

Diagnosing Frontotemporal Dementia: Reviewing Apathy as a Distinguishing Clinical Marker

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ABSTRACT: Neurodegenerative diseases often share similar symptoms, resulting in frequent misdiagnosis, especially for lesser-known diseases. Frontotemporal dementia (FTD) is one of the most commonly misdiagnosed, with misdiagnosis rates of up to 70%. This review evaluates the extent to which apathy can serve as a key factor in distinguishing FTD, specifically behavioral variant FTD (bvFTD), from other FTD subtypes and Alzheimer's dementia. Recent research has begun to discover differences in apathy in FTD compared to other neurodegenerative diseases. An analysis of clinical and neuroimaging evidence suggests that apathy, particularly in bvFTD, has the potential to serve as a distinguishing marker for early diagnosis, given its notable differences across neurodegenerative diseases. When implemented, it can improve the accuracy of future diagnoses, allowing patients to receive targeted treatment and complete necessary preparations earlier.

KEYWORDS: Behavioral and Social Sciences, Neuroscience, Behavioral Variant Frontotemporal Dementia, Apathy in the Diagnosis of bvFTD.

■ Introduction

Frontotemporal dementia is a progressive neurodegenerative disease that targets the frontal and temporal lobes. It is also speculated to be a main cause of young-onset dementia, which typically affects individuals 45-60 years old. However, due to a gap in resources and knowledge, patients with lesser-known diseases such as FTD often receive less attention and proper, quality care than those with other diseases. A survey involving over 900 participants found that the annual cost of a patient with frontotemporal dementia (FTD) totals around \$119654, nearly twice the cost of Alzheimer's disease (AD), which has received far more attention from researchers.

FTD is categorized into two main groups: behavioral variant frontotemporal dementia (bvFTD) and primary progressive aphasia (PPA), further sorted into either semantic dementia (svPPA or SD) and progressive non-fluent aphasia (PNFA).^{1,2} Apathy is common in FTD patients and has also been shown to be especially strongly associated with bvFTD, making it a promising marker for improved diagnoses. Despite growing knowledge on apathy and frontotemporal dementia, FTD remains frequently misdiagnosed,³ since its symptoms are similar to other more well-known diseases, especially Alzheimer's disease (AD). Thus, novel uses of apathy in diagnostic or clinical applications are underexplored.

Since there is no known cure for FTD, current "treatments" mainly target symptom management. Even so, early diagnosis can reduce economic burden, improve patient and caregiver quality of life, and reduce stress on caregivers and patients. Furthermore, it allows patients to make financial, emotional, and legal plans while they retain cognitive function.^{4,5} Establishing apathy as a potential key marker for distinguishing bvFTD from other neurodegenerative diseases can lead to increased diagnostic accuracy, allowing earlier delivery of treatment and improved quality of life for both patients and caregivers.

This review critically examines the role of apathy in bvFTD, with a focus on its diagnostic potential. It explores the neuroanatomical correlates of apathy, analyzes differences in comparison to other dementias, and discusses current limitations and future directions, as well as possible applications aimed at improving diagnostic accuracy and quality of life for patients and caregivers.

Methods:

The literature in this review was found using scholarly databases such as ScienceDirect, Wiley Online Library, and PubMed with the following keywords: 'frontotemporal dementia FTD', 'apathy', 'diagnosis', 'economic burden', 'distinguish', 'classify', 'MRI', 'neuroimaging', 'alzheimer's', 'behavioral variant frontotemporal dementia bvFTD'. Articles were included if they were peer-reviewed and published between 2005 and 2025 to ensure recent and reputable information. Only records written in English were included due to feasibility constraints. The review methodology is summarized in Figure 1.

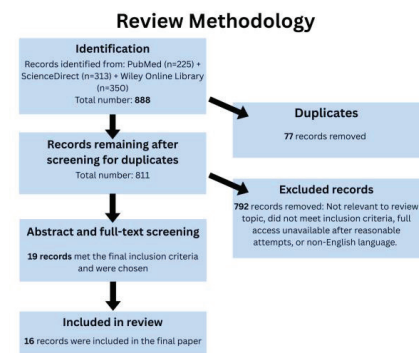


Figure 1: PRISMA diagram highlighting review methodology. The systematic screening process narrowed the literature from 888 initial records to the 16 eligible records analyzed in this review. Created using Canva (Canva, 110 Kippax St, Surry Hills NSW 2010, Australia).

■ Discussion

1. Utilizing apathy as a key marker in clinical diagnosis of bvFTD:

Apathy, now recognized as a multidimensional symptom, is slowly gaining more attention, especially as it is present in many illnesses and disorders. Noting the unique characteristics of a patient's apathy can help determine and accurately diagnose their condition. Commonly described as a reduction of the completion of self-initiated behaviors and an inability to judge or weigh the available options, people with apathy frequently have reduced goal-directed behavior, a lack of motivation/initiation, and less emotional expression.^{6,7}

1.1. Apathy as a Multidimensional Symptom:

Apathy is mainly categorized into three dimensions: cognitive, emotional-affective, and auto-activation/behavioral. Each dimension, or "subtype", is associated with unique symptoms. For instance, patients with cognitive apathy frequently have impaired goal-directed behavior and executive function, while those with emotional-affective apathy frequently have diminished emotional expression and processing. The auto-activation subtype is recognized by researchers and clinicians as one of the most severe forms, since patients are unable to initiate behavior/actions.⁷

As our understanding of apathy evolves and changes, our framework for diagnosing apathy has also been updated. To reflect our improved understanding and avoid confusion with other common symptoms in neurocognitive disorders (NCD), a consensus panel in 2021 revised and created 4 criteria for diagnosing apathy in NCD, including dementias such as FTD and Alzheimer's,⁸ illustrated in Figure 2.

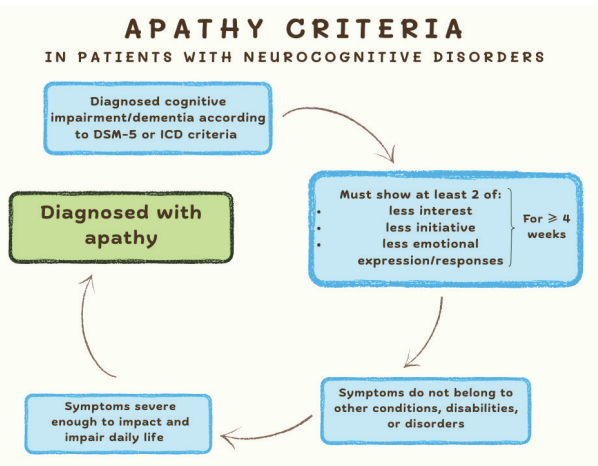


Figure 2: Diagnostic criteria for apathy in patients with neurocognitive disorders. These criteria emphasize persistent symptoms that are distinct and independent from other conditions. Adapted from Table 1 in Miller *et al.*⁸ using Canva (Canva, 110 Kippax St, Surry Hills NSW 2010, Australia).

This review considers studies that diagnose apathy through different scales such as the Dimensional Apathy Scale (DAS), the Apathy Evaluation Scale (AES), or the Neuropsychiatric Inventory (NPI). While the scales used may differ, the findings of the articles are generally in agreement with each other.

1.2. Distinguishing bvFTD from PNEA and svPPA:

Apathy can be a key factor that differentiates bvFTD from other FTD subtypes such as PPA. Studies have shown that, unlike the rates of disinhibition and stereotypical behavior, more patients (71.5%) with bvFTD had severe apathy compared to other subtypes of FTD, such as svPPA, in which 33% had severe apathy.⁹ This difference reveals the severity of apathy as a useful clinical distinguisher, but it is important to consider the challenge in determining severity objectively and consistently when using different scales. For instance, the AES's definition of severe can differ from another scale's, and this difference may affect the diagnosis of a patient.

Furthermore, the subtypes of apathy can also help distinguish between the different FTD subtypes. Studies show that a majority (75%) of patients with bvFTD were impaired in emotional apathy compared to 41.7% of PPA patients. Although the study had small bvFTD and PPA cohort sizes, which may limit the generalizability of its findings, the findings seem reasonable.¹⁰ Since bvFTD targets the areas controlling social and reward processing earlier on and more severely, it follows that they would have more emotional apathy compared to other groups. Understanding the subtype of apathy a patient has can therefore help determine which subtype of FTD they have.

1.3. Severity of Apathy as a Differential Factor of bvFTD from Alzheimer's Disease:

Aside from distinguishing between FTD subtypes, apathy can also distinguish between other similar neurodegenerative diseases, including AD. A study found that significantly more bvFTD patients (84%) were reported to have apathy compared to AD patients (60%), along with more severe apathy compared to AD patients.¹¹ The severity of one's apathy can help distinguish bvFTD from AD, in addition to other FTD subtypes mentioned earlier. Studies also found that the bvFTD group had severe emotional-affective apathy compared to the AD group, and they were also less aware of it.^{10,11} Despite using different apathy measurement scales, both studies had similar results, suggesting the reliability of using the severity of apathy in FTD's diagnosis.

The more severe apathy and higher emotional apathy seen in bvFTD can not only help distinguish it from other FTD subtypes like PPA, but it can also distinguish bvFTD from AD. By acknowledging the differences in characteristics of apathy, it could potentially achieve a more accurate clinical diagnosis for bvFTD.

2. Structural and Neurobiological Components of FTD and Apathy:

While useful, clinically distinguishing apathy tends to be difficult; most scales hinge on patient and or caregiver reports, but their perspectives may differ, and patients may lack insight into the presence and severity of their symptoms. There remains no objective way to determine one's apathetic condition, so apathy diagnosis can sometimes be unreliable. Therefore, a more concrete way to analyze and diagnose apathy would be

incredibly useful. Having examined the clinical component of apathy in FTD, we now consider its biological aspect.

2.1. Typical Structural Changes Associated with Apathy and FTD:

Apathy usually manifests after damage to the basal ganglia or the prefrontal cortex (PFC), and subtypes of apathy are also associated with a decrease in gray matter intensity in different regions. Lesions in the orbitofrontal PFC, areas that are involved in evaluating reward and emotions, are correlated with emotional-affective apathy, while the dorsolateral PFC (DLPFC) could be associated with cognitive inertia apathy. It is worth noting that, unlike the results of Levy and Dubois's lesion analysis,⁷ Kumfor and her colleagues found that cognitive apathy was associated with lower gray matter in a more medial region, and mentioned that patients with dorsomedial PFC lesions had somewhat preserved cognition levels. Although Kumfor's study has a relatively large bvFTD cohort size for neuroimaging analysis, the selection of younger patients for MRI could impact generalizability. Additionally, the utilization of two carer-completed apathy scales could introduce bias, as carers may misinterpret or inaccurately report symptoms, highlighting the importance of the patient's perspective.¹¹ Additionally, the utilization of two carer-completed apathy scales could introduce bias, as the carer may misinterpret or inaccurately report symptoms, highlighting the importance of incorporating the patient's perspective. More research in these areas may reveal further information about observing apathy through neuroimaging techniques, a crucial step towards developing more early diagnosis. Lastly, research suggests that lesions in specific areas of the basal ganglia could lead to reduced self-initiated actions, a core symptom of auto-activation apathy.⁷

As for gray matter loss, emotional apathy is associated with reduced gray matter intensity in ventral PFC regions and the left temporal poles, while cognitive apathy is associated with more dorsal PFC regions, left orbitofrontal and subcallosal regions. Kumfor and her colleagues found that auto-activation/behavioral apathy is associated with atrophy in the frontal pole and subcortical regions (such as the basal ganglia),¹¹ while Massimo and her colleagues found that it was associated with the anterior cingulate cortex. The latter study is well-designed, with clear diagnostic criteria and exclusion of patients with potential confounding factors. However, its small sample size and gender imbalance (5 females out of 18) may limit its generalizability.¹² Given the differences in these findings and possible limitations, it is reasonable to infer that apathy, especially auto-activation apathy, can be correlated with multiple regions, due to its complicated nature. These studies suggest that apathy is not purely clinical; it can be seen neurobiologically, which can possibly be a more objective view than clinical diagnoses.

To discover objective signs of FTD, we can focus on structural differences in FTD compared to healthy controls. Patients with FTD are commonly noted to have bilateral atrophy in the mediofrontal and orbitofrontal regions, anteromedial temporal areas, insula, and basal ganglia. Similarly, apathy in FTD

patients is associated with atrophy in frontotemporal areas such as the dorsolateral, ventromedial, and orbitofrontal PFC, basal ganglia, insula, and the anterior cingulate cortex (ACC), depending on severity.¹²⁻¹⁴

2.2. Atrophy and Apathy in bvFTD Compared to Other Conditions:

Many of the aforementioned regions are highly interconnected with each other, suggesting that key atrophy regions in FTD patients are connected to apathy. Moreover, compared to AD, bvFTD showed more atrophy in the ventromedial PFC (VMPFC), ACC, caudate, thalamus, and bilateral temporal poles.¹¹ Most of these regions are both targeted early on in FTD and are also commonly affected by apathy, explaining the increased severity of apathy in FTD patients.

Furthermore, since apathy severity is correlated with atrophy in frontotemporal areas, it holds potential as an indicator of the progression of frontotemporal degeneration, especially as longitudinal studies show an increase in apathy severity as the disease progresses.^{9,13}

Across multiple sources, apathy was associated with atrophy in key neuroanatomical regions that differentiate bvFTD from AD, as well as in a majority of primary regions targeted by FTD, including the DLPFC and the VMPFC. While more research and larger-scale experiments would be necessary in validating and confirming these preliminary findings, these results strengthen the argument that apathy has neurobiological aspects that may help diagnose bvFTD. Furthermore, these neuroimaging studies suggest that atrophy in frontotemporal regions corresponds with increased severity of apathy, supporting its potential as a clinical marker.

3. Establishing More Reliable Diagnoses:

Notably, current diagnoses have much room for improvement; one study shows that only 12% of FTD patients were properly diagnosed by the first doctor they visited, and in another, around half saw 3 or more doctors before their FTD diagnosis. Additionally, despite displaying common symptoms such as behavioral or personality changes early on, a study found that 58% of initial diagnoses were incorrect.^{15,16}

Improper or misdiagnosis has many negative consequences. Other than the direct effects, such as improper care and treatment for patients, it also places greater stress and anxiety on both the patient and loved ones and or caregivers.^{4,16} Apart from psychological effects, treatments and care are expensive; family or caretakers might end up spending money without any benefits, which is a predicament, especially since surveys have found that household income decreases after diagnosis.⁴ This decrease in household income can be possibly attributed to costs associated with diagnosis or treatment, caregiving expenses, or may reflect decisions to prioritize patient care over work obligations. Furthermore, a later diagnosis prevents families from making the necessary preparations, including emotional or legal, while the patient still retains cognitive function. Therefore, improving diagnostic accuracy in bvFTD is crucial and can be achieved through the discovery of reliable

key markers, such as apathy, that help distinguish bvFTD from other neurodegenerative diseases.

3.1. Limitations:

There are a few limitations to using apathy as a marker for FTD. For one, severity is relative, and different scales use different measurements of severity. While this is acknowledged to be an issue, specifying one's severity can still be clinically useful. As mentioned earlier, despite using different scales, studies still found bvFTD to have more severe apathy and higher emotional apathy in comparison to other neurodegenerative diseases. In addition, while apathy may become a core factor in diagnosing bvFTD, it should still be considered as a component among other factors, such as neurobiological or clinical/behavioral symptoms; therefore, the diagnostic outcome will not be heavily affected by this inconsistency. To resolve any inconsistency issues, clinical diagnoses for apathy could aim to agree to use as few scales as possible to avoid any complications, since different scales might complicate comparison and diagnosis. When determining which scale to use, it's important to consider its reliability, ability to consider multiple perspectives (including both the patient and family/caregivers), as well as acknowledging apathy as a multidimensional aspect. Another important consideration is that more research and studies are needed; while there has been an increase in attention to apathy in FTD, it is hard to conclusively establish apathy as a key early diagnosis factor based on a few articles. However, since the articles are generally in agreement, this represents a promising direction for future research.

3.2. Potential Applications and Future Directions:

Although apathy is included in the bvFTD criteria,² there are many improvements to be made, as seen in the statistics above. Additionally, as apathy appears in more than 90% of FTD cases, many would benefit if it could become a distinguishing factor. One novel way to apply apathy in diagnosis would be having a test, along with the current diagnosis tests, specifically designed to analyze one's apathy severity and subtype. After comparing and considering it with other symptoms and signs, the clinician could use apathy to determine a more conclusive result. For instance, if it's unclear whether a person has Alzheimer's or frontotemporal dementia, a psychiatrist or neurologist could note that the patient has extremely high emotional apathy and more severe apathy in general, distinguishing and solidifying their diagnosis of frontotemporal dementia. Furthermore, identifying atrophy in certain areas correlated with apathy may also hold diagnostic potential, serving as a more "evidence-based" component.

As frontotemporal degeneration progresses, apathy increases. Longitudinal evidence suggests that apathy can precede cognitive decline in FTD; a study found that apathy severity increases annually in presymptomatic carriers.¹³ By introducing the idea that apathy manifests before cognitive decline in FTD, this article establishes the possibility that apathy can be a reflection of early dysfunction in frontotemporal areas, not just a symptom of FTD. However, since the study targeted a genetically predisposed cohort, further research is needed to

confirm whether apathy can truly signal atrophy and or cognitive decline in all FTD cases, including sporadic (non-genetic) ones. Aside from conducting future research to confirm these findings, one idea is to attempt to utilize this result to consider regularly testing for apathy as people age in order to determine susceptibility and detect early signs of FTD. While not fully concrete, these results hold a lot of potential towards apathy becoming an early diagnostic marker for frontotemporal dementia.

■ Conclusion

Studies suggest apathy's characteristics, including severity and subtype, may aid in more accurate early diagnosis for FTD, though future validