.1177/0963721420964095

Fedorenko, E., Ryskin, R., & Gibson, E. (2023). Agrammatic output in non-fluent, including Broca's, aphasia as a rational behavior. *Aphasiology, 37*(12), 1981–2000. https://doi.org/10.1080/02687038.2022.2143233

Feng, Y., Dyson, B., & Ballard, K. J. (2024). Assessment of agrammatism in individuals with Broca's aphasia and non-fluent variant primary progressive aphasia: A scoping review [conference presentation]. In *Paper presented at the speech pathology Australia conference* (Perth, WA, Australia).

Fischl, B., & Dale, A. M. (2000). Measuring the thickness of the human cerebral cortex from magnetic resonance images. *Proceedings of the National Academy of Sciences, 97*(20), 11050–11055. https://doi.org/10.1073/pnas.200033797

Fischl, B., van der Kouwe, A., Destrieux, C., Halgren, E., Ségonne, F., Salat, D. H., Busa, E., Seidman, L. J., Goldstein, J., Kennedy, D., Caviness, V., Makris, N., Rosen, B., & Dale, A. M. (2004). Automatically parcellating the human cerebral cortex. *Cerebral Cortex, 14*(1), 11–22. https://doi.org/10.1093/cercor/bhg087

Fraser, K. C., Meltzer, J. A., Graham, N. L., Leonard, C., Hirst, G., Black, S. E., & Rochon, E. (2014). Automated classification of primary progressive aphasia subtypes from narrative speech transcripts. *Cortex, 55*, 43–60. https://doi.org/10.1016/j.cortex.2012.12.006

Gallant, M., Lavoie, M., Hudon, C., & Monetta, L. (2019). Analysis of naming errors in healthy aging, mild cognitive impairment, and Alzheimer's disease. *Canadian Journal of Speech-Language Pathology and Audiology, 43*(2), 95–108.

Goodglass, H., Kaplan, E., & Weintraub, S. (2001). *BDAE: The Boston diagnostic aphasia examination*. Philadelphia, PA: Lippincott Williams & Wilkins.

Gorno-Tempini, M. L., Hillis, A. E., Weintraub, S., Kertesz, A., Mendez, M., Cappa, S. F., Ogar, J. M., Rohrer, J. D., Black, S., Boeve, B. F., Manes, F., Dronkers, N. F., Vandenberghe, R., Rascovsky, K., Patterson, K., Miller, B. L., Knopman, D. S., Hodges, J. R., Mesulam, M. M., & Grossman, M. (2011). Classification of primary progressive aphasia and its variants. *Neurology, 76*(11), 1006–1014. https://doi.org/10.1212/wnl.0b013e31821103e6

Graham, N. L., Leonard, C., Tang-Wai, D. F., Black, S., Chow, T. W., Scott, C. J. M., McNeely, A. A., Masellis, M., & Rochon, E. (2016). Lack of frank agrammatism in the nonfluent agrammatic variant of primary progressive aphasia. *Dementia and Geriatric Cognitive Disorders Extra, 6*(3), 407–423. https://doi.org/10.1159/000448944

Greenslade, K. J., Bogart, E., Gyory, J., Jaskolka, S., & Ramage, A. E. (2024). Story grammar analyses capture discourse improvement in the first 2 years following a severe traumatic brain injury. *American Journal of Speech and Language Pathology, 33*(2), 1004–1020. https://doi.org/10.1044/2023_ajslp-23-00269

Gunawardena, D., Ash, S., McMillan, C., Avants, B., Gee, J., & Grossman, M. (2010). Why are patients with progressive nonfluent aphasia nonfluent? *Neurology, 75*(7), 588–594. https://doi.org/10.1212/WNL.0b013e3181ed.9c7d

Hardy, S. M., Segaert, K., & Wheeldon, L. (2020). Healthy aging and sentence production: Disrupted lexical access in the context of intact syntactic planning. *Frontiers in Psychology, 11*, 257. https://doi.org/10.3389/fpsyg.2020.00257

Harrag, C., Sabil, A., Conceição, M. C., & Radvansky, G. A. (2024). Propositional density: Cognitive impairment and aging. *Frontiers in Psychology, 15*, Article 1434506. https://doi.org/10.3389/fpsyg.2024.1434506

