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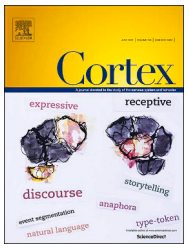

Research Report

Unravelling cognition–Emotion mechanisms in Alzheimer's disease and frontotemporal dementia: A network analysis approach

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ABSTRACT

Emotion processing deficits are common in Alzheimer's disease (AD) and behavioural-variant frontotemporal dementia (bvFTD), yet few studies have examined how specific cognitive domains relate to emotion processing in these syndromes. This study investigated these relationships using network psychometrics. A total of 209 participants (56 AD, 55 bvFTD, 98 healthy controls) completed neuropsychological testing. Cognitive functions were assessed with the Addenbrooke's Cognitive Examination-III and the Sydney Language Battery, while emotion processing was measured with the Facial Affect Selection Task. Network models were estimated for each group and compared using the Network Comparison Test (NCT). Both patient groups showed impaired emotion recognition versus controls. Network density was highest in bvFTD (.62), followed by AD (.44), with controls showing the sparsest network (.16). In AD, emotion processing was associated with semantic comprehension and semantic knowledge, whereas in bvFTD, emotion processing related more closely to verbal fluency. Centrality indices supported these syndrome-specific patterns. NCT results showed no significant global differences in overall strength ($St = .55$, $p = .411$) or structure ($Mt = .34$, $p = .529$) between AD and bvFTD networks, indicating comparable overall organization despite differing functional roles of specific nodes. These findings suggest disease-specific cognitive-emotion associations: semantic mechanisms in AD and fluency-related mechanisms in bvFTD. Importantly, however, contributions of other cognitive processes, not measured here, are also plausible. Overall, the study highlights both shared and distinct patterns characterizing emotion processing impairments across dementias, offering insights for developing tailored interventions targeting syndrome-specific cognitive profiles.

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1. Introduction

Emotion processing involves a range of cognitive processes, including the recognition of other people's emotions, empathy, and affect regulation. Central to this system is facial emotion recognition, the ability to perceive and comprehend emotional information derived from facial signals successfully. This complex process relies on several cognitive processes. Attention allows the detection of appropriate emotional stimuli (Phelps et al., 2006); memory contributes by pulling on prior experience to guide interpretation (LaBar & Cabeza, 2006); and executive functions, including cognitive control, allow production of context-appropriate responses and emotion moderation in intricate social contexts (Zelazo & Cunningham, 2007). Language and semantic knowledge also play a fundamental role in processing emotionally-valenced information, including emotion identification and appropriate interpretation of social signals. Semantic knowledge disturbances impact emotion perception (Souter et al., 2021) and categorization (Lindquist et al., 2014), which are important variables modulating facial emotion perception (Lee & Satpute, 2024). While distinct, language (the system of communication) and semantic knowledge (the understanding of concepts and their relationships) overlap. Semantic knowledge facilitates the categorization and interpretation of emotions, often mediated through language. Evidence from semantic dementia, a younger-onset dementia characterised by progressive semantic loss, demonstrates that semantic deficits disrupt discrete emotion perception, while emotion concept knowledge shapes perceptual representations of facial expressions and modulates neural decoding of emotional cues (Lindquist et al., 2014; Liu et al., 2024).

Constructivist theories of emotion provide a useful framework to understand how emotion recognition relies on the integration of perceptual information from facial expressions with semantic and conceptual emotion knowledge, which together shape how emotional cues are decoded and understood (Brooks & Freeman, 2018; Eickers, 2025). According to these theories, emotion recognition functions as a classification system which unites sensory data with semantic and linguistic conceptual frameworks (Barrett, 2017; Lindquist et al., 2014). The breakdown of emotional sense-making abilities in dementia patients results from both sensory processing deficits and deterioration of emotional conceptual framework, particularly where semantic knowledge and conceptual integrity are compromised (Chen et al., 2025).

At the neural level, emotion processing is supported by a distributed network of brain regions (Pessoa, 2018). Importantly, core affective structures such as the amygdala play a crucial role in valence detection and emotional learning, and amygdalar dysfunction has been linked to emotion recognition impairments across a range of neurological and psychiatric conditions. In addition to the amygdala, other key structures include insula (involved in interoception and subjective feeling states), anterior cingulate cortex (monitoring and control), orbitofrontal cortex (valuation), and temporal lobe regions that are engaged in semantic and face processing (Banks et al., 2007; Goghari et al., 2011; Murray & Izquierdo, 2007; Šimić et al., 2021). Many of these regions also support attention, memory, executive control, and social cognition (Dolcos et al., 2017; Raschle et al., 2017; Salzman & Fusi, 2010), demonstrating the intrinsically integrated nature of emotion and cognition. As such, emotion processing deficits may arise not only from disruptions to semantic and linguistic conceptual systems, but also from primary affective processing abnormalities within limbic networks.

This interdependence is particularly evident in neurodegenerative diseases. We focus here on the two dementia types that are most common in younger-onset dementia (i.e., before the age of 65 years): Alzheimer's disease (AD) and behavioural-variant frontotemporal dementia (bvFTD), which together represent the most frequent younger-onset dementias (Rivas et al., 2025). These two disorders show distinct cognitive and social-emotional characteristics that are associated with their specific brain damage patterns. The main cognitive deficit in Alzheimer's disease (AD) involves episodic memory loss (McKhann et al., 2011). Disturbances of emotion processing (including emotion recognition) tend to emerge later in the disease process and are often secondary to progressive cognitive decline, manifesting as reduced social engagement and difficulty understanding others' mental states (da Silva et al., 2021; Li et al., 2014). In contrast, the primary symptoms of bvFTD include major personality and social conduct changes, which result in severe emotional recognition deficits during the early stages of the disease (Piguet & Kumfor, 2020; Rascovsky et al., 2011). In this syndrome, emotion recognition deficits occur alongside broad social cognitive disturbances, including diminished empathy, loss of social awareness, and impaired theory of mind (Boeve et al., 2022; Piguet & Hodges, 2013).

The observed behavioural changes are associated with different patterns of brain atrophy. In AD, early changes are found in the hippocampus and the medial temporal and parietal lobes bilaterally (Du et al., 2007; Eskildsen et al., 2015; Landin-Romero et al., 2017). It is thought that the resulting memory and attention problems subsequently affect emotion recognition (Miller et al., 2012). The main brain region affected in bvFTD include the prefrontal, anterior cingulate, and anterior temporal cortices bilaterally (Bejanin et al., 2020; Landin-Romero et al., 2017; Staffaroni et al., 2019). These brain regions have been implicated in aspects of social cognition and affect regulation (Hutchings et al., 2018), as well as in physiological feedback processing (Hazelton et al., 2023).

As such, existing evidence suggests a close interaction between the processing of socioemotional and cognitive information and that emergence of socioemotional and cognitive symptoms occurs along a continuum involving these different dimensions rather than caused by discrete and independent systems (Ramanan et al., 2025; Rouse et al., 2024, 2025). Given their different clinical profiles, a direct comparison between AD and bvFTD would identify the unique and common cognitive pathways that link emotion to cognition and provide an integrated model of emotion processing in these neurodegenerative brain conditions, addressing a gap in the literature.

In sum, this study investigated facial emotion recognition in individuals diagnosed with AD or with bvFTD. The aim of the project was to determine how facial emotion recognition performance is reliant on the integrity of other cognitive

functions, including attention, memory, and language abilities in these syndromes. To achieve this goal, we applied novel network analysis methods that allow researchers to examine the complex relationships between multiple variables without requiring linear or unidirectional causal connections (Costantini et al., 2015). These analyses will provide integrated models of facial emotion recognition in the two most common younger-onset dementia syndromes by revealing their emotional-cognitive connection structure.

2. Methods

2.1. Participants

Two hundred and nine participants from FRONTIER, the frontotemporal dementia clinical research group at the University of Sydney, Australia, were included in this study. These comprised 56 individuals diagnosed with typical (amnestic) AD, 55 diagnosed with probable bvFTD (McKhann et al., 2011; Rascovsky et al., 2011) and 98 healthy controls (HC). All participants underwent comprehensive neurological examination, neuropsychological evaluation, and structural MRI brain scans. For patients with dementia, diagnosis was established by consensus among a multidisciplinary clinical team. The HC were age- and education-matched and recruited from a panel of healthy volunteers. These individuals had no significant medical history and scored >88/100 on the Addenbrooke's Cognitive Examination–III (ACE–III) (Hsieh et al., 2013). Additional exclusion criteria for all participants included a history of other neurological or psychiatric disorders, traumatic brain injury with loss of consciousness >5 min, use of neuroleptics or other drugs with central nervous system side effects, or substance abuse. Patients with dementia were also excluded if their score on the ACE–III was lower than 50/100.

2.2. Measures

2.2.1. Cognition and emotion processing examination

Cognitive function was assessed using the ACE-III (Hsieh et al., 2013) and the Sydney Language Battery (SYDBAT) (Savage et al., 2013). The ACE-III measures the integrity of five cognitive domains with specific maximum scores for each subdomain: Attention (maximum 18 points, assessing orientation and sustained attention), Memory (maximum 26 points, assessing anterograde and retrograde memory through encoding, recall, and recognition tasks), Fluency (maximum 14 points, assessing letter and category fluency), Language (maximum 26 points, assessing naming, comprehension, repetition, reading, and writing), and Visuospatial abilities (maximum 16 points, assessing copying, drawing, and perceptual matching). Maximum total score is 100 points with scores below 88/100 indicative of cognitive deficits (Carrick et al., 2025; Hsieh et al., 2013).

SYDBAT measures the integrity of four aspects of language: naming, comprehension, repetition, and semantic association. All the subtests of SYDBAT use the same corpus of 30 polysyllabic words which include living and non-living objects and have variable word frequencies. Each subtest assesses different language functions: the naming subtest requires participants to verbally label pictured objects; the comprehension subtest requires participants to point to the correct picture when given a spoken word; the repetition subtest requires participants to repeat words spoken by the examiner; and the semantic association subtest requires participants to match items based on conceptual relationships (for example, matching an image to the word that matches it based on shared category membership or functional similarity). One point is given for each correct item, with a maximum of 30 possible points for each subtest (Savage et al., 2013).

2.2.2. Emotion processing

All participants were also assessed on the emotional expression recognition task of the Facial Affect Selection Task (FAST) (Kumfor et al., 2014; Miller et al., 2012). In this task, participants were asked to point to the face that matched the emotion with instructions (e.g., "Point to the HAPPY face") from an array of faces showing the six basic emotions (happiness, surprise, disgust, anger, fear, and sadness) and a neutral expression. This task consisted of 42 trials. The participants were not timed or given feedback. One point was given for a correct answer, and zero for an incorrect answer. The faces were selected from the NimStim Stimulus Set (Tottenham et al., 2009; www.macbrain.org). Non-facial features such as hair and shoulders were removed by cropping the images. Faces were displayed on a grayscale on a computer screen.

The Facial Affect Selection Task (FAST) was selected because it minimizes confounds from other cognitive functions. Unlike naming-based emotion tasks, the FAST requires participants to select the correct emotional expression rather than verbalize it, thereby reducing the impact of language deficits. Additionally, the task did not involve time constraints, controlling for attentional and processing speed variations. Other emotion processing tasks, such as the Ekman 60 Faces Test or the Reading the Mind in the Eyes Test, may introduce greater verbal or executive demands. By using FAST, we aimed to isolate emotion recognition while mitigating the influence of other cognitive deficits common in AD and bvFTD.

2.3. Statistical analyses

Chi-squared tests were used to assess group differences in categorical variables (sex). An independent samples *t*-test was used to assess the differences between AD and bvFTD on continuous variables (i.e., disease duration). One-way ANOVAs were used to assess group effects (AD, bvFTD, and HC) on cognitive function and emotion processing. For all analyses, the significance level was set at $p < .05$ to test against the null hypothesis. Exact *p*-values smaller than .001 are also reported to indicate stronger evidence.

