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Understanding Theory of Mind–pragmatics relationship through the spectrum of neurodegenerative diseases

Olga Ivanova
University of Salamanca
ORCID: 0000-0002-9657-5380

Abstract

Our understanding of how pragmatic skills change in ageing is not systematic. Even less is known about the effects of pathological ageing—particularly in the context of neurodegenerative diseases—on pragmatic competence, changes in Theory of Mind and the potential inter-relationship between these domains. Given that pragmatic competence relies on a wide range of cognitive functions, examining pragmatic changes in speakers with neurodegenerative diseases may clarify whether these alterations are inherently pragmatic or arise from deficits in other cognitive domains, such as impairments in Theory of Mind. In this work, I inquire into how eight neurodegenerative conditions (behavioural variant frontotemporal dementia, semantic dementia, Alzheimer's disease, amnesic mild cognitive impairment, Parkinson's disease, Lewy body dementia, cerebellar neurodegenerative disorders and right temporal degeneration) compromise pragmatic abilities and explore whether such impairments are linked to changes in Theory of Mind. Based on a narrative review of available experimental evidence, I come to several conclusions. First, only one neurodegenerative disease (behavioural variant frontotemporal dementia) shows up with pragmatic deficits directly linked to impairment in Theory of Mind. In the rest of the conditions, pragmatic deficits, although linked to Theory of Mind, derive from impairments in other cognitive functions (such as working memory or executive functions). Overall, pragmatic abilities are better predicted by their internal gradation (which abilities impose more cognitive load) than by the neural pathways of the diseases.

1. Introduction

How do pragmatic skills change during late adulthood, and how are these changes related to impairments in Theory of Mind (ToM)? Viewed one-way, pragmatic competence—considered a component of crystallized intelligence—shows notable robustness and resilience during ageing, predictably owing to the expertise acquired over the lifespan and the need to

compensate for declines in fluid abilities (Baltes, 1993; Daniluk & Borkowska, 2020; Staudinger et al., 1989; Verschueren et al., 2006). Unlike fluid abilities—defined as the capacity to solve a wide range of problems quickly and effectively and thus considered to be an 'adaptative behaviour'—crystallized intelligence is measured as a factor of accumulated knowledge (Hills, 2025; Salthouse, 2012), which frequently becomes stronger in late adulthood.

Alternatively, there is evidence that some pragmatic skills in late adulthood are compromised (cf. Messer, 2015). One possible explanation for why only some pragmatic skills change in ageing while others do not is supported by the analysis of general cognitive decline. As ageing mainly affects cognitive functions involved in cognitive flexibility, it might lead to impairments in those pragmatic skills that are most dependent on them (cf. Bambini et al., 2021; Saryazdi et al., 2022). For example, ageing speakers present with decline (and even impairment) in the pragmatic use of language (mainly, inferences, figurative language, humour, prosody adjustment or conversation organization), non-verbal and paralinguistic pragmatics (including facial expressions, gestures or their integration with speech), and social and contextual pragmatics (supporting conversational rules or speech act selection) (Hilviu et al., 2022).

A central question in current research is what causes these changes in pragmatic ability during ageing. It remains unclear whether ageing damages pragmatic competence itself or whether it predominantly affects the cognitive functions supporting pragmatics (e.g. ToM), thereby leading to secondary alteration. One way to learn more about pragmatic change in late adulthood is to approach it by focusing on speakers with attested cognitive impairments. If pragmatic impairment in ageing is cognition-dependent, then pragmatic abilities will be compromised as much as the cognitive functions underpinning them are, by supporting the theoretical dichotomy between fluid and crystallized knowledge (cf. Burke, 2006).

In this context, changes in ToM may provide unique insights into how ageing affects pragmatic abilities. ToM, often referred to as mentalizing, is considered central to pragmatics, primarily owing to its crucial role in meaning reconstruction (cf. Grigoroglou & Papafragou, 2019). Although it is risky to equate ToM solely with mentalizing—an issue thoroughly discussed by Quesque and colleagues (Quesque et al. 2024)—in this paper, I adopt this perspective given its widespread use in research on lifelong development and, more specifically, ageing (Lecce et al., 2019; Peters & Schulz, 2022). Thus, under the definition adopted in this paper, ToM is understood as a cognitive capacity that enables speakers to appropriately direct their behaviour in communication by facilitating the attribution of mental states. In recent years, several

attempts have been made to untangle how both ageing-related changes (Bambini et al., 2021; Buschetti et al., 2023; Calso et al., 2020) and clinical conditions in adulthood (Bosco et al., 2017; Champagne-Lavau & Joanette, 2009; Ouerchefani et al., 2024; Pluta et al., 2017; Tsolakopoulos et al., 2023).

An important clarification to be made here is that pragmatic abilities and ToM cannot be conceptually conflated nor can pragmatic tasks be regarded as direct measures of ToM (Bosco et al., 2018). However, there is an important relationship between pragmatic abilities and ToM that underpins efficient communication. Pragmatic abilities require speakers to interpret explicit and implicit contextual clues surrounding them continuously during communication and adapt their response, whether verbal or non-verbal, accordingly (Ibañez & Manes, 2012). As such, pragmatic abilities require speakers to manage multiple phenomena, from speech act and genre selection or turn-talking organization to more meaning-related ones (identification and interpretation of non-literal meanings, implicatures, humour or references (cf. Airenti, 2017)), adequately selecting and using different language tools in line with the contextual cues (Turkstra et al., 2017). To be activated and put into practice, pragmatic abilities substantially depend on the speaker's ability to engage a range of cognitive processes, not excluding motor or sensorimotor abilities. The former involve such general cognitive functions as selective attention, short-term and long-term memory, and perception, but also other cognitive functions specifically supporting pragmatic processes and responsible for mental and affective state attribution: mainly, ToM and executive functions (EF) (Cummings, 2017; Saryazdi et al., 2022). Thus, although pragmatic abilities and ToM are two distinct faculties (cf. Bosco et al., 2018), both play a crucial role in effective communication—a relationship that is particularly evident in the context of language disorders, where ToM deficits are frequently associated with impairments in pragmatic functioning (Cummings, 2013).

With this in mind, this work focuses on the relationship between ToM and the implication of its pragmatic impairment across various ageing patterns associated with neurodegenerative processes. Research on the relationship between cognitive impairment in neurodegenerative diseases, pragmatic ability and ToM has been limited. A pioneering study by Cuerva et al. (2001) identified a relationship between pragmatic deficit and ToM in Alzheimer's disease (AD). However, the study also revealed that not all speakers with AD presented with such deficits and that their severity correlated with impairments in other cognitive functions (such as memory, comprehension, thinking or naming). More recently, a comparative study by Shany-Ur et al. (2012), examining pragmatic impairment and ToM across four

neurodegenerative profiles—behavioural variant frontotemporal dementia (bvFTD), AD, progressive supranuclear palsy and vascular cognitive impairment—yielded two significant conclusions. On one hand, the authors observed a pronounced impairment in ToM and pragmatic abilities in individuals with bvFTD specifically. This severe impairment was likely associated with the focal degeneration in brain areas responsible for ToM. On the other hand, they found that in all other neurodegenerative conditions, the degree of pragmatic performance was predicted by the overall degree of cognitive impairment. Consistent with this observation, a comparative study by Tsentidou et al. (2021) of ageing speakers with vascular risk profile and mild cognitive impairment (MCI) found that both groups exhibited similar impairments in ToM-based pragmatic abilities too. Taken together, these findings suggest that ToM-based pragmatic abilities may impose a particularly high cognitive load, thereby becoming among the first functions to fail across the neurodegenerative spectrum. Against this background, the aim of the present work is to expand upon previously reported findings by comparingthe ToM–pragmatics relationship across eight neurodegenerative patterns with different cognitive profiles.

2. Neurodegenerative diseases as a window to understanding Theory of Mind–pragmatics relationship

2.1. Neurodegenerative diseases, dementia and pragmatic abilities

To explore ToM–pragmatics impairment across the spectrum of neurodegenerations, in this paper, I will employ a narrative review of recent literature on the intersection of cognitive impairments in neurodegenerative ageing conditions and associated pragmatic impairments. The term ‘neurodegenerative disease’ refers to any disorder defined by a progressive loss and dysfunction of neuronal populations, usually occurring in localized brain areas as a result of protein depositions (Kovacs, 2018). Typically, neurodegenerative diseases result in progressive, mild-to-severe cognitive and/or motor impairments (Sumien et al., 2021). It is generally assumed that age is one of the strongest risk factors for the onset of a neurodegenerative disease (Armstrong, 2020; Farooqui & Farooqui, 2009), but recent consensus reports (see Livingston et al., 2024) highlight the crucial role of other protective and risk factors with a direct impact on when and how deterioration begins. With this in mind, a predominant part of neurodegenerative diseases, despite their heterogeneity, develop during ageing or in the near-ageing stage (Hou et al., 2019; Sumien et al., 2021).