Hickok, G., & Poeppel, D. (2004). Dorsal and ventral streams: A framework for understanding aspects of the functional anatomy of language. *Cognition, 92*(1–2), 67–99. https://doi.org/10.1016/j.cognition.2003.10.011

Hsieh, S., Schubert, S., Hoon, C., Mioshi, E., & Hodges, J. R. (2013). Validation of the Addenbrooke's cognitive examination III in frontotemporal dementia and Alzheimer's disease. *Dementia and Geriatric Cognitive Disorders, 36*(3–4), 242–250. https://doi.org/10.1159/000351671

Hughes, C. P., Berg, L., Danziger, W. L., Coben, L. A., & Martin, R. L. (1982). A new clinical scale for the staging of dementia. *The British Journal of Psychiatry: the Journal of Mental Science, 140*, 566–572. https://doi.org/10.1192/bjp.140.6.566

Husa, R. A., Gordon, B. A., Cochran, M. M., Bertolin, M., Bond, D. N., & Kirchhoff, B. A. (2017). Left caudal middle frontal gray matter volume mediates the effect of age on self-initiated elaborative encoding strategies. *Neuropsychologia, 106*, 341–349. https://doi.org/10.1016/j.neuropsychologia.2017.10.004

Kemmerer, D. (2022). Revisiting the relation between syntax, action, and left BA44. *Frontiers in Human Neuroscience, 16*, Article 923022. https://doi.org/10.3389/fnhum.2022.923022

Kemper, S., Marquis, J., & Thompson, M. (2001). Longitudinal change in language production: Effects of aging and dementia on grammatical complexity and propositional content. *Psychology and Aging, 16*(4), 600–614. https://doi.org/10.1037//0882-7974.16.4.600

Kielar, A., Milman, L., Bonakdarpour, B., & Thompson, C. K. (2011). Neural correlates of covert and overt production of tense and agreement morphology: Evidence from fMRI. *Journal of Neurolinguistics, 24*(2), 183–201. https://doi.org/10.1016/j.jneuroling.2010.02.008

Kintz, S. (2023). Discourse characteristics in aphasia. In A. P.-K. Kong (Ed.), *Spoken discourse impairments in the neurogenic populations: A state-of-ther-art, comtemporary approach* (pp. 23–36). Springer Nature. https://doi.org/10.1007/978-3-031-45190-4. Ch. 2.

Kong, A. P.-H. (2023). In *Analysis of neurogenic disordered discourse production: Theories, assessment and treatment* (2nd ed.). Routledge. https://doi.org/10.4324/9781003254775.

Kong, A. P.-H., Cheung, R. T. H., Wong, G. H. Y., Choy, J. C. P., Dai, R., & Spector, A. (2023). Spoken discourse in episodic autobiographical and verbal short-term memory in Chinese people with dementia: The roles of global coherence and informativeness [Original Research]. *Frontiers in Psychology, 14*. https://doi.org/10.3389/fpsyg.2023.1124477

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

Landin-Romero, R., Liang, C. T., Monroe, P. A., Higashiyama, Y., Leyton, C. E., Hodges, J. R., Piguet, O., & Ballard, K. J. (2021). Brain changes underlying progression of speech motor programming impairment. *Brain Communications, 3*(3), fcab205. https://doi.org/10.1093/braincomms/fcab205. fcab205.

Landin-Romero, R., & Piguet, O. (2017). Recent advances in longitudinal structural neuroimaging of younger-onset dementias. *Neurodegenerative Disease Management, 7*(6), 349–352. https://doi.org/10.2217/nmt-2017-0057

Lavoie, M., Black, S. E., Tang-Wai, D. F., Graham, N. L., Stewart, S., Leonard, C., & Rochon, E. (2021). Description of connected speech across different elicitation tasks in the logopenic

variant of primary progressive aphasia. *International Journal of Language & Communication Disorders, 56*(5), 1074–1085. https://doi.org/10.1111/1460-6984.12660

Le Heron, C., Apps, M. A. J., & Husain, M. (2018). The anatomy of apathy: A neurocognitive framework for amotivated behaviour. *Neuropsychologia, 118*(Pt B), 54–67. https://doi.org/10.1016/j.neuropsychologia.2017.07.003

Leech, R., & Sharp, D. J. (2014). The role of the posterior cingulate cortex in cognition and disease. *Brain, 137*(Pt 1), 12–32. https://doi.org/10.1093/brain/awt162

