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Multiple types of dyslexia in primary progressive aphasia

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ABSTRACT

Background: Primary progressive aphasia (PPA) is a neurocognitive syndrome hallmarked by decline in language-related cognitive abilities. Although reading is an essential cognitive ability for communication and everyday activities, reading impairment, dyslexia, is under-researched in PPA compared to speech production and comprehension. Dyslexia research in PPA focuses on only two types: surface and phonological dyslexia, even though many more types are known to occur in non-neurodegenerative brain disease.
Aims: To investigate the frequency and types of dyslexia in a case series of patients with PPA.
Methods: Forty PPA participants completed a comprehensive reading assessment examining reading lists of words, nonwords, and word-pairs that are sensitive to the identification of at least 13 dyslexia types. Types of reading errors were analyzed to determine the dyslexia type for each participant with dyslexia. The participants also underwent comprehensive language and neurocognitive assessment.
Results: Dyslexia was identified in 83% of the PPA participants, manifesting in 11 distinct dyslexia types: surface, attentional, phonological output buffer, orthographic input buffer, phonological conversion, vowel, letter-position, visual, voicing, morphological-conversion, and deep dyslexias. Five of these types of dyslexia have not been previously reported in PPA or other dementias. We found a double dissociation between surface dyslexia and semantic impairment and dissociations between phonological dyslexia and a phonological impairment in production.
Conclusions: The results underscore the need for refined assessment tools for diagnosing dyslexia in PPA, based on a theory of reading, that is able to identify the various dyslexia types. Accurate identification of identifies dyslexia types can guide targeted individualized interventions.

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1. Introduction

1.1. Dyslexia in PPA

Primary progressive aphasia (PPA), the neurocognitive syndrome hallmarked by predominant decline in language and communication abilities, has been long known to present with reading impairment, dyslexia. In his seminal descriptions of what later became PPA and frontotemporal dementia (FTD), Arnold Pick detailed the struggle of his patient August H. in reading aloud – "a task he executed slowly, laboriously, with occasional reading errors" (Pick, 1892/1994) . Mesulam (1982), in his initial description of PPA, reported four of the six patients had reading impairments.

Whereas these earliest reports of progressive aphasia examined dyslexia, current research into PPA predominantly focuses on speech production and comprehension, and less on reading. Reading tasks in PPA research batteries usually aim to identify two types of dyslexia: surface and phonological dyslexia (Brambati et al., 2009; Gorno-Tempini et al., 2008, 2011; Staffaroni et al., 2021). These two types of dyslexia are attributed each to a different variant of PPA: surface dyslexia is a diagnostic criterion for the semantic variant (svPPA), and phonological dyslexia is often attributed to the logopenic variant (lvPPA), although it is not included in the core diagnostic criteria (Gorno-Tempini et al., 2011).[1] Consequently, tasks employed to investigate reading in PPA usually rely on the regular-irregular distinction (to identify surface dyslexia), or the word-nonword distinction (to identify phonological dyslexia) (Brambati et al., 2009; Gorno-Tempini et al., 2011; Luzzatti et al., 2016; Matías-Guiu et al., 2017). This methodology creates an unfortunate gap, as it leads to a situation in which many other dyslexia types are not tested and therefore not identified, perpetuating a paradigm in which PPA can only present with phonological or surface dyslexia. This paradigm is in stark contrast to dyslexia research in aphasia caused by cerebrovascular and other brain diseases and in developmental disorders, where a broad range of dyslexia types are reported, each explainable in a cognitive neuropsychological model of the reading process from the visual-orthographic analysis stage to the word's production and comprehension. Identifying the exact difficulties each patient faces in reading is a crucial step toward designing efficient intervention and treatment for their reading impairment (see Nickels et al., 2010 for an overview on the bidirectional relations between theory and treatment in cognitive neuropsychology).

1.2. The process of reading single words and nonwords

Research on aphasia caused by cerebrovascular and other brain diseases, as well as research on developmental disorders revealed and described many types of dyslexia, each explained by a deficit in a different cognitive component or multiple components in the reading process (Brunsdon et al., 2006; Coltheart et al., 2001; Friedmann & Coltheart, 2018; Marshall & Newcombe, 1973; Shallice, 1988) as introduced in the model depicted in Figure 1.

The model describes the flow of information from an input written word to an output speech and to comprehension. The process begins with orthographic analysis of the letter string (marked A in Figure 1), which includes identifying individual letters, encoding their relative position within the word, and binding letters to the word in which they appear

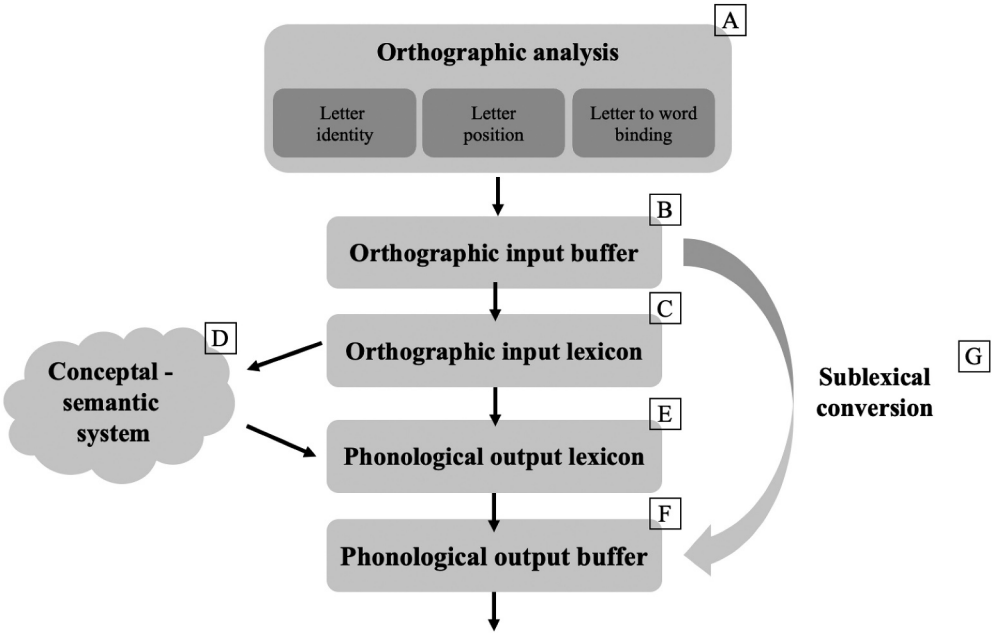

Figure 1. A neuropsychological model of single word and nonword reading.

(Coltheart, 1981; Friedmann & Coltheart, 2018; Humphreys et al., 1990; Peressotti & Grainger, 1995).

The output of the visual-orthographic letter analysis processes is temporarily stored in the orthographic input buffer (B in Figure 1) (Tainturier & Rapp, 2003) and subsequently funneled through two routes: the lexical route (C→E) and the sublexical route (G) (Marshall & Newcombe, 1973).

The lexical route utilizes two stores of the reader's previous knowledge: the orthographic input lexicon (C), which stores orthographic representations of written words, and the phonological output lexicon (E), which stores their corresponding sound. This direct connection enables rapid and accurate reading of words that are already known to the reader, but it cannot be used to read nonwords or new words. The orthographic input lexicon is also connected to the conceptual-semantic system (D), enabling written word comprehension (Castles et al., 2010; Coltheart et al., 2001), but crucially, this is a separate route, and accessing conceptual-semantic information is not necessary for reading aloud in this model.

Nonwords and new words, which are absent from the lexicons, cannot be read through the efficient lexical route, therefore they must be read through the sublexical route (G). This route converts each letter (or multi-letter units such as *ch*, *ea*) to their corresponding sounds (Coltheart et al., 2001).

The final stage of both routes is the phonological output buffer (F), which holds phonological information until it is fully produced, and assembles words from smaller units such as phonemes, syllables, and morphemes (Caramazza et al., 1986; Dotan & Friedmann, 2015; Shallice et al., 2000). Skilled readers employ both routes simultaneously

while reading aloud. Importantly, different types of dyslexia result from impairments in different components of this model.

1.2.1. Examples for information flow in the model

Example 1: Use of the lexical route for reading the word "Talk". In the orthographic analysis stage the letters *t,a,l*, and *k* are identified, and their relative position is encoded: *t* is the first letter, *k* is the final one, and *a* and *l* are in the middle. Each of the four letters is ascribed to the same word. These ordered letters are held for a short time in the orthographic input buffer, and then identified as a familiar string in the orthographic input lexicon. The entry of this word in the orthographic input lexicon is connected to the sound string /tɑ:k/ in the phonological store. These sounds are held for a short time in the phonological output buffer until speech production is completed.

Example 2: Use of the sublexical route for reading the work "Tlak". A nonword undergoes the same orthographic analysis stage, where the letters are identified, ordered, and bound, and then held for a short time. Then, the lexical route is unavailable because this letter string is not previously known. Hence the sublexical conversion route is used, where each letter is converted to its corresponding sound. Each of these sounds is sent to the phonological working memory component, where they are assembled to a sound string and then produced.

1.3. Types of dyslexia

In a cognitive neuropsychological framework, each type of dyslexia is defined by a selective impairment to a component in the cognitive process of unimpaired reading. The type of dyslexia is identified by the disturbance that occurs in the process of reading, i.e., in the types of stimuli that pose difficulties to the patient, and in the type of errors that they make. Dyslexia types can emerge from a deficit in the visual-orthographic analysis stages (A, B) (sometimes termed peripheral dyslexias), or from a deficit in later stage of the reading process (C, D, E, F, G) (sometimes termed central dyslexia) (Ellis, 2016; Ellis & Young, 1996). The types of dyslexia derived by the neuropsychological model presented in Figure 1, the sensitive stimuli, and the characteristic error pattern for each type are presented in Table 1.

1.4. The current study – hypotheses

We hypothesized that various types of dyslexia exist in PPA, as have been previously described in other brain diseases, and as predicted by cognitive neuropsychological models of reading. We thus aimed to evaluate reading abilities in individuals with PPA, as well as to accurately describe the relation between dyslexia and impairment in speech production and comprehension. Since conceptual/semantic representations are not part of the lexical route in this model, and therefore accessing conceptual/semantic information is not necessary for reading aloud (including reading of irregular words), we predict a double dissociation between semantic impairment and surface dyslexia. From the

Table 1. Sensitive stimuli and characteristic errors type for each types of dyslexia.

Dyslexia	Impairment (Impaired component in Figure 1)	Symptoms and error types	Sensitive stimuli	Error example
Letter identity dyslexia, visual letter agnosia[2]	Letter identification (A) or access to it	Letter substitution, omission, addition	Words and nonwords with orthographic neighbors	cat →*bat*
Letter position dyslexia	Letter position encoding (A)	Letter transposition	Migratable words	slime →*smile*
Attentional dyslexia	Letter-to-word binding (A)	Letter migration between words	Migratable word-pairs	form work → *fork worm*
Visual dyslexia	All three functions of orthographic analysis (A)	Letter substitution, omission, addition, transposition, and migration between words	Words, nonwords, migratable word pairs	drink→ *drove*
Orthographic buffer dyslexia	Orthographic input buffer (B)	Letter substitution, omission, addition, transpositions, and migration between words. morphological errors. Length effect	Words, nonwords, long words, morphologically complex words	international→ *intention*
Neglect dyslexia (word level)	Orthographic-attention (A)	Letter substitution, omission, addition on the neglected side	Words and nonwords	disorganized→ *reorganized*
Surface dyslexia	Lexical route (C→E)	Regularization of unpredictable and irregular words	Unpredictable and irregular words	now → *know*
Phonological output buffer impairment	phonological output buffer (F)	Impaired reading aloud, repetition, and production of nonwords, long words, morphological errors in morphologically complex words	Nonwords, long words, morphologically complex words	*stradivate→*vading*
Phonological dyslexia	Letter-to-sound conversion (G)	Letter substitution, omission, addition in nonwords	Nonwords (and words when surface dyslexia is also present)	*snet → *snel*
Vowel dyslexia	A vowel-selective deficit in letter-to-sound conversion (G)	Vowel letter transposition, omission, substitution, and addition in nonwords	Nonwords (and words when surface dyslexia is also present)	*slop → *slip*
Voicing dyslexia (*dyzlegzia)*	A voicing-feature-selective deficit in letter-to-sound conversion (G)	Substitution of graphemes representing voiced consonants with their voiceless counterparts and vice-versa	Nonwords (and words when surface dyslexia is also present)	*vade → *fade*
Morphological dyslexia	A deficit in the morphological sublexical route (morpheme to sound conversion) (G)	Morphological substitutions – inflectional errors (and derivational when reading sublexically)	Morphologically complex words and nonwords, function words	*spawless→ *spawly*
Deep dyslexia	A deficit in the lexical and sublexical routes (C→E & G)	Semantic substitutions, morphological errors, inability to read nonwords, low-imageability words, and function words	Morphologically complex words, low-imageability words, function words, nonwords	wave → *sea*

*indicates a nonword stimulus.

model we also draw that phonological dyslexia can be of two types: a general phonological impairment that also affects production and repetition, and a selective impairment to the letter-to-sound conversion in the sublexical route that does not cause an impairment in production. Thus, we predict that phonological dyslexia can occur without a phonological impairment in production, which defines the logopenic variant.

