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Literature Review

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Rethinking apathy assessment in neurological disorders: conceptual and clinical challenges

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ABSTRACT

Objective: Apathy is a prevalent and clinically consequential neuropsychiatric syndrome across neurological disorders, yet its assessment remains conceptually and methodologically challenging. This narrative review synthesizes current knowledge on the measurement of apathy, with particular emphasis on apathy-specific instruments validated in neurological populations and on the theoretical and practical factors that constrain their clinical utility.

Method: Literature was identified through PubMed searches, citation tracking, and review of influential consensus statements and neurocognitive models. Instruments were critically examined with regard to their conceptual foundations, psychometric properties, and ecological validity in clinical neuropsychological contexts.

Results: Despite broad agreement that apathy is a multidimensional construct, many widely used instruments retain predominantly unidimensional structures or show limited alignment with contemporary models, contributing to suboptimal discriminant validity—particularly in relation to depression—and inconsistent agreement across self-, informant-, and clinician-based assessments. Additional sources of bias include contextual and sensorimotor constraints, such as institutional environments and motor disability, which may inflate behavioral ratings and obscure primary motivational deficits.

Conclusions: Recent advances, including dimensional and context-sensitive approaches, represent promising developments; however, improving diagnostic precision and clinical decision-making requires integrating standardized rating scales with complementary paradigms targeting core motivational processes, such as effort-based decision-making, reward anticipation, and emotional responsiveness. Greater conceptual consensus, clearer construct boundaries, improved cross-cultural adaptation, and more consistent reporting practices are essential to enhance comparability across studies and to support clinical neuropsychologists involved in differential diagnosis, longitudinal monitoring, and treatment planning in neurological populations.

Keywords: apathy, motivation, neuropsychological assessment, clinical neuropsychology, neurological disorders

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Introduction

Apathy is a prevalent neurobehavioral syndrome observed across a wide range of neurological and psychiatric conditions. It is characterized by a clinically significant reduction in goal-directed activity—behavioral, cognitive, emotional, or social—relative to an individual's previous level of functioning and cannot be explained by altered consciousness, substance effects, or major contextual changes (Robert et al., 2018, Table 1). Although not recognized as a distinct diagnosis in the *Diagnostic and Statistical Manual of Mental Disorders, Fifth Edition* (*DSM-5*), apathy is frequently encountered in clinical practice and often subsumed under other neuropsychiatric labels such as depression or behavioral symptoms of dementia, contributing to under-recognition and diagnostic ambiguity. Empirical evidence suggests that this diagnostic ambiguity contributes to under-recognition in routine clinical practice. For example, in a cohort of patients with Parkinson's disease, more than half of those presenting with primary apathy received no formal psychiatric diagnosis (Ratajska et al., 2025).

Epidemiological evidence indicates that apathy affects a substantial proportion of individuals with stroke, traumatic brain injury, Alzheimer's disease, Parkinson's disease, frontotemporal dementia, amyotrophic lateral sclerosis, and Huntington's disease (Caeiro et al., 2013; den Brok et al., 2015; Kutlubaev et al., 2023; Matmati et al., 2022; Staekenborg et al., 2010; Starkstein & Pahissa, 2014; Zhao et al., 2016). It is also observed in schizophrenia and major depression (Yazbek et al., 2014; Yuen et al., 2015), in community-dwelling older adults (Brodaty et al., 2010), and in association with pharmacological treatments such as selective serotonin reuptake inhibitors (SSRIs; Masdrakis et al., 2023). Beyond its high prevalence, apathy is consistently associated with adverse outcomes, including accelerated cognitive decline, functional deterioration, poorer rehabilitation response, caregiver burden, and increased mortality (Arnould et al., 2015; Clarke et al., 2010; Dujardin et al., 2007; Hama et al., 2007; Starkstein et al., 2006; van Reekum et al., 2005). In individuals with mild cognitive impairment, apathy predicts conversion to dementia independently of depressive symptoms (Delrieu et al., 2015; Ismail et al., 2016; Vicini Chilovi et al., 2009). These findings underscore its prognostic value and its relevance for treatment planning and stratification in neuropsychological care.

Despite its clinical importance, the assessment of apathy remains conceptually and methodologically challenging. Early clinical formulations described apathy as a syndrome characterized by reductions in cognitive engagement, emotional responsiveness, and behavioral initiative relative to an individual's previous level of functioning (Marin, 1991). Marin's framework emphasized the multidimensional nature of apathy and established it as a clinically meaningful syndrome distinct from depression or cognitive decline.

Subsequent neuroanatomical accounts further refined this conceptualization. Levy and Dubois (2006) proposed that apathy reflects a reduction in goal-directed behavior that may arise from disruptions in partially dissociable fronto-striatal circuits. Their model distinguished three mechanisms—emotional–affective, cognitive, and auto-activation deficits—each linked to dysfunction in specific prefrontal–basal ganglia pathways. Importantly, not all of these mechanisms reflect primary motivational disturbances; some involve impairments in the cognitive control or initiation processes required to translate intention into action.

More recent neurocognitive accounts frame apathy within effort-based decision-making and reward-processing frameworks, highlighting impairments in effort allocation, outcome valuation, and action initiation (Husain & Roiser, 2018; Le Heron et al., 2018). Contemporary perspectives have further proposed computational and active inference models, suggesting that apathy may reflect diminished precision in action–outcome expectations or reduced confidence in the efficacy of goal-directed behavior (Williams & Rowe, 2025).

The present narrative review was conducted following the principles of the Scale for the Assessment of Narrative Review Articles. Literature was identified through PubMed searches and citation tracking, prioritizing apathy-specific instruments in neurological populations, conceptual frameworks, diagnostic proposals, and relevant systematic reviews. Our aim is to critically examine the principal instruments developed for the assessment of apathy, with particular attention to their theoretical foundations, psychometric robustness, and clinical applicability. We further analyze how neurobiological evidence, motivational models, and contextual influences shape current measurement practices and discuss the need for more integrative approaches that extend beyond observable behavior to include core motivational and neurocognitive mechanisms (Husain & Roiser, 2018).

Critical clinical determinants in apathy assessment

Apathy results from a complex interaction between biological, cognitive, emotional, and environmental factors. While neurological dysfunction often accounts for the onset of the condition, its severity and progression are modulated by individual and contextual determinants. These factors not only influence symptom severity but also introduce challenges for accurate assessment, affecting both measurement validity and clinical interpretation. The following sections outline key considerations in evaluating apathy, particularly in neurological populations.

Neurobiological and clinical correlates of apathy

Understanding apathy requires considering neurological and medical conditions that can shape or mimic motivational deficits. Although apathy is defined as a multidimensional syndrome involving reduced initiative, interest, and emotional engagement, its expression is frequently modulated by neurobiological factors and broader clinical context. Therefore, clinicians must evaluate these correlates to differentiate primary motivational syndromes from secondary or confounding conditions.

Neuroanatomical substrates

Converging evidence from lesion studies, structural and functional neuroimaging, and transdiagnostic analyses indicates that apathy is closely linked to disruptions within fronto-striatal and fronto-limbic circuits. Lesions involving the anterior cingulate cortex (ACC), orbitofrontal cortex (OFC), medial prefrontal cortex, and ventral striatum have been

Table 1 Apathy diagnostic criteria (adapted from Robert et al., 2018)

CRITERION A: A quantitative reduction of goal-directed activity either in behavioral, cognitive, emotional or social dimensions in comparison to the patient's previous level of functioning in these areas. These changes may be reported by the patient himself/herself or by observation of others.

CRITERION B: The presence of at least 2 of the 3 following dimensions for a period of at least four weeks and present most of the time.

B1. BEHAVIOUR AND COGNITION

Loss of, or diminished, goal-directed behaviour or cognitive activity as evidenced by at least one of the following:
General level of activity: the patient has a reduced level of activity either at home or work, makes less effort to initiate or accomplish tasks spontaneously, or needs to be prompted to perform them.
Persistence of activity: He/she is less persistent in maintaining an activity or conversation, finding solutions to problems or thinking of alternative ways to accomplish them if they become difficult.
Making choices: He/she has less interest or takes longer to make choices when different alternatives exist (e.g., selecting TV programs, preparing meals, choosing from a menu, etc.)
Interest in external issue: He/she has less interest in or reacts less to news, either good or bad, or has less interest in doing new things.
Personal wellbeing: He/she is less interested in his/her own health and wellbeing or personal image (general appearance, grooming, clothes, etc.)

B2. EMOTION

Loss of, or diminished, emotion as evidenced by at least one of the following:
Spontaneous emotions: the patient shows less spontaneous (self-generated) emotions regarding their own affairs, or appears less interested in events that should matter to him/her or to people that he/she knows well.
Emotional reactions to environment: He/she expresses less emotional reaction in response to positive or negative events in his/her environment that affect him/her or people he/she knows well (e.g., when things go well or bad, responding to jokes, or events on a TV program or a movie, or when disturbed or prompted to do things he/she would prefer not to do).
Impact on others: He/she is less concerned about the impact of his/her actions or feelings on the people around him/her
Empathy: He/she shows less empathy to the emotions or feelings of others (e.g., becoming happy or sad when someone is happy or sad, or being moved when others need help).
Verbal or physical expressions: He/she shows less verbal or physical reactions that reveal his/her emotional states.

B3. SOCIAL INTERACTION

Loss of, or diminished engagement in social interaction as evidenced by at least one of the following:
Spontaneous social initiative: the patient takes less initiative in spontaneously proposing social or leisure activities to family or others.
Environmentally stimulated social interaction: He/she participates less, or is less comfortable or more indifferent to social or leisure activities suggested by people around him/her.
Relationship with family members: He/she shows less interest in family members (e.g., to know what is happening to them, to meet them or make arrangements to contact them)
Verbal interaction: He/she is less likely to initiate a conversation, or he/she withdraws soon from it
Homebound: He /She prefers to stay at home more frequently or longer than usual and shows less interest in getting out to meet people.

CRITERION D: These symptoms (A–B) cause clinically significant impairment in personal, social, occupational, or other important areas of functioning.

CRITERION E: The symptoms (A–B) are not exclusively explained or due to physical disabilities (e.g., blindness and loss of hearing), to motor disabilities, to a diminished level of consciousness, to the direct physiological effects of a substance (e.g., drug of abuse, medication), or to major changes in the patient's environment

consistently associated with diminished initiative, impaired reward valuation, and reduced goal-directed behavior (Bonelli & Cummings, 2007; Levy & Dubois, 2006).

Meta-analytic and comparative reviews suggest that apathy across neurodegenerative, vascular, and psychiatric conditions shares partially overlapping neural substrates, particularly involving medial frontal and subcortical networks (Kos et al., 2016; Starkstein & Brockman, 2018). However, the syndrome does not appear neuroanatomically uniform. Emerging evidence supports partial dissociations between apathy dimensions. Reduced initiation and executive apathy have been associated more strongly with dorsal anterior cingulate and dorsolateral prefrontal dysfunction, whereas affective blunting has been linked to ventromedial prefrontal and orbitofrontal regions implicated in emotional valuation (Levy & Dubois, 2006; Stanton et al., 2013). Such findings

support the view that distinct motivational components may rely on partially separable, though interacting, neural circuits.

Recent multimodal and network-based approaches further emphasize that apathy reflects distributed circuit dysfunction rather than focal lesions alone. Disruptions in systems supporting effort allocation, value computation, and action–outcome integration appear to represent a transdiagnostic core mechanism (Gazes et al., 2025; Husain & Roiser, 2018). At the same time, disease-specific patterns modulate clinical presentation. For example, mesocorticolimbic dopaminergic dysfunction is central in Parkinson's disease (Pagonabarraga et al., 2015), whereas medial frontal and anterior temporal degeneration contribute prominently in frontotemporal dementia (Massimo et al., 2018).

From a clinical neuropsychological perspective, recognizing these neural distinctions is critical. Different neuroanatomical profiles may underlie superficially similar behavioral presentations, influencing prognosis and responsiveness to dopaminergic, cognitive, or behavioral interventions. Incorporating neurobiological knowledge into assessment therefore enhances interpretative precision and supports individualized clinical decision-making.

Pharmacotherapy and neurochemical influences

Medications affecting dopaminergic, serotonergic, or cholinergic systems may induce or exacerbate apathetic symptoms. Dopamine agonists or antagonists, as well as SSRIs, have been associated with reduced motivation, diminished emotional reactivity, and "emotional blunting" syndromes that can phenotypically resemble primary apathy (Costello et al., 2024; Le Heron et al., 2019; Masdrakis et al., 2023).

Given the central role of dopaminergic signaling in effort allocation and reward anticipation (Husain & Roiser, 2018; Le Heron et al., 2018), pharmacological modulation of mesocorticolimbic pathways may alter goal-directed behavior independently of cognitive status. In neurological populations, this issue may be particularly relevant given the high prevalence of psychiatric comorbidity and consequent psychotropic medication use. For example, studies in Parkinson's disease have reported higher apathy scores among patients treated with SSRIs, even after controlling for depressive symptoms and demographic factors (Schade et al., 2025; Zahodne et al., 2012).

Distinguishing primary apathy from medication-induced motivational changes is therefore critical during clinical evaluation, as the latter reflects neurochemical modulation rather than intrinsic disruption of motivational circuitry.

Motor and sensorimotor constraints in clinical expression

In neurological populations, reduced motor or sensory capacity may constrain observable goal-directed behavior and complicate differentiation between primary motivational deficits and physical incapacity. These difficulties are common in disorders such as Parkinson's disease, Huntington's disease, or progressive supranuclear palsy (Flavell & Nestor, 2022; Pagonabarraga et al., 2015; Paulsen, 2001). Consequently, assessment tools must be sensitive to these limitations. For instance, the Dimensional Apathy Scale (DAS) (Radakovic & Abrahams, 2014, 2018) was designed to evaluate apathy in patients with severe motor deficits, as seen in amyotrophic lateral sclerosis (ALS). Unlike traditional tools such as the Apathy Evaluation Scale (AES) by Marin (1991), the DAS avoids overreliance on activity frequency or productivity, thus reducing the risk of motor-related assessment bias.

Contextual and psychosocial modulators of apathy

While neurological and clinical factors provide a foundational understanding of apathy's underlying mechanisms, they do not operate in isolation. Apathy is often shaped, modulated, or even misinterpreted in light of the patient's psychosocial history and immediate environment. Contextual variables such as premorbid personality, life events, social support, and environmental stimulation can profoundly influence the manifestation of motivational deficits. Therefore, an ecologically valid assessment of apathy must incorporate these broader psychosocial and environmental dimensions to distinguish between primary apathy and reactive or context-dependent disengagement.

Psychosocial history and premorbid motivation

Understanding baseline motivation and coping strategies is essential, as variability arises from life experiences, socioeconomic factors, cultural background, and age (Arnould et al., 2013; Marin & Wilkosz, 2005). For example, older age has been associated with higher prevalence of apathetic symptoms even in otherwise healthy populations, potentially reflecting changes in dopaminergic motivation systems, reduced environmental stimulation, or shifts in social roles following retirement or functional decline (Brodaty et al., 2010; Lanctôt et al., 2017). Without this context, clinicians may overestimate the severity of apathy or misattribute longstanding behavioral patterns to neuropsychiatric causes.

Reactive emotional phenomena

Life events such as bereavement or trauma can produce emotional withdrawal resembling apathy, yet often reflect adaptive coping rather than neurological dysfunction (Marin & Wilkosz, 2005). Distinguishing transient emotional responses from persistent apathy is crucial, particularly in populations with comorbid depression or recent psychosocial stressors.