2.4. Network estimation

We estimated undirected psychological networks separately for each group (AD, bvFTD, and HC) using the Extended Bayesian Information Criterion Graphical Lasso (EBICglasso; Chen & Chen, 2008; Blanken et al., 2022; Aunger et al., 2025)

within a Pairwise Markov Random Field (PMRF) framework (Costantini et al., 2015; Epskamp et al., 2022). This approach corresponds to a Gaussian graphical model (GGM), an undirected graphical model in which edges represent partial correlations ranging from −1 to 1, reflecting the remaining association between two nodes after conditioning on all other variables (Epskamp, Borsboom, & Fried, 2018; Epskamp, Waldorp, et al., 2018; Ferguson, 2021). This procedure applies graphical LASSO regularization with a tuning parameter of .25, shrinking weaker edges toward zero to enhance interpretability while balancing sensitivity and specificity in model selection (Epskamp & Fried, 2018; Isvoranu & Epskamp, 2023). In the resulting networks, nodes represent measures of emotion processing and cognitive function, and edges reflect regularized partial correlations, indicating direct associations after conditioning on all other variables. Analyses were performed using the *qgraph* package (Epskamp et al., 2012) in R (R Core Team, 2025) via the Positron application (Posit Software & PBC, 2025).

Bootstrapped 95% confidence intervals (CIs) were computed for edge weights to assess their precision and stability, indicating how reliably each connection was estimated across resamples. Importantly, these CIs are not used for hypothesis testing or determining statistical significance, in contrast to traditional regression CIs. The correlation stability (CS) coefficient was calculated only for node centrality indices to determine their robustness across bootstrap samples, with centrality estimates interpreted as stable and reliable only when CS $\geq$ .50 (Epskamp, Borsboom, & Fried, 2018; Epskamp & Fried, 2018).

2.4.1. *Descriptive statistics and transformation*

All variables were examined using descriptive statistics, including mean, standard deviation, median, skewness, and kurtosis, computed for each variable in each group (AD, bvFTD, and HC), following APA reporting guidelines (Appelbaum et al., 2018). For continuous data deviating from normality, a nonparanormal (npn) transformation was applied prior to estimating the Gaussian Graphical Model (Deserno et al., 2022; Epskamp & Fried, 2018; Liu et al., 2012) using the *huge* package, which also allows tuning parameter selection via cross-validation or EBIC (Zhao et al., 2012). The nonparanormal (npn) transformation was applied to mitigate deviations from multivariate normality. Summary statistics before and after transformation are presented in Table 2, and distributional plots for raw and transformed data are provided in Supplementary Material A.

2.4.2. *Confirmatory network model evaluation*

To further evaluate the robustness of the estimated network structure, we conducted a complementary confirmatory network analysis using the *psychonetrics* package (Epskamp, 2025). This approach allows the estimation of Gaussian graphical models within a structural equation modeling framework and enables formal evaluation of model fit using multiple fit indices. Network structure evaluation was performed using full-information maximum likelihood estimation (FIML), which accommodates missing data under the assumption of missing at random (MAR). Model fit was assessed using several commonly reported indices, including the Comparative Fit Index (CFI), Tucker–Lewis Index (TLI), and the Root Mean Square Error of Approximation (RMSEA) with 95% confidence intervals. Model fit was considered acceptable when RMSEA $\leq$.08 and CFI and TLI $\geq$.90 (Wang & Wang, 2020).

The selection of these three fit indices was guided by both methodological considerations and software implementation constraints. Specifically, the *psychonetrics* package provides conventional global fit indices commonly used in structural equation modeling, namely CFI, TLI, and RMSEA, in addition to the χ^2 statistic, whereas other indices such as SRMR are not implemented for confirmatory network models within this framework (Epskamp, 2025). Moreover, prior methodological literature has emphasized that the interpretation and cutoff values of RMSEA, CFI, and TLI are largely rooted in normal-theory maximum likelihood estimation with continuous data (Xia & Yang, 2019), and these indices remain the most frequently reported and discussed global fit measures in applied SEM research (Kyriazos & Poga-Kyriazou, 2023).

At the same time, it is important to acknowledge that the use of ML estimation under conditions of extreme non-normality may affect the behaviour of these fit indices, including spuriously inflated χ^2 values leading to model over-rejection (Bollen, 1989; Brown, 2015), slight underestimation of CFI and TLI, and potential overestimation of RMSEA (Byrne, 2012). For this reason, the χ^2 test statistic (and the χ^2-to-degrees-of-freedom ratio) was reported for completeness, although it was not treated as the primary criterion for model evaluation due to its well-documented sensitivity to sample size. This reporting strategy aligns with contemporary recommendations for transparent and comprehensive model fit reporting in SEM and related modeling frameworks (Fenn et al., 2020; Kline, 2015, 2023).

Model selection followed a stepwise procedure. First, a saturated network model was estimated in which all possible edges were freely estimated. Next, statistically non-significant edges were removed using a step-down pruning procedure with false discovery rate (FDR) correction based on the Benjamini–Hochberg method (Benjamini & Hochberg, 1995) at $\alpha = .05$, resulting in a pruned network model. Subsequently, a step-up procedure was applied in which edges were iteratively added if their inclusion led to improved model parsimony, as indicated by a reduction in the Bayesian Information Criterion (BIC). This procedure terminated when no further improvement in BIC was observed, yielding an optimized network model.

To compare competing models, we followed the approach proposed by Asparouhov and Muthén (2019) to evaluate whether models were nested. Once nestedness was established, chi-square difference tests were conducted. A significant chi-square difference ($p < .05$) indicates that the more complex model provides a significantly better fit to the data than the more constrained model (Satorra & Bentler, 2010). In addition, BIC values were used as an information-theoretic criterion, with smaller BIC values indicating a network structure that more parsimoniously and accurately represents the underlying data-generating process (Kline, 2023). This confirmatory psychonetrics-based analysis was intended as a model validation and sensitivity analysis, complementing the primary EBICglasso network estimation. By demonstrating convergence across regularized and confirmatory modelling

approaches, this procedure strengthens confidence in the stability and interpretability of the identified network structure.

2.4.3. Model selection, estimation and regularisation

Networks were estimated using the EBICglasso procedure with *cor_auto* correlations, and no arbitrary edge threshold was applied. All non-zero edges retained by EBICglasso regularization were reported to produce a sparse network structure with relatively few edges (Epskamp & Fried, 2018). This approach uses the least absolute shrinkage and selection operator (lasso) to shrink edge weights toward zero, with some set exactly to zero (Tibshirani, 1996). The EBIC hyperparameter, set to .25, balances sensitivity and specificity, optimizing psychometric properties. For visualization purposes, supplementary figures highlight edges stronger than .10.

Network stability was evaluated using nonparametric bootstrapping with 2,000 resamples. The correlation stability (CS) coefficient was calculated only for node strength centrality, in line with recommended practice (Borsboom et al., 2021; Epskamp, Borsboom, & Fried, 2018). Edge-weight stability was assessed descriptively using bootstrapped confidence intervals rather than CS estimation. Node strength was calculated as the sum of absolute partial correlation values connected to each node. All centrality indices were computed on unthresholded network estimates to avoid artefactual zero values. Additionally, descriptive network metrics—including edge density, total number of non-zero edges, and minimum, maximum, and mean edge weights—were reported to summarize global network structure. Between-group network comparisons were conducted using the Network Comparison Test (NCT; van Borkulo et al., 2023), assessing global strength, network structure invariance, and edge strength differences.

2.4.4. Simulation studies

To evaluate the robustness of the EBICglasso estimator and its hyperparameter (γ), we conducted three simulation studies for each group (AD, bvFTD, HC). The simulations used the true underlying network structure estimated from the empirical data as the ground truth. Sample sizes of 100, 200, and 500 were applied to systematically assess estimator performance across low-to-moderate sample regimes, even though the empirical groups included ~60 participants. This approach is consistent with previous simulation studies, where sample sizes larger than those in the empirical dataset are used to ensure stable evaluation of estimator performance (Ferguson, 2021).

Each simulation followed a five-step procedure: (1) generating random data from the true underlying network, (2) applying different EBIC hyperparameter values (0, .25, .5), (3) estimating networks from the simulated data, (4) comparing estimated networks to the input network, and (5) repeating the procedure 1,000 times. Key properties examined included sensitivity, specificity, and the correlation between estimated and true networks. Simulations were conducted using the *bootnet* package (Epskamp, Borsboom, & Fried, 2018). Results indicated stable performance of the estimator across conditions, with EBIC $\gamma = .25$ maintaining specificity without substantially reducing sensitivity. These simulations were intended solely to inform hyperparameter selection and do not affect the substantive network findings, with detailed plots visualizing the simulation outcomes reported in Supplementary Material B.

2.4.5. Post-hoc investigations

Edge Weight Stability; Non-parametric bootstrapping was conducted to account for sampling variability and assess the reliability of network estimates for each group (AD, bvFTD, and HC) using the *bootnet* package (Epskamp, Borsboom, et al., 2018). For each network, 2,000 bootstrap samples were generated across 8 cores, and the following statistics were computed: edge weights, strength, closeness, betweenness, and expected influence. Specifically, the procedure involved computing network statistics in the original dataset, resampling with replacement to generate new datasets, recalculating statistics in each resampled dataset, and repeating these steps 2,000 times. Bootstrapped distributions were then used to estimate 95% confidence intervals for edge weights and centrality metrics. Complete results are reported in Supplementary Material C.

Edge Density; The number of edges observed in each network was calculated and compared to the number of possible edges. The number of possible edges was determined using formula $P(P-1)/2$, where P represents the number of nodes in the network (Epskamp et al., 2017). In this study, each network consisted of 10 nodes, resulting in 45 possible edges.

Comparison of Network Structures; The Network Comparison Test (NCT) was used to examine the invariance of network structure and global strength across the AD, bvFTD, and HC networks. Network structure refers to the specific pattern of edges connecting nodes, while global strength refers to the absolute sum of all edges (Opsahl et al., 2010; van Borkulo et al., 2023). The NCT is a non-parametric permutation-based test that repeatedly redistributes participants between groups to generate a null distribution, allowing the observed differences in network structure or strength to be compared against chance. For this study, the NCT was conducted for the comparisons AD versus bvFTD, AD versus HC, and bvFTD versus HC, and p values were interpreted as exploratory indices of deviation from the null hypothesis. In addition to examining overall network structure and global strength, the NCT also allowed for edge-specific comparisons to explore whether particular connections between cognition and emotion nodes significantly differed across groups. For each pairwise group comparison (AD *vs* bvFTD, AD *vs* HC, bvFTD *vs* HC), 1000 permutations were conducted to generate empirical null distributions. Edge weights that significantly deviated from these distributions were flagged for exploratory interpretation.

Centrality Indices; Node strength and betweenness centrality were calculated for each network and reported as standardized z scores. Prior to interpretation, the stability of these centrality estimates was assessed using case-dropping bootstrapping, which employs subsampling without replacement and diminishing sample sizes rather than resampling with replacement and constant sample sizes. These methods generated a centrality stability coefficient (CS), which quantifies, with 95% confidence, the largest proportion of participants that could be dropped while preserving a correlation

greater than .70 with the original centrality values (Epskamp & Fried, 2018). A CS of .50 or higher indicates a stable centrality estimate, whereas a CS below .25 should not be interpreted. In the present analyses, only the strength index met the minimum criterion for stability and was therefore interpreted. Betweenness and closeness centrality estimates did not reach the stability threshold and are reported in the Supplementary Material D for completeness but are not interpreted. Expected influence is presented for reference, with interpretation limited to nodes meeting stability criteria. Node centrality correlations, row-complexity, and squared multiple correlations (SMC) are reported in the Results, with detailed stability analyses and tables of centrality and predictability indices (R^2) provided in Supplementary Material D.

2.4.6. *Node-level network metrics*

To characterize the structural properties of the FAST–Cognition networks, we computed several node-level indices. Strength centrality was the primary metric due to its robustness in psychological networks. While betweenness and closeness were also calculated, they were not interpreted because their case-dropping stability coefficients (CS) were below .25 (Fig. D2–D4). We also included row-complexity, which quantifies how evenly a node distributes its connections across the network, with higher values indicating more diffuse connectivity, and Squared Multiple Correlation (SMC), which indexes the proportion of variance of each node explained by all other nodes in the network. Finally, predictability (R^2) was computed to indicate the proportion of variance in each node accounted for by its immediate neighbours, offering a measure of node-level determinism. These metrics collectively provide insight into how cognitive and emotion-processing nodes are organized and interact across diagnostic groups. Detailed tables, correlation matrices, and stability plots are provided in Supplementary Material D (Table D, Fig. D1–D4).

2.5. *Sensitivity analyses: regression, correlation, and moderation tests*

To evaluate the robustness of the network findings, sensitivity analyses were conducted using multiple linear regression and correlation checks. The FAST Emotion total score was treated as the outcome variable, while the nine remaining network variables (ACE–III Attention, ACE–III Memory, ACE–III Fluency, ACE–III Language, ACE–III Visuospatial, SYDBAT Comprehension, SYDBAT Naming, SYDBAT Semantic, and SYDBAT Repetition) were entered as predictors. Linear regression models were fitted separately for each group (AD, bvFTD, and HC) to examine whether the pattern of relationships observed in the network analyses was consistent when tested with conventional parametric approaches.

To formally assess multicollinearity among predictors, Variance Inflation Factors (VIFs) were computed for all regression models. Following standard guidelines, VIF values below 3.5–5 (Hair et al., 2019) or 10 (Field, 2024) are generally considered acceptable and indicate that multicollinearity is unlikely to bias the regression estimates. Pearson correlation coefficients among all predictors and between each predictor and the outcome were also computed to cross-check the strength and direction of associations indicated by the network models. Significant relationships were interpreted as complementary evidence supporting the network-estimated associations.

To further examine relationships involving emotion processing, we also conducted additional bivariate correlations and group-wise regression models to statistically compare the strength of associations between emotion processing and other tasks (e.g., verbal fluency, semantic comprehension). These analyses were performed alongside the network approach to determine whether observed differences were statistically significant. In addition, we computed a correlation similarity matrix for all tasks within each group to illustrate the degree of overlap between tasks such as semantic processing, comprehension, and verbal fluency. The matrices are presented in Supplementary Material E (see Tables E4–E6, and Fig. E3a–E3c). To formally test whether the associations between cognitive measures and emotion processing differed across groups, we also fitted regression models including predictor interaction terms x group (AD, bvFTD, and HC) for all cognitive predictors. This moderation framework allowed us to directly assess whether the strength of the association between each cognitive variable and FAST Emotion varied across AD, bvFTD, and HC groups. Significant interactions would indicate that the effect of a cognitive predictor on emotion processing is moderated by group membership.

3. Results

3.1. *Demographic characteristics*

The demographic characteristics are reported in Table 1. No significant group differences were observed for sex ($X^2_{(2)} = 1.750$, $p = .417$), education ($F_{(2,\ 206)} = 4.211$, $p = .016$), or disease duration ($t_{(49)} = .910$, $p = .367$). However, a significant main effect of age was present ($F_{(2,\ 206)} = 12.980$, $p < .001$), and post hoc Tukey's HSD test showing that the bvFTD group was significantly younger than both the AD ($p = .014$) and HC ($p < .001$) groups. The difference between the AD and HC groups was not significant ($p = .135$).

3.2. *Cognition and emotion*

Analysis of cognitive performance revealed significant group effects on global cognition (ACE total; $F_{(2,\ 206)} = 123.400$, $p < .001$), with HC outperforming both dementia groups (both $p < .001$) and bvFTD performing better than AD ($p < .001$). Similar patterns were found across ACE-III subdomains (Table 1), where HC consistently showed superior performance. Between the dementia groups, bvFTD demonstrated higher scores than AD on Attention, Memory, and Visuospatial domains (all p values $< .001$), with no differences on Fluency ($p = .316$) and Language ($p = .999$).

A significant group effect was observed on the following SYDBAT Language tests: SYDBAT Naming, $F_{(2,\ 206)} = 46.230$, $p < .001$; SYDBAT Semantic, $F_{(2,\ 206)} = 36.200$, $p < .001$; SYDBAT Comprehension, $F_{(2,\ 206)} = 45.190$, $p < .001$; and SYDBAT Repetition, $F_{(2,\ 206)} = 8.765$, $p < .001$ (Table 1). Post hoc comparisons showed that the HC group obtained significantly

Table 1 – Demographic and neuropsychological characteristics of the dementia groups and HC.

Variables	AD ($n = 56$)	bvFTD ($n = 55$)	HC ($n = 98$)	Statistics	p	*Post hoc*
Demographic and clinical characteristics						
Sex (M:F)	34:22	31:24	49:49	1.750 (χ^2)	.417	N/A
Age (Y)	69.1 ± 9.5	64.3 ± 7.9	71.9 ± 9.0	12.980	<.001	bvFTD < AD = HC
Education (Y)	12.1 ± 3.2	12.2 ± 2.7	13.4 ± 3.1	4.211	.016	AD = bvFTD < HC
Disease duration (months)	37.7 ± 24.8	31.3 ± 19.6	N/A	.910(t)	.367	N/A
ACE-III total (/100)	67.9 ± 13.1	76.0 ± 15.6	94.5 ± 3.4	123.400	<.001	AD < bvFTD < HC
FRS	.7 ± 1.5	−.3 ± 1.3	N/A	3.927(t)	<.001	N/A
Cognitive measures						
ACE-III subdomains						
Attention (/18)	12.9 ± 3.2	14.9 ± 3.1	17.2 ± .9	60.600	<.001	AD < bvFTD < HC
Memory (/26)	13.3 ± 5.0	18.2 ± 5.4	24.4 ± 1.7	146.400	<.001	AD < bvFTD < HC
Fluency (/14)	7.5 ± 2.9	6.8 ± 3.9	12.1 ± 1.6	88.530	<.001	AD = bvFTD < HC
Language (/26)	22.1 ± 3.1	22.1 ± 4.5	25.2 ± 1.1	29.350	<.001	AD = bvFTD < HC
Visuospatial (/16)	12.5 ± 3.1	14.0 ± 2.1	15.5 ± .9	40.720	<.001	AD < bvFTD < HC
SYDBAT						
Naming (/30)	21.2 ± 5.0	22.7 ± 4.3	26.7 ± 2.2	46.230	<.001	AD = bvFTD < HC
Semantic (/30)	24.6 ± 3.1	24.9 ± 3.8	28.0 ± 1.6	36.200	<.001	AD = bvFTD < HC
Repetition (/30)	29.2 ± 1.5	28.8 ± 2.5	29.8 ± .4	8.765	<.001	AD = bvFTD < HC
Comprehension (/30)	25.5 ± 3.4	26.9 ± 2.5	29.1 ± 1.2	45.190	<.001	AD < bvFTD < HC
FAST (/42)	31.3 ± 5.4	29.2 ± 7.5	38.1 ± 3.1	61.280	<.001	AD = bvFTD < HC

Notes. Values are raw scores except for Rasch score (FRS). Values represent the mean ± standard deviation. AD, Alzheimer's disease; ACE–III, Addenbrooke's Cognitive Examination–III; bvFTD, behavioural-variant frontotemporal dementia; N/A, not available; FAST, Facial Affect Selection Task; FRS, Functional Rating Scale (Rasch score); HC, healthy controls; SYDBAT, Sydney Language Battery test.

higher scores than both AD and bvFTD across all measures (all p values < .001), except for Repetition, where only the HC–AD ($p = .026$) and HC–bvFTD ($p < .001$) contrasts were significant. Between the dementia groups, bvFTD outperformed AD on the Comprehension task ($p = .003$). A significant group effect was also found for the emotion processing task (FAST total: $F_{(2, 206)} = 61.280$, $p < .001$), with HC showing higher scores than both AD and bvFTD (both p values < .001), whereas the dementia groups did not differ significantly ($p = .083$).

3.3. *Descriptive statistics and data transformation*

Summary statistics for all variables used in the network analyses, including mean, standard deviation, median, skewness, and kurtosis, before and after nonparanormal (npn) transformation, are presented in Table 2. Across raw data, several variables exhibited deviations from normality, as indicated by Shapiro–Wilk p values below .05 (e.g., ACE–III Attention in HC, SYDBAT Repetition in bvFTD). After nonparanormal (npn) transformation, the distributions of all variables improved, with skewness and kurtosis values approaching zero and Shapiro–Wilk p values indicating approximate normality for most measures, and these transformed variables were subsequently used for estimating the Gaussian Graphical Models (GGM).

3.4. *Network model fit and selection*

To determine the most appropriate network representation for each diagnostic group, we first evaluated and compared the global fit and relative performance of three competing network models: a saturated model (Model A), a pruned model (Model B), and an optimized model (Model C) (Table 3). Across all diagnostic groups, the saturated network model (Model A) served as a baseline reference and, as expected, showed perfect fit indices due to its zero degrees of freedom. However, this model yielded the largest BIC values in all groups, indicating poor parsimony despite its nominally perfect fit. For the Alzheimer's disease (AD) group, the pruned network model (Model B) demonstrated acceptable incremental fit (CFI = .92; TLI = .89) but showed marginal absolute fit, as reflected by an elevated RMSEA (.09). In contrast, the optimized network model (Model C) achieved excellent global fit, with CFI and TLI values exceeding 1.00 and an RMSEA effectively equal to zero. Importantly, Model C also resulted in a significantly lower BIC compared to Model B, and the chi-square difference test confirmed a significant improvement in fit ($\Delta\chi^2_{(2)} = 20.70$, $p < .001$), supporting the superiority of the optimized model for the AD group.

A similar pattern emerged in the bvFTD group. The pruned network model showed poor model fit, characterized by very low incremental fit indices (CFI = .29; TLI = .26) and a substantially inflated RMSEA (.29). In contrast, the optimized network model demonstrated excellent fit across all indices (CFI = 1.00; TLI = 1.01; RMSEA = .00) and yielded a markedly lower BIC value. The chi-square difference test further indicated that Model C provided a significantly better fit than Model B ($\Delta\chi^2_{(9)} = 207.08$, $p < .001$), underscoring the necessity of the step-up optimization procedure for this group. For the healthy control group (HC), the pruned model again exhibited suboptimal fit, with moderate incremental fit indices (CFI = .56; TLI = .52) and elevated RMSEA (.13). The optimized network model substantially improved model fit, achieving near-perfect incremental fit (CFI = .99; TLI = .99) and low RMSEA (.02). Consistent with the AD and bvFTD groups, Model C produced the smallest BIC value and significantly

Table 2 – Descriptive statistics of network variables before and after transformation.

Node	n	Raw Data						Transformed Data					
		M	SD	Md	Skew	Kurt	*p*	M	SD	Md	Skew	Kurt	*p*
AD													
FAST_emotion	56	31.34	5.37	32.50	−.71	.09	.017	.00	1.00	−.04	−.01	−.54	.901
ACEIII_attention	56	12.91	3.24	13.00	−.40	−.67	.023	.00	.99	−.12	−.04	−.65	.512
ACEIII_memory	56	13.27	4.98	12.00	.50	−.29	.100	.00	1.00	−.09	−.01	−.58	.890
ACEIII_fluency	56	7.50	2.94	8.00	−.60	−.72	<.001	.00	.98	−.07	−.02	−.66	.182
ACEIII_language	56	22.14	3.12	23.00	−.81	−.02	<.001	.00	1.00	.07	−.04	−.62	.765
ACEIII_visuospatial	56	12.48	3.11	14.00	−.48	−1.12	<.001	−.01	.97	.14	−.12	−.80	.088
SYDBAT_comprehension	56	25.48	3.42	26.00	−1.39	2.13	<.001	.00	.99	−.09	−.05	−.60	.404
SYDBAT_naming	56	21.21	4.98	22.00	−.98	1.11	<.001	.00	1.00	.07	−.02	−.58	.871
SYDBAT_semantic	56	24.63	3.10	25.00	−.85	1.05	<.001	.00	.99	−.05	−.01	−.57	.445
SYDBAT_repetition	56	29.18	1.45	30.00	−1.74	1.84	<.001	−.07	.79	.46	−1.05	−.39	<.001
bvFTD													
FAST_emotion	55	29.22	7.52	31.00	−.83	−.06	.003	.00	1.00	−.02	−.01	−.58	.973
ACEIII_attention	55	14.88	3.06	15.38	−1.50	1.91	<.001	−.01	.98	−.02	−.10	−.72	.246
ACEIII_memory	55	18.16	5.44	19.00	−.81	−.14	.001	.00	.99	−.05	.02	−.73	.365
ACEIII_fluency	55	6.75	3.93	7.00	−.21	−1.01	.036	.01	.98	.00	.09	−.73	.241
ACEIII_language	55	22.13	4.45	23.98	−1.58	2.44	<.001	−.02	.97	.00	−.19	−.82	.043
ACEIII_visuospatial	55	14.03	2.14	15.00	−1.50	2.15	<.001	−.01	.98	.14	−.08	−.71	.149
SYDBAT_comprehension	55	26.95	2.54	27.00	−1.13	1.10	<.001	−.01	.96	−.19	−.13	−.84	.014
SYDBAT_naming	55	22.67	4.29	24.00	−.57	−.56	.012	.00	.99	.02	−.02	−.61	.727
SYDBAT_semantic	55	24.93	3.78	26.00	−1.04	.91	.001	.00	.99	.05	−.06	−.64	.399
SYDBAT_repetition	55	28.82	2.53	30.00	−3.55	14.94	<.001	−.07	.80	.49	−1.00	−.39	<.001
HC													
FAST_emotion	98	38.13	3.14	39.00	−1.27	2.54	<.001	−.01	1.00	.18	−.18	−.65	.003
ACEIII_attention	98	17.20	.85	17.39	−2.20	6.67	<.001	−.03	.95	.10	−.35	−.55	<.001
ACEIII_memory	98	24.39	1.68	25.00	−.78	−.39	<.001	−.04	.92	.10	−.47	−.85	<.001
ACEIII_fluency	98	12.14	1.55	12.50	−.76	−.25	<.001	−.02	.97	.06	−.23	−.67	<.001
ACEIII_language	98	25.20	1.08	25.93	−1.57	2.21	<.001	−.04	.94	.16	−.43	−.75	<.001
ACEIII_visuospatial	98	15.53	.87	16.00	−2.19	4.82	<.001	−.04	.93	−.03	−.44	−.70	<.001
SYDBAT_comprehension	98	29.11	1.23	30.00	−1.67	3.00	<.001	−.06	.87	.69	−.76	−.55	<.001
SYDBAT_naming	98	26.74	2.16	27.00	−.64	.10	.001	−.01	1.01	−.03	−.10	−.55	.010
SYDBAT_semantic	98	27.99	1.56	28.00	−.78	.34	<.001	−.02	.98	−.12	−.21	−.62	<.001
SYDBAT_repetition	98	29.85	.44	30.00	−2.90	7.86	<.001	−.06	.62	.16	−2.42	4.22	<.001

Notes. AD, Alzheimer's disease; ACE–III, Addenbrooke's Cognitive Examination–III; bvFTD, behavioural-variant frontotemporal dementia; FAST, Facial Affect Selection Task; HC, healthy controls; *M*, mean; *SD*, standard deviation; *Md*, median; *Skew*, skewness; *Kurt*, kurtosis; SYDBAT, Sydney Language Battery test, *p* values reported above correspond to Shapiro–Wilk tests for normality.

outperformed the pruned model based on the chi-square difference test ($\Delta\chi^2_{(7)} = 76.27$, $p < .001$). Taken together, these results indicate that, across all diagnostic groups, the optimized network model (Model C) provides the best balance between model fit and parsimony. Accordingly, all subsequent network visualizations and substantive interpretations are based on the optimized network models.

3.5. *Network psychometrics of Cognition–Emotion mechanisms*

Figs. 1–3 present the network plots for the AD, bvFTD, and HC groups, respectively. These plots display the associations among the emotion and cognition variables, with FAST Emotion highlighted as the target node across all groups. Across groups, networks were relatively sparse. The AD network comprised 20 non-zero edges (mean edge weight = .20, range = .03–.47), the bvFTD network comprised 28 non-zero edges (mean edge weight = .15, range = −.16–.48), and the HC network comprised 7 non-zero edges (mean edge weight = .27, range = .19–.37). Complete edge weight estimates with bootstrapped confidence intervals are reported in Supplementary Tables C1–C3. In the network analyses, bootstrap confidence intervals reflect the precision and stability of edge estimates and are not used to determine conventional statistical significance. Consequently, edges whose CIs do not cross zero are not formally interpreted as significant, unlike in traditional regression analyses. All identified associations with FAST Emotion were positive in both the AD and bvFTD networks.

The bootstrapped stability analyses further indicated that edge-weight patterns were consistent across resamples. Case-dropping bootstraps revealed that centrality stability was limited. CS coefficients for node-strength centrality were .00 in AD, .055 in bvFTD, and .133 in the HC network, with betweenness and closeness showing similarly low or zero stability across groups. Such values fall below the recommended interpretability threshold (CS $\geq$ .50) but are widely observed in psychological network studies with modest sample sizes. This pattern is consistent with prior reports that centrality metrics are less reliable in small samples or sparse networks (Bringmann et al., 2019; Epskamp, Borsboom, &

Table 3 – Network model fit and comparisons across diagnostic groups.

Model	Model Test		Incremental Fit Measures		Absolute Fit Measure		Nested Test		BIC
	$\chi2_{(df)}$	*p*	CFI	TLI	RMSEA [95% CI]	*p*	$\Delta\chi2_{(df)}$	*p*	
AD									
Model A	.00 (0)	1.000	1.00	NaN	N/A	N/A	–	–	1561.00
Model B	51.29 (35)	.037	.92	.89	.09 [.02, .12]	.119	51.29 (35)	.037	1471.40
Model C	30.59 (33)	.588	1.00	1.02	.00 [.00, .09]	.769	20.70 (2)	<.001	1458.75
bvFTD									
Model A	.00 (0)	1.000	1.00	NaN	N/A	N/A	–	–	1447.64
Model B	238.02 (43)	<.001	.29	.26	.29 [.25, .32]	<.001	238.02 (43)	<.001	1513.35
Model C	30.94 (34)	.619	1.00	1.01	.00 [.00, .08]	.792	207.08 (9)	<.001	1342.33
HC									
Model A	.00 (0)	1.000	1.00	NaN	N/A	N/A	–	–	2684.02
Model B	111.37 (41)	<.001	.56	.52	.13 [.10, .16]	<.001	111.37 (41)	<.001	2607.41
Model C	35.10 (34)	.416	.99	.99	.02 [.00, .08]	.753	76.27 (7)	<.001	2563.23

Notes. AD, Alzheimer's disease; ACE–III, Addenbrooke's Cognitive Examination–III; bvFTD, behavioural-variant frontotemporal dementia; HC, healthy controls. Estimator: Full-information maximum likelihood estimation (FIML). CFI, Comparative fit index; TLI, Tucker–Lewis index; RMSEA, Root mean square error of approximation; BIC, Bayesian information criterion; CI, Confidence interval; NaN, not a number; N/A, not available. Model A (Saturated network model): all possible edges among variables were freely estimated without any constraints, providing a fully connected baseline network. Model B (Pruned network model): statistically non-significant edges were removed using a false discovery rate (FDR; Benjamini–Hochberg) correction at $\alpha = .05$, resulting in a more parsimonious network containing only significant associations. Model C (Optimized network model): starting from the pruned network, edges were iteratively added using a step-up procedure based on the Bayesian Information Criterion (BIC) at $\alpha = .05$, until no further improvement in model fit was achieved.

Fried, 2018; Williams & Rast, 2020). Accordingly, we restricted interpretation to edge-level associations and overall network density, while considering centrality-based findings as exploratory. These patterns reflect general properties of centrality estimation in small or sparse networks rather than weaknesses specific to the present models (Fried & Cramer, 2017). In accordance with these methodological constraints, centrality-based findings are reported in Supplementary

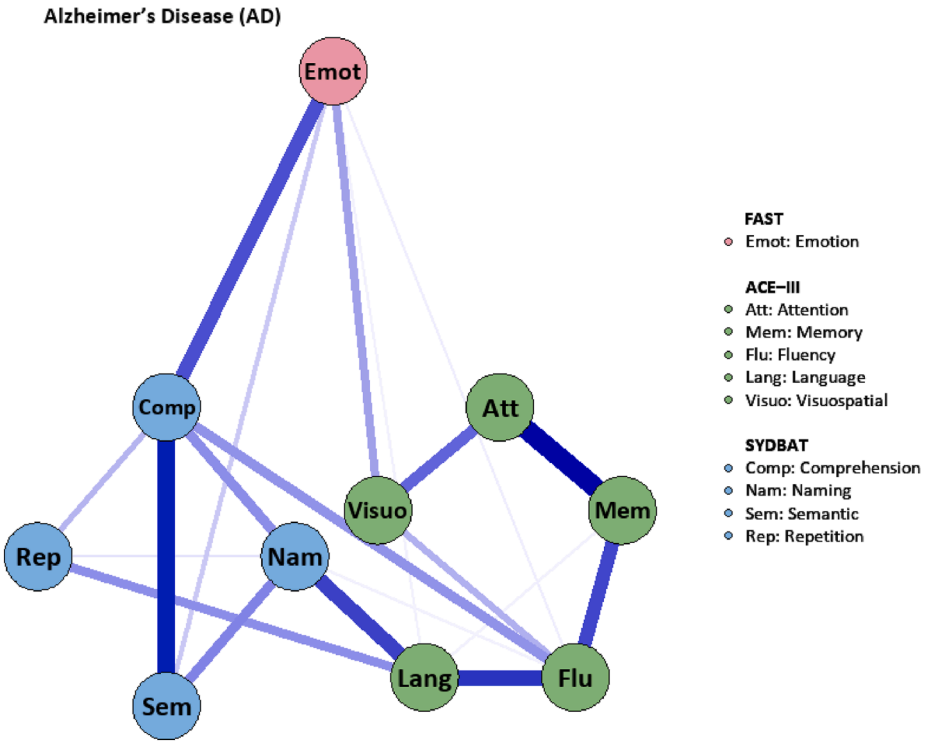

Fig. 1 – Network model of cognition–emotion mechanisms in Alzheimer's disease (AD).
Note: ACE–III, Addenbrooke's Cognitive Examination–III; FAST, Facial Affect Selection Task; SYDBAT, Sydney Language Battery test. Edge thickness represents the magnitude of partial correlation (edge weight), with thicker lines indicating stronger associations. Blue edges indicate positive correlations, whereas red edges indicate negative correlations.

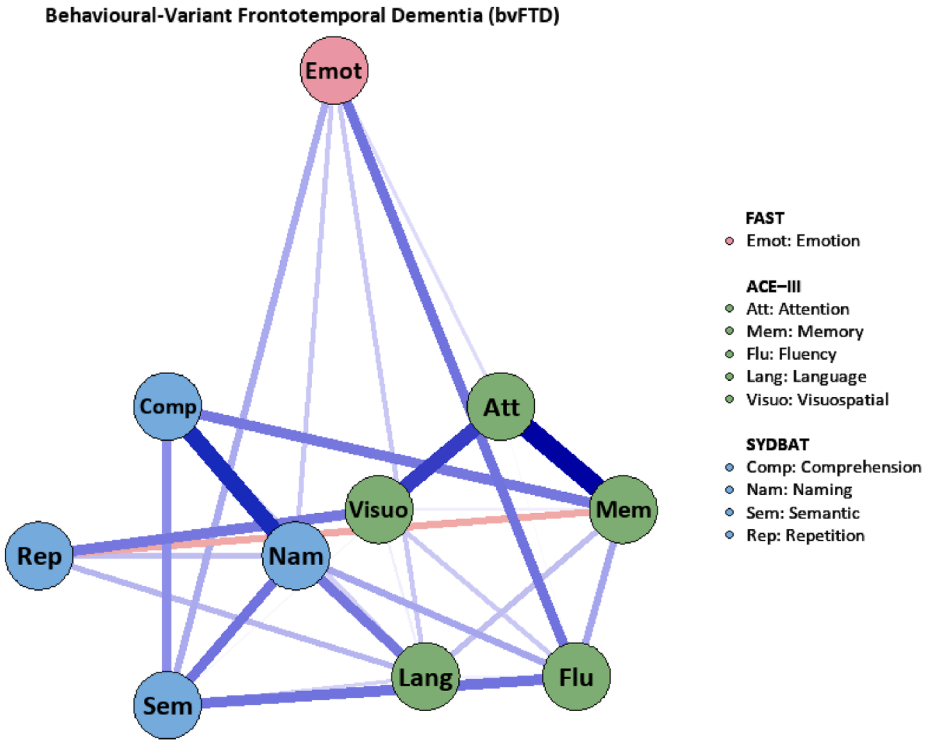

Fig. 2 – Network model of cognition–emotion mechanisms in behavioural-variant frontotemporal dementia (bvFTD). Note: ACE–III, Addenbrooke's Cognitive Examination–III; FAST, Facial Affect Selection Task; SYDBAT, Sydney Language Battery test. Edge thickness represents the magnitude of partial correlation (edge weight), with thicker lines indicating stronger associations. Blue edges indicate positive correlations, whereas red edges indicate negative correlations.

Fig. D2–D4 for completeness. Bootstrap distributions, centrality plots, and predictability indices (R^2) are also provided in the supplementary materials to support full transparency.

Bootstrap analyses further indicated that apparent differences in node-strength indices should not be interpreted as statistically meaningful, as the corresponding bootstrap confidence intervals overlapped extensively across nodes (Supplementary Fig. C2). Consistent with the low CS coefficients reported for all three diagnostic groups, centrality estimates are presented for completeness and considered exploratory rather than inferential. Edge-weight stability analyses revealed substantial variability in several edges, with many bootstrap confidence intervals crossing zero (Supplementary Tables C1–C3). To provide an objective indicator of variability, we report the mean bootstrap standard deviation across all edges for each group: AD (bootstrapped mean edge weight = .09, SD = .10, 95% CI [-.08, .29]), bvFTD (bootstrapped mean edge weight = .09, SD = .10, 95% CI [-.08, .29]), and HC (bootstrapped mean edge weight = .06, SD = .07, 95% CI [-.04, .21]). These values reflect moderate dispersion and align with prior observations that edge-weight estimates in small networks often show wide confidence intervals yet remain interpretable at the pattern level. Accordingly, interpretations focus on overall network structure and consistent edge-level relations rather than isolated edge estimates.

3.5.1. *Alzheimer's disease (AD)*

The AD network model was relatively sparse (Fig. 1, edge density = .44), indicating fewer robust associations among cognition–emotion measures compared to cognitively healthy groups. The most prominent edges were concentrated within cognitive domains, such as ACE-III attention–memory (bootstrapped mean edge weight = .44, 95% CI [.25, .68]) and ACE-III fluency–language (bootstrapped mean edge weight = .31, 95% CI [.17, .57]). Additional clustering was observed within the SYDBAT, including comprehension–semantic links (bootstrapped mean edge weight = .38, 95% CI [.21, .62]), and between language and naming tasks (bootstrapped mean edge weight = .33, 95% CI [.09, .59]). These patterns suggest that the AD network is organized primarily around within-domain clustering across memory, language, and semantic processes.

Beyond these cognitive domains, FAST Emotion demonstrated selective but meaningful links with other functions. Specifically, it was positively associated with visuospatial ability (bootstrapped mean edge weight = .16, 95% CI [−.08, .42]) and language comprehension (bootstrapped mean edge weight = .28, 95% CI [.08, .56]). Although modest, these edges highlight that emotion recognition in AD relies on broader cognitive resources—particularly visuospatial integration and semantic–linguistic processing—rather than forming widespread or uniform connections across all cognitive domains.

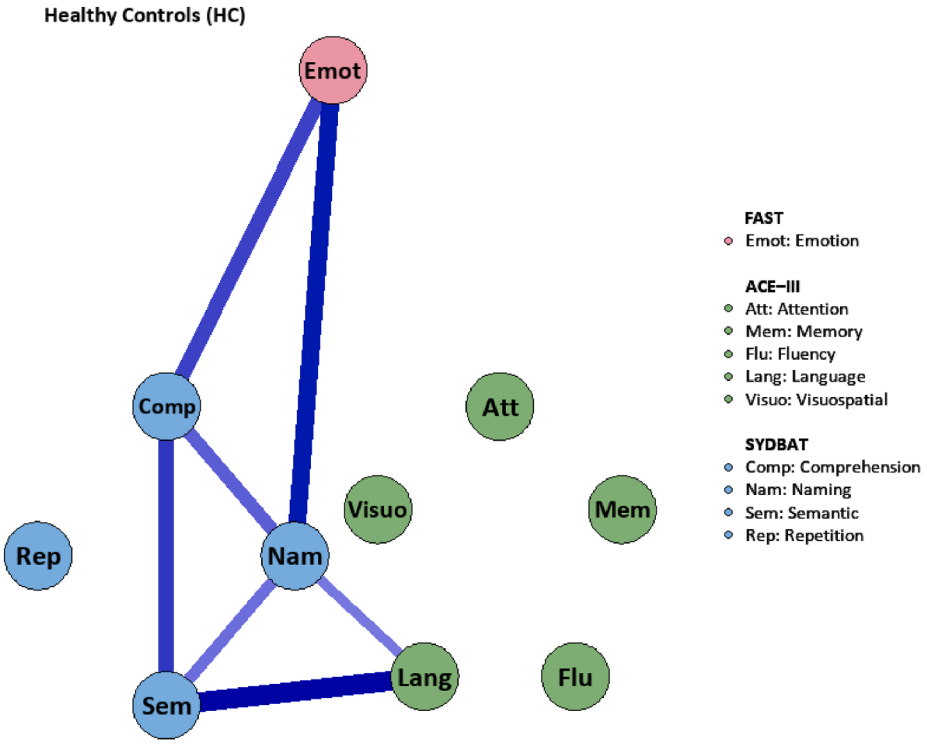

Fig. 3 – Network model of cognition–emotion mechanisms in healthy controls (HC).
Note: ACE–III, Addenbrooke's Cognitive Examination–III; FAST, Facial Affect Selection Task; SYDBAT, Sydney Language Battery test. Edge thickness represents the magnitude of partial correlation (edge weight), with thicker lines indicating stronger associations. Blue edges indicate positive correlations, whereas red edges indicate negative correlations.

Centrality indices provided further insight into network organization. SYDBAT comprehension (strength = 1.28, $R^2 = .63$) and ACE-III fluency (strength = 1.11, $R^2 = .59$) emerged as the most central and predictable nodes, serving as integrative hubs across domains. By contrast, FAST Emotion showed relatively lower centrality (strength = .65, $R^2 = .37$), underscoring its peripheral role in the AD network. Taken together, these results indicate that in AD, emotion processing is shaped by core cognitive hubs—particularly comprehension and fluency—while the overall network remains dominated by within-domain clustering.

3.5.2. *Behavioural-variant frontotemporal dementia (bvFTD)*
The bvFTD network was denser compared to AD (see Fig. 2., edge density = .62), reflecting widespread connections among cognitive and emotion-related nodes. Within-domain links were observed, such as between ACE-III attention and memory (bootstrapped mean edge weight = .45, 95% CI [.26, .69]) and ACE-III attention and visuospatial ability (bootstrapped mean edge weight = .32, 95% CI [.15, .58]). Language-related clustering was also evident, including ACE-III language–SYDBAT naming (bootstrapped mean edge weight = .23, 95% CI [.06, .43]) and SYDBAT comprehension–naming (bootstrapped mean edge weight = .40, 95% CI [.18, .62]). These results indicate that in bvFTD, the network is characterized by broad and integrated clustering across cognitive subdomains, particularly between attention, memory, and language functions.

FAST Emotion showed higher cross-domain connectivity in bvFTD relative to AD. It was positively associated with ACE-III fluency (bootstrapped mean edge weight = .24, 95% CI [.01, .51]) and SYDBAT semantic processing (bootstrapped mean edge weight = .15, 95% CI [−.07, .38]), with additional modest edges to ACE-III language (bootstrapped mean edge weight = .11, 95% CI [−.11, .31]) and naming (bootstrapped mean = .13, 95%CI [−.12, .33]). These findings indicate that emotion recognition in bvFTD patients is more closely tied to expressive and semantic–linguistic functions, rather than visuospatial integration as observed in AD. The network pattern thus reflects that impairments in socio-emotional processing in bvFTD may stem from disruptions in fluency and semantic systems central to communication and meaning.

Centrality and predictability analyses reinforced this interpretation. SYDBAT naming (strength = 1.29, $R^2 = .74$) and ACE-III memory (strength = 1.20, $R^2 = .63$) emerged as highly central and predictable nodes, acting as key hubs that organize network connectivity. FAST Emotion also displayed higher centrality in bvFTD (strength = .68, $R^2 = .46$) compared to AD, underscoring its more integrated role within the cognitive–emotional system. In contrast, SYDBAT repetition showed low centrality and predictability (strength = .67, $R^2 = .19$), suggesting a limited contribution to the overall network structure. Collectively, these findings suggest that bvFTD is marked by a denser network in which emotion

processing is more directly embedded within core cognitive domains, particularly fluency, memory, and semantic language processes.

3.5.3. Healthy controls (HC)

The HC network was markedly sparse (Fig. 3, edge density = .16), indicating far fewer robust associations among cognitive and emotion-related nodes compared to both dementia groups. Most estimated edges were weak and not statistically reliable, with confidence intervals overlapping zero. Nevertheless, a small number of meaningful associations emerged, particularly within the language domain. For example, ACE-III language showed robust links with SYDBAT semantic processing (bootstrapped mean edge weight = .34, 95% CI [.18, .56]) and naming (bootstrapped mean edge weight = .14, 95% CI [.00, .38]). Additional clustering was observed among SYDBAT tasks, including comprehension–semantic (bootstrapped mean edge weight = .24, 95% CI [.07, .50]) and comprehension–naming (bootstrapped mean edge weight = .21, 95% CI [.04, .42]). These findings suggest that, in HC, network connectivity is concentrated within language and semantic systems, reflecting the intact organization of communication-related processes in the absence of neurodegeneration. Centrality values for the HC network were recomputed on the unthresholded network, yielding small but non-zero scores (see Supplementary Table D).

Emotion–cognition links in HC were limited. FAST Emotion was positively associated with SYDBAT comprehension (bootstrapped mean edge weight = .22, 95% CI [.04, .50]) and naming (bootstrapped mean edge weight = .28, 95% CI [.15, .52]), while its associations with other ACE-III subtests (e.g., memory, attention, visuospatial ability) were weak or non-significant. This selective coupling indicates that, in healthy adults, emotion recognition primarily depends on semantic and lexical functions rather than broader cognitive domains. Such a pattern aligns with the view that efficient socio-emotional processing in healthy individuals is scaffolded by preserved linguistic and semantic resources, in contrast to the more diffuse and compensatory connectivity seen in dementia groups.

Centrality indices further clarified these patterns. The most central and predictable nodes in HC were SYDBAT naming (strength = .96, R^2 = .44), SYDBAT semantic (strength = .86, R^2 = .41), and SYDBAT comprehension (strength = .78, R^2 = .38), underscoring the central role of semantic–linguistic systems. FAST Emotion showed modest strength centrality (strength = .60, R^2 = .32), indicating moderate connectivity with other cognition–emotion nodes. Note that strength centrality reflects the overall magnitude of connections and does not specify which neighbouring nodes drive the associations. As such, language-related patterns were interpreted primarily based on the strongest edges in the network rather than relying solely on node-level centrality metrics. By contrast, ACE-III domains such as attention, memory, fluency, and visuospatial ability showed negligible centrality, suggesting limited contributions to the overall HC network.

3.6. Comparison of Network Structures

Pairwise network comparisons using the NCT revealed no significant differences in overall global strength between AD and bvFTD (*St* = .55, *p* = .411), AD and HC (*St* = 2.19, *p* = .534), or bvFTD and HC (*St* = 2.74, *p* = .300). Similarly, network structure comparisons did not yield statistically significant differences for AD versus bvFTD (*Mt* = .34, *p* = .529), AD versus HC (*Mt* = .47, *p* = .057, trend-level), or bvFTD versus HC (*Mt* = .47, *p* = .043, exploratory significance). Taken together, these findings indicate that although bvFTD shows denser local clustering—particularly in emotion–language couplings—the broader architecture of cognition–emotion interactions remains largely comparable across groups. The relative sparsity observed in HC networks is likely influenced by restricted variance due to ceiling effects (Šimkovic & Träuble, 2019), which may limit the detectability of underlying associations in healthy populations. Table 4 presents a summary of NCT results, including global strength, structural differences, and exploratory edge-level comparisons.

3.7. Network integration and node properties

The analysis of correlations among centrality indices, row-complexity, and squared multiple correlation (SMC) revealed distinct patterns across the networks (Fig. 4). These metrics collectively provide insight into how cognitive and emotion-processing functions are organized and integrated. In the AD network, strength and expected influence were highly associated with row-complexity and SMC, indicating that nodes with broader variance, such as SYDBAT_semantic and SYDBAT_comprehension, tended to occupy central positions and exert greater influence over other network nodes. By contrast, betweenness was negatively correlated with row-complexity and SMC, reflecting a more marginal structural role. These results suggest that in AD, language-related nodes are highly integrated and central to the interplay between cognitive and emotion-processing functions.

In bvFTD, strength and closeness were most clearly linked to row-complexity and SMC, highlighting the pivotal role of nodes that bridge cognitive and emotional domains, particularly verbal fluency and FAST_emotion. Negative correlations of betweenness and expected influence with SMC further indicate that different centrality metrics capture complementary aspects of network structure, illustrating the multidimensionality of node centrality in bvFTD. This pattern supports the idea that while the bvFTD network has some central hubs, overall integration is more distributed compared with AD. The HC network exhibited a distinct pattern: strength and expected influence showed positive associations with row-complexity, whereas closeness and SMC were negatively related. This indicates that only a limited subset of nodes—particularly SYDBAT_naming and SYDBAT_comprehension—contributed meaningfully to network organization, consistent with lower density and weaker integration. Predictability was also reduced, suggesting that nodes in HC were less determined by their neighbours relative to clinical groups.

Overall, these findings indicate that network integration and node-level determinism are syndrome-specific, with more pronounced integration in AD and bvFTD than in HC. Importantly, nodes related to language and memory consistently emerged as central, reinforcing their critical role in cognitive-emotion interactions. This provides robust support for the study's central hypothesis that language-related processes shape emotion processing across clinical syndromes.

Table 4 – Edge weights and NCT comparisons for FAST–cognition links.

Node	Edge Weight			*Network Comparison Test* (NCT)		
	AD ($M \pm SD$)	bvFTD ($M \pm SD$)	HC ($M \pm SD$)	p (AD *vs* bvFTD)	p (AD *vs* HC)	p (bvFTD *vs* HC)
FAST_emotion ↔ ACEIII_attention	.02 ± .01	.07 ± .09	−.00 ± .04	.416	1.000	.066
FAST_emotion ↔ ACEIII_memory	.05 ± .08	−.02 ± .10	.08 ± .10	.613	.170	.082
FAST_emotion ↔ ACEIII_fluency	.03 ± .06	.24 ± .13	.04 ± .07	1.000	1.000	1.000
FAST_emotion ↔ ACEIII_language	.03 ± .06	.11 ± .11	.01 ± .04	.215	.593	.054
FAST_emotion ↔ ACEIII_visuospatial	.17 ± .12	.02 ± .10	.02 ± .06	.331	.142	1.000
FAST_emotion ↔ SYDBAT_comprehension	.28 ± .12	−.02 ± .08	.22 ± .12	.021	.893	.074
FAST_emotion ↔ SYDBAT_naming	.00 ± .07	.13 ± .11	.28 ± .09	.312	.018	.376
FAST_emotion ↔ SYDBAT_semantic	.13 ± .12	.15 ± .12	.06 ± .07	1.000	1.000	1.000
FAST_emotion ↔ SYDBAT_repetition	.03 ± .07	−.04 ± .12	.01 ± .03	.780	.508	.306

Notes. Edge weights represent mean partial correlation ± standard deviation from 2,000 bootstrap resamples. NCT p-values indicate significance of differences in edge strength between groups (AD, bvFTD, and HC). AD, Alzheimer's disease; ACE–III, Addenbrooke's Cognitive Examination–III; bvFTD, behavioural-variant frontotemporal dementia; FAST, Facial Affect Selection Task; HC, healthy controls; *M*, mean; *SD*, standard deviation; SYDBAT, Sydney Language Battery test.

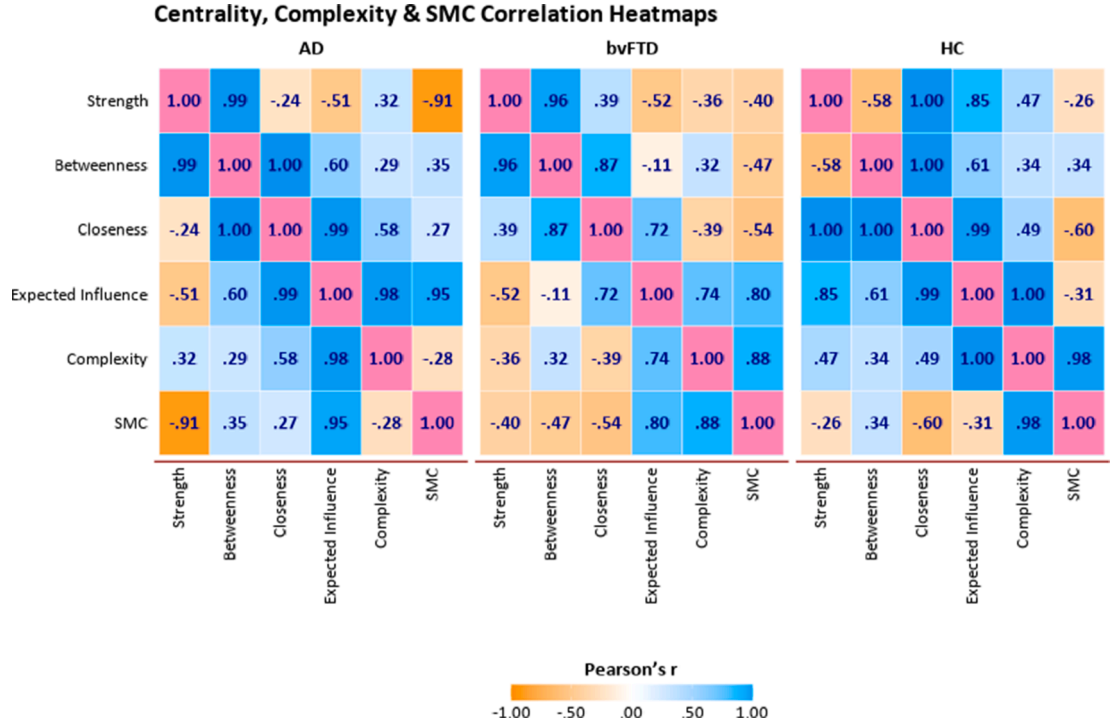

Fig. 4 – Correlation of node centralities, row-complexity and squared multiple correlation (SMC).
Note: Pearson correlations are reported below and above the diagonals. Complexity, Hofmann's row-complexity index; SMC, squared multiple correlation. AD, Alzheimer's disease; bvFTD, behavioural-variant frontotemporal dementia; HC, healthy controls.

3.8. *Robustness checks using regression, correlation, and moderation models*

Before conducting sensitivity analyses, multicollinearity among predictors was assessed using Variance Inflation Factors (VIFs). As reported in Supplementary Tables E1–E2, all VIF values were below 5, indicating that multicollinearity was not likely to bias the regression estimates. Pearson correlation coefficients among predictors confirmed the direction and strength of associations consistent with the network results.

The regression-based sensitivity analyses corroborated the network findings, showing that language-related measures remained the most prominent predictors of emotion processing across groups (Fig. 5; see Supplementary Material E for

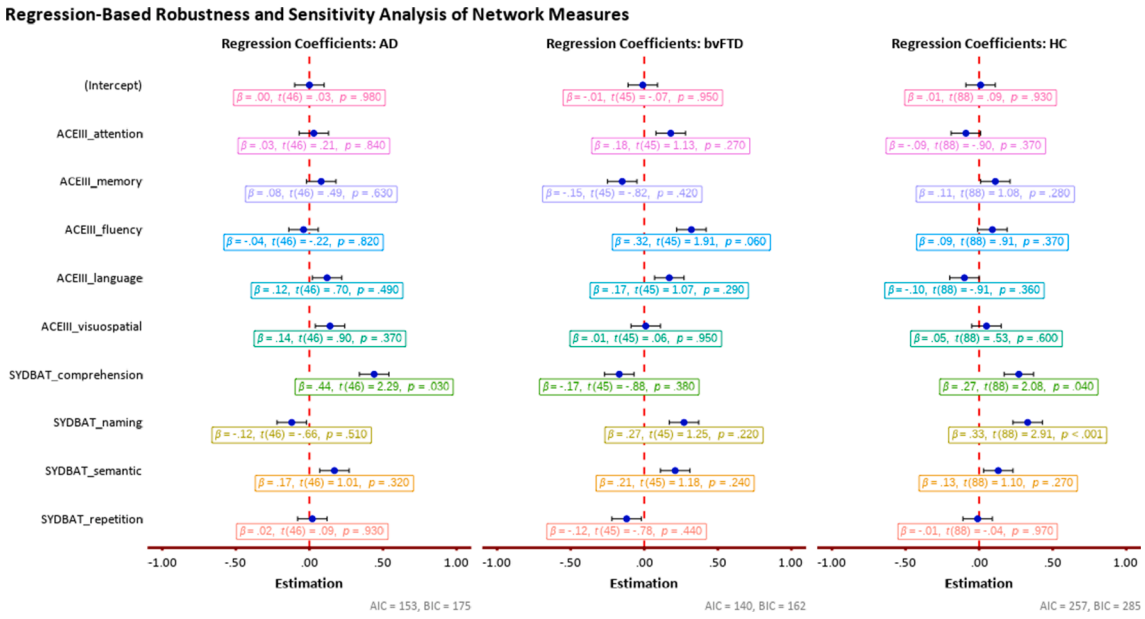

Fig. 5 – Regression-based sensitivity analysis of emotion processing predictors across groups (AD, bvFTD, and HC). Note: Points represent standardized regression coefficients (β) from the linear regression models. Horizontal error bars indicate ±1 standard error (SE) of the estimates rather than 95% confidence intervals. The vertical dashed line indicates zero. Predictors whose error bars cross zero are not statistically significant at $\alpha = .05$. AD, Alzheimer's disease; ACE–III, Addenbrooke's Cognitive Examination–III; bvFTD, behavioural-variant frontotemporal dementia; FAST, Facial Affect Selection Task; HC, healthy controls; SYDBAT, Sydney Language Battery test.

full results). In AD, SYDBAT_comprehension emerged as the only significant predictor of emotion processing, consistent with the network-derived associations. In bvFTD, none of the predictors reached conventional significance, although verbal fluency showed a marginal trend in line with its centrality in the network. In HC, both SYDBAT_comprehension and SYDBAT_naming significantly predicted emotion processing, again converging with the network findings that highlighted the role of language functions.

Overall, these results indicate that the observed associations are not merely artifacts of the network estimation but are substantiated by conventional regression modeling. By converging across analytic approaches, the findings reinforce the robustness of the main conclusion that language-related functions play a central role in shaping emotion processing, with syndrome-specific patterns in AD, bvFTD, and HC.

To formally evaluate whether the strength of these associations differed across diagnostic groups, we conducted moderation analyses including predictor × group interaction terms for all cognitive variables. As shown in Supplementary Tables E2–E3, most interactions were not significant; however, a few interactions reached significance (SYDBAT_naming × Group AD, ACEIII_attention × Group bvFTD, and ACEIII_fluency × Group bvFTD; all p values < .05). These results indicate that some group-specific effects exist, particularly for naming, attention, and fluency, although the majority of cognitive predictors did not show differential slopes across groups. Therefore, while visual differences in slope steepness can be observed (Fig. 6), the overall pattern of associations between cognitive performance and emotion processing remains largely comparable across AD, bvFTD, and HC. Hence, group-specific patterns observed in the networks likely reflect differences in network structure rather than differential regression slopes.