The current study focused on Hebrew, constituting the first time that dyslexia in PPA has been examined in this language. Most research on reading in PPA has been carried out with English speakers and readers, although there is some pioneering work on other languages including French (Teichmann et al., 2019), Spanish (Wilson & Martínez-Cuitiño, 2012), Portuguese (Senaha et al., 2006), and Chinese (Liu et al., 2022, for a review). While functional impairments that give rise to dyslexia can appear in speakers of any language, the specific manifestations of the impairment may differ depending on the characteristics of the language and its orthography. Hebrew is particularly informative in this respect, since the mapping between different dyslexia presentations and underlying functional impairments has been extensively studied in post-stroke aphasia and developmental disorders, and sensitive tasks are available that can reliably distinguish between different dyslexia types. Hebrew is also a good testing ground for various kinds of dyslexia because of the properties of its language and orthography, such as its rich morphological structure, the under-specification of vowel letters and high rate of homophonic letters which lead to unpredictability in word reading (orthographic depth), and the dense orthographic neighborhood (Friedmann & Lukov, 2008; Khentov-Kraus & Friedmann, 2018).

To test our hypotheses we prospectively tested a cohort of 40 patients with PPA using a test battery of words, nonwords, and word pairs, which was sensitive to identifying at least 13 types of dyslexia and analyzed the types of reading errors each participant made, performing a detailed investigation of dyslexia type for each participant. Additionally, we administered comprehensive language testing, and research-grade cognitive neurological and neuroimaging assessment.

2. Methods

2.1. Participant recruitment

The study's design was prospective, cross-sectional case series. Participants with PPA were recruited as part of a larger study called DIASPORA (Dementia research in Israelis of Adverse Social health determinants and Populations Of underRepresented Ancestry) in Rabin Medical Center, Israel, between January 2021 and March 2024. The study recruitment was designed to meet a community-based cohort through referrals from memory clinics, and its inclusion criteria included 1) age between 40–80 years,[3] 2) new cognitive decline limiting daily activities or independence, and 3) a basic workup to exclude non-neurodegenerative etiology: recent brain imaging (computed tomography or magnetic resonance imaging – MRI) and labs such as a complete blood count, blood chemistry, thyroid stimulating hormone and vitamin B-12. Three additional participants were assessed outside of the DIASPORA study, and were directly referred to reading and language assessment. These participants were diagnosed with PPA after memory clinic comprehensive neurocognitive assessment, brief cognitive testing, and brain neuroimaging. Their records were reviewed in a consensus meeting by two experienced behavioral

neurologists (O.K and A.G). These participants were included in the current study since their PPA diagnosis was confirmed using the same standards as the larger study and they underwent the same reading and language assessment as the rest of the participants. The only difference was that they did not participate in the other components of the larger study.

All participants provided written informed consent for research. The study adheres to the Declaration of Helsinki and was approved by the ethics committees of the Rabin Medical Center (protocol #0128–14-RMC) and Tel Aviv University (protocol #0003893-3).

2.2. Neurocognitive and neuropsychological assessment

The DIASPORA study participants underwent a structured research protocol including informant interview, standardized caregiver self-administered questionnaires, social and neuro-developmental history, childhood learning and reading disabilities, neurological exam, comprehensive neuropsychological assessment, and brain MRI. Neuropsychological assessment included tests of global cognition (MoCA), as well as tests of verbal memory, executive, and visuospatial domains. Language tests included assessment of syntactic production and comprehension, lexical retrieval, and phonological working memory. Decline in independence or instrumental activities of daily living (I-ADL), were measured using the Functional Activity's Questionnaire (FAQ) (Pfeffer et al., 1982), and the Clinical Dementia Rating (CDR). This was supplemented with CDR-FTLD subscales of language (CDR-Language, Knopman et al., 2008).

2.3. Diagnosis of PPA clinical syndrome

All participants met the diagnostic criteria for PPA (Gorno-Tempini et al., 2011; Mesulam, 2001). Aphasia was determined on the basis of the results of the comprehensive language testing and spontaneous speech by two researchers experienced in aphasia and language disorders (N.F and Y.Z.K). Aphasia was determined as the predominantly impaired cognitive domain affecting daily living on consensus by two experienced behavioral neurologists (O.K. and A.G) based on a neurological exam, an informant interview, and results of neuropsychological testing. Alternative etiologies for the disorder, including cognitive, psychiatric, neurological , and other medical conditions, were excluded.

Ten participants with questionable aphasia (CDR-Language subcomponent of 0.5) were excluded, and remaining participants were diagnosed with PPA. Forty participants met core criteria for the diagnosis of PPA, 39 were Hebrew-readers and one was an English-reader. Clinical and demographic information about the participants is presented in Table 2. Although the present study adopts a cognitive neuropsychological approach that goes beyond the tripartite variant classification, participants' classification according to the criteria of Gorno-Tempini et al. (2011) is provided in Appendix B (see Supplementary file) for reference.

Brain MRI scans were initially evaluated independently and then reviewed in a consensus meeting by two experienced neurologists (O.K. and A.G). MRI scans were used to exclude non-neurodegenerative etiologies for aphasia and as an exploratory step toward examining potential relationships between dyslexia type and atrophy patterns in future work. The Harper brain atrophy visual rating scale was applied to each scan to

Table 2. Demographic and cognitive information on participants.