To a varying degree, most ageing-related neurodegenerative diseases lead to dementia, i.e. to cognitive and functional disorders affecting the speaker's cognitive abilities (such as memory, thinking, attention, etc.), task performance, personality or behaviour (cf. McKhann et al., 2011). Although an outdated term (following recommendations from various expert groups, DMS-5 replaced 'dementia' with 'major neurocognitive disorder' (MNCD) to describe the condition previously encompassed by the former term and to update its typological and aetiological criteria (cf. Sachdev et al., 2014), 'dementia' remains widely used in both specialized literatura and broader society. Specifically, it continues to be a key term for tracing descriptions in research, particularly in earlier studies, and both for this reason and to maintain consistency, I will use 'dementia' throughout this paper to refer to cognitive syndromic presentations arising from any neurodegenerative condition (drawing on the recent Framework for Dementia Nomenclature from Petersen et al. (2023)).

Since dementia can result from different neurodegenerative patterns, it is commonly used as an umbrella term to refer to any impairment caused by brain disorders. However, it is also acknowledged that different dementias often exhibit similar cognitive presentations (Keenan et al., 2016). The most common neurodegenerative causes of dementia are AD, Parkinson's disease (PD) (although, in the case of PD, it would be necessary to distinguish between the condition affecting the cognitive domain and those affecting only the motor domain), vascular dementia, FTD and its subtypes, mixed dementia, Lewy body dementia (LBD) and Huntington's disease. Dementia can also develop, although with a lower frequency, in neurological conditions such as amyotrophic lateral sclerosis and motor neuron disease (MND) or metabolic conditions such as diabetes mellitus. Yet what the mechanisms underlying the association of non-degenerative causes and cognitive impairment are is still a debated question. In this paper, I will only focus on the neurodegenerative causes of dementia and their relationship with pragmatic impairment.

It has been traditionally assumed that pragmatic impairment is more pronounced in clinical conditions involving right-hemisphere (RH) damage (Frank, 2018; Martin & McDonald, 2003). This idea has been supported by neuroimaging evidence suggesting that RH focal alterations correlate with pragmatic difficulties (Foldi et al., 1983; Siegal et al., 1996). Yet, more recent evidence suggests that the neural underpinnings of pragmatic abilities are much more complex and dynamic (as pragmatic abilities themselves) and that they recruit an extensive network in both the RH and left hemisphere (LH) (Blake, 2017; Stemmer, 2017). In this scenario, the

neural underpinnings of ToM are specifically engaged in a distributional left–right neural network, which partially coincides with the language neural network (Kobayashi et al., 2008; Wakusawa et al., 2007), the intention processing network supporting communicative intention processing (Enrici et al., 2019) and the pragmatic language neural network (Reyes-Aguilar et al., 2018). Importantly, evidence from neurodegenerative conditions suggests that ToM impairment correlates with abnormal connectivity within the default mode network, the frontoparietal network and the salience network, pointing to their roles in social and adaptative behaviour (Schimmelpfennig et al., 2023; Trojsi et al., 2017).

The neurodegenerative diseases causing dementia affect different neural pathways or loci, with pragmatic ability potentially being affected differently too. In addition, while some dementias directly affect language, others are non-language-led. This is a crucial point since changes in pragmatic abilities (including ToM-related abilities) can be both independent of language abilities (Fittipaldi et al., 2019; Van Herwegen et al., 2013) and correlate with them (Cavallini et al., 2013; Smogorzewska et al., 2018).

2.2. Understanding the relationship between dementia, Theory of Mind and pragmatic impairment: about this study

To address the proposed question, I will rely on scientific evidence located in PubMed through a search formula including the following keywords: Theory of Mind OR ToM; language OR pragmatic; aging OR neurodegenerative OR neurodegeneration OR dementia OR cognitive impairment. In selecting these keywords, I aimed to include all terms that could cover the máximum amount of published research, as being the most generic and widely used so far (such as the term ‘dementia’, as opposed to MNCD).

After obtaining 217 results, I selected 25 papers based on the following inclusion criteria: (i) studies addressing human ageing speakers at any neurodegenerative condition in ageing and (ii) studies reporting original experimental research. Studies addressing other lifespan groups, healthy populations or non-humans (e.g. primates) or reporting meta-analysis or reviews were excluded. Furthermore, I put no limitations regarding the year (studies range from 2001 to 2024) and the language of publication.

Selected studies address AD (n = 6), amnesic MCI (aMCI) (n = 2), bvFTD (n = 7) and semantic dementia (SD) as its specific subtype (n = 2), PD (n = 5) (all of them considered speakers with

compromised cognitive functions), LBD (n = 1), cerebellar neurodegenerative disorders (CND) (n = 1) and right temporal degeneration (RTD) (n = 1) (see electronic supplementary material for a detailed list of selected studies). For the analysis, I shall group them according to clinical criteria: (i) bvFTD and SD; (ii) AD and aMCI; (iii) PD; and (iv) low-prevalence conditions, including LBD, CND and RTD. I complemented the selected studies with additional sources identified through the screening of reference lists.

3. Theory of Mind–pragmatics relationship in neurodegenerative pathways in ageing

3.1. Frontotemporal dementia and its types

FTD is a neurodegenerative syndrome that causes changes in personality and social cognition (Neary et al., 2005). It leads to disinhibited behaviour, which can be either compulsive or apathetic and often includes stereotyped actions (Torralva et al., 2009). FTD is characterized by difficulties in ToM, although the pattern and severity of such impairment depend on the FTD type, usually subdivided into bvFTD and SD.

Speakers with bvFTD are not usually impaired in language and memory; contrastingly, impairments in social cognition, ToM and decision-making are key clinical characteristics. These cognitive impairments expectedly correlate with the pattern of neural atrophy in bvFTD, which primarily occurs in prefrontal and frontal areas (Gregory et al., 2002) that are particularly involved in supporting ToM (Irish et al., 2014). Impairments in social cognition and ToM lead speakers with bvFTD to struggle with affective interpretation and the adequate use of communicative and pragmatic norms. They are typically reported to have difficulty in recognizing facial emotions and adjusting empathically (Oliver et al., 2015). Furthermore, they exhibit deficits in understanding figurative language and adhering to interpersonal and conversational norms (Ibañez & Manes, 2012). In pragmatic performance, these deficits lead to several dysfunctions too. On the one hand, speakers with bvFTD struggle to identify sarcasm, deception and certain types of lies (Kosmidis et al., 2008; Shany-Ur et al., 2012), and they generally tend towards literal and concrete comprehension (Lee et al., 2012). As a result, they literally interpret scalar implicatures (Spotorno et al., 2015) and, at times, cannot comprehend indirect speech acts (Healey et al., 2021) or the discourse meaning itself (Luzzi et al., 2020).

Against bvFTD, speakers with SD, who are defined by lesions in the temporal lobes and are significantly affected in language (mainly, in semantic tasks) and socioemotional abilities (mainly, in empathy) (Irish et al., 2014), have received little attention concerning their ToM–pragmatic abilities because it is still not clear whether ToM is essentially impaired in SD. The very few available studies suggest that speakers with SD can be thematically and formally irrelevant in communication, including stereotypic, unrespectful and disinhibited participations (Harciarek & Cosentino, 2013), but it is not clear whether this type of pragmatic impairment can be attributed to ToM disruption.

3.2. Alzheimer's disease and amnesic mild cognitive impairment

AD is the most common cause of dementia, leading to changes in episodic memory, semantic memory, language and speech (Lindeboom & Weinstein, 2004; Verma & Howard, 2012). The specific neurodegenerative pattern of AD does not significantly affect the ToM network, with overlap occurring only in the temporal parietal junction (TPJ) and posterior cingulate (Laisney et al., 2013). Consequently, AD is not characterized by a primary deficit in ToM; instead, ToM functions are disrupted in AD when related cognitive functions (such as memory or EF) are primarily compromised (Laisney et al., 2013). In fact, disruptions in cognitive functions, including not only memory but also inhibition or processing, predict both interactive and meaning aspects of pragmatic abilities in AD.

On an interactive level, AD speakers may have difficulties in adequately interpreting social relations and behaving appropriately. Simm et al. (2017) attributed these difficulties to deficits in self-monitoring and self-reflection, which stem from primary deficits in processing speed, learning abilities and inhibition. Disruption in metalinguistic abilities (Rosell-Clari & González Valles, 2016) could also be related to these cognitive dysfunctions. Interestingly, Schild et al. (2021) suggested that the cognitive load imposed by a task predicts how well AD speakers would perform it. Considering that performance on affective ToM is less impaired in AD (cf. Baksh et al., 2024) and that disruptions in cognitive ToM mainly affect the most complex tasks (e.g. making inferences) (Laisney et al., 2013), it seems plausible to propose a gradational, non-primary affection of ToM–pragmatics in AD.

Despite a less pronounced impairment of ToM, speakers with AD exhibit difficulties in understanding metaphors and sarcastic expressions, preferring literal interpretations (Maki et al., 2013). Evidence on which types of metaphors are impaired in AD is not systematic,

suggesting that either only novel metaphors (Amanzio et al., 2008) or both novel and conventional metaphors are disrupted (Maki et al., 2013). Maki et al. (2013) explained the disruption in conventional metaphors by the decrease in their use in pathological ageing, leading to their processing as novel metaphors. On its part, the tendency towards literal interpretation would be attributed to the deficit in inhibiting competing (i.e. literal) meanings.

Crucially, this tendency towards non-literality is already observed in aMCI, the most probable precursor of AD (Lindeboom & Weinstein, 2004). Since speakers with aMCI have difficulties in understanding irony (Gaudreau et al., 2013), a function that already declines in healthy ageing and further deteriorates in AD, it is plausible to suggest a gradual, cognitive-driven disruption of ToM–pragmatics in AD. This is supported by studies (e.g. Bora et al., 2015) suggesting a correlation between the degree of cognitive impairment and ToM impairment in AD.

3.3. Parkinson's disease

Unlike FTD and AD, PD is characterized not only by cognitive but also by motor impairment. The neural underpinning of these impairments includes dopamine depletion that affects the correct functions of basal ganglia and frontostriatal circuits supporting affective, motor and cognitive abilities (Baraldi et al., 2021; Poletti et al., 2011). Some areas affected by PD, including sections of the orbitofrontal cortex (OFC) and the anterior cingulate cortex, are also involved in ToM.

Expectedly, ToM impairment leads to pragmatic difficulties in PD. Already at the early stages, PD speakers exhibit compromised general pragmatic production and comprehension (Baraldi et al., 2021). These impairments in social cognition are primarily related to deficits in ToM (Alonso-Recio et al., 2021).

Specific pragmatic impairments in PD affect communicative behaviour, discourse and conversational organization, discourse tasks, non-verbal language, inferential ability and humour comprehension (Baraldi et al., 2021; Montemurro et al., 2019). However, evidence regarding figurative language is inconsistent. Montemurro et al. (2019) report no impairment in non-literality, while Baraldi et al. (2021) observe difficulties with proverbs, idioms and novel metaphors in more than half of their sample. One possibility is that samples recruited PD speakers at different stages of cognitive impairment. Indeed, PD speakers with lower or no

cognitive impairment are neither impaired on ToM nor pragmatic tasks (Fernández-Fernández et al., 2024), with ToM abilities being predicted by overall cognition and other cognitive functions, such as language, memory or EF (Maggi et al., 2024).

3.4. Low-prevalence neurodegenerative conditions

Low-prevalence dementias have received little attention in research on ToM. The three clinical conditions with available data are LBD, CND and RTD.

Speakers with LBD are impaired in ToM, as LBD-related atrophy affects frontal brain areas, specifically the medial prefrontal cortex (MPFC), the OFC, the right TPJ and the insula (Heitz et al., 2016). However, this disturbance does not appear to directly affect pragmatic abilities (Kemp et al., 2017). Similar to PD, ToM impairment in LBD correlates with deficits in other cognitive domains, including EF, memory, or visuospatial abilities.

Speakers with CND present with atrophy in the cerebellum, which is involved in social cognition. Unlike LBD, neuropathological pathways in CND suggest that social cognitive impairment might be more direct in this clinical condition (Tamaš et al., 2021), although it is still unclear to what extent (and how) pragmatic abilities would be compromised.

RTD runs without affecting frontal lobes and predominantly affects sufferers' empathy and semantic knowledge (Younes et al., 2022). Apparently, there is no remarkable effect on pragmatics.

4. Discussion

The present overview of ToM–pragmatic affection across eight neurodegenerative conditions in ageing reveals two interesting ideas. First, the pattern of neurodegeneration predicts not only ToM affection but also the order in which ToM and pragmatic abilities are affected within the overall cognition. Second, ToM–pragmatic affection in neurodegenerations is considerably modulated by contextual, cognitive and individual factors, plausibly owing to the complex nature of ageing as a long-experienced life stage.

4.1. Patterns of neurodegeneration and effects on Theory of Mind–pragmatics affection

Distinct networking patterns underlying different neurodegenerative conditions predictably guide the patterns of ToM impairment in ageing. As a cognitive function, and from the structural point of view, ToM relies on a network involving MPFC, anterior paracingulate cortex, anterior temporal poles, posterior superior temporal sulci, precuneus and bilateral TPJ (cf. Irish et al., 2014; Lee et al., 2012; Michel et al., 2013) (figure 1A). Consequently, the ToM neural network differentially overlaps with other cognitive and language-supporting areas. The vulnerability of ToM to different patterns of neurodegeneration (figure 1B) is in line with recent models suggesting a large structural network for its support.

[FIGURES 1A AND 1B HERE]

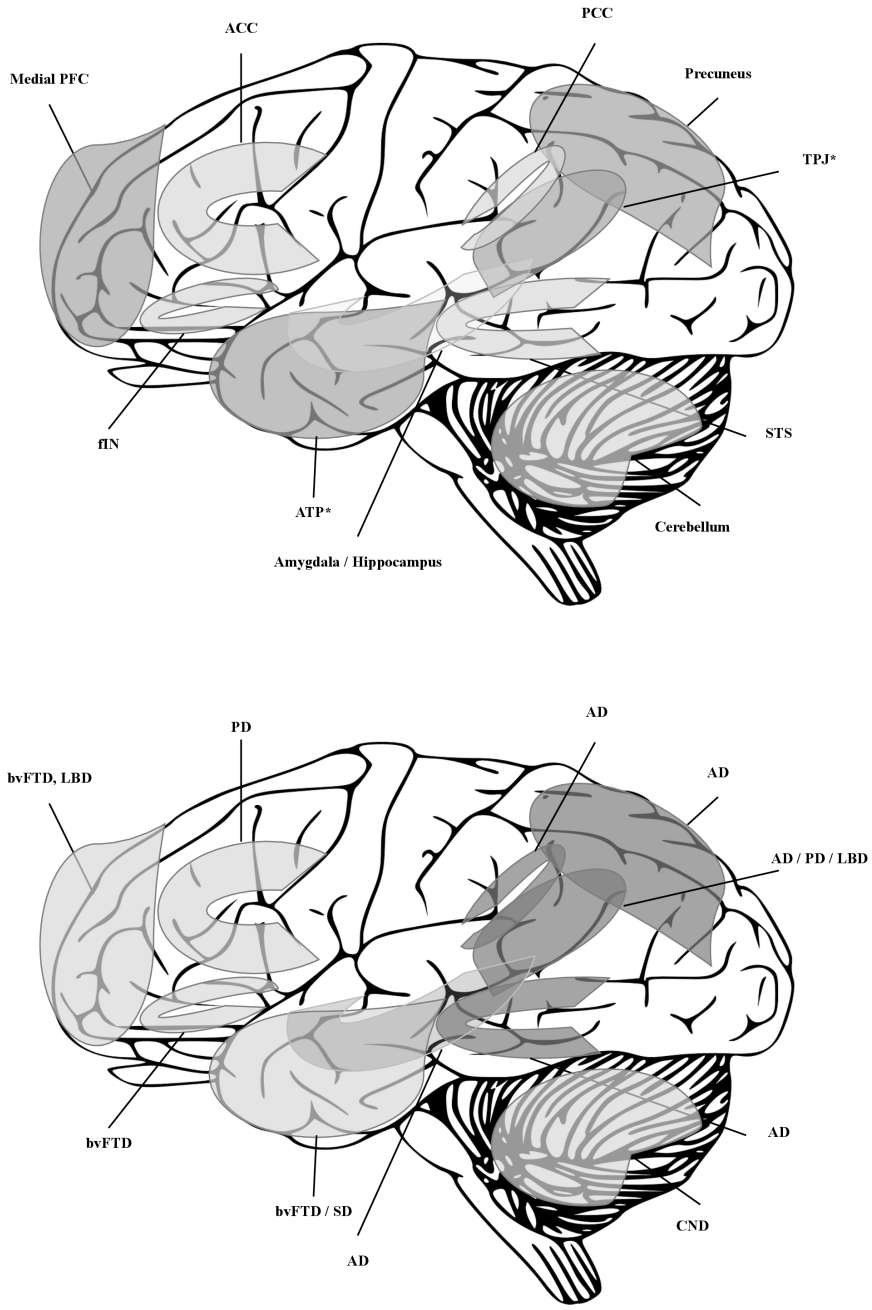

Figure 1. (A) Neural network involved in ToM. (B). Neural networks and loci disrupted in different neurodegenerative diseases. Note. ACC, anterior cingulate cortex; ATP, anterior temporal pole; fIN, frontoinsular; Medial PFC, medial prefrontal cortex; PCC, posterior cingulate cortex; STS, superior temporal sulci; TPJ, temporoparietal junction. *Bilateral implication. Figure based on data from Fittipaldi et al. (2019), Frank (2018), Seelaar et al. (2011) and essentially Strikwerda-Brown et al. (2019).

Likewise, the extensive functional distribution of the ToM-supporting network can explain why ToM and pragmatic impairments may show up differently within the same neurodegenerative condition. Frequent overlaps between ToM network and areas involved in other cognitive or

language functions may contribute to variations in how specific ToM-related functions are impaired. For instance, lesions in TPJ, supporting semantic access, can impair the ability to access mental states associated with specific concepts or memories (Michel et al., 2013). However, these lesions are not the only mechanisms responsible for ToM deficits (Irish et al., 2014).

4.2. Theory of Mind–pragmatics phenomena and their modulation

A comparative overview of ToM–pragmatic impairment in different neurodegenerative conditions suggests that only one condition (bvFTD) is responsible for pragmatic impairments directly related to ToM impairment. In other dementias, ToM–pragmatic impairments are second order; i.e. they are predicted by cognitive impairments other than ToM (figure 2A,B).

[FIGURES 2A AND 2B HERE]

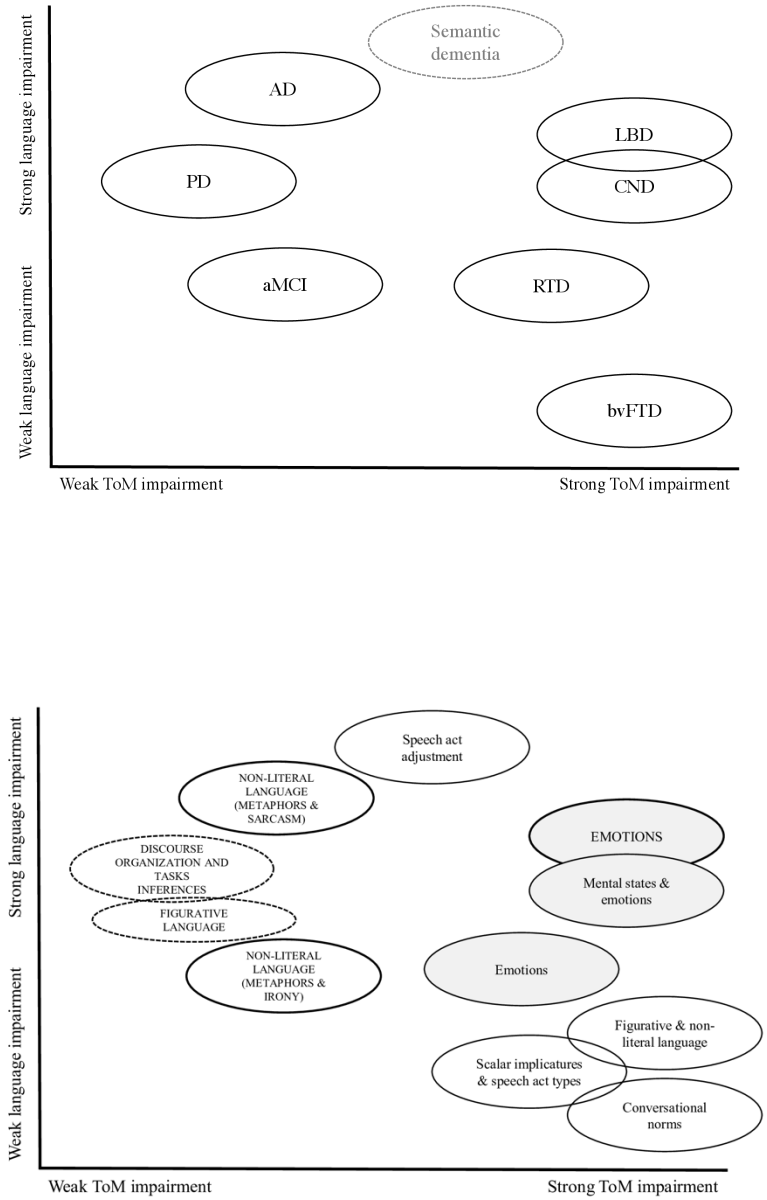

Figure 2. (A) Distribution of neurodegenerative diseases on ToM–language interface. (B). Distribution of associated pragmatic impairments on ToM–language interface. Note. Capital letters marks the second-order cognitively driven impairments. Shaded forms represent non-language-related deficits.

This observation is supported by evidence indicating that pragmatic impairments in pathological ageing can be modulated by contextual and cognitive cues. Research shows that providing additional or enriched contextual information or presenting alternative options can help compensate for pragmatic difficulties and enhance ToM-related performance in speakers with dementia (Ibañez & Manes, 2012; Kosmidis et al., 2008; Spotorno et al., 2015; Laisney et

al., 2013). This suggests that late-adulthood diseases can benefit from overlearned skills. For instance, some ToM-related tasks can be successfully completed by individuals with dementia without relying on complex mentalizing processes (e.g. when they successfully perform non-verbal ToM-related tasks) (cf. Michel et al., 2013). It is thus not unreasonable to propose an internal gradation of ToM-related pragmatic abilities in late adulthood cognition, in line with the suggestion for healthy ageing (cf. Messer, 2015). An illustrative example is that while the comprehension of metaphors (first-order ToM) remains similar between healthy older adults and young adults, understanding irony or sarcasm (second-order ToM) declines with age (Maki et al., 2013). This decline is further exacerbated by cognitive impairment, as evidenced in conditions such as PD (Monetta et al., 2009). Data in figure 2B also indicate that, despite differing underlying causes, ToM-related pragmatic deficits predominantly involve second-order phenomena.

Since affective ToM is generally less affected than cognitive ToM in dementia (Poletti et al., 2011), it is crucial to consider the specific cognitive pathways involved in different neurodegenerative conditions when assessing ToM–pragmatic abilities. Further research agenda should focus on how compensatory strategies impact ToM performance and account for external variables such as education level (Laisney et al., 2013), as well as internal variations within clinical conditions (Simm et al., 2017).

In this context, it is worth noting that any impairment in ToM (or pragmatic abilities in general) can have devastating effects on the speaker's everyday life. The loss of the ability to understand non-literal messages—such as sarcasm, metaphors or insincere communication (cf. Shany-Ur et al., 2012)—or the tendency to misinterpret the meaning of the message itself, significantly reduces one's capacity for meaningful and effective communication. This, in turn, can lead to impoverished relationships and diminished social interactions in daily life (Moreau et al., 2015).

Before concluding, the limitations of this work should be acknowledged. First, its design does not account for the effect sizes of the reviewed studies, limiting the robustness of the conclusions and making the results more coarse-grained. Second, the study overlooks variables that may influence dementia-related cognitive symptoms, ranging from social to highly individual factors. It would also be important for future studies to take a step towards integrating such narrative-based findings with evidence from functional neuroimaging research. So, since dementia progression is shaped by various elements, the trends presented here should be viewed as overarching patterns. Despite these limitations, this study provides valuable insights into the

relationship between cognitive functions and ToM, paving the way for future experimental research.

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