Physical and social environment

Environments that are under-stimulating or lack frequent social interaction—such as institutional settings—can diminish opportunities for engagement, thereby reinforcing passive behavior (Ballard et al., 2001; Jao et al., 2019; Massimo et al., 2018). Moreover, limited interpersonal contact or monotonous routines can suppress initiative and interest. Current diagnostic criteria explicitly advise ruling out contextual explanations (Robert et al., 2018). Importantly, structured and personalized activity programs can mitigate symptoms in institutionalized settings (Choi & Medalia, 2005; Kolanowski et al., 2011).

Family and caregiver dynamics

Family and caregiver behaviors may also play a substantial role in the emergence or perpetuation of apathetic behaviors. Overprotection, excessive structuring of daily routines,

or lowered expectations may inadvertently limit autonomy and diminish initiative (Massimo et al., 2018). Additionally, caregivers' attributions about the patient's abilities—especially when pessimistic or misinformed—can shape interaction patterns that reinforce disengagement. Exploring caregiver beliefs and interaction patterns offers valuable insight for assessment and intervention.

Motivational processes and mechanisms

Apathy is not simply the absence of behavior but reflects disruptions in the processes that generate, sustain, or direct goal-oriented action. Clinical neuropsychological assessment therefore requires understanding motivation as a dynamic, multi-level construct shaped by internal capacities, personal beliefs, and environmental contingencies.

Motivation encompasses intrinsic and extrinsic components (Burke et al., 2024), and disruption of either may manifest as reduced initiative or emotional engagement. Diminished reward sensitivity, altered effort–cost evaluation, or impaired outcome valuation—frequently observed in neurodegenerative conditions—can compromise both anticipation and pursuit of rewarding activities (Le Heron et al., 2018). These processes rely on frontostriatal circuitry, particularly the anterior cingulate cortex and ventral striatum (Husain & Roiser, 2018; Le Heron et al., 2019).

Psychological factors such as self-efficacy and perceived control further modulate engagement. When action is experienced as futile or unlikely to succeed, disengagement may follow (Arnould et al., 2013; Bandura, 1997). Importantly, cognitive impairments affecting planning, working memory, or monitoring can disrupt the execution of intention even when desire remains intact. Exploring subjective goals, perceived barriers, and prior reward experiences therefore supports differentiation between primary motivational deficits and secondary cognitive or contextual influences. A critical clinical implication of these motivational models concerns the distinction between impaired willingness to act and reduced capacity to translate intentions into behavior.

Capacity constraints and volitional drive deficits

A central challenge in apathy assessment is distinguishing primary deficits in volitional drive from limitations in cognitive or motor capacity that constrain the expression of goal-directed behavior. Although both may present as reduced initiative, their mechanisms—and clinical implications—differ.

Volitional drive deficits involve impairments in reward sensitivity, effort–cost computation, or action–outcome expectation, leading to reduced willingness to initiate or sustain behavior despite preserved capacity (Le Heron et al., 2018; Le Heron et al., 2019). Contemporary accounts further suggest that reduced confidence in the impact of one's actions may weaken motivational engagement (Williams & Rowe, 2025). In contrast, capacity constraints arise when executive dysfunction, slowed processing, or physical limitations interfere with translating intention into action (Husain & Roiser, 2018). Patients may endorse interest in activities yet struggle with initiation, sequencing, or sustained performance.

Clinical differentiation often depends on contextual variability. Improved engagement under structured conditions or external cueing may indicate preserved motivational drive with impaired self-initiation. Conversely, persistent disengagement even when cognitive and motor demands are minimized may suggest a primary disturbance of volitional systems. Effort-based paradigms that normalize demands to individual capacity further illustrate the importance of separating willingness to exert effort from absolute performance limitations (Gazes et al., 2025).

Failure to distinguish these mechanisms risks diagnostic imprecision and suboptimal intervention. Capacity-related disengagement may respond to cognitive scaffolding or environmental structuring, whereas primary motivational disturbances may require targeted pharmacological or behavioral strategies. Incorporating this distinction into neuropsychological assessment enhances interpretative accuracy and supports individualized treatment planning.

Apathy and depression: overlap and differentiation in clinical evaluation

Apathy and depression frequently co-occur in neurological disorders, with reported overlap rates ranging from 14% to 38% in neurocognitive conditions (Lanctôt et al., 2023; Starkstein & Brockman, 2018). Despite this comorbidity, converging clinical, neuropsychological, and neurobiological evidence supports their partial dissociation. Failure to differentiate these syndromes may obscure prognosis, bias measurement, and lead to inappropriate treatment decisions.

Phenomenological differentiation

Apathy reflects diminished motivation or volition without pervasive dysphoria, whereas depression is primarily a mood disorder characterized by sadness, hopelessness, and affective distress (Marin, 1991; Lanctôt et al., 2023).

While both syndromes share reduced interest and withdrawal, their subjective experience diverges. Individuals with apathy often report indifference or a lack of drive without suffering, whereas those with depression describe emotional pain and demoralization (Lanctôt et al., 2023). Vegetative symptoms—such as appetite disturbance, sleep disruption, and pervasive dysphoria—are characteristic of depression but are not defining features of apathy.

A particular source of diagnostic confusion is anhedonia. In depression, anhedonia reflects reduced capacity to experience pleasure ("liking"), whereas apathy more centrally involves diminished motivation to initiate behavior ("wanting") (Husain & Roiser, 2018; Lanctôt et al., 2023). Although these constructs overlap, they rely on partially distinct psychological and neurobiological mechanisms.

Importantly, many commonly used depression scales (e.g., GDS, HAM-D) include apathy-related items such as "loss of interest," contributing to symptom conflation and potential overdiagnosis of depression in neurologically apathetic patients (Lanctôt et al., 2023; Starkstein & Brockman, 2018).

Neurocognitive differences

Neuropsychological evidence suggests partially distinct cognitive profiles. Apathy shows robust associations with frontal-executive dysfunction, including impairments in planning, mental flexibility, inhibitory control, and working memory (Lanctôt et al., 2023; Oliveri et al., 2026). Notably, apathy is

specifically linked to deficits in language initiative—such as reduced verbal fluency and anomia—supporting the hypothesis that motivational failure involves impaired initiation within frontal–subcortical circuits (Oliveri et al., 2026).

In contrast, depression is more frequently associated with attentional inefficiency, slowed processing speed, and short-term verbal memory deficits, including retrieval difficulties sometimes described as "pseudodementia" in older adults (Lanctôt et al., 2023; Oliveri et al., 2026).

Longitudinal studies further differentiate outcomes: apathy independently predicts accelerated cognitive decline and increased risk of conversion from mild cognitive impairment to dementia, whereas depression is more strongly associated with loss of daily autonomy and affective burden (Clarke et al., 2007; Delrieu et al., 2015; Oliveri et al., 2026). These distinct trajectories underscore the prognostic importance of accurate differentiation.

Neurobiological distinctions

Neuroimaging studies support partially dissociable neural correlates. Apathy is consistently linked to dysfunction in fronto-striatal–subcortical circuits, particularly involving the anterior cingulate cortex (ACC), dorsolateral prefrontal cortex, and basal ganglia (Kos et al., 2016; Oliveri et al., 2026; Starkstein & Brockman, 2018). Greater apathy severity has been associated with frontal white matter hyperintensities and cortical thinning in frontal and temporoparietal regions (Oliveri et al., 2026).

Depression, by contrast, more prominently involves limbic and serotonergic networks, including subgenual and orbitofrontal regions implicated in affect regulation (Lanctôt et al., 2023). Distinct patterns of white matter involvement have also been reported, with depressive symptoms showing greater association with right parietal white matter changes compared to the frontal predominance observed in apathy (Oliveri et al., 2026).

Although overlap exists—particularly in dopaminergic and serotonergic systems—the weight of evidence suggests that apathy reflects primary disruption of motivational circuitry, whereas depression involves affective dysregulation and mood disturbance. These partially dissociable neural profiles reinforce the importance of assessment tools capable of distinguishing motivational circuit dysfunction from affective symptomatology.