Additional correlation-based sensitivity analyses are presented in Fig. 7, which displays the full Pearson and Fisher's z–transformed correlation matrices for each diagnostic group (AD, bvFTD, and HC). The two correlation metrics yielded identical patterns in terms of direction and relative magnitude of associations, indicating that the observed relationships were not driven by scale-related artifacts or nonlinearity. Importantly, the values shown in Fig. 7 correspond directly to those reported in Supplementary Tables E4–E6, confirming consistency between the graphical and tabular presentations. Across groups, language-related measures—particularly SYDBAT comprehension, naming, attention, and fluency—showed the strongest correlations with FAST emotion processing, mirroring the central edges observed in the network models.

Together with the full correlation matrices for each group (Supplementary Tables E4–E6) and the regression-based robustness checks (Fig. 5), these results strengthen the network findings and indicate that language-related measures—particularly comprehension, naming, attention, and fluency—play a central role in shaping emotion processing across groups, while syndrome-specific differences are

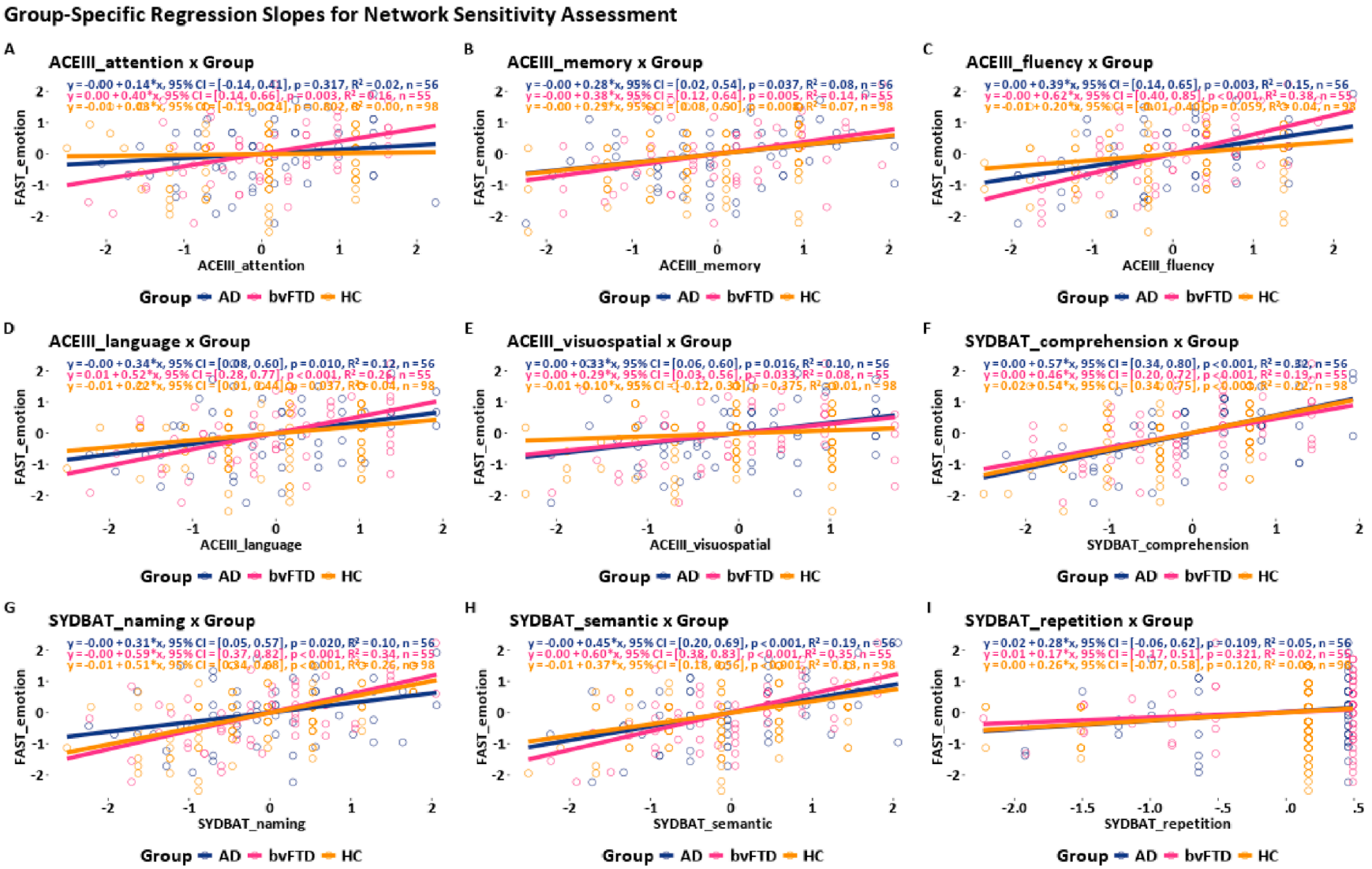

Fig. 6 – Simple slope plots illustrating associations between cognitive predictors and FAST Emotion across diagnostic groups (AD, bvFTD, and HC).
Note: Each panel presents fitted regression lines for AD, bvFTD, and HC along with 95% confidence intervals. Some interactions reached significance (e.g., SYDBAT_naming × Group AD, ACEIII_attention × Group bvFTD, ACEIII_fluency × Group bvFTD; all $p < .05$), highlighting group-specific effects for these predictors, while most associations were broadly consistent across groups. AD, Alzheimer's disease; ACE–III, Addenbrooke's Cognitive Examination–III; bvFTD, behavioural-variant frontotemporal dementia; FAST, Facial Affect Selection Task; HC, healthy controls; SYDBAT, Sydney Language Battery test.

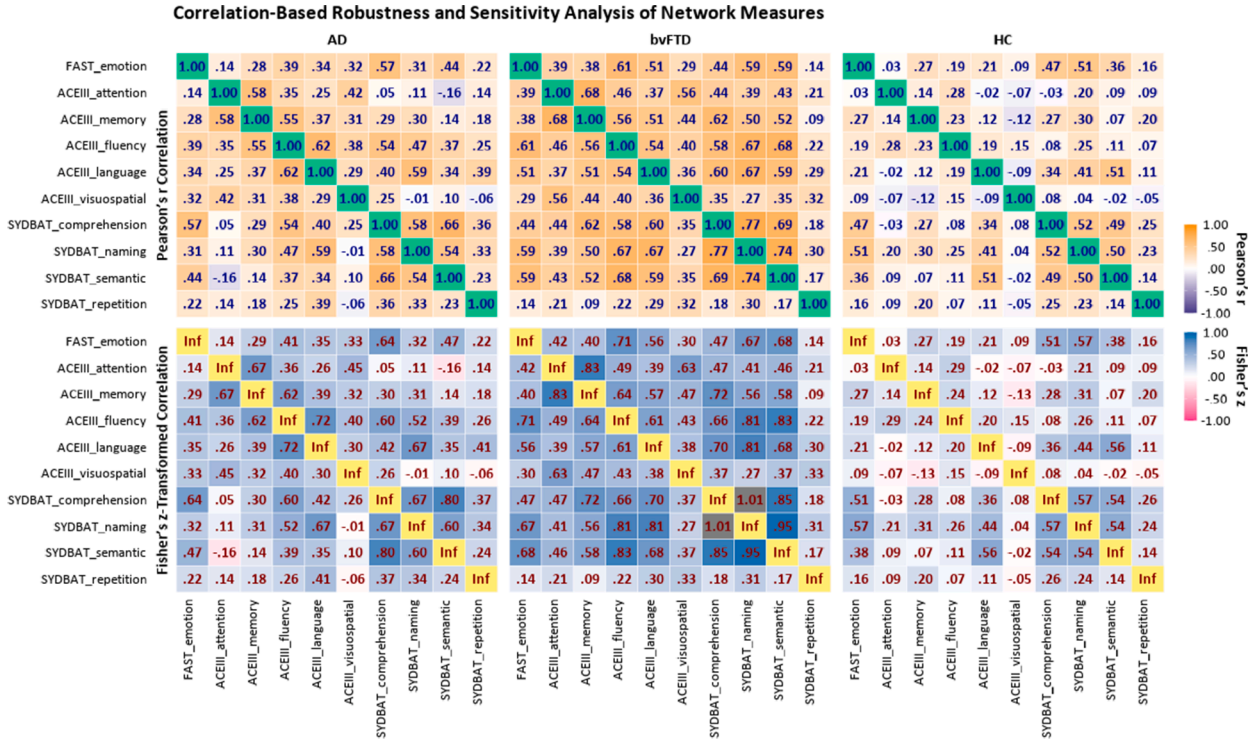

Fig. 7 – Correlation-based sensitivity analysis of emotion processing predictors across groups (AD, bvFTD, and HC). Note: AD, Alzheimer's disease; ACE–III, Addenbrooke's Cognitive Examination–III; bvFTD, behavioural-variant frontotemporal dementia; FAST, Facial Affect Selection Task; HC, healthy controls; SYDBAT, Sydney Language Battery test.

primarily reflected in the strength of a few interactions rather than in widespread differential regression slopes.

4. Discussion

The research used network psychometrics to analyse how basic cognitive abilities relate to facial emotion recognition abilities in patients diagnosed with AD and bvFTD and in healthy control participants. Both dementia syndromes showed significant deficits in emotion recognition compared to controls but their cognitive-emotional integration patterns followed different patterns which matched their unique clinical characteristics. In AD, emotion processing was supported primarily by semantic comprehension and visuospatial abilities, whereas it was predominantly associated with verbal fluency performance in bvFTD.

4.1. *Disease-specific network patterns*

Network analyses revealed patterns of associations between cognitive and emotion processing variables that were syndrome specific. The AD network outlined emotion recognition as an element which functioned inside a system dedicated to semantic understanding and visuospatial processing. The connection to visuospatial abilities stems from the need to process complex facial information which requires perceptual abilities that are supported by parietal lobe structures, brain regions undergoing early pathological changes in AD (Zamboni et al., 2024). The link to semantic processing indicates that emotion recognition deficit in this group is also determined by the disturbance in semantic memory systems and conceptual knowledge integrity, which are supported by temporal lobe regions, other common sites of atrophy in AD (Grossman et al., 2003; Rouse et al., 2024; Stam et al., 2023). In contrast, a relatively pronounced relationship between emotion recognition and verbal fluency ability was identified in bvFTD. Verbal fluency is supported by executive control processes that combine word retrieval with task initiation, strategic word searching and mental flexibility. These processes are predominantly underpinned by prefrontal cortices, sites of early atrophy in bvFTD (Delgado-Álvarez et al., 2022).

Network Comparison Tests showed no major differences in global strength or overall structural invariance between AD and bvFTD networks but both dementia groups showed higher network density than healthy controls. Research findings show that cognitive-emotional network architecture keeps its general structure between syndromes yet shows distinct functional patterns between different conditions. In other words, it appears that with disease worsening, as reflected by the progression of brain pathology, cognitive processes undergo network reorganization, where some specialized networks become less efficient or new connections emerge, possibly as compensatory mechanisms (Kanishka & Jha, 2023; Watanabe et al., 2021).

Importantly, similarities in specific network associations between AD and healthy controls should not be interpreted as

reflecting equivalent functional integrity in these groups. Preserved edges likely indicate that normative dependencies between cognitive and emotional processes remain detectable in AD despite overall declines in task performance. In contrast, the absence or reconfiguration of comparable associations in bvFTD suggests a qualitative disruption of cognitive–emotional coupling, consistent with early degeneration of frontal and anterior temporal networks. Thus, network edges primarily reflect patterns of functional coupling rather than absolute performance levels, allowing preserved associations to coexist with impaired performance in neurodegenerative conditions.

4.2. *Neuroanatomical and theoretical integration*

The cognitive networks identified in this study align well with the known neuroanatomical patterns of atrophy observed these younger-onset dementia syndromes known to support these cognitive processes, namely medial temporal and parietal regions in AD (Du et al., 2007; Landin-Romero et al., 2017), and prefrontal, anterior cingulate and anterior temporal regions in bvFTD (Bejanin et al., 2020; Staffaroni et al., 2019). Our research findings further support constructivist theories of emotion processing which posit that effective emotion recognition performance relies on the integration of sensory information with semantic and linguistic conceptual systems (Barrett, 2017; Lindquist et al., 2014). Our results confirm previous findings which showed that semantic degradation may disrupt emotion recognition without affecting visual perception abilities (Bertoux et al., 2020).