Patient	Sex	Education (years)	Age (years)[a]	Years since onset[a]	Regional grey matter atrophy[b]	loss of independence and severity[c]	CDR-L[d]	MoCA
A.D.	M	>15	53	2	No Atrophy	Mild	1	19
A.E.	F	11	56	5	Anterior Temporal, Parietal, Frontoinsular	Moderate	2	8
A.N.	M	>15	54	0.5	No Atrophy	Mild	1	23
A.U.	F	10	60	2	Frontoinsular	Moderate	2	16
C.O.	F	9	60	0.8	Frontoinsular	Moderate	1	11
D.E.	M	15	65	1	Frontoinsular, Anterior Temporal	Severe	N/A	N/A
D.I.	F	12	75	1	Frontoinsular	Mild	1	10
D.M.	M	12	59	1	Right Parietal (Left Handed)	Mild	1	21
D.P.	M	13	79	3	Frontoinsular	Mild	1	11
E.C.	F	>15	73	3	Anterior Temporal	Moderate	1	N/A
E.M.	M	12	48	5	No Atrophy	Moderate	2	13
E.N.	M	12	67	1.7	Parietal, Frontoinsular	Mild	1	N/A
E.P.	F	12	73	2	Parietal, Frontoinsular	Moderate	1	17
E.T.	F	11	77	5	Frontoinsular	Mild	2	15
E.V.	F	>15	66	2	Posterior Perisylvian/ Parietal, Frontoinsular	Mild	1	15
F.T.	F	12	66	6	Parietal	Mild	1	24
G.H.	M	10	70	2	Posterior Perisylvian/ Parietal, Frontoinsular	Mild	1	16
H.E.	M	9	78	2	Anterior Temporal	Severe	1	14
I.L.	M	10	65	2.3	Parietal, Frontoinsular	Moderate	1	15
I.N.	M	>15	75	2	No Atrophy	Mild	1	N/A
I.V.	F	N/A	65	2.5	Frontoinsular	Moderate	2	16
M.B.	F	15	61	3	No Atrophy	Severe	2	N/A
M.G.	M	12	65	0.3	Parietal, Frontoinsular	Moderate	1	17
N.D.	M	11	55	3	N/A	Severe	2	11
N.E.	M	15	79	4	Posterior Perisylvian/ Parietal	Mild	1	13
N.M.	F	13	77	4	Anterior Temporal, Parietal, Frontoinsular	Severe	N/A	20
P.E.	M	>15	55	2	Anterior Temporal	Mild	1	21
R.E.	F	>15	69	4	No Atrophy	Mild	1	14
S.A.	F	12	62	3	No Atrophy	Mild	1	16
S.E.	M	14	64	4.5	Frontoinsular	Severe	1	9
S.R.	F	>15	61	6	Anterior Temporal	Mild	1	22
S.T.	M	10	59	2	Parietal, Frontoinsular	Mild	1	19
T.E.	M	13	72	2	Parietal, Frontoinsular	Moderate	2	4
T.O.	F	12	64	3	Parietal, Frontoinsular	Moderate	2	10
T.R.	F	N/A	83	0.5	Frontoinsular, Temporal, Parietal	Mild	1	18
T.S.	F	>15	59	2	No Atrophy	Mild	1	17
T.U.	F	13	72	2	No Atrophy	Moderate	2	11
V.D.	M	14	66	2	No Atrophy	Mild	1	13
Y.D.	F	>15	67	5.5	Parietal, Frontoinsular	Mild	2	27
Y.E.	F	15	58	0.7	Frontoinsular	Mild	2	17

Abbreviations: M–Male F–Female, N/A–not available, CDR–Clinical Dementia Rating (L–Language subscale), MoCA–Montreal Cognitive Assessment.

[a]At assessment.

[b]Regions are left hemisphere unless noted otherwise.

[c]Severity of decline in independence on Instrumental activities of daily living (IADL): Mild – Independent or with subjective difficulties performing IADL. Moderate – Requires assistance or supervision on IADL. Severe – Is dependent on others for most IADL.

[d]CDR language impairment levels: 0 = None, 0.5 = Questionable, 1 = Mild, 2 = Moderate, 3 = Severe.

assess brain atrophy (Harper et al., 2016). This scale provides a semi-quantitative measure of atrophy across 12 brain regions (six per hemisphere), with scores ranging from 0 to 3 for the frontal, insular, and parietal regions, and from 0 to 4 for the anterior and medial temporal regions. The Harper scale has been extensively validated for assessing neurodegenerative conditions and clinical syndromes, including FTD and PPA (Falgàs et al., 2024; Illán-Gala et al., 2021). Regional atrophy was classified as positive if the score was ≥ 2 in the anterior insular and parietal regions or ≥ 3 in the anterior temporal regions. Additionally, significant atrophy in the inferior frontal gyrus was also considered positive.

2.4. Comprehensive dyslexia assessment

The identification of multiple types of dyslexia was enabled by a combination of two factors: the use of tests with sensitive stimuli that allowed for the manifestation of the various dyslexia types, and a classification and quantitative analysis of the various types of reading errors (Table 1). These together reflect on the cognitive components within the reading model (Figure 1) that were impaired for each participant, and hence – on the type of dyslexia that the participant had.

2.4.1. Reading tests

The participants were tested with the Hebrew dyslexia screening test called TILTAN (Friedmann & Gvion, 2003). This test includes reading aloud of 136 single Hebrew words, 40 nonwords, and 60 words presented in pairs. The bilingual English speaking participant was tested with a similar English test called FriCasKo with 140 words, 30 nonwords, 45 word-pairs (Friedmann et al., 2010).

Importantly, these reading screening tests were designed with stimuli that uncover each of the various known types of dyslexia (e.g., for detecting letter position dyslexia, the test includes words like "slime" in which an error of transposition of middle letters creates another existing word →"smile", see Table 1). Without such sensitive stimuli, dyslexias can be missed (e.g., a dyslexia manifesting difficulty reading long nonwords would be missed unless such stimuli are presented).

All stimuli were unrelated to each other and presented in isolation, one above the other (not in a sentence, to eliminate the possibility of using syntactic and semantic cues to compensate for reading impairment). Non-words were used to isolate letter analysis and letter-to-sound conversion and avoid the support from the lexicons. Word pairs included two unrelated words and were constructed so that a migration of each letter from one word to the neighboring word (keeping the relative position within the word) created another existing word, to test between-word letter migrations, a symptom of attentional dyslexia (e.g., goat coal->goal coat).

Participants who screened positive for dyslexia were administered additional reading tasks to better characterize their dyslexia type to the extent possible given available time and testing conditions. These additional tests included two single word reading tasks (POTENTIOPHONES – reading of 78 single-words sensitive to surface dyslexia, Friedmann & Gvion, 2003; MIVRAG reading of 160 morphologically complex words; Stark et al., 2018) a word pair reading task (WOPI reading of 32 migratable word pairs, Toledano & Friedmann, 2023), and a nonword repetition task (BLIP – repetition of 48 nonwords, Friedmann, 2003).

The tests were presented to each participant over the desk, printed on a white page. The participant was requested to read the words aloud as accurately as possible. All the sessions were audio-recorded and the two experimenters (authors N.F and Y.Z.K) listened to the recordings after the sessions, and checked, corrected or completed the transcription from the session using the recordings. Words were scored for both correctness, and the type of error made. Error classification was performed by the two experimenters individually and by an additional expert in dyslexia classification. Any discrepancies were then discussed and resolved by consensus. Normative data were obtained from the Language and Brain Lab database, based on Hebrew-speaking healthy volunteers without language impairments tested using the exact same tests and procedure used for the participants with PPA in this study (766 participants for the main screening test; 40 participants for the MIVRAG test; 122 participants for the POTENTIOPHONES test; 62 participants for the WOPI test).

2.4.2. Reading error analysis

Each dyslexia manifests itself in a characteristic pattern of errors, as summarized in Table 1. Therefore, to determine the type of dyslexia of the participants we coded all the error types for all the errors all participants made. For each participant we counted the number of characteristic errors of each type, in each list (words, nonwords, word-pairs, additional tests) and compared it to the normative threshold. The threshold was computed using the case-control comparison t-test (Crawford & Garthwaite, 2002; Crawford & Howell, 1998), at $p < .01$. When a participant had more errors of a specific type than the impairment threshold (meaning they had significantly more errors of a specific type than the norm), in specific types of stimuli, they were classified as having the corresponding type of dyslexia. The types of errors and effects on reading that were the criteria according to which we classified each dyslexia type are summarized in Table 1. For example, a patient who made significantly more letter position errors than the norm was classiged as having letter position dyslexia; a patient who made regularization errors in irregular and unpredictable words was classified with surface dyslexia; a patient who made letter migrations between words was classified with attentional dyslexia. If participants had more errors than the threshold in several error types, they were classified as having several types of dyslexia.[4]

2.5. Additional language assessment

To establish aphasia and to examine the relation between surface dyslexia and semantic impairment, all participants underwent a systematic comprehensive language assessment (beyond the reading assessment), in which they were assessed for syntax, morphology, lexical retrieval, semantic knowledge, conceptual abilities, and phonological working-memory. This assessment, carried out by N.F and Y.Z.K in the Language and Brain Lab, used tests assessing core linguistic abilities including naming (picture naming, Biran & Friedmann, 2004), single-word comprehension (word-picture matching, Friedmann, 2015), object knowledge (picture association, Biran & Friedmann, 2007), sentence repetition (Friedmann, 2000), and sentence production (Friedmann & Novogrodsky, 2002). Motor speech impairment (apraxia of speech) was determined based on spontaneous speech and nonword repetition (Friedmann, 2003). Performance in each task was

compared to normative thresholds generated by the case-control t-test (Crawford & Garthwaite, 2002; Crawford & Howell, 1998). Since we adopt a cognitive neuropsychological approach aimed at identifying the precise linguistic difficulties of each patient according to a cognitive model of reading, we base our classification on the analyses of error types and performance in tasks. This allows us to pinpoint for each participant the exact locus of functional deficit in the reading process described as discrete impairments in specific language functions in the cognitive model, rather than only grouping patients into the prevalent three variants (Gorno-Tempini et al., 2011).

This approach enables us to uncover the full range of dyslexia types in PPA, rather than limiting ourselves to the two types typically identified in the syndromic approach – surface and phonological dyslexia. Moreover, it allows us to empirically test whether surface dyslexia is associated with semantic impairment, without assuming it in our initial classification.

2.6. Statistical analysis

Statistical analyses were conducted using point bi-serial correlation and χ^2-tests to test relations between presence of dyslexia and demographic data. The reading and language tests are all norm-referenced, and the number of each type of error and the performance of each patient was compared to the impairment thresholds in these test (which are based on the case-control comparison method Crawford & Garthwaite, 2002; Crawford & Howell, 1998).

3. Results

3.1. Distribution of dyslexia in PPA

Of the 40 participants with PPA, 33 participants (83%) had some form of dyslexia. No correlation was found between the presence of dyslexia and age ($r_{pb} = .045$, $p = .78$), sex (χ^2 (1, $N = 40$) = .91, $p = .34$), or time since aphasia started ($r_{pb} = .03$, $p = .86$). Only one participant (Y.E.) reported learning difficulties in early childhood and elementary school, which may indicate that she had undiagnosed developmental language or reading disorder.

3.2. Types of dyslexia in PPA

Participants with dyslexia were classified as having one or more types. The following dyslexia types were identified in our cohort: surface dyslexia ($N = 23$), attentional dyslexia ($N = 11$), phonological output buffer dyslexia ($N = 6$), orthographic buffer dyslexia ($N = 5$), phonological (conversion) dyslexia ($N = 3$), vowel dyslexia ($N = 3$), letter-position dyslexia ($N = 2$), visual dyslexia ($N = 2$), voicing dyslexia ($N = 1$), morphological-conversion dyslexia ($N = 1$), and deep dyslexia ($N = 1$). Table 3 details the type(s) of dyslexia for each participant. The number and type of errors for each participant with dyslexia in each task compared to normative data are detailed in Appendix A, Table A1 (see Supplementary file).

Table 3. Type(s) of dyslexia of each PPA participant.

Participant	Type(s) of dyslexia
A.D.	Spared reading
A.E.	Orthographic buffer, surface
A.N.	Spared reading
A.U.	Surface, attentional, phonological buffer
C.O.	Visual
D.E.	Spared reading
D.I.	Surface, phonological conversion
D.M.	Surface
D.P.	Letter position, attentional, surface
E.C.	Attentional, surface
E.M.	Orthographic buffer, surface
E.N.	Attentional, surface
E.P.	Letter position, surface
E.T.	Spared reading
E.V.	Surface
F.T.	Spared reading
G.H.	Voicing
H.E.	Phonological conversion, surface
I.L.	Orthographic buffer, surface
I.N.	Spared reading
I.V.	Spared reading
M.B.	Attentional, phonological buffer, surface
M.G.	Surface
N.D.	Phonological buffer, surface
N.E.	Attentional, phonological conversion
N.M.	Orthographic buffer, surface
P.E.	Surface
R.E.[a]	Attentional
S.A.	Surface
S.E.	Surface, attentional, vowel, morphological conversion
S.R.	Surface
S.T.	Attentional, visual
T.E.	Deep
T.O.	Vowel, surface
T.R.	Attentional, surface
T.S.	Attentional, surface, phonological buffer
T.U.	Attentional
V.D.	Phonological buffer
Y.D.	Vowel
Y.E.	Phonological buffer

[a]R.E. was tested with the English battery.

3.3. Surface dyslexia and semantic impairment

Surface dyslexia, defined by regularization errors in reading aloud in a rate that was significantly higher than that of the control group, was found in 23 participants. We proceeded to testing whether there is a double dissociation between surface dyslexia and semantic/conceptual impairment, as predicted by the model in Figure 1. The model includes a direct lexical route connecting the orthographic input lexicon and the phonological output lexicon, which does not run through the semantic system, therefore, reading via the direct lexical route may be impaired with intact semantic system, and vice versa: the semantic system may be impaired without affecting the lexical route and hence without surface dyslexia. To avoid circularity, given that the diagnostic criteria for the svPPA variant involves surface dyslexia, semantic/conceptual impairment was defined using cognitive neuropsychological measures rather than by the diagnostic criteria of svPPA. Participants were classified as having

a semantic/conceptual impairment if their performance was significantly below controls in naming, together with and impairment in either word comprehension (word-picture matching task) or object knowledge (picture association task), compared to normative data. Only 9 of the participants with surface dyslexia met these criteria for semantic/conceptual impairment, whereas 14 participants presented with a dissociation: surface dyslexia without semantic impairment (A.U., D.I., D.P., E.C., E.N., E.P., E.V., M.B., M.G., P.E., S.A., S.E., T.R., T.S.). On the flip side of the dissociation, 5 participants presented with semantic impairment without surface dyslexia (H.E., N.E., S.T., T.E., Y.E.). Therefore, there is a double dissociation between semantic impairment and surface dyslexia, both defined by cognitive neuropsychological classifications. Considering the full set of features of svPPA (Gorno-Tempini et al., 2011), none of the participants met the criteria to receive a classification. Compliance of participants with surface dyslexia with diagnostic criteria for svPPA is presented in Supplementary Table 1.

3.4. Phonological dyslexia and phonological impairment

Phonological dyslexia is defined by a reading impairment that disproportionally affects nonword reading compared to existing words. A total of 13 participants showed dyslexia that affects nonword reading significantly more than existing words. Our participants showed 5 different kinds of dyslexia especially affecting nonwords: 6 had phonological output buffer impairment, and 7 had impairment in the sublexical conversion itself: 3 had phonological conversion dyslexia (one of them did not have an additional impairment in the phonological output buffer), 3 had vowel dyslexia (one of them also had morphological conversion dyslexia).

Importantly, two of the patients (G.H., N.E.) who showed phonological dyslexia in sublexical conversion (one with phonological conversion dyslexia and one with voicing dyslexia) did not show a concurrent deficit in the phonological output buffer which affects production and repetition and were not classified as having a phonological output buffer impairment in Katz et al. (2026), in which the lexical retrieval abilities of the same cohort was tested. One of them (G.H.) had a deficit in access to the phonological output lexicon from the semantic lexicon, and the other had an impairment in the semantic lexicon, both of which are lexical components that do not affect the reading of nonwords. Namely, at least two of the participants with a disproportionate specific deficit in reading nonwords (aka "phonological dyslexia") did not have a phonological output buffer deficit, which is considered the core impairment in the logopenic variant of PPA.

We also found the other direction of dissociation between phonological production impairment and no phonological dyslexia: In naming, based on the classification in Katz et al. (2026), 18 participants had an impairment in the phonological output lexicon or access to it from the semantic lexicon, impairments that affect lexical retrieval with possible phonological errors and therefore could be classified as lvPPA. Of these patients, only 4 had some type of phonological dyslexia. Therefore, we found a double dissociation between a phonological impairment that affects production (and hence lvPPA) and phonological dyslexia.

Phonological dyslexia is not a part of the diagnostic criteria for lvPPA, but impaired nonword reading is mentioned by Gorno-Tempini et al. (2011) as a possible symptom (see Footnote 1). Examining the full set of diagnostic criteria for the logopenic variant, only two participants with some type of phonological dyslexia met the proposed criteria for

lvPPA. Compliance of participants with phonological dyslexias with diagnostic criteria for lvPPA is presented in Supplementary Table 2.

The diagnostic criteria for lvPPA encompass both impairment to the phonological output buffer, which causes difficulties in nonword reading, and impairment to the phonological output lexicon, which does not. Therefore, it is expected that some patients diagnosed with lvPPA will not have phonological dyslexia. The criteria for lvPPA also include two exclusion criteria ("spared single-word comprehension and object knowledge"; "absence of frank agrammatism") which may be concurrent to a phonological impairment. This may lead to underclassification of lvPPA in patients with a phonological impairment (and possibly phonological dyslexia) who have an additional impairment in the conceptual system, the semantic lexicon, or syntax.

4. Discussion

Using assessment tools that are sensitive to at least 13 types of dyslexia, our results indicate that dyslexia is highly prevalent and diverse in PPA. Of 40 participants with PPA, 33 (83%) had dyslexia. Furthermore, our results indicate that different participants presented with different types of dyslexia. We identified 11 dyslexia types: five types that have never been described in PPA or another dementia, and four types that were only described in case reports of dementia (Caccappolo-van Vliet et al., 2004; Crutch & Warrington, 2007; Saffran & Coslett, 1996; Snowden et al., 2012).

Lastly, we found a double dissociation between surface dyslexia and semantic impairment, and between phonological dyslexia and phonological production impairment.

4.1. Eleven types of dyslexia identified in PPA, five of them for the first time

Our results bring forward a novel perspective on dyslexia in PPA by discovering the frequency and variability of dyslexia typology in PPA. Whereas previous reports focused on surface and phonological dyslexia (Brambati et al., 2009; Gorno-Tempini et al., 2008, 2011; Luzzatti et al., 2016), our results indicate that there are at least 11 different types of dyslexia in PPA. We have identified five dyslexia types never reported in PPA or any type of dementia: letter position dyslexia, orthographic buffer dyslexia, and three selective impairments in the sublexical route: morphological conversion dyslexia, voicing dyslexia, and vowel dyslexia. In addition, we found in our cohort four dyslexia types that were previously described in case reports of individuals with dementia, but are otherwise under-reported in PPA: attentional dyslexia (Saffran & Coslett, 1996), phonological conversion dyslexia (Caccappolo-van Vliet et al., 2004), deep dyslexia (Snowden et al., 2012), and visual dyslexia (Crutch & Warrington, 2007). We also found surface dyslexia and phonological dyslexia, which have been widely studied in PPA.

Whereas these results are novel in PPA, they are consistent with previous research on aphasia and dyslexia in non-neurodegenerative diseases such as cerebrovascular or other brain diseases. These studies document all of our identified dyslexia types and across various languages: English, Italian, French, Arabic, Bengali, Portuguese, Turkish, Spanish, Hebrew, and more (Coltheart, 1981; Friedmann & Coltheart, 2018; Friedmann & Gvion, 2001; Friedmann et al., 2010, 2012; Güven & Friedmann, 2021; Gvion & Friedmann, 2010; Humphreys et al., 1990; Johnston, 1983; Khentov-Kraus & Friedmann, 2018; Potier Watkins

et al., 2023; Shallice & Warrington, 1977). The similarity of our findings to those found in non-neurodegenerative diseases is not surprising, as reading is a cognitive ability and thus localizes to neuroanatomical networks regardless of the underlying pathology (e.g., vascular vs. neurodegenerative).

We ascribe the difference in our study from previous PPA research to the methodology employed in assessing dyslexia, in particular to two factors: test sensitivity and error analysis. We used tasks designed for the identification of various types of dyslexia based on a theoretical model, using stimuli sensitive to different types of dyslexia. These tasks have been previously used to study dyslexia in non-neurodegenerative diseases. We then employed error analysis for each incorrect response each patient made. This allowed us to not only identify that the patient has a reading impairment, but also to pinpoint the locus of the deficit in the reading process.

4.2. Surface dyslexia and semantic impairment

We found a double dissociation between semantic impairment and surface dyslexia (both defined by cognitive neuropsychological measures): there were 14 participants with surface dyslexia without a semantic impairment, and 5 participants with a semantic impairment without surface dyslexia.

Surface dyslexia results from a deficit in the lexical route which, as depicted in the neuropsychological model in Figure 1, comprises of the orthographic lexicon and the phonological lexicon. Individuals with impairment in each of these components (or the connection between them) present with surface dyslexia (Friedmann & Lukov, 2008). If the deficit is in the phonological lexicon, it will present with both naming deficits and surface dyslexia (Gvion & Friedmann, 2016). However, this naming impairment is not due to a semantic deficit, as it does not involve a deficit in the semantic lexicon and hence no deficit in word comprehension.

Accessing semantic/conceptual information about a word is not a part of the lexical route for reading aloud in our model, and is therefore not necessary for reading irregular words (Friedmann et al., 2017). Thus, as we hypothesized, we found individuals with surface dyslexia without a semantic deficit (as they may be impaired in the orthographic/phonological lexicons without a deficit in the semantic system), and individuals with a semantic deficit without surface dyslexia (as they are able to use the direct lexical route, bypassing the semantic lexicon). A similar conclusion was reached by Teichmann et al. (2019), who found that surface dyslexia in French-speaking patients with svPPA is related to their lexical abilities rather than their semantic abilities. While this result is predicted by the reading model in Figure 1, other models of reading and semantic processing posit a semantic contribution to reading irregular words, and therefore they may predict that semantic impairment will cause surface dyslexia. In the triangle model of reading (Plaut et al., 1996) and its implementation for semantic dementia (Woollams et al., 2007) infrequent irregular words are read with support of the semantic system, and therefore damage to the semantic system is predicted to cause surface dyslexia. Similarly, in the summation approach (Hillis & Caramazza, 1991, 1995) semantics plays a role in reading irregular words as the output component sums the information received from the sub-lexical route and from the semantic component, which together activate the correct representation in the phonological output lexicon. The findings that some individuals

have a semantic impairment without surface dyslexia suggest that irregular words can be read without the involvement of semantic components, at least for some readers, contrary to the prediction of the triangle model and the summation approach.

Hebrew is an ideal testing ground for surface dyslexia and its relations to other linguistic abilities since its orthography underspecifies stress position and most vowels in mid-word positions, and because of its high rate of homophonic letters. Therefore, none of the words can be read accurately relying only on grapheme-to-phoneme conversion. In this sense all Hebrew words are unpredictable. Thus, the orthography of Hebrew makes it easier to identify surface dyslexia, and hence to explore and detect the dissociation between surface dyslexia and semantic impairment.

The inclusion of surface dyslexia as a symptom of semantic variant PPA dates to research on Semantic Dementia. Several studies have found an association between Semantic Dementia and surface dyslexia (Hodges et al., 1992; Patterson & Hodges, 1992; Snowden et al., 1992; Warrington, 1975; Woollams et al., 2007). However, many other studies have previously found a dissociation, i.e., semantic impairment without surface dyslexia, in semantic dementia in English (Blazely et al., 2005; Cipolotti & Warrington, 1995), Spanish (Wilson & Martínez-Cuitiño, 2012), and Portuguese (Senaha et al., 2006), as well as in Alzheimer's disease (Lambon Ralph et al., 1995). There are also previous reports of surface dyslexia without a semantic impairment (Watt et al., 1997), and the current study corroborates these findings.

To conclude, we propose that the finding that there is a double dissociation between surface dyslexia and semantic impairment is not specific to Hebrew and its orthography which involves significant unpredictable mapping between written and spoken form of words, as both sides of the dissociation have previously been reported for other languages. While certain languages like Hebrew, in which almost every word is unpredictable, make surface dyslexia easier to detect, we propose that the dissociation between surface dyslexia and semantic impairment reflects a testing advantage for these languages rather than a true cross-linguistic difference in how semantic impairment presents.

The finding is readily explainable under the neuropsychological model we used, as a direct route for reading words exists, which does not require semantics (Figure 1 C→E). Thus, whereas a deficit in the lexical route leading to surface dyslexia may occur concurrently with a semantic deficit, functionally, they are separate and do not need to occur together.

None of the participants with surface dyslexia (in fact, no participant in the study) met the diagnostic criteria for svPPA, even though 18 participants had a clear semantic or conceptual impairment (Katz et al., 2026). We propose that this is largely due to the exclusionary nature of the supporting criteria. Two of the four supporting features for svPPA (of which at least three must be present) are "intact repetition" and "intact speech production". These criteria do not characterize the semantic impairment itself but rather exclude patients with additional phonological or syntactic deficits. This is problematic for two reasons. First, phonological or syntactic impairments can co-occur with semantic impairment. Since neurodegeneration often produces several deficits at once, a patient with a genuine semantic impairment may be excluded from the svPPA classification on the basis of an additional deficit. While this may be useful for identifying "pure" cases of svPPA, a cognitive-neuropsychological standpoint, such as the one we adopt here,

focuses on functional impairments (which can be multiple) rather than on prototypical syndrome profiles. Second, semantic impairment itself can disrupt production and repetition through its direct effect on lexical retrieval, which is essential for both production and repetition.

Our central claim here, however, is not about the characterization of the semantic variant, but about the cognitive mechanisms underlying the reading of irregular words and their independence from semantic abilities.

4.3. Phonological dyslexia and phonological impairment

"Phonological dyslexia" is defined as a disproportionate deficit in reading nonwords in comparison to existing words (Ellis & Young, 1996). Such deficit may arise either from a deficit in the phonological output buffer, or from a deficit in letter-to-sound conversion in the sublexical route (Barbieri et al., 2011; Friedmann & Coltheart, 2018). Whereas the phonological output buffer is also involved in phonological production in spontaneous speech, naming, and repetition, the sublexical conversion route is specific only to reading. In our study, at least two of the PPA patients who were diagnosed with phonological dyslexia that results from a deficit in letter-to-sound conversion did not show a deficit in the phonological output buffer. These two participants, then, show phonological dyslexia without a phonological output deficit. Phonological dyslexia without phonological impairment was previously described in a patient with probable Alzheimer's, who had phonological dyslexia caused by impaired letter to sound conversion Caccappolo-van Vliet et al.

Another reason that phonological dyslexia is not always associated with a phonological impairment to production is that there are several different types of phonological impairments in production. Namely, the logopenic variant of PPA encompasses not only impairment to the phonological output buffer, but also impairment to the phonological lexicon and access to it from the semantic lexicon (Katz et al., 2026). When the phonological lexicon or the access to it is impaired, patients may be diagnosed with a phonological impairment (i.e., they have impaired naming, possibly with phonological errors), but they will not necessarily have phonological dyslexia. In the current cohort, 18 participants had impairment in the phonological output lexicon or access to it from the semantic lexicon (based on impaired naming with characteristic error types, such as phonological errors and self-corrected semantic errors). Of them, only four had some type of phonological dyslexia, meaning that 14 patients had a phonological output lexicon impairment without phonological dyslexia. In fact, individuals with a deficit in the phonological output lexicon, which is part of the lexical route, may show surface dyslexia (Coltheart & Funnell, 1987; Friedmann & Lukov, 2008; Gvion & Friedmann, 2016).