Treatment implications

Differentiating apathy from depression has direct therapeutic consequences. Selective serotonin reuptake inhibitors (SSRIs), which are effective for affective symptoms, show limited efficacy in improving apathy in neurocognitive disorders and may, in some cases, exacerbate motivational deficits. This so-called "SSRI-induced apathy syndrome" has been attributed to serotonergic modulation of dopaminergic reward pathways, potentially dampening incentive salience and behavioral activation (Lanctôt et al., 2023; Masdrakis et al., 2023).

In contrast, apathy associated with fronto-striatal dysfunction appears more closely linked to dopaminergic disruption. In Parkinson's disease and related disorders, motivational deficits have been associated with mesocorticolimbic dopamine depletion, suggesting that dopaminergic strategies may preferentially target reward sensitivity and effort allocation mechanisms (Oliveri et al., 2026; Pagonabarraga et al., 2015). However, increasing dopamine may enhance behavioral vigor or capacity for high-effort tasks without necessarily restoring baseline motivational drive for everyday activities (Le Heron et al., 2019), highlighting the importance of distinguishing capacity from volitional impairment.

From a computational perspective, recent models suggest that apathy may reflect reduced precision in action–outcome expectations rather than solely diminished reward magnitude (Williams & Rowe, 2025). This implies that interventions aimed at enhancing environmental salience, structured cueing, or behavioral activation may compensate for reduced motivational precision even when pharmacological response is limited.

Neuromodulatory approaches, including repetitive transcranial magnetic stimulation targeting dorsolateral prefrontal regions, have shown preliminary promise in modulating motivational circuitry, although evidence remains limited and methodologically heterogeneous (Lanctôt et al., 2023).

Overall, failure to differentiate apathy from depression may lead to inappropriate antidepressant prescription or misinterpretation of treatment response. A neuropsychological assessment that integrates motivational profiling, executive testing, contextual analysis, and medication review is therefore essential for guiding individualized intervention.

The psychometric assessment of apathy

Clinicians often rely on qualitative, observational evaluations of apathy, partly because apathetic patients are not always able to complete formal assessments (Sanz & Ruiz-Sánchez, 2021). However, psychometric tools allow exploration of the dimensional structure of apathy and comparison of its manifestations across diseases and lesion patterns (Ang et al., 2017; Marin et al., 1991; Radakovic & Abrahams, 2014). A variety of scales, inventories, and questionnaires are available to quantify apathy through clinician-, informant-, or self-report formats (Table 2).

Recent work has also examined how commonly used apathy scales align with the updated diagnostic criteria for apathy in neurocognitive disorders (DCA-NCD; Miller et al., 2021). Sankhe et al. (2025) evaluated the extent to which items from widely used instruments map onto the three diagnostic dimensions of apathy—diminished initiative, diminished interest, and diminished emotional expression—and found that the Neuropsychiatric Inventory–Clinician apathy domain (NPI-C; de Medeiros et al., 2010) showed stronger alignment with the diagnostic framework than the AES (Marin et al., 1991). These findings highlight the importance of considering how well existing instruments capture the multidimensional structure of apathy when selecting assessment tools for clinical or research purposes.

At the same time, many instruments that include motivational or initiative-related items were not originally designed to assess apathy as a distinct syndrome. For example, scales such as the Frontal Systems Behavior Scale (FrSBe; Grace et al., 1999) or the NPI (Cummings et al., 1994) include

Table 2 Main instruments for the assessment of apathy in neurological disorders

Instrument and authors	Type of measure	Dimensions assessed/ identified factors	Number of items and scoring	Validated populations	Internal consistency	Reliability	Validity	Optimal cut-off	Advantages, disadvantages, and recommendations
AES (Marin et al., 1991)	Self-report (AES-S) Informant version (AES-I) Structured clinical interview (AES-C)	Cognitive (lack of interest), Emotional (blunted affect), Behavioral (lack of activity) 3 factors: general apathy, novelty seeking, lack of concern/ need for structure	18 items 0–72 pints	CVA, TBI, PD, AD, MDD, schizophrenia	$\alpha = 0.86$–0.95 (depending on version/sample)	Test–retest (2 weeks): AES-S = 0.76, AES-I = 0.44 (AD only), AES-C = 0.99, AES-I = 0.94 Inter-rater reliability: 0.94	Convergent (NPI): AES-I = 0.49 ($p < .01$), AES-C = 0.27 ($p < .01$), AES-S = 0.22 ($p < .01$) Divergent (NPI-Depression): AES-I = 0.22 ($p < .05$), AES-C = 0.18 (NS), AES-S = 0.23 ($p < .05$)	34–37	Highly recommended in clinical practice and research due to its psychometric robustness and various validations. Widely used in longitudinal research. AES-I scale is less discriminatory of depression than the other two variants. Possible cultural biases may affect this discrimination.
AI (Robert et al., 2002)	Structured interview with patient and informant	Initiative, Interest, Emotional blunting	3 items (includes frequency and severity) 0–36 points	MCI, PD, AD	α (informant) =0.84	Inter-rater agreement (video-based rating by 26 evaluators): $\kappa = 0.99$	Convergent (NPI): affective flattening = 0.01 (NS), lack of initiative = 0.23 ($p < .01$), lack of interest = 0.63 ($p < .001$)		Recommended as a brief screening tool, but not for diagnosis or in-depth evaluation. Limited evidence from large samples.
AMI/AMI-CG (Ang et al., 2017; Klar et al., 2022)	Self-report (AMI) Informant report (AMI-CG	Behavioral Activation (BA), Social Motivation (SM), Emotional Sensitivity (ES) 3 factors: BA, SM, ES	18 items 0–72 points	Healthy population, neurological diseases	α total = 0.77 α BA = 0.79 α SM = 0.75 α ES = 0.75	Test–Retest r total = 0.83 r BA = 0.88 r SM = 0.84 r ES = 0.72	Convergent (AES): r = 0.61 ($p < .01$) Divergent: BA and SM correlated with depression (BDI: $\rho = 0.26$, $p < .01$) and fatigue (MFI: r = 0.19, $p < .01$); ES subscale showed better discriminant power		Includes a social dimension. Promising tool for research and identification of motivational profiles. Subscales of social motivation and behavioral activation correlate with depression and apathy, but not the emotional sensitivity scale.

(continued)

Table 2 Continued.

Instrument and authors	Type of measure	Dimensions assessed/ identified factors	Number of items and scoring	Validated populations	Internal consistency	Reliability	Validity	Optimal cut-off	Advantages, disadvantages, and recommendations
APADEM-NH (Agüera-Ortiz et al., 2015)	Professional caregiver interview	Cognitive, Emotional, Behavioral 3 factors: cognitive, emotional, behavioral	26 items 0–78 points	Institutionalized dementia	$\alpha = 0.83$–0.88	Test–retest (7–25 days): κ ranged from: 0.48–0.92 Interrater reliability: $\kappa = 0.84$–1.00	Convergent validity (AI): rs = 0.33, ($p = .004$) NPI-apathy: rs = 0.31, ($p = .008$) Discriminant validity: NPI-depression: rs = −0.003 (NS)		Excellent for institutionalized severe dementia. High discriminant validity. Limited use outside institutional settings.
AS (Starkstein et al., 1992)	Self-report Informant Structured interview (caregiver)	Cognitive, Emotional, Behavioral	14 items 0–42 points	CVA, PD, AD	$\alpha = 0.76$	Test–retest correlation coefficient (1 week): r = 0.90, ($p < .01$) Inte-rater reliability (two-raters, two different days): r = 0.81, ($p < .01$)	Convergent validity (Neurologist blind rating): rated six apathetic and six non-apathetic, with apathetic patients having significantly higher SAS score (apathetic SAS) mean ± SD = 14.8 ± 5.7; non-apathetic SAS mean = 5.5 ± 2.2; t(10) = 3.70, ($p < .001$)	14	Cutoff point of 14 is a gold standard for discriminating apathy (Clarke et al. 2011), with 66% sensitivity and 100% specificity. Some items, such as item 3 ("*Are you worried about your condition?*"), are ambiguous and negatively influence internal consistency.
DAIR (Strauss & Sperry, 2002)	Structured informant interview	Social interaction, Interests, Self-initiation	16 items 0–48 points	AD	$\alpha = 0.89$	Test–retest (same day, different clinician): $\kappa = 0.96$–0.99	Convergent validity: (Clinical ratings; Nurse, Physician and Neuropsychologist): r = 0.40 (NS), 0.31 ($p < .05$) and 0.46 ($p < .01$), respectively Discriminant (BRSD-depression): r = 0.08 (NS)		Excellent psychometrics for institutionalized AD. High apathy specificity, low depression overlap. Limited use beyond AD context.

