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Investigating the Multidimensional Structure of Apathy in Cognitive Decline: An Exploratory Network Analysis of the Apathy Evaluation Scale

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ABSTRACT

Objective: *Apathy is a multidimensional syndrome present in neurodegenerative disorders such as Alzheimer's disease and mild cognitive impairment, distinct from depression. It requires targeted assessment tools able to explore and measure it as a complex system. This study aimed to explore the latent structure of apathy in individuals with cognitive decline using Exploratory Graph Analysis (EGA) applied to the Apathy Evaluation Scale – Clinician version (AES-C), and to compare this structure with traditional factorial models.* ***Methods:*** *A total of 214 individuals, including patients with mild cognitive impairment (MCI), Alzheimer's disease (AD), and healthy controls, completed the AES-C and a standardized neuropsychological battery. EGA was used to estimate the network-based factor structure of the AES-C. Confirmatory factor analysis compared the EGA-derived model with both the original structure proposed by Marin et al. and a model derived through exploratory factor analysis (EFA).* ***Results:*** *EGA revealed a four-factor structure: interest and motivation, autonomy, novelty, and social apathy. This solution demonstrated better fit,*

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parsimony, and conceptual clarity than both the original and EFA-derived models. The EGA model showed good internal consistency, convergent and discriminant validity, and performed consistently across MCI and AD subgroups. ***Conclusions:*** *EGA provided a clearer understanding of apathy by identifying specific, clinically relevant, and objective dimensions that differ in disease progression. This precise characterization facilitates targeted and timely interventions and has the potential to initiate the development of new test batteries fully validated through network methods, allowing more complex analysis of apathy symptoms.* (Am J Geriatr Psychiatry 2026; 34:450–461)

Highlights

- **What is the primary question addressed by this study?**
  Can exploratory graph analysis (EGA) provide a more clinically informative structure of apathy in cognitive decline compared to traditional models?
- **What is the main finding of this study?**
  EGA identified a four-factor structure of apathy—interest and motivation, autonomy, novelty, and social apathy—which outperformed both Marin's original model and the exploratory factor analysis-derived solution in terms of model fit and conceptual clarity. This structure demonstrated good internal consistency and validity across individuals with mild cognitive impairment and Alzheimer's disease.
- **What is the meaning of the finding?**
  Network-based methods can enhance apathy assessment by identifying specific, actionable, and clinically relevant dimensions that may manifest differently across neurodegenerative disorders, thus supporting more targeted interventions. These findings may also promote the broader adoption of network psychometrics in the development, refinement, and calibration of neuropsychological tests, enabling more nuanced assessment of complex syndromes such as apathy.

BACKGROUND

Apathy is a frequent and debilitating symptom in neurocognitive disorders, particularly in Alzheimer's disease (AD) and mild cognitive impairment (MCI).[1] Clinically, it is defined as a quantifiable reduction in goal-directed activity, with impairment for at least 4 weeks in at least two domains: behavior/cognition, emotion, and social interaction.[2] It is considered distinct from depression, as it is not attributable to reduced consciousness, cognitive decline, or emotional distress.[3] Contemporary research supports a view of apathy as a multidimensional syndrome, with evidence from several neurological conditions, including Parkinson's disease,[4] multiple sclerosis,[5] and various forms of dementia.[6]

In MCI and AD, apathy is associated with faster cognitive decline,[7] reduced treatment response,[8] increased mortality,[9] and a higher risk of conversion to dementia.[10] Notably, only a portion of apathy's variance is explained by cognitive or affective factors, reinforcing its role as an independent, complex construct.[11] Recent studies using machine learning have also shown that apathy enhances diagnostic classification across cognitive stages, further confirming its clinical relevance.[12]

These findings underscore the need to investigate apathy through models that reflect its internal complexity. Among the proposed frameworks, the ABC model of apathy—encompassing affective, behavioral, and cognitive components—has gained traction.[13] This model posits that apathy arises from impairments in emotional responsiveness (affective), initiation of actions (behavioral), and cognitive engagement (cognitive). Crucially, this tripartition does not simply label different manifestations of a unified trait but rather defines apathy as a network of interconnected but non-redundant elements.

Several instruments have been developed from this perspective. Among these, the Apathy Evaluation Scale (AES), developed by Marin et al., stands as the earliest and potentially the most significant formalized scale.[14] The clinician-rated version (AES-C) demonstrated its validity across different populations and has been recently standardized in AD samples.[15]

Most validation studies of the AES have employed traditional latent variable models, such as exploratory factor analysis (EFA), which assume that observable symptoms stem from a single latent trait. However, this assumption is increasingly challenged by both theoretical models and clinical findings.[16] A recent work suggests that apathy components vary in salience across disease stages: for example, cognitive apathy (abulia) is more central in AD, while all three domains are equally relevant in cognitively healthy individuals.[11] These dynamic patterns are not well captured by classical factor models.