In sum, the association of lvPPA with phonological dyslexia has two major shortcomings: phonological dyslexia may not involve phonological production and may be caused by an impairment to the sublexical conversion route; and phonological production impairments can stem from either the phonological output lexicon or the phonological output buffer, and only the latter causes impairment in reading nonwords. Additionally, the supporting criteria for lvPPA, like the ones for svPPA, might exclude participants who have a syntactic impairment in addition to their phonological impairment (through the

"absence of frank agrammatism" feature), leading to fewer patients diagnosed with lvPPA when the criteria are taken at face value.

This study showed a double dissociation between a deficit in phonological output and phonological dyslexia: phonological output deficits (in the phonological lexicon) without phonological dyslexia, and intact phonological output with phonological dyslexia (in the sublexical conversion process).

4.4. Strength and limitations

The current study has several important strengths. A rigorously selected large PPA cohort diagnosed using research-grade neurocognitive and neuroimaging assessment. All participants underwent a comprehensive language battery, including tests designed to identify dyslexia type. All errors underwent further meticulous error analysis. The stimuli used in the reading tests and the neuropsychological-theory-motivated error analysis allowed for the diagnosis of a wide range of dyslexia types. This methodology is not novel, and is widely used to study dyslexia in non-neurodegenerative diseases, and across multiple languages.

Our study has several possible limitations. First, since dyslexia commonly occurs as a neuro-developmental disorder, it is possible that some of our participants had pre-existing undiagnosed developmental dyslexia. However, this is unlikely due to several reasons. Only one participant disclosed childhood learning difficulty compatible with dyslexia in the severity we asses for. Moreover developmental dyslexia is much less prevalent (4%-7%, Lishman, 2003) than in our PPA cohort.

Secondly, we did not test impairments in earlier visual-spatial stages that affect reading only under certain visual conditions, reported in the literature on non-neurodegenerative conditions, such as crowding dyslexia (Daini et al., 2021, 2025). These types of dyslexia should be directly tested in future research by including relevant stimuli in reading tests (e.g., spacing manipulation for identifying crowding dyslexia).

Another suggested limitation is testing PPA participants who are native Hebrew speakers. Hebrew is a Semitic language with marked differences from European languages. This may, in theory, limit the generalization of our results. Nevertheless, we believe our findings of multiple types of dyslexia and of the high prevalence of dyslexia are unlikely to result from testing Hebrew native speakers. All of the dyslexia types we identified in PPA were previously reported in non-neurodegenerative diseases in English and other languages (Coltheart, 1981; Friedmann & Coltheart, 2018; Friedmann & Gvion, 2001; Friedmann et al., 2010, 2012; Gvion & Friedmann, 2010; Humphreys et al., 1990; Johnston, 1983; Khentov-Kraus & Friedmann, 2018; Shallice & Warrington, 1977). It is highly unlikely that dyslexia types are manifested so broadly across brain diseases speaker (R.E.) was found to have attentional dyslexia, previously under-described in PPA. Indeed, the orthographic and linguistic properties of each language necessitate choosing specialized stimuli that will uncover potential reading impairments in that language. This approach has been shown to accurately identify previously unreported dyslexia in Turkish, Italian, French, and Arabic. Extending aphasia research into diverse languages helped support or modify neuropsychological models of all language related tasks. In this regard, the inclusion of Hebrew or any other diverse language should be viewed as a strength (Franzen et al., 2023).

Finally, our cohort included no participant who met the full diagnostic criteria for svPPA. This cannot be ascribed to inability to detect semantic impairments using our tools, nor to absence of patients with semantic impairment in our cohort, as our cohort did include 18 patients with conceptual/semantic deficits. As discussed in Section 4.2, they did not receive the svPPA classification primarily due to the exclusionary nature of the supporting criteria, so that patients who did have a semantic deficit but also showed additional difficulties in sentence repetition and production could not receive the svPPA diagnosis. This does not affect our claim about a double dissociation between surface dyslexia and semantic impairment, which was determined on the basis of the existence and absence of a semantic deficit in comprehension and production, and of regularization errors in the reading aloud of unpredictable words. Such analysis was not based on the diagnostic criteria for PPA variants but rather pertained to the cognitive mechanisms underlying reading and lexical/conceptual processing, showing that reading aloud of words can proceed without semantics, and semantics can be preserved even when the lexical route for reading is impaired.

5. Conclusions

In summary, we show that individuals with PPA are likely to present with dyslexia alongside an impairment in language production and comprehension, that dyslexia types in PPA can vary greatly and are not limited to the surface or phonological types, and that over-reliance on surface and phonological dyslexia as a criterion for semantic or phonological impairment can lead to misclassification of the underlying cognitive impairment. Future research should aim to replicate our findings in additional languages by using the relevant stimuli, to assess the prevalence of different types of dyslexia in other neurodegenerative syndromes, and to further examine the relation between dyslexia and the production and comprehension impairments different PPA profiles. Reading, as a cognitive task, has a central role in daily activities, in communication and independence. Careful assessment of reading impairments in clinical settings, informed by the range of dyslexia types that can appear in PPA, has several advantages. First, reading tests are relatively easy to administer: they involve simple instructions and take little time to administer. Given the high prevalence of diverse dyslexia types in PPA, dyslexia screening could serve as an efficient initial screening tool for PPA. Second, knowing each patient's exact difficulty in reading may improve written-communication with the patient by caregivers and care-providers. Lastly, an accurate diagnosis may lead to interventions tailored for the specific type of dyslexia (e.g., Coltheart & Byng, 1989; Friedmann & Rahamim, 2014; Lott et al., 1994; Nickels, 1995; Shvimer et al., 2009), which is expected to mitigate communication difficulties and positively affect quality of life.

Notes

1. Phonological errors in reading are mentioned by Gorno-Tempini et al. (2011) as a symptom of lvPPA in Table 5 titled "Tasks that may be used to assess speech and language functions in PPA" under the reading/spelling task (p. 1011).
2. Some dyslexia types have overlapping error patterns, with the characteristic errors of one type forming a subset of those of another. For example, both letter-position dyslexia and

orthographic input buffer dyslexia may involve letter transpositions within words, but only the latter also includes additional error types such as letter migrations between words and morphological errors. When the participant exhibited the more inclusive (superset) error pattern, they were classified with the more inclusive dyslexia type only. In the example presented here, if the participant exhibited letter transpositions within words as well as letter migrations between words and morphological errors, they would be classified as having orthographic input buffer dyslexia rather than letter-position dyslexia.

3. Another term that has been used in the literature is "pure alexia", which refers to several different patterns of impairments or their combination. It may correspond to visual letter agnosia (Miozzo & Caramazza, 1998), word-form dyslexia (Shallice, 1988; Warrington & Shallice, 1988), or simultanagnosia for letters (Farah, 1990).
4. A lower bound was set as a heuristic to reduce the likelihood of non-neurodegenerative causes for language impairment, in practice the youngest participant was 48 years old, and there was one participant outside of the larger study who was older than 80.

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Data availability statement

The authors confirm that the data supporting the findings of this study are available within the article.

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