(continued)

Table 2 Continued.

Instrument and authors	Type of measure	Dimensions assessed/ identified factors	Number of items and scoring	Validated populations	Internal consistency	Reliability	Validity	Optimal cut-off	Advantages, disadvantages, and recommendations
DAS/b-DAS (Radakovic & Abrahams, 2014; Radakovic et al., 2020)	Self-report Informant Short form (b-DAS)	Executive, Emotional-Affective, Cognitive Inertia 4 factors: executive, emotional, cognitive initiation, behavioral initiation	24 items 0–72 points b-DAS: 9 items + self-awareness item 0–27 points	PD, AD, ALS, MS, healthy population	$\alpha = 0.83$ (executive); $\alpha = 0.93$ (initiation) $\alpha = 0.64$ (emotional)	Test–Retest ICC (b-DAS) = 0.84	Convergent validity (AES): Self: r = 0.75, ($p < .001$) Carer: r = 0.75, ($p < .001$) Discriminant validity (GDS–15): Self: r = 0.52, ($p < .001$) Carer: r = 0.36, ($p < .001$)		Recommended for subtype profiling and dimensional research. Useful for severe motor impairment. Low consistency of self-rated emotional subscale.
E-RAS Shamsalinia et al. (2021)	Self-report	Behavioral Engagement, Emotional Responsiveness, Initiative	18 items, 4-point Likert scale	Epilepsy	$\alpha = 0.93$	Not reported	Convergent validity with AES and BDI-II	Not specified	Specifically designed for epilepsy; strong psychometric properties; fills a gap in population-specific assessment
GAS Yi et al. (2024)	Self-report	Cognition and Social Motivation, Emotional Response and Expression, Spontaneous Behavioral Activation	16 items, 4-point Likert scale	Older adults with PD and AD (Chinese population)	$\alpha = 0.862$	Test–retest ICC = 0.767	Moderate convergent validity with AES (r = 0.639); low correlation with GDS-15	15.5 for PD; 12.5 for AD	Culturally sensitive; multidimensional; suitable for neurodegenerative populations; good validity and reliability

(continued)

Table 2 Continued.

Instrument and authors	Type of measure	Dimensions assessed/ identified factors	Number of items and scoring	Validated populations	Internal consistency	Reliability	Validity	Optimal cut-off	Advantages, disadvantages, and recommendations
LARS (Sockeel et al., 2006)	Structured interview (patient/ informant)	9 subscales: productivity, interest, initiative, novelty seeking, emotional blunting, concern, social life, self-awareness. 4 factors: intellectual curiosity, emotion, action initiation, self-awareness	33 items −36 a +36 puntos	PD, ALS	$\alpha = 0.928$–0.940	Test–retest (maximum of 15 days) $r = 0.94$, ($p < .001$)	Concurrent validity (AES): Total score, $r = 0.87$. Intellectual curiosity, $r = 0.84$. Action initiation, $r = 0.65$. Emotion, $r = 0.44$. Self-awareness, $r = 0.15$. Divergent validity (NPI-depression) $r = 0.37$ ($p = .029$) ICC = 0.99, $r = 0.97$ ($p < .001$)	−9 −16 (PD)	Strong psychometric properties, especially in PD. Incorporates awareness and curiosity subscales. Long evaluation time (extensive scale) Dichotomous scoring may reduce sensitivity to changes. More studies needed on discriminant validity.
PEAR (Jao et al., 2016)	Direct clinical observation	Apathy and environmental stimulation (physical and social)	12 items (2 subscales) 6–24 points each.	Institutionalized dementia patients	Apathy subscale $\alpha = 0.85$	Interrater reliability (two independently trained raters, one week later): 63.5%–85.4% agreement, weighted $\kappa = 0.66$–0.86 Intra-rater reliability (48/96) 75.0%–89.6% agreement, weighted $\kappa = 0.74$ – 0.89	Convergent validity: (PDS): $r = 0.81$, ($p < .001$) (NPI-apathy): $\rho r = 0.71$, ($p < .001$) Discriminant validity (NPI-depression): $r = 0.46$, ($p < .001$)		Only instrument considering environmental stimulation impact. Based on video observation (ecological). Good inter-rater reliability, though lower on some items (facial expression, eye contact).

Note: AD: Alzheimer's disease. AES: Apathy Evaluation Scale. AI: Apathy Inventory. ALS: amyotrophic lateral sclerosis. AMI: Apathy Motivation Index. AMI-CG: Apathy Motivation Index–Caregiver. APADEM-NH: Apathy in Dementia Nursing Home Scale. AS: Apathy Scale. CVA: cerebrovascular accident. DAIR: Dementia Apathy Interview and Rating. DAS: Dimensional Apathy Scale. GAS: Geriatric Apathy Scale. E-RAS: Epilepsy-Related Apathy Scale. ICC: intraclass correlation coefficient. Internal consistency: Related to reliability of the tool, assesses the extent to which items on a test are measuring the same thing. High internal consistency = Cronbach's alpha (α) closest to 1. LARS: Lille Apathy Rating Scale. MCI: mild cognitive impairment. MDD: major depressive disorder. MS: multiple sclerosis. NPI: Neuropsychiatric Inventory. NS: non-significant. Optimal cut-off: A value in a test that maximizes both sensitivity and specificity. *Sensitivity*: Ability of a test to correctly identify an individual with the disease state (the true-positive rating). *Specificity*: Ability of a test to correctly identify an individual without the disease state (the true negative rate). PD: Parkinson's disease. PEAR: Person-Environment Apathy Rating. Reliability: Consistency of the measurement. *Interrater reliability*: Measures how scores may differ when two or more raters administer the same tool. *Test–retest reliability:* At least two administrations of the tool in order to assess if similar results are obtained over time. Validity: How well a test measures the outcome of interest. *Convergent validity*: Two different measures of the same concept yielding similar results and having the results correlate with well-validated measures. *Divergent/Discriminant validity:* Used to show that two constructs, which are known to be unrelated, are in fact dissociated from one another and show little to no correlation with one another. *Note:* Psychometric values (e.g., internal consistency, validity) are representative of available validation studies, primarily drawn from research in neurodegenerative conditions and across diverse clinical and linguistic contexts. Variability should be interpreted accordingly.

behaviors relevant to apathy but were developed for broader behavioral assessment. Similarly, instruments measuring negative symptoms in psychiatric disorders (e.g., PANSS, SANS; Andreasen, 1989; Kay et al., 1987) include apathy-related domains such as avolition yet do not provide a theory-based assessment of apathy as an independent construct. Their conceptual overlap with related dimensions (e.g., anhedonia, blunted affect) and their differing diagnostic aims limit their usefulness for capturing the specific phenomenology of apathy. Consistent with these concerns, COSMIN-based evidence suggests insufficient construct validity for the original NPI apathy subscale, supporting its use primarily as a screening tool rather than as an outcome measure (Burgon et al., 2021).