4.3. *Emotion recognition as an integrated cognitive system*

The consistent association between emotion recognition and language–semantic performance across groups also has important implications for understanding emotion processing in health and neurodegenerative disease. In healthy individuals, this pattern supports the view facial emotion recognition is not solely a perceptual process but also depends on conceptual knowledge systems that help individuals interpret and categorize emotional signals. From this perspective, semantic and linguistic processes provide the conceptual scaffolding that enables sensory facial cues to be mapped onto meaningful emotional categories, consistent with constructivist accounts of emotion (Barrett, 2017; Lindquist et al., 2014). Indeed, recent empirical work indicates that conceptual and linguistic knowledge can shape the perceptual representation of emotional expressions and facilitate emotion inference from facial cues (Liu et al., 2024; Ventura-Bort et al., 2023). In neurodegenerative conditions, the persistence of these associations despite overall cognitive decline suggests that the structural organization linking cognitive and emotional processes may remain partially preserved even when absolute performance deteriorates. Rather than reflecting isolated impairments, emotion recognition difficulties in dementia therefore appear to emerge from disruptions within an integrated cognitive–emotional system. Consequently, deficits in semantic or language systems may indirectly compromise emotion recognition by limiting the conceptual resources required to interpret emotional expressions.

4.4. *Socio-cognitive contributions to emotion processing*

Beyond the network-level findings, the regression analyses further indicate that the socio-cognitive variables examined in this study explain a substantial proportion of the variance in emotion recognition performance. This result underscores that emotion recognition is not an isolated perceptual skill but rather is embedded within a broad socio-cognitive architecture. The predictive contributions of language–semantic abilities, attentional control, and executive processes suggest that successful interpretation of facial expressions depends on the coordinated interaction of multiple cognitive systems involved in perceptual analysis, conceptual categorization, and goal-directed evaluation of social signals. This interpretation is consistent with contemporary models of emotion perception which emphasize that emotion recognition emerges from distributed cognitive and neural systems rather than from a single specialized module (Adolphs, 2017; Kober et al., 2008; Lindquist et al., 2012). Importantly, the relatively high explanatory value of the regression models indicates that socio-cognitive abilities provide meaningful insights into the cognitive architecture supporting emotion recognition in both healthy individuals and neurodegenerative conditions. Nevertheless, the present models were necessarily limited to the cognitive domains assessed in the available neuropsychological battery. Future research incorporating additional socio-cognitive constructs—such as mentalizing, social reasoning, and broader semantic knowledge—may further clarify how different cognitive systems interact to support emotion recognition across health and disease.

4.5. *Syndrome-specific cognitive pathways*

Further insights emerge from the interaction effects observed in the regression analyses, which suggest that the relationship between specific cognitive domains and emotion recognition differs across diagnostic groups. Notably, the stronger association between verbal fluency and emotion recognition observed in the patient groups may indicate that individuals with neurodegenerative conditions increasingly rely on executive–linguistic strategies to interpret emotional expressions when other cognitive resources deteriorate. Recent studies in AD have similarly shown that emotion recognition performance is closely linked to executive functions rather than to dementia severity itself (Buçgün et al., 2024). The strong association between attention and emotion recognition in bvFTD further highlights syndrome-specific patterns of cognitive–emotional integration. Given the prominent involvement of frontal networks in bvFTD, attentional control mechanisms may play a critical role in supporting the processing of socially relevant stimuli, consistent with evidence that attention can mediate the relationship between executive functioning and emotion recognition in neurodegenerative conditions (Chiang et al., 2024). In contrast, the weaker association between naming and emotion recognition in AD compared with bvFTD and healthy controls may reflect the

characteristic degradation of semantic memory systems in AD, which disrupts the conceptual representations necessary for linking perceptual facial cues with emotion categories. Supporting this interpretation, recent neuroimaging studies in individuals with cognitive impairment have shown that emotion recognition performance is associated with broad neurocognitive and structural brain changes underlying social cognition (Gandia-Ferrero et al., 2024). Taken together, these interaction patterns indicate that although emotion recognition deficits occur across neurodegenerative syndromes, the cognitive pathways supporting this ability may differ, potentially reflecting disease-specific neural degeneration as well as compensatory recruitment of remaining cognitive systems.

4.6. *Directions for future research*

Taken together, the present findings also open several directions for future research. The syndrome-specific patterns observed in the cognitive–emotional networks suggest that emotion recognition impairments in neurodegenerative disorders arise through different cognitive pathways rather than from a single disrupted mechanism. Future studies could therefore test more explicit mechanistic models examining how executive, attentional, and semantic systems interact to support emotion recognition across different stages of disease progression. This perspective aligns with recent research emphasizing that social and emotional cognition emerges from large-scale distributed brain systems supporting both perceptual and higher-order cognitive processes (Olsson et al., 2020; Schurz et al., 2021). In addition, the interaction effects observed in the regression analyses raise the possibility that certain cognitive domains may partially compensate for declining socio-emotional abilities in neurodegenerative conditions. Longitudinal research designs will be particularly important for determining whether the stronger associations observed in patient groups reflect adaptive compensatory recruitment or progressive reorganization of cognitive networks as pathology advances. Finally, integrating network psychometrics with multimodal approaches such as structural or functional neuroimaging may help clarify how disease-specific patterns of neural degeneration shape the neural architecture of social cognition and emotion recognition (Dilcher et al., 2023).

4.7. *Limitations and future directions*

Several methodological considerations contextualize the interpretation of these findings. First, the network structures and centrality patterns identified here are inherently dependent on the specific assessment battery employed. The prominence of language-related nodes reflects both the genuine functional importance of semantic and linguistic processes in emotion recognition, as well as the composition of our test battery, which included multiple language measures (ACE–III Language, ACE–III Fluency, SYDBAT Naming, SYDBAT Comprehension, SYDBAT Semantic). Arguably, had we incorporated different executive function or visuospatial tasks instead, the resulting network topology and centrality rankings would likely differ. As such, the networks presented here need to be understood as characterizing the relationships among these specific measures employed rather than representing absolute ground truth architectures. The stability analyses revealed that edge weights were reliable across bootstrap resamples, but centrality indices showed limited stability, consistent with prior research demonstrating that these metrics are less robust in smaller samples or sparse networks (Epskamp, Borsboom, & Fried, 2018). Accordingly, our interpretations focused primarily on edge-level associations and network density patterns, while node-level metrics were interpreted cautiously. Nevertheless, node centrality metrics were retained to provide exploratory insights into the relative positioning of cognitive domains within the cognition–emotion networks. Even when stability is limited, these indices still offer descriptive information regarding which cognitive functions tend to occupy more interconnected positions within the network architecture. In the present study, centrality patterns therefore serve primarily as heuristic indicators of potentially influential cognitive domains rather than definitive evidence of causal importance. As such, they complement the edge-level findings by highlighting candidate processes that may play a more integrative role in the cognition–emotion system and that may warrant further investigation in larger or longitudinal samples.

Second, the cross-sectional design arguably prevents us from inferring causal effects within the networks that we identified; in other words, we cannot say whether changes in specific cognitive functions are directly responsible for emotion recognition performance or whether the observed associations reflect co-occurring decline driven by global disease progression. Given the progressive nature of these neurodegenerative conditions, multiple (socio) cognitive functions are impacted simultaneously, resulting in correlations through shared variance related to overall disease progression rather than direct functional dependencies. The sparse network observed in healthy controls, where ceiling effects restrict variance, further supports this interpretation by demonstrating that associations may emerge primarily when pathological processes increase variability across measures. However, the syndrome-specific patterns we identified—semantic comprehension links in AD versus verbal fluency links in bvFTD—suggest that domain-specific mechanisms contribute beyond simple global severity effects. Definitive resolution of these competing interpretations will require longitudinal designs that are able to track the temporal dynamics of cognitive-emotional changes and disentangle disease progression effects from specific functional relationships.

Finally, the use of a single facial emotion recognition task, while methodologically appropriate to minimise confounds from other cognitive demands, captures only one aspect of the broad emotion processing system. While the FAST was selected to minimise confounding effects related to language production demands, performance may still be influenced by language comprehension difficulties, particularly in participants with semantic impairments. Future research incorporating multiple emotion tasks across different modalities, including non-verbal measures, will provide a comprehensive assessment of socio-emotional functioning. Similarly, while our neuroanatomical interpretations align with established patterns of regional atrophy in AD and bvFTD, the absence of neuroimaging data in the current study limits our ability

to directly link behavioural network patterns to underlying brain structures. Integration of structural and functional neuroimaging with network psychometrics represents an important direction for future investigation.

Despite these constraints, our findings demonstrate that emotion recognition impairments in these two younger-onset dementia syndromes reflect integrated cognitive-emotional systems with syndrome-specific organizational patterns. The network approach extends traditional group comparison methods by revealing how disease affects not only individual domains but also the functional architecture linking cognitive and emotional processes. Future research should employ longitudinal designs to examine how these network patterns evolve during disease progression, incorporate neuroimaging to establish brain-behaviour correspondences, and extend the approach to additional neurodegenerative syndromes to determine whether the patterns observed represent syndrome-specific features or more general principles of cognitive-emotional integration in neurodegeneration.

5. Conclusion

Network analysis methods uncovered syndrome-specific patterns of cognitive-emotional integration in AD and bvFTD. In AD, emotion recognition was most strongly associated with semantic comprehension and visuospatial processing, reflecting the involvement of temporal and parietal brain systems. In bvFTD, emotion recognition was primarily linked to verbal fluency, highlighting the contribution of prefrontal executive processes. While these patterns align with established profiles of brain atrophy in these syndromes, alternative mechanisms—such as the role of the amygdala, anterior temporal lobe, or orbitofrontal cortex—may also support emotion recognition. Overall, our findings suggest that emotion recognition deficits in dementia emerge from disruptions within integrated cognitive-emotional systems, with syndrome-specific organizational patterns. These insights underscore the utility of network approaches in understanding cognitive-emotional interactions and may inform the development of targeted interventions for socioemotional impairments in neurodegenerative conditions.

CRediT authorship contribution statement

Andi Tri Supratno Musrah: Writing – review & editing, Writing – original draft, Investigation, Formal analysis. **Dwi Yan Nugraha:** Writing – review & editing, Methodology, Formal analysis. **Fiona Kumfor:** Writing – review & editing, Supervision, Resources, Investigation. **Olivier Piguet:** Writing – review & editing, Validation, Supervision, Investigation, Funding acquisition, Formal analysis, Conceptualization.

Consent to participate

Participants and or the Person Responsible were provided written informed consent in accordance with the standard informed consent of the Declaration of Helsinki.

Ethical consideration

The study was approved by The South Eastern Sydney Local Health District, The University of New South Wales, and The University of Sydney ethics committees (HREC 10/126, HREC 13/177, HREC 10/092, and HREC 2020/224). All participants who volunteered were financially reimbursed for their time and travel costs.

Consent for publication

Not applicable.

Data availability statement

Processed experimental data are available for open access: https://osf.io/8c3pm/

Transparency statement

DATA: All processed data supporting this research are publicly available: https://osf.io/8c3pm/

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Declaration of competing interest

The author(s) declared no potential conflicts of interest with respect to the research, authorship, and/or publication of this article.

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Scientific transparency statement

DATA: Some raw and processed data supporting this research are publicly available, while some are subject to restrictions: https://osf.io/8c3pm/

CODE: All analysis code supporting this research is publicly available: https://osf.io/8c3pm/

MATERIALS: Some study materials supporting this research are publicly available, while some are subject to restrictions: https://www.sydney.edu.au/brain-mind/our-clinics/dementia-test.html.

DESIGN: This article reports, for all studies, how the author(s) determined all sample sizes, all data exclusions, all data inclusion and exclusion criteria, and whether inclusion and exclusion criteria were established prior to data analysis.

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

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

Supplementary data

Supplementary data to this article can be found online at https://doi.org/10.1016/j.cortex.2026.03.009.

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