Lerch, J. P., & Evans, A. C. (2005). Cortical thickness analysis examined through power analysis and a population simulation. *Neuroimage, 24*(1), 163–173. https://doi.org/10.1016/j.neuroimage.2004.07.045

Leyton, C. E., Hodges, J. R., McLean, C. A., Kril, J. J., Piguet, O., & Ballard, K. J. (2015). Is the logopenic-variant of primary progressive aphasia a unitary disorder? *Cortex, 67*, 122–133. https://doi.org/10.1016/j.cortex.2015.03.011

Leyton, C. E., Landin-Romero, R., Liang, C. T., Burrell, J. R., Kumfor, F., Hodges, J. R., & Piguet, O. (2019). Correlates of anomia in non-semantic variants of primary progressive aphasia converge over time. *Cortex, 120*, 201–211. https://doi.org/10.1016/j.cortex.2019.06.008

Leyton, C. E., Villemagne, V. L., Savage, S., Pike, K. E., Ballard, K. J., Piguet, O., Burrell, J. R., Rowe, C. C., & Hodges, J. R. (2011). Subtypes of progressive aphasia: Application of the international consensus criteria and validation using β-amyloid imaging. *Brain, 134*(10), 3030–3043. https://doi.org/10.1093/brain/awr216

Lorca-Puls, D. L., Gajardo-Vidal, A., Mandelli, M. L., Illán-Gala, I., Ezzes, Z., Wauters, L. D., Battistella, G., Bogley, R., Ratnasiri, B., Licata, A. E., Battista, P., García, A. M., Tee, B. L., Lukic, S., Boxer, A. L., Rosen, H. J., Seeley, W. W., Grinberg, L. T., Spina, S., … Gorno-Tempini, M. L. (2023). Neural basis of speech and grammar symptoms in non-fluent variant primary progressive aphasia spectrum. *Brain*. https://doi.org/10.1093/brain/awad327

Mack, J. E., Barbieri, E., Weintraub, S., Mesulam, M. M., & Thompson, C. K. (2021). Quantifying grammatical impairments in primary progressive aphasia: Structured language tests and narrative language production. *Neuropsychologia, 151*, 107713. https://doi.org/10.1016/j.neuropsychologia.2020.107713, 107713.

MacWhinney, B. (2000). In *The Childes project: Tools for analyzing talk* (3rd ed.). Lawrence Erlbaum Associates.

Mandelli, M. L., Lorca-Puls, D. L., Lukic, S., Montembeault, M., Gajardo-Vidal, A., Licata, A., Scheffler, A., Battistella, G., Grasso, S. M., Bogley, R., Ratnasiri, B. M., La Joie, R., Mundada, N. S., Europa, E., Rabinovici, G., Miller, B. L., De Leon, J., Henry, M. L., Miller, Z., & Gorno-Tempini, M. L. (2023). Network anatomy in logopenic variant of primary progressive aphasia. *Human Brain Mapping, 44*(11), 4390–4406. https://doi.org/10.1002/hbm.26388

Marshall, J. (2011). Disorders of sentence processing in aphasia. In I. Papathanasiou, P. Coppens, & C. Potagas (Eds.), *Aphasia and related neurogenic communication disorders* (1st ed., pp. 197–213). Jones & Bartlett Learning.

Mayer, M. (1969). *Frog, where are you?* Penguin Books.

Mesulam, M. M. (2013). Primary progressive aphasia and the language network: The 2013 H. Houston Merritt lecture. *Neurology, 81*(5), 456–462. https://doi.org/10.1212/WNL.0b013e31829d87df

Mesulam, M. M., Coventry, C., Bigio, E. H., Geula, C., Thompson, C., Bonakdarpour, B., Gefen, T., Rogalski, E. J., & Weintraub, S. (2021). In *Nosology of primary progressive aphasia and the neuropathology of language* (pp. 33–49). Springer International Publishing. https://doi.org/10.1007/978-3-030-51140-1_3.