To address this, network-based approaches have been proposed. The network theory of psychopathology views symptoms as mutually interacting elements that may reinforce one another over time.[17] In this framework, psychopathological constructs are better understood as emergent properties of these interactions, rather than manifestations of hidden causes.[18] This approach has been successfully applied to a range of disorders, including unipolar and bipolar depression,[19–21] schizophrenia,[22] and neurodegenerative diseases.[23]

Exploratory Graph Analysis (EGA) is a recent development within network psychometrics.[24] Unlike EFA, which assumes underlying latent variables, EGA uses partial correlations to identify clusters of items—interpreted as dimensions—within a network structure. The method is based on Gaussian graphical models estimated via the graphical LASSO algorithm and includes data-driven detection of item communities.[25] This allows dimensions to emerge organically from the data, reducing researcher bias and enhancing interpretability.

EGA also offers several advantages: it provides a visual representation of item relationships, facilitates the exploration of multidimensional constructs, and has shown superior accuracy in detecting the correct number of dimensions in simulations and real-world data.[26,27] Despite these strengths, EGA has not yet been applied to apathy questionnaires, especially in populations with MCI or AD.

Given the complexity of apathy and the theoretical limitations of existing methods, this study aimed to explore the dimensional structure of the AES-C using EGA and to compare it with both the original model by Marin et al. and a factor solution obtained through EFA.[14] This comparison will help determine whether network-based models offer a better framework to capture the multifaceted nature of apathy in neurodegenerative disorders.

METHODS

Participants

A total of 214 subjects were recruited and divided into three different groups. The experimental group consisted of 107 patients, including 34 males and 73 females, selected at the UOS Alzheimer's Centre and Psychogeriatrics of the ASP3 DSM in Catania, Italy. The new diagnostic criteria of the National Institute of Aging and the Alzheimer's Association for patients with probable AD and those with amnestic MCI due to AD were applied.[28] In this case, AD was defined as "probable" because the diagnosis was made only by relying on clinical signs/symptoms, and not on biomarkers, while MCI was defined as "amnesic" according to Petersen distinction,[29] because patients with mainly single-domain memory deficits were selected. The criteria for probable AD and for amnestic MCI due to AD are reported elsewhere.[28,30]

For screening the general cognitive functioning, the standardised Italian version of the Mini Mental State Examination (MMSE) was used, including patients with a total score, adjusted for age and education, between 18 (inclusive) and 28. The choice of MMSE and thresholds was made according to the Italian guidelines for cognitive deterioration staging (AIFA, 2023)[31]. Of the 107 patients, 77 were diagnosed with MCI due to AD and 30 with mild AD.

The control group consisted of 107 healthy subjects, 29 males and 78 females, who obtained MMSE scores of 28 or higher. Coherently with the cited guidelines, their anamnesis, cognitive performance, and daily autonomy were considered to ascertain their healthy status.

The study was conducted in accordance with the Declaration of Helsinki and its later amendments. The protocol was approved by the Internal Ethical

Review Committee of the Department of Educational Sciences (Psychology Section) of the University of Catania (Ierb-Edunict-2023.05.23/02, 23/05/2023). The research procedures followed the guidelines of the Italian Psychology Association (AIP) and the relevant Ethics Council. All participants provided informed written consent to participation and publication of the results of this study.

Materials

The neuropsychological assessment was carried out by licensed psychologists in a controlled setting. The following tests were administered to both the patient and the control group:

- The Apathy Evaluation Scale − Clinician version (AES-C)[14] is an 18-item rating scale, structured on a 4-point Likert answer modality with a total score that ranges from 18 to 72. Ratings are determined by the clinician's evaluation of the patient's self-reports. In other words, aside from the self-evaluation items which are coded exclusively on the subject's rating of severity, the scores for the AES-C are based on the clinician's best judgment (or "objective" assessment) of the patient's motivational state. In the context of this study, the scale was administered by a clinician following a semi-structured interview with the patient to ensure that symptoms were accurately reported.
- The Mini Mental State Examination is a screening tool for cognitive impairment, recommended for deterioration staging by the Italian guidelines cited previously. It explores several cognitive domains, with a maximum score of 30. Corrections for age and level of schooling is required.
- The Montreal Cognitive Assessment (MoCA) is another psychometric tool for general cognitive functioning, with a specific sensitivity for MCI. It measures different cognitive areas and requires age and schooling final adjustment. The maximum total score is equal to 30, and specific cut-offs for Southern Italian older people are available.[32]
- The Italian version of the Frontal Assessment Battery (FAB) measures executive deficits, whose maximum score is equal to 18 and adjustments for age and schooling are required. It examines different areas of executive functions, such as conceptualization, fluency, interference sensitivity, and so on.
- The Italian form of the Hamilton Depression Rating Scale (HDRS) is a semi-structured interview for measuring depressive symptoms. A score <7 indicates no depression; between 8 and 17 indicates mild depression; between 18 and 24 moderate depressions; > 24 severe depression.

Statistical Analysis

Quantitative analyses were conducted on the data collected from both clinical and control participants. Descriptive statistics—including means, standard deviations, and percentages—were calculated to characterize the sample.

Given the ordinal nature of the AES-C items, Spearman's rank-order correlations were computed. To investigate the latent dimensional structure of the AES-C, EGA was conducted using the *EGA package in R. A* graphical lasso (glasso) model was employed to prevent overfitting, and the walktrap algorithm was applied to optimize modularity detection. The resulting network structure and dimensions were visualized using the *qgraph* package and centrality indices were calculated to assess the relative importance of each item within the network.

Following EGA, a confirmatory factor analysis was conducted on the extracted structure. The model was estimated using a weighted least squares mean and variance adjusted (WLSMV) estimator, which is more appropriate than maximum likelihood (ML) estimation when dealing with ordinal data and non-normal distributions.[33,34]

Additionally, an exploratory factor analysis (EFA) was performed to further examine the latent structure of the AES-C. Prior to EFA, sampling adequacy was verified using the Kaiser-Meyer-Olkin (KMO) measure and Bartlett's test of sphericity. Three models—the EGA-derived structure, the EFA-derived structure, and the original Marin model—were fitted using the *lavaan* package in R. Model fit was assessed using several fit indices: chi-square (χ^2), root mean square error of approximation (RMSEA), comparative fit index (CFI), standardized root mean square residual (SRMR), and Tucker-Lewis index (TLI). Comparisons between models were performed using the Satorra-Bentler scaled chi-square difference test,[35] and the

most parsimonious and best-fitting model was selected. The internal consistency of the identified subscales was evaluated using Cronbach's alpha.

Subsequently, the EGA-derived model was tested in a multiple-group confirmatory factor analysis (MGCFA) framework to assess its invariance across participants with Mild Cognitive Impairment (MCI) and Alzheimer's Disease (AD). Model fit was again evaluated using CFA metrics.

Normality of the EGA-derived factor scores was assessed using the Shapiro-Wilk test. To examine convergent and discriminant validity, Spearman's correlations were calculated between AES-C subscales and established clinical measures, including the MMSE, MoCA, FAB, and HDRS.

Finally, to test group differences in AES-C subscale scores across the three diagnostic groups, Kruskal-Wallis tests were conducted. When statistically significant differences were found, Dunn's post-hoc tests were performed to identify specific between-group contrasts. In addition, to assess whether depressive symptoms influenced apathy scores across groups, a multivariate general linear model (GLM) was performed. The four AES-C factor scores were entered as dependent variables, diagnostic group (Control, MCI, AD) was used as a fixed factor, and HDRS total score was included as a covariate. Pairwise comparisons were adjusted using the Sidak correction.

RESULTS

The descriptive statistics for the three groups are presented in Table 1.

The EGA identified four factors that rank the items of the AES. These factors are displayed in Figure 1, and each has been renamed based on the thematic content of the items they group together: Factor 1 − Interests and Motivation (items 1, 3, 6, 7, 9, 10, 11, 15, 17, 18); Factor 2 − Social Apathy (items 12, 13); Factor 3 − Autonomy (items 2, 8, 14, 16); Factor 4 − Novelty (items 4, 5). Edges between nodes represent partial correlations between items, controlling for associations with all other variables in the network; no negative edges were found.

The model's CFA fit indices indicate that the model fits the data well: χ^2 = 117.901; df = 129; p-value = 0.749; SRMR = 0.057; CFI scaled = 0.984; RMSEA scaled = 0.047; TLI scaled = 0.981. The reliability of each resulting factor was assessed using

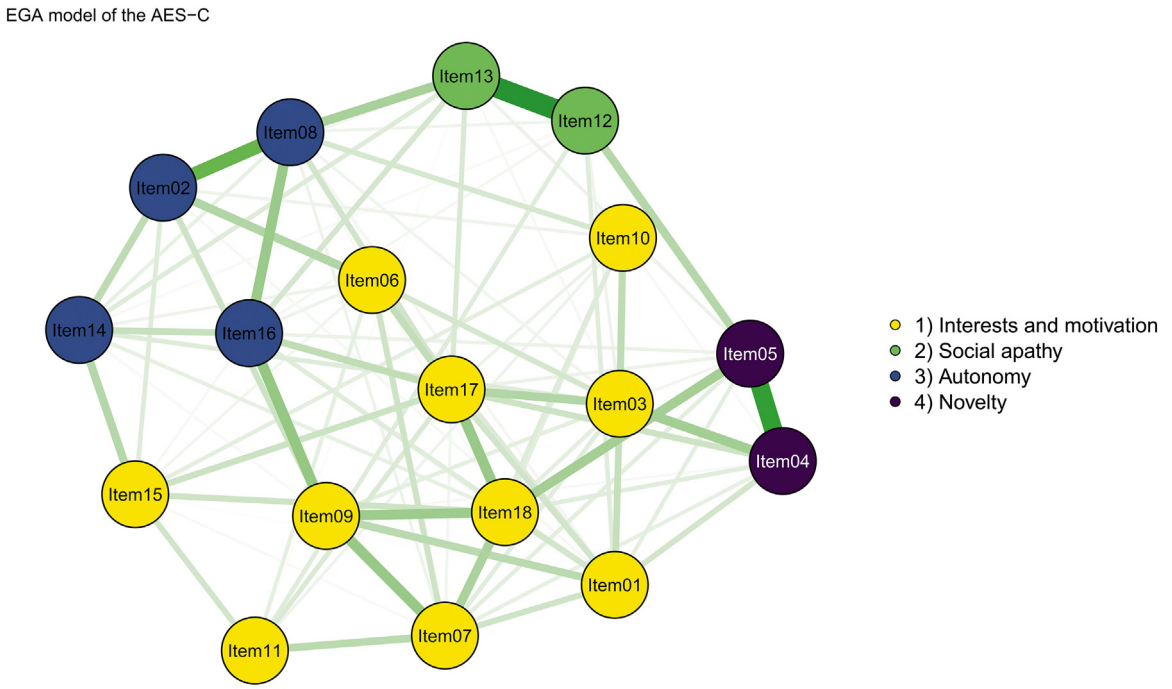
TABLE 1. Demographic Characteristics and Neuropsychological Test Scores for the 3 Diagnostic Groups Included in the study: Cognitively Healthy Controls, Individuals With Mild Cognitive Impairment, and Patients with Alzheimer's Disease

Demographics	Control Group		Mild Cognitive Impairment		Alzheimer's Disease	
	Sample	Percentage	Sample	Percentage	Sample	Percentage
Sample size	107	100	77	100	30	100
Females	78	72.9	54	70.1	19	63.3
Males	29	27.1	23	29.9	11	36.7
Mean Age	74.06 ± 6.8		75.53 ± 7.3		76.33 ± 6.4	
EDUCATION						
- Mean education	10.29 ± 5.7	8.56 ± 4.4	6.87 ± 4.2			
- Primary school	42	39.1	35	45.5	17	56.5
- Secondary school	14	13.1	18	23.4	7	23.4
- High School	22	20.5	15	19.5	4	13.3
- University and more	29	27.1	9	11.7	2	6.6
NEUROPSYCHOLOGICAL ASSESSMENT						
- Mini-Mental State Examination	29.35 (0.84)		24.95 (3.42)		22.00 (2.92)	
- Montreal Cognitive Assessment	26.98 (1.38)		16.55 (4.80)		11.77 (3.51)	
- Frontal Assessment Battery	15.56 (1.88)		10.7 (3.54)		8.97 (3.42)	
- Hamilton Depression Rating Scale	4.37 (4.58)		11.66 (7.81)		6.67 (4.46)	
- Apathy Evaluation Scale	25.42 (7.11)		32.32 (9.46)		38.23 (9.72)	

For Each Group, the table reports sample size, gender distribution, mean age, and level of education (both as mean years and categorical levels). neuropsychological measures include the mini-mental state examination, Montreal cognitive assessment, frontal assessment battery, Hamilton depression rating scale, and apathy evaluation scale − clinician version.

Values are presented as means with standard deviations in parentheses, or as absolute frequencies with corresponding percentages.

FIGURE 1. Exploratory Graph Analysis model of the Apathy Evaluation Scale – Clinician version. Each node represents one questionnaire item, while edges indicate positive Spearman partial correlations between items, with edge thickness corresponding to the strength of the association.



Cronbach's Alpha: Factor 1 alpha = 0.87; Factor 2 alpha = 0.80; Factor 3 alpha = 0.74; Factor 4 alpha = 0.79. In summary, the model demonstrates optimal fit and strong reliability across multiple indices. The high Cronbach's alpha indicates good internal consistency for both the identified factors.

Before comparing the new EGA model of the scale with Marin's model, an EFA was performed, and the EGA model was compared with the factor structure resulting from the EFA. To ensure the suitability of the dataset for factor analysis, the Kaiser-Meyer-Olkin test was conducted and yielded an overall measure of sampling adequacy of 0.91. Bartlett's test was also significant (χ^2 = 1,730.13, df = 153, p < 0.00001), confirming the appropriateness of conducting factor analysis in our data.

The parallel analysis suggested retaining four factors in the EFA. These four factors explained 49% of the total variance (Factor 1: 24%, Factor 2: 12%, Factor 3: 7%, Factor 4: 7%) and showed moderate intercorrelations. However, Factor 4 contained only one item, making its internal consistency incalculable. Furthermore, Item 10 did not independently cluster with any factor and was assigned to Factor 1, with which it showed the highest correlation.

A subsequent EFA was conducted using a more parsimonious model with three factors instead of four. In this model (3-factor-EFA), two items (10 and 11) were again not clearly associated with any factor. After examining their individual loadings, each item was assigned to the factor with which it showed the highest correlation, as done previously.

CFA were conducted on both the 3-factor-EFA and the EGA models. While the EGA model showed excellent fit (as reported above), the EFA-based model yielded slightly weaker fit indices: χ^2 = 156.30; df = 132; p-value = 0.073; SRMR = 0.066; CFI scaled = 0.974; RMSEA scaled = 0.060; TLI scaled = 0.969. The Satorra-Bentler test confirmed that the EGA model provided a significantly better fit than the EFA model ($\Delta\chi^2$ (3) = 20.998, p < 0.001). This was further supported by lower information criteria for the EGA model (AIC = 8,689.38; BIC = 8,830.75) compared to the EFA model (AIC = 8,736.89; BIC = 8,868.16),

indicating superior parsimony and model adequacy for the EGA-based solution.

Finally, the Marin's model was tested to assess its fit with the current data. The model showed considerably poorer fit compared to both the EGA- and EFA-based models: χ^2 (87) = 248.63, p < 0.001; SRMR = 0.090; CFI scaled = 0.916; RMSEA scaled = 0.111; TLI scaled = 0.899. These values fall below commonly accepted thresholds for good model fit, particularly in terms of RMSEA and SRMR.

The EGA-derived model was further tested separately in the MCI and AD patient groups to assess its generalizability across clinical subpopulations. In the MCI group (n = 77), the model showed acceptable fit: χ^2 (129) = 181.88, p = 0.002; SRMR = 0.081; CFI = 0.895; RMSEA = 0.073; TLI = 0.875. The indices indicated a reasonably good fit to the data. In the AD group (n = 30), fit indices were weaker overall: χ^2 (129) = 190.37, p < 0.001; SRMR = 0.111; CFI = 0.789; RMSEA = 0.126; TLI = 0.750. While these values fall below conventional cutoffs, it is important to consider the limited sample size, which is known to compromise fit estimates, especially for RMSEA and SRMR.[36] The Multigroup Confirmatory Factor Analysis (MGCFA) supported configural and metric invariance, indicating a stable factor structure and comparable factor loadings across groups. Although scalar invariance was only marginally supported (p = 0.088), this suggests that observed mean differences should be interpreted with caution. Overall, these findings suggest that the EGA-derived structure is conceptually stable across different stages of cognitive decline, even if its performance is constrained in smaller clinical samples such as the AD group.

For improved clarity and direct comparison, Table 2 summarizes the fit indices for all tested models.

The convergent and discriminant validity of the new subscales of the AES was also assessed by analysing correlations with MMSE, MoCA, FAB and HDRS. The negative correlations between the new EGA model subscales and the cognitive tests (MMSE, MoCA, and FAB), along with the positive correlations with diagnostic groups and HDRS, reflect findings from the existing literature. They indicate an association between higher apathy and cognitive decline, while also showing that, although related, apathy remains distinct from depressive affect. The correlation table can be seen in Figure 2.

The Kruskal-Wallis tests reveal significant differences among the groups for all variables. The subsequent Dunn's post-hoc tests indicate which specific group comparisons are significantly different. All pairwise comparisons between Control and other groups (MCI and AD) show significant differences, while comparisons between MCI and AD were significant for Interest and motivation, and Novelty. The specific group means, H and p values of the tests are reported in Table 3.

To further examine whether depressive symptoms affected the differences observed across groups, a multivariate GLM was performed with the four AES-C subscales as dependent variables and HDRS as a covariate. The overall effect of the Group variable remained significant for all subscales (p < 0.001 for Interest and Motivation, Autonomy, and Novelty; p = 0.002 for Social Apathy), confirming that the observed differences were not explained by

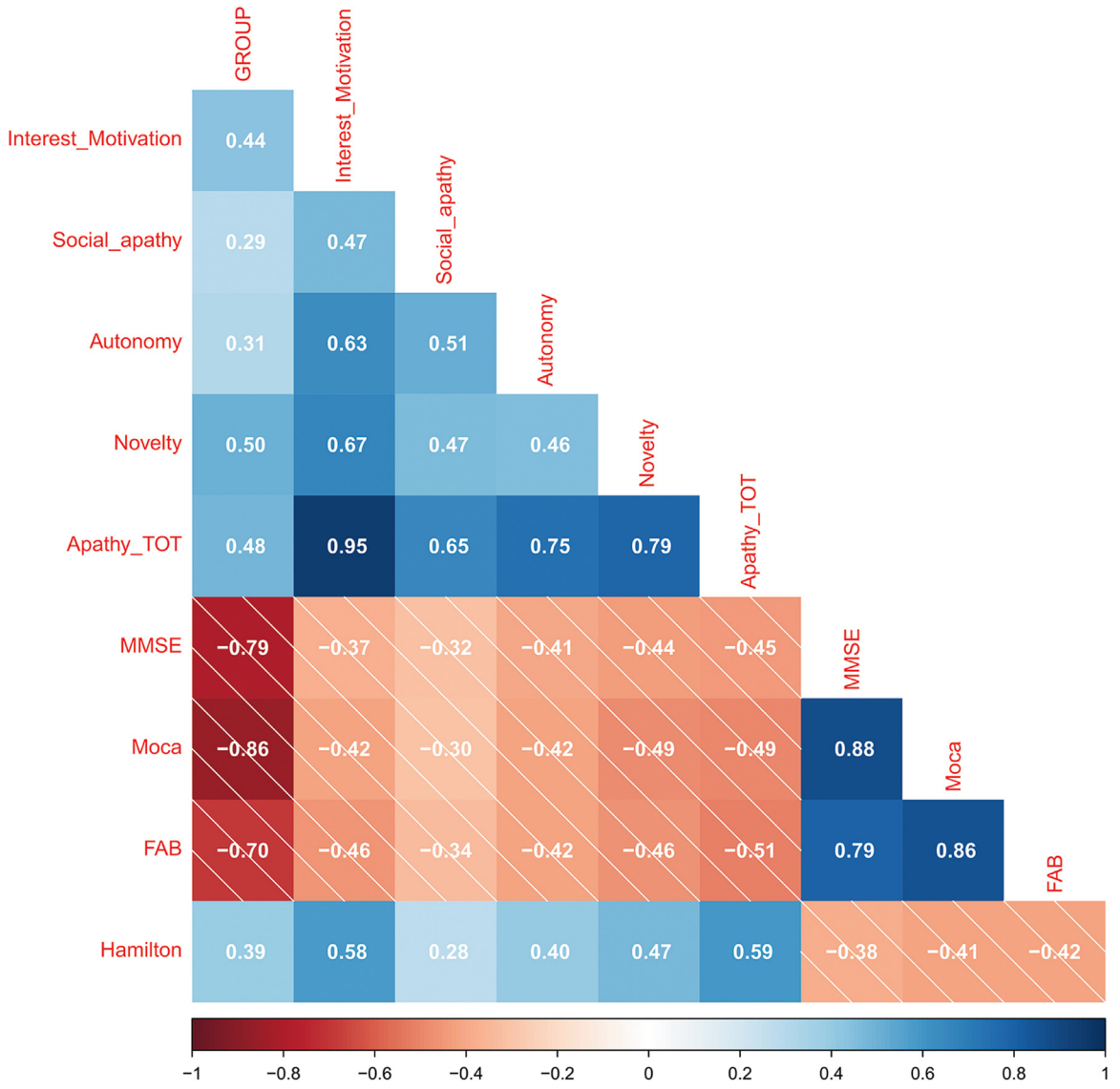
TABLE 2. Key Model Fit Indices for the Competing CFA Models Tested: The Exploratory Graph Analysis (EGA)-Derived Model, the Exploratory Factor Analysis (EFA)-Based Model, and the Original Model By Marin et al

Model	χ^2 (df)	p-Value	SRMR	CFI	RMSEA	TLI	AIC	BIC
EGA-derived model	**117.90 (129)**	**0.749**	**0.057**	**0.984**	**0.047**	**0.981**	**8.689.38**	**8.830.75**
EFA-based model	156.30 (132)	0.073	0.066	0.974	0.060	0.969	8.736.89	8.868.16
Marin's original model	248.63 (87)	< 0.001	0.090	0.916	0.111	0.899	\	\
EGA - Multigroup Confirmatory Factor Analysis								
EGA model – MCI group	181.88 (129)	0.002	0.081	0.895	0.073	0.875	\	\
EGA model – AD group	190.37 (129)	< 0.001	0.111	0.789	0.126	0.750	\	\

Additionally, the performance of the EGA Model is reported separately in the MCI and AD clinical subgroups. Fit Indices Include Chi-Square (χ^2), Degrees of Freedom (df), Standardized Root Mean Square Residual (SRMR), Comparative Fit Index (CFI), Root Mean Square Error of Approximation (RMSEA), Tucker–Lewis Index (TLI), Akaike Information Criterion (AIC), and Bayesian Information Criterion (BIC). AIC and BIC values are only reported for the models directly compared via Satorra–Bentler scaled Chi-Square tests.

Bolded entries indicate the best-performing model (EGA) across the main fit indices.

FIGURE 2. Spearman correlation matrix between the four EGA-derived subscales (Interests and motivation, Social apathy, Autonomy, Novelty) and the apathy evaluation scale total score, in relation to clinical and cognitive variables. These include the diagnostic group (categorical: 1 = Control, 2 = Mild Cognitive Impairment, 3 = Alzheimer's Disease), Mini-Mental State Examination (MMSE), Montreal Cognitive Assessment (MoCA), Frontal Assessment Battery (FAB), and Hamilton Depression Rating Scale (HDRS).



depression. Pairwise comparisons, performed after controlling for HDRS, confirmed that AD patients had significantly higher scores than MCI patients on the Interest and Motivation subscale (mean difference = 6.21, SE = 1.08, $p < 0.001$) and the Novelty subscale (mean difference = 1.84, SE = 0.39, $p < 0.001$). Furthermore, only after controlling for HDRS, the Autonomy subscale showed significantly higher scores in AD patients compared to MCI patients (mean difference = 1.4, SE = 0.46, $p = 0.007$). Multiple comparisons and significance levels across all subscales are reported in Figure 3.

DISCUSSION

The aim of this study was to apply exploratory graph analysis to the AES-C and compare the

TABLE 3. The Table Reports the Results of Kruskal-Wallis Tests (χ^2 and p-values) and Dunn's Post-Hoc Pairwise Comparisons (Mean Differences, Z-Scores, and Adjusted p-values) for the 4 Exploratory Graph Analysis Derived Factors of the Apathy Evaluation Scale-Clinician Version, Across Diagnostic groups: Healthy Subjects (control), Mild Cognitive Impairment (MCI), and Alzheimer's Disease (AD) Patients

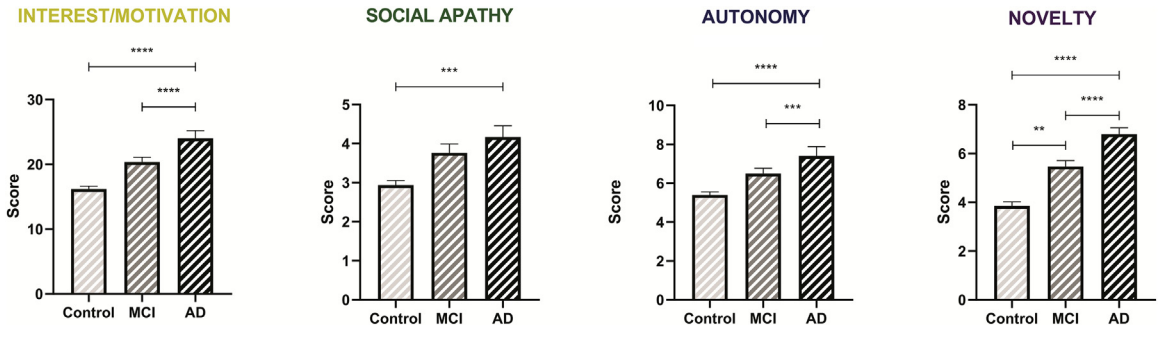
EGA Factor	χ^2 (p)	Comparison	Mean Difference	z	p-Value
Interest and motivation	41.31 (<0.0001)	Control vs MCI	−4.186	−40.61	<0.0001
		Control vs AD	−7.855	−74.08	<0,0001
		MCI vs AD	−3.669	−33.47	0.0356
Social apathy	18.5 (<0.0001)	Control vs MCI	−0.831	−23.5	0.0234
		Control vs AD	−1.232	−49.3	0.0002
		MCI vs AD	−0.4	−25.8	0.1289
Autonomy	20.7 (<0001)	Control vs MCI	−1.101	−28.2	0.0053
		Control vs AD	−2.007	−51.2	0.0001
		MCI vs AD	−0.906	−23	0.2321
Novelty	51.7 (<0.0001)	Control vs MCI	−1.617	−46.2	<0.0001
		Control vs AD	−2.949	−84.2	<0.0001
		MCI vs AD	−1.332	−38	0.0115

resulting structure with the original model proposed by Marin et al., and one derived through EFA. This approach aimed to evaluate whether a network-based methodology could offer a novel and clinically informative representation of apathy, particularly in populations affected by cognitive decline, such as individuals with MCI and AD.

EGA identified a four-factor structure—interest and motivation, social apathy, autonomy, and novelty—which outperformed both the EFA-derived and Marin's models in terms of model fit, parsimony, and psychometric robustness. The EFA solution included an unstable factor with poorly defined item loadings, while Marin's model showed inadequate fit to our clinical sample. In contrast, the EGA-derived solution demonstrated good internal consistency, acceptable fit indices across subgroups, and conceptual clarity. Notably, all four factors align with current views of apathy as a multidimensional construct. From a clinical perspective, a network-based approach may enhance the assessment of apathy by capturing more specific symptom profiles—such as reduced motivation, limited engagement in novel activities, or social disengagement—thereby informing more targeted interventions. This may be particularly valuable in neurodegenerative conditions, where early and domain-specific identification of apathy can contribute to maintaining patient autonomy, improving quality of life, and alleviating caregiver burden.

The multidimensional structure derived from EGA resonates with the broader literature describing apathy as a syndrome that encompasses affective, behavioral, cognitive, and social components.[2] Previous studies have suggested subtypes such as emotional

FIGURE 3. Multiple comparisons between the four-factor derived from the Exploratory graph analysis in the three study groups: control, MCI (mild cognitive impairment) and AD (Alzheimer's disease). The significance between the various groups was indicated according to the following scheme: p < 0.05 *; p < 0.01 **; p <0.001 *; p < 0.0001 ****. The color of each title matches the group's colors in Figure 1.**



blunting, executive dysfunction, and behavioral inertia, with apathy manifesting not only as a lack of motivation but also as impairments in daily routines, social interactions, and cognitive functioning.[37] Our findings offer an empirically grounded framework that parallels and refines these classifications by delineating clinically relevant and actionable dimensions.

The identification of four distinct apathy dimensions—Interest and Motivation, Social Apathy, Autonomy, and Novelty—not only advances the psychometric understanding of the construct but also provides a theoretical framework for future research. These components may serve as specific phenotypic markers in longitudinal studies tracking disease progression and support the development of novel assessment tools grounded in network-based approaches, which are better suited to capture the complexity of symptoms in neurodegenerative conditions.

The factor Interest and motivation highlights the motivational deficits and disruptions in habitual behaviours.[38] Autonomy reflects a diminished sense of agency and self-directed behavior, which often leads to dependence on others by affecting personal initiative. Novelty refers to reduced involvement in new experiences, a hallmark of apathy. Finally, Social apathy describes the lack of interest in social interactions, underscoring the social withdrawal often observed affected individuals.[39,40]

Our results compare favorably with recent literature. For instance, the best-fitting model in Furneri et al. (Model 1) had good fit indices (SRMR = 0.05; RMSEA = 0.064; CFI = 0.93; TLI = 0.92) but still performed less well than the current EGA-derived model.[15] This comparison highlights the added value of network-based approaches in psychometric modelling.

The EGA model showed promising indications of generalizability across clinical subgroups, including individuals with MCI and AD. While the SRMR was slightly elevated in these subgroups, most other fit indices fell within acceptable ranges, suggesting a reasonably good fit. However, these findings should be interpreted cautiously, given the sample size and the exploratory nature of the analysis. Convergent and discriminant validity were supported by significant correlations with cognitive and affective measures: higher apathy scores were associated with worse cognitive performance and higher depressive symptoms. Moreover, AD patients exhibited significantly more severe apathy than both controls and MCI patients, particularly on the interest and motivation, and novelty subscales. This supports the hypothesis that apathy severity increases along the continuum of cognitive decline and may serve as a transdiagnostic marker of neurodegeneration.

Notably, while all four EGA-derived dimensions distinguished patients from healthy controls, only two—Interest and Motivation, and Novelty—showed significant differences between AD and MCI groups. This suggests that these specific aspects of apathy may be particularly sensitive to the disease progression and may help delineate distinct stages of neurodegeneration.[41,42] For example, individuals scoring high on the Novelty factor may benefit from behavioral activation programs that emphasize exposure to new experiences and stimulation of interest, while those with prominent Social Apathy may require interventions focused on enhancing interpersonal connection and social participation. This dimensional profiling may aid in differentiating primary apathy, characterized by intrinsic motivational deficits, from secondary apathy linked to depression or cognitive impairment. This distinction is especially relevant in neurodegenerative conditions, where overlapping symptoms can obscure clinical judgment.[43] By addressing domain-specific manifestations, clinicians may improve diagnostic clarity and prognosis, ultimately enhancing quality of life, and alleviating caregiver burden.

These results also align with the network analysis by Sarti et al., which identified abulia and lack of initiative as central symptoms in AD, closely associated with diminished novelty seeking—a dimension consistently impaired in AD.[22]

Despite the strengths of this study, several limitations should be acknowledged. First, the social apathy and novelty dimensions consisted of only two items each. While their internal consistency was acceptable—and even the original AES-C model by Marin et al. includes a two-item domain—this brevity may constrain content coverage and limit dimensional stability. Future studies should enrich these domains by incorporating additional items specifically designed to capture their nuanced manifestations. Second, the sample sizes of the clinical subgroups were unequal, with the AD group being smaller than the MCI group. This imbalance may have reduced the statistical

power of between-group comparisons. Third, although the model demonstrated an overall good fit across subgroups, slightly elevated SRMR values suggest minor misfit, warranting further refinement and cross-validation. Finally, the present sample was drawn from a single country and ethnic background was not reported; therefore, the model's generalizability to more diverse populations remains untested. Validation across different cultural and demographic contexts is essential to confirm the robustness and applicability of the model.

In conclusion, applying EGA to the AES-C enabled a deeper exploration of apathy, expanding it beyond a loss of motivation in behavioral, cognitive, and emotional dimensions. The analysis revealed that apathy affects multiple domains, including autonomy, social relationships, and intrinsic motivation, providing a clearer and more actionable operationalization of the construct. The validation of the EGA model was further supported by strong Cronbach's Alpha values, confirmatory factor analysis, and analysis of convergent and discriminant validity, all of which demonstrated superior statistical fit. Future research should aim to expand the number of items for each factor identified by the EGA to enhance the scale's reliability and validity, ensuring a more balanced representation of all components.

CONSENT STATEMENT

All human participants signed informed consent to participation in the study described.

AUTHOR CONTRIBUTIONS

Conceptualization: CSG, GAP, SV, PS, JMCB, FC; Data curation: PS, FMB, VT, MS; Formal analysis: PS, JMCB; Visualization: PS, CSG; Writing—original draft: CSG, GAP, SV, Writing—review and editing: CSG, PS, JMCB, SDN, FC, MS, GF, CP, SC; Supervision: FC, SC, SDN, CP, GF, MS.

DATA STATEMENT

The data has not been previously presented orally or by poster at scientific meetings.

DISCLOSURES

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