This section focuses exclusively on instruments explicitly developed to assess apathy and validated across various clinical populations. Essential information is summarized in Table 2, followed by a critical review of each instrument's conceptual foundation, psychometric robustness, and clinical relevance. Given the frequent overlap with depressive symptoms, discriminant validity and item content are central considerations when selecting apathy instruments in neurocognitive disorders (Lanctôt et al., 2023). Where available, we incorporate COSMIN-based evidence to appraise the methodological quality supporting each instrument. The instruments are discussed in the following subsections according to their conceptual origins and primary clinical contexts, beginning with scales derived from Marin's original conceptualization of apathy and then considering subsequent instruments developed for specific theoretical frameworks or clinical populations.

Classical Marin-derived scales

These scales derive directly from Marin's original framework and prioritize feasibility and broad syndrome-level severity, often at the expense of subtype differentiation and mechanistic specificity.

Apathy Evaluation Scale

The AES (Marin et al., 1991) was the first instrument developed to assess apathy as a distinct neuropsychiatric construct. Grounded in Marin's conceptualization of apathy as a disorder of goal-directed behavior, it includes items addressing cognitive interest, behavioral initiation, and emotional responsiveness. The AES is available in clinician-rated, self-report, and informant-rated formats and has been widely used in populations with Alzheimer's disease, stroke, and schizophrenia.

Despite its enduring clinical utility, the AES has been described as a predominantly unidimensional measure. Some items—particularly in the informant-rated version—overlap with depressive symptoms such as reduced initiative or emotional flatness, raising concerns about discriminant validity. However, the clinician-rated version (AES-C) distinguishes apathy from depression and independently predicts cognitive and functional decline (Clarke et al., 2007). This is particularly relevant given the frequent overlap between apathy and depressive symptoms in neurocognitive disorders (Lanctôt et al., 2023). Nonetheless, more recent models of apathy highlight its multidimensional nature, encompassing executive, emotional-affective, and social components (Radakovic & Abrahams, 2014), which the AES may only partially address. As such, although the AES remains a widely adopted tool, its limitations in capturing domain-specific profiles of apathy have led to the development of newer, theoretically informed instruments.

Evidence quality (COSMIN). In a COSMIN-based systematic review of apathy measures in older adults and people with dementia, the AES showed *sufficient* content validity (largely based on reviewer ratings due to limited development reporting), *moderate-quality* evidence supporting structural validity and internal consistency, and *moderate-to-low quality* evidence for reliability (Burgon et al., 2021). Evidence for measurement error and responsiveness was limited or absent, underscoring the need for more rigorous longitudinal evaluation.

Apathy Scale

The Apathy Scale (AS) (Starkstein et al., 1992) is a 14-item adaptation of the AES developed to enhance feasibility in clinical practice. It focuses primarily on behavioral and cognitive features and has been widely used in Parkinson's disease and other neurodegenerative conditions. The AS demonstrates sensitivity to change in some Parkinson's disease cohorts (Pedersen et al., 2012).

Like the AES, it has a largely unidimensional structure and limited capacity to differentiate apathy subtypes or separate apathy from affective symptoms (Burgon et al., 2021; Radakovic & Abrahams, 2014). Its simplicity favors screening purposes but limits detailed clinical profiling.

Evidence quality (COSMIN). COSMIN evaluation found *inconsistent* evidence for structural validity across studies of the AS, despite some high-quality individual studies, and *very low* evidence for sufficient content validity and reliability (Burgon et al., 2021). Internal consistency and measurement error were frequently inconclusive, supporting use primarily as a practical screening measure rather than for fine-grained phenotyping.

Lille Apathy Rating Scale

The Lille Apathy Rating Scale (LARS) (Sockeel et al., 2006) is a structured interview-based instrument originally developed for Parkinson's disease. It assesses multiple domains, including daily activities, interests, emotional responsiveness, self-awareness, and hygiene concerns. A key strength of the LARS is its ability to distinguish between active disengagement and environmentally determined inactivity, an issue particularly relevant in patients with motor impairments. Furthermore, it includes items specifically addressing self-awareness of apathy, which is often overlooked in other instruments.

However, its rigid structure and complex scoring may hinder routine use in busy clinical settings (Burgon et al., 2021). Validation beyond Parkinson's disease remains limited, although subsequent applications suggest broader potential in specialized clinical settings.

Evidence quality (COSMIN). COSMIN review identified *sufficient* content validity (often based on reviewer ratings due to limited development reporting), *low-to-moderate* evidence for sufficient reliability, and *high-quality* evidence supporting hypothesis testing for construct validity. As a *formative* measure, internal consistency/structural validity criteria are

not directly applicable, and measurement error evidence was absent in eligible studies (Burgon et al., 2021).

Dimensional scales

These instruments aim to capture apathy as a multidimensional construct, improving profiling of motivational components relevant to neuropsychological interpretation and treatment planning.

Dimensional Apathy Scale

The DAS (Radakovic & Abrahams, 2014) reflects a shift toward theoretically grounded, multidimensional assessment. It distinguishes executive, emotional-affective, and initiation apathy, enabling identification of distinct motivational profiles relevant for individualized intervention.

The DAS is particularly sensitive in populations with motor impairment, such as ALS, where traditional measures may confound reduced activity with apathy. Its psychometric robustness has been supported in neurodegenerative disease and stroke (Myhre et al., 2022; Radakovic et al., 2016). Overall, the DAS stands out for its dimensional precision and its ability to minimize common assessment confounds.

Evidence quality (COSMIN). In the COSMIN review, the DAS was treated as a *formative* measure (limiting the applicability of internal consistency criteria) and showed *moderate-to-high quality* evidence for hypothesis testing of construct validity, while evidence for content validity and reliability was *very low* and based on a small study base (Burgon et al., 2021). Measurement error and responsiveness data were not established in the eligible literature.

Apathy Motivation Index

The Apathy Motivation Index (AMI) (Ang et al., 2017) was developed to assess apathy as a multidimensional construct grounded in contemporary motivational theories. It includes three domains—emotional, behavioral, and social apathy—and was originally proposed as a self-report measure, showing good internal consistency and construct validity in healthy, aging, and subclinical populations.

A limitation of the original version was its reliance on self-report, which restricts applicability in individuals with cognitive impairment or reduced insight. To address this issue, a caregiver-rated version (AMI-CG) was subsequently developed and validated (Klar et al., 2022), preserving the original factorial structure and demonstrating good convergent validity with interview-based measures such as the Lille Apathy Rating Scale. Despite these strengths, overlap between subscales—particularly within the social domain—has been reported. Overall, the AMI offers a theoretically informed alternative to classical apathy scales and is particularly suited to the assessment of early or subsyndromal apathy.

Evidence quality (COSMIN). In the COSMIN review, traditional internal consistency criteria are not directly applicable, and evidence for construct validity (hypothesis testing) was rated *insufficient* with low/very low evidence quality in the included older-adult/dementia literature (Burgon et al., 2021). These findings suggest that broader cross-diagnostic validation and longitudinal responsiveness data are still needed.

Dementia-focused tools

Dementia-oriented measures emphasize informant report and observable behavior, often incorporating premorbid comparison to mitigate confounding by cognitive impairment.

Apathy Inventory

The Apathy Inventory (AI) (Robert et al., 2002) was developed as a brief informant-based tool to assess apathy in dementia, particularly Alzheimer's disease. It evaluates three domains—emotional blunting, loss of initiative, and loss of interest—derived from the Neuropsychiatric Inventory. Its brevity facilitates routine clinical use; however, its limited item depth can constrain dimensional profiling in broader neuropsychological assessment.

Evidence quality (COSMIN). COSMIN synthesis reported *inconsistent* evidence for content validity and hypothesis-testing for construct validity, with *no conclusive* evidence for structural validity or internal consistency across eligible studies (Burgon et al., 2021). Reliability evidence was rated as sufficient, but overall quality was limited by methodological weaknesses, supporting cautious interpretation when used beyond brief screening.

Dementia Apathy Interview and Rating

The Dementia Apathy Interview and Rating (DAIR) (Strauss & Sperry, 2002) is a caregiver-rated instrument designed to minimize confounding with cognitive impairment. It emphasizes observable motivational changes and requires comparison with premorbid functioning, a key strength in dementia assessment.

Although internal consistency and inter-rater reliability are adequate in some cohorts, convergent validity with clinician ratings has been modest. Its limited consideration of environmental context may restrict sensitivity to situational influences. Nonetheless, the DAIR remains a useful option in long-term care and has informed subsequent dementia-focused apathy measures.

Evidence quality (COSMIN). COSMIN synthesis reported *inconsistent* evidence for content validity in both older adults and people with dementia, with *very low to moderate* evidence supporting structural validity and internal consistency, and *very low* evidence for test–retest reliability and measurement error (Burgon et al., 2021). The eligible evidence base was small (single included study), highlighting the need for independent replication and more robust methodology.

Apathy in Dementia–Nursing Home version

The Apathy in Dementia–Nursing Home version (APADEM-NH; Agüera-Ortiz et al., 2015) is a caregiver-rated scale designed for institutionalized older adults with dementia. It focuses on observable behaviors in routine, interpersonal, and affective contexts, making it suitable for individuals unable to complete self-report measures. The scale shows good internal consistency ($\alpha = 0.84$) and inter-rater reliability in Spanish-speaking samples.

A key strength is its ecological validity in long-term care settings. However, it does not assess internal motivational states or distinguish apathy subtypes, and contextual factors may influence ratings. Accordingly, the APADEM-NH is best

viewed as a complementary screening tool rather than a comprehensive apathy assessment.

Evidence is concentrated in a limited number of institutional cohorts and linguistic contexts, and independent replication and cross-cultural invariance testing remain limited. As a proxy-based observational rating, scores may be sensitive to environmental stimulation and caregiver expectations, which should be explicitly considered when interpreting change over time (Agüera-Ortiz et al., 2015).

Person–Environment Apathy Rating

The Person–Environment Apathy Rating (PEAR; Jao et al., 2016) is an observational tool designed for institutionalized older adults, emphasizing the interaction between individual capacity and environmental stimulation. This contextual focus distinguishes it from traditional apathy measures and supports identification of modifiable environmental barriers.

While reliable, its specificity to long-term care settings and reliance on observer judgment limit generalizability. Conceptually, the PEAR aligns with ecological and person–environment frameworks in gerontology and rehabilitation, which emphasize that behavioral engagement reflects the dynamic interaction between individual capacities and environmental affordances rather than motivation alone (Jao et al., 2016; Lawton & Nahemow, 1973). Nevertheless, the PEAR is valuable for guiding environmental interventions aimed at enhancing engagement. This aligns with broader concerns about contextual confounds in apathy assessment (Lane-Brown & Tate, 2009).

Psychometric support is currently anchored in institutional samples and observer-based designs. Further work is needed on discriminant validity relative to depression and on generalizability to community settings, where environmental structure and opportunity differ substantially (Jao et al., 2016).

Population-specific tools

Population-specific tools address context-dependent manifestations and improve face validity in targeted groups, although independent replication is typically limited.

Epilepsy-Related Apathy Scale

The Epilepsy-Related Apathy Scale (E-RAS; Shamsalinia et al., 2021) is the first instrument specifically developed for individuals with epilepsy. It comprises 18 self-report items across behavioral engagement, emotional responsiveness, and initiative. The scale demonstrates excellent internal consistency ($\alpha = 0.93$) and moderate to strong convergent validity with the AES and Beck Depression Inventory-II (BDI-II).

Limitations include the absence of normative cut-offs and reliance on self-report, which may constrain use in patients with cognitive impairment. Nevertheless, the E-RAS represents a meaningful advance in population-specific apathy assessment.

Evidence is currently concentrated in initial validation work, and independent replication (including test–retest reliability, measurement error, and responsiveness) is needed before routine use as an outcome measure across epilepsy subgroups (Shamsalinia et al., 2021).

Geriatric Apathy Scale

The Geriatric Apathy Scale (GAS; Yi et al., 2024) is a 16-item self-report instrument developed for older adults, incorporating three domains: cognitive–social motivation, emotional responsiveness, and behavioral activation. It demonstrates good internal consistency ($\alpha = 0.86$) and test–retest reliability.

A distinctive feature is its incorporation of sociocultural factors relevant to Chinese populations. However, generalizability beyond East Asian samples remains untested, and the self-report format may limit applicability in advanced cognitive decline.

At present, evidence derives primarily from development/validation in a single cultural context, and cross-cultural invariance, discriminant validity against depression, and responsiveness to intervention have not yet been established (Yi et al., 2024).

Across apathy-specific measures, COSMIN-based syntheses highlight recurrent methodological constraints, including incomplete reporting of item development (content validity), inconsistent evidence for structural validity across populations, and scarce data on measurement error and responsiveness to change (Burgon et al., 2021). Proxy-based instruments may be vulnerable to caregiver and contextual bias, whereas self-report tools can be affected by reduced insight in neurocognitive disorders.

Accordingly, scale selection should be guided not only by feasibility and population fit but also by the intended purpose (screening, dimensional profiling, or outcome monitoring) and the strength of the supporting evidence. The following section outlines the principal challenges inherent to psychometric apathy assessment and their implications for clinical interpretation.

Limitations in the current assessment of apathy

The psychometric evaluation of apathy remains constrained by methodological, conceptual, and contextual challenges that complicate interpretation in clinical neuropsychology, particularly when observable behavior is used as a proxy for underlying processes. These limitations extend beyond measurement format and reflect deeper tensions between theoretical models, observable behavior, and underlying motivational mechanisms.

Challenges in measuring complex psychological constructs are not unique to apathy. Similar issues have been widely discussed in the assessment of depression and other psychiatric syndromes, where symptom expression varies across contexts and measurement modalities (Fried, 2017). In response, contemporary clinical research increasingly emphasizes multimodal assessment strategies that combine symptom rating scales with functional outcomes and contextual information rather than relying on a single instrument (Rush et al., 2006). Ecologically oriented approaches, including ecological momentary assessment, have also been proposed to enhance external validity by capturing symptoms as they occur in everyday life (Colombo et al., 2020). Within this framework, apathy assessment may benefit from prioritizing

complementary sources of information—standardized scales, neuropsychological profiling, and contextual evaluation—rather than assuming that any single measure can fully capture the complexity of motivational disturbances. From a clinical standpoint, this implies that apathy measurement should be interpreted within a multimethod framework in which psychometric instruments provide structured symptom quantification, but diagnostic interpretation is guided by converging evidence from clinical interview, neuropsychological assessment, and contextual analysis.

Informant and insight bias

Self-report, informant-report, and clinician-rated instruments each introduce specific sources of bias. Reduced insight—particularly in neurocognitive disorders—limits the reliability of self-report measures (Agüera-Ortiz et al., 2010; Klar et al., 2022). Greater cognitive impairment is consistently associated with underestimation of apathy severity and reduced awareness of motivational change.

Caregivers, in contrast, tend to rate apathy as more severe than patients themselves, as reported in TBI (van Zomeren & van den Burg, 1985), Huntington's disease (Chatterjee et al., 2005), Parkinson's disease (McKinlay et al., 2008; Valentino et al., 2018), and Alzheimer's disease (Seltzer et al., 1997, 2001). While this may reflect patient underreporting, caregiver burden and dependency-related stress may also inflate ratings (Pfeifer et al., 2017). These explanations are likely complementary rather than mutually exclusive (Klar et al., 2022).

Importantly, caregiver-based scales may increase the risk of conflating apathy with depression, particularly when items assess withdrawal or reduced engagement without probing affective distress (Lanctôt et al., 2023; Lee et al., 2020). Given that motivational deficits are inherently linked to subjective internal states (Lane-Brown & Tate, 2009), reliance on single-informant approaches may produce systematic distortions.

For these reasons, multi-informant integration and contextual triangulation are recommended (Arnould et al., 2013).

Contextual and motor confounds

Many apathy scales operationalize motivational deficits through observable behavior (e.g., engagement in activities, task initiation). However, such items are inherently sensitive to contextual opportunity and motor capacity. Institutionalized individuals or those living in impoverished environments may show reduced engagement due to environmental constraint rather than primary motivational impairment (Jao et al., 2016).

Similarly, motor and sensorimotor impairments—common in Parkinson's disease, ALS, or stroke—can limit behavioral expression of intact motivational intent (Radakovic & Abrahams, 2014). This confound parallels the distinction between capacity constraints and volitional drive deficits discussed earlier (Le Heron et al., 2019; Williams & Rowe, 2025). Without careful clinical interpretation, reduced activity may be misclassified as motivational deficit when it reflects executive dysfunction, physical limitation, or environmental deprivation.

Although instruments such as the DAS or PEAR attempt to mitigate these confounds, context-sensitive bias remains a pervasive limitation.

Factorial instability and construct fragmentation

While contemporary models conceptualize apathy as multidimensional (Levy & Dubois, 2006; Robert et al., 2018), empirical factor-analytic findings have not consistently replicated stable domain structures across populations.

Importantly, while factor analytic approaches identify dimensions based on shared variance among items, they do not necessarily reflect etiopathogenic mechanisms or neurobiological dissociations. The statistical structure of a scale should therefore not be assumed to directly reflect the underlying organization of motivational systems.

Evidence from clinical profiling supports partial orthogonality of the emotional dimension. In patients with traumatic brain injury, Arnould et al. (2015) identified three apathy profiles: (a) reduced initiative and interest, (b) affective blunting, and (c) global severe apathy. Reduced initiative and interest were associated with greater psychosocial dysfunction, whereas affective blunting—particularly when combined with low initiative—was linked to increased caregiver burden. These findings suggest that diminished initiative and reduced interest may share common mechanisms, while affective blunting may be partially dissociable.

Neuroanatomical studies further support this possibility. Stanton and coworkers (2013) reported partially distinct correlates for emotional and behavioral apathy components, indicating that affective blunting may arise from disruptions different from those underlying goal-directed behavior. Subsequent work has documented dissociations between emotional apathy and cognitive or behavioral components (Ang et al., 2017; Petitet et al., 2021; Thompson et al., 2023), as well as between emotional apathy and fatigue (Daumas et al., 2022) or dysphoric symptoms (Ineichen & Baumann-Vogel, 2021).

The fragmentation of the emotional component has also been observed in schizophrenia, where affective blunting has been linked more closely to deficits in emotional expression and regulation than to reduced subjective emotional experience (Henry et al., 2007; Lee et al., 2014). Such findings suggest that emotional apathy may reflect disruptions in affective signaling or expression rather than a primary inability to experience pleasure.

Finally, recent diagnostic proposals have emphasized a social dimension of apathy (Robert et al., 2018), and specific instruments have operationalized this domain (Ang et al., 2017). However, debate persists as to whether social withdrawal constitutes an independent motivational dimension or a contextual manifestation of generalized goal-directed impairment (Dickson & Husain, 2022; Strauss & Cohen, 2017).

Discriminant validity problems

A central limitation in the psychometric assessment of apathy concerns discriminant validity, particularly in relation to depression. Many apathy items assess reduced interest, diminished initiative, or behavioral withdrawal—features that overlap with depressive symptom scales and may artificially inflate comorbidity estimates (Lanctôt et al., 2023).

This issue may be particularly pronounced in caregiver-rated instruments, where observable inactivity or social withdrawal can be interpreted as depressive disengagement rather than primary motivational failure (Lanctôt et al., 2023).

Despite this symptomatic overlap, converging evidence suggests that apathy and depression differ in phenomenology, cognitive correlates, and longitudinal outcomes. Apathy is primarily characterized by diminished motivation and reduced goal-directed behavior in the absence of pervasive emotional distress, whereas depression involves negative affect, hopelessness, guilt, rumination, and affective suffering (Marin, 1991; Lanctôt et al., 2023). Vegetative symptoms and dysphoria are central to depression but not defining features of apathy.

Failure to adequately differentiate these constructs has important prognostic implications. Apathy independently predicts accelerated cognitive decline and increased risk of conversion from mild cognitive impairment to dementia, whereas depression alone does not consistently demonstrate the same risk profile (Clarke et al., 2007; Delrieu et al., 2015). Neuropsychologically, apathy shows stronger associations with frontal–executive dysfunction and impaired initiation, while depression is more frequently linked to attentional inefficiency, slowed processing speed, and affective disturbance (Lanctôt et al., 2023; Oliveri et al., 2026).

From a motivational neuroscience perspective, further refinement of this distinction is possible. Depression-related anhedonia is typically conceptualized as reduced reward valuation or diminished hedonic experience ("liking"), whereas apathy may reflect impairments in effort allocation, incentive salience, or the precision of action–outcome expectations ("wanting") (Husain & Roiser, 2018; Williams & Rowe, 2025). These processes are computationally and neurobiologically distinguishable, yet traditional item-based scales rarely differentiate them explicitly, as both may manifest behaviorally as "loss of interest" or reduced engagement.

Consequently, psychometric scores alone may be insufficient to disentangle primary motivational inertia from affective dysphoria or executive limitation. Accurate differentiation requires integration of structured clinical interview, neuropsychological profiling, and contextual analysis, particularly in neurocognitive populations where apathy and depression frequently co-occur.

Evidence quality and sampling limitations

Beyond conceptual concerns, methodological rigor remains uneven across instruments. COSMIN-based syntheses reveal recurrent issues including incomplete reporting of item development, inconsistent structural validity across populations, limited replication in independent samples, and scarce evidence for measurement error or responsiveness (Burgon et al., 2021).

Many validation studies rely on modest sample sizes, single diagnostic groups, or cross-sectional designs, limiting generalizability. Cross-cultural invariance testing is infrequent, and longitudinal sensitivity to intervention effects remains insufficiently established.

These limitations are particularly relevant in neuropsychology, where apathy often serves as both a diagnostic marker and a treatment outcome. Without stronger methodological foundations, the risk of measurement bias and interpretive overreach persists.

Conclusion

This review has examined the principal instruments used in the assessment of apathy, highlighting their theoretical foundations, clinical applicability, and inherent limitations. Despite growing consensus that apathy is a multidimensional syndrome, important challenges remain regarding discriminant validity, factorial stability, contextual sensitivity, and the differentiation of primary motivational deficits from capacity constraints or affective syndromes.

A central implication of this analysis is that psychometric scales—while indispensable—primarily capture observable manifestations of reduced goal-directed behavior. They often provide limited access to the underlying mechanisms that generate or sustain motivational engagement. As a result, similar behavioral profiles may arise from distinct processes, including volitional drive disruption, executive dysfunction, emotional blunting, or environmental constraint.

To address these limitations, future efforts should prioritize more integrative approaches to assessment—combining structured psychometric tools with performance-based and experimental paradigms aimed at probing core motivational mechanisms. Measures of reward anticipation, reinforcement learning, emotional reactivity, and effort-based decision-making (Husain & Roiser, 2018; Le Heron et al., 2018) may offer complementary insights that extend beyond behavioral manifestations of disengagement and enhance mechanistic specificity.

At the same time, progress does not necessarily require the proliferation of new instruments. Rather, the field would benefit from greater consensus-building around existing tools—harmonizing conceptual definitions, clarifying domain boundaries, strengthening discriminant validity (particularly in relation to depression), and improving cross-cultural and cross-diagnostic applicability. Such convergence would enhance comparability across studies and increase the clinical interpretability of apathy measures.

Until stronger alignment between theory, measurement, and neurobiology is achieved, clinicians must approach apathy assessment as a multidimensional and context-sensitive process. Structured scales should be interpreted in conjunction with clinical interview, neuropsychological profiling, contextual analysis, and careful differentiation from affective and executive syndromes. Only through this multifaceted, theory-informed approach will it be possible to accurately delineate motivational disturbances and guide targeted, mechanism-sensitive interventions.

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