Mesulam, M. M., Coventry, C. A., Rader, B. M., Kuang, A., Sridhar, J., Martersteck, A., Zhang, H., Thompson, C. K., Weintraub, S., & Rogalski, E. J. (2021). Modularity and granularity across the language network: A primary progressive aphasia perspective. *Cortex, 141*, 482–496. https://doi.org/10.1016/j.cortex.2021.05.002

Meteyard, L., & Patterson, K. (2009). The relation between content and structure in language production: An analysis of speech errors in semantic dementia. *Brain and Language, 110*(3), 121–134. https://doi.org/10.1016/j.bandl.2009.03.007

Mioshi, E., Dawson, K., Mitchell, J., Arnold, R., & Hodges, J. R. (2006). The Addenbrooke's cognitive examination revised (ACE-R): A brief cognitive test battery for dementia screening. *International Journal of Geriatric Psychiatry, 21*(11), 1078–1085. https://doi.org/10.1002/gps.1610

Montembeault, M., Brambati, S. M., Gorno-Tempini, M. L., & Migliaccio, R. (2018). Clinical, anatomical, and pathological features in the three variants of primary progressive aphasia: A review. *Frontiers in Neurology, 9*. https://doi.org/10.3389/fneur.2018.00692

Mueller, K. D. (2023). Using discourse as a measure of early cognitive decline associated with Alzheimer's disease biomarkers. In A. P.-K. Kong (Ed.), *Spoken discourse impairments in the neurogenic populations: A state-of-ther-art, contemporary approach* (pp. 53–64). Springer Nature. https://doi.org/10.1007/978-3-031-45190-4. Ch. 4.

Newman, A. J., Pancheva, R., Ozawa, K., Neville, H. J., & Ullman, M. T. (2001). An event-related fMRI study of syntactic and semantic violations. *Journal of Psycholinguistic Research, 30*(3), 339–364. https://doi.org/10.1023/a:1010499119393

Ni, W., Constable, R. T., Mencl, W. E., Pugh, K. R., Fulbright, R. K., Shaywitz, S. E., Shaywitz, B. A., Gore, J. C., & Shankweiler, D. (2000). An event-related neuroimaging study distinguishing form and content in sentence processing. *Journal of Cognitive Neuroscience, 12*(1), 120–133. https://doi.org/10.1162/08989290051137648

Oberhuber, M., Hope, T. M. H., Seghier, M. L., Parker Jones, O., Prejawa, S., Green, D. W., & Price, C. J. (2016). Four functionally distinct regions in the left supramarginal gyrus support word processing. *Cerebral Cortex, 26*(11), 4212–4226. https://doi.org/10.1093/cercor/bhw251

Payne, B. R., & Silcox, J. W. (2019). Chapter seven: Aging, context processing, and comprehension. In K. D. Federmeier (Ed.), *Psychology of learning and motivation* (Vol. 71, pp. 215–264). https://doi.org/10.1016/bs.plm.2019.07.001

Raichle, M. E., Macleod, A. M., Snyder, A. Z., Powers, W. J., Gusnard, D. A., & Shulman, G. L. (2001). A default mode of brain function. *Proceedings of the National Academy of Sciences, 98*(2), 676–682. https://doi.org/10.1073/pnas.98.2.676

Reuter, M., & Fischl, B. (2011). Avoiding asymmetry-induced bias in longitudinal image processing. *Neuroimage, 57*(1), 19–21. https://doi.org/10.1016/j.neuroimage.2011.02.076

Reuter, M., Rosas, H. D., & Fischl, B. (2010). Highly accurate inverse consistent registration: A robust approach. *Neuroimage, 53*(4), 1181–1196. https://doi.org/10.1016/j.neuroimage.2010.07.020

Rice, G. E., Lambon Ralph, M. A., & Hoffman, P. (2015). The roles of left versus right anterior temporal lobes in conceptual knowledge: An ALE meta-analysis of 97 functional neuroimaging studies. *Cerebral Cortex, 25*(11), 4374–4391. https://doi.org/10.1093/cercor/bhv024

Ridderinkhof, K. R., Ullsperger, M., Crone, E. A., & Nieuwenhuis, S. (2004). The role of the medial frontal cortex in cognitive control. *Science, 306*(5695), 443–447. https://doi.org/10.1126/science.1100301

Rogalski, E., Cobia, D., Harrison, T. M., Wieneke, C., Thompson, C. K., Weintraub, S., & Mesulam, M. M. (2011). Anatomy of language impairments in primary progressive aphasia. *The Journal of Neuroscience, 31*(9), 3344–3350. https://doi.org/10.1523/JNEUROSCI.5544-10.2011

Rohrer, J. D., Ridgway, G. R., Crutch, S. J., Hailstone, J., Goll, J. C., Clarkson, M. J., Mead, S., Beck, J., Mummery, C., Ourselin, S., Warrington, E. K., Rossor, M. N., & Warren, J. D. (2010). Progressive logopenic/phonological aphasia: Erosion of the language network. *Neuroimage, 49*(1), 984–993. https://doi.org/10.1016/j.neuroimage.2009.08.002

Rohrer, J. D., Warren, J. D., Modat, M., Ridgway, G. R., Douiri, A., Rossor, M. N., Ourselin, S., & Fox, N. C. (2009). Patterns of cortical thinning in the language variants of frontotemporal lobar degeneration. *Neurology, 72*(18), 1562–1569. https://doi.org/10.1212/wnl.0b013e3181a4124e

Rosen, H. J., Allison, S. C., Schauer, G. F., Gorno-Tempini, M. L., Weiner, M. W., & Miller, B. L. (2005). Neuroanatomical correlates of behavioural disorders in dementia. *Brain, 128*(Pt 11), 2612–2625. https://doi.org/10.1093/brain/awh628

Sajjadi, S. A., Patterson, K., Tomek, M., & Nestor, P. J. (2012). Abnormalities of connected speech in the non-semantic variants of primary progressive aphasia. *Aphasiology, 26*(10), 1219–1237. https://doi.org/10.1080/02687038.2012.710318

Santi, G. C., Conca, F., Esposito, V., Polito, C., Caminiti, S. P., Boccalini, C., Morinelli, C., Berti, V., Mazzeo, S., Bessi, V., Marcone, A., Iannaccone, S., Kim, S.-K., Sorbi, S., Perani, D., Cappa, S. F., & Catricalà, E. (2024). Heterogeneity and overlap in the continuum of linguistic profile of logopenic and semantic variants of primary progressive aphasia: A profile analysis based on multidimensional scaling study. *Alzheimer's Research & Therapy, 16*(1), 49. https://doi.org/10.1186/s13195-024-01403-0

Savage, S., Hsieh, S., Leslie, F., Foxe, D., Piguet, O., & Hodges, J. R. (2013). Distinguishing subtypes in primary progressive aphasia: Application of the Sydney language battery. *Dementia and Geriatric Cognitive Disorders, 35*(3–4), 208–218. https://doi.org/10.1159/000346389

Shetreet, E., & Friedmann, N. (2014). The processing of different syntactic structures: fMRI investigation of the linguistic distinction between wh-movement and verb movement. *Journal of neurolinguistics, 27*(1), 1–17. https://doi.org/10.1016/j.jneuroling.2013.06.003

So, M., Foxe, D., Kumfor, F., Murray, C., Hsieh, S., Savage, G., Ahmed, R. M., Burrell, J. R., Hodges, J. R., Irish, M., & Piguet, O. (2018). Addenbrooke's cognitive examination III: Psychometric characteristics and relations to functional ability in dementia. *Journal of the International Neuropsychological Society, 24*(8), 854–863. https://doi.org/10.1017/s1355617718000541

Stark, B. C., Basilakos, A., Hickok, G., Rorden, C., Bonilha, L., & Fridriksson, J. (2019). Neural organization of speech production: A lesion-based study of error patterns in connected speech. *Cortex, 117*, 228–246. https://doi.org/10.1016/j.cortex.2019.02.029

Tetzloff, K. A., Duffy, J. R., Clark, H. M., Strand, E. A., Machulda, M. M., Schwarz, C. G., Senjem, M. L., Reid, R. I., Spychalla, A. J., Tosakulwong, N., Lowe, V. J., Jack, J. C. R., Josephs, K. A., & Whitwell, J. L. (2018). Longitudinal structural and molecular neuroimaging in agrammatic primary progressive aphasia. *Brain, 141*(1), 302–317. https://doi.org/10.1093/brain/awx293

Themistocleous, C. (2023). Discourse and conversation impairments in patients with dementia. In A. P.-K. Kong (Ed.), *Spoken discourse impairments in the neurogenic populations: A state-of-the-art, contemporary approach* (pp. 37–52). Springer Nature. https://doi.org/10.1007/978-3-031-45190-4. Ch. 3.

Themistocleous, C., Ficek, B., Webster, K., Den Ouden, D.-B., Hillis, A. E., & Tsapkini, K. (2021). Automatic subtyping of individuals with primary progressive aphasia. *Journal of Alzheimer's Disease, 79*(3), 1185–1194. https://doi.org/10.3233/jad-201101

Thompson, C. K., Ballard, K. J., Tait, M. E., Weintraub, S., & Mesulam, M. (1997). Patterns of language decline in non-fluent primary progressive aphasia. *Aphasiology, 11*(4–5), 297–321. https://doi.org/10.1080/02687039708248473

Thompson, C. K., Cho, S., Hsu, C.-J., Wieneke, C., Rademaker, A., Weitner, B. B., Mesulam, M. M., & Weintraub, S. (2012). Dissociations between fluency and agrammatism in primary progressive aphasia. *Aphasiology, 26*(1), 20–43. https://doi.org/10.1080/02687038.2011.584691

Thompson, C. K., & Mack, J. E. (2014). Grammatical impairments in PPA. *Aphasiology, 28*(8–9), 1018–1037. https://doi.org/10.1080/02687038.2014.912744

Thompson, C. K., Meltzer-Asscher, A., Cho, S., Lee, J., Wieneke, C., Weintraub, S., & Mesulam, M. M. (2013). Syntactic and morphosyntactic processing in stroke-induced and primary progressive aphasia. *Behavioural Neurology, 26*(1–2), 35–54. https://doi.org/10.3233/ben-2012-110220

Vergis, M. K., Ballard, K. J., Duffy, J. R., McNeil, M. R., Scholl, D., & Layfield, C. (2014). An acoustic measure of lexical stress differentiates aphasia and aphasia plus apraxia of speech after stroke. *Aphasiology, 28*(5), 554–575. https://doi.org/10.1080/02687038.2014.889275

Wauters, L. D., Croot, K., Dial, H. R., Duffy, J. R., Grasso, S. M., Kim, E., Mendez, K. S., Ballard, K. J., Clark, H. M., Kohley, L., Murray, L. L., Rogalski, E. J., Figeys, M., Milman, L., & Henry, M. L. (2024). Behavioral treatment for speech and language in primary progressive aphasia and primary progressive apraxia of speech: A systematic review. *Neuropsychology Review, 34*(3), 882–923. https://doi.org/10.1007/s11065-023-09607-1

Whitwell, J. L., Jones, D. T., Duffy, J. R., Strand, E. A., Machulda, M. M., Przybelski, S. A., Vemuri, P., Gregg, B. E., Gunter, J. L., Senjem, M. L., Petersen, R. C., Jack, C. R., & Josephs, K. A. (2015). Working memory and language network dysfunctions in logopenic aphasia: A task-free fMRI comparison with Alzheimer's dementia. *Neurobiology of Aging, 36*(3), 1245–1252. https://doi.org/10.1016/j.neurobiolaging.2014.12.013

Wilson, S. M., Dronkers, N. F., Ogar, J. M., Jang, J., Growdon, M. E., Agosta, F., Henry, M. L., Miller, B. L., & Gorno-Tempini, M. L. (2010). Neural correlates of syntactic processing in the nonfluent variant of primary progressive aphasia. *The Journal of Neuroscience, 30*(50), 16845–16854. https://doi.org/10.1523/jneurosci.2547-10.2010

Wilson, S. M., Galantucci, S., Tartaglia, M. C., & Gorno-Tempini, M. L. (2012). The neural basis of syntactic deficits in primary progressive aphasia. *Brain and Language, 122*(3), 190–198. https://doi.org/10.1016/j.bandl.2012.04.005

Wilson, S. M., Henry, M. L., Besbris, M., Ogar, J. M., Dronkers, N. F., Jarrold, W., Miller, B. L., & Gorno-Tempini, M. L. (2010). Connected speech production in three variants of primary progressive aphasia. *Brain, 133*(7), 2069–2088. https://doi.org/10.1093/brain/awq129

Zamboni, G., Huey, E. D., Krueger, F., Nichelli, P. F., & Grafman, J. (2008). Apathy and disinhibition in frontotemporal dementia. *Neurology, 71*(10), 736–742. https://doi.org/10.1212/01.wnl.0000324920.96835.95

Zhang, S., Ide, J. S., & Li, C.-s. R. (2011). Resting-state functional connectivity of the medial superior frontal cortex. *Cerebral Cortex, 22*(1), 99–111. https://doi.org/10.1093/cercor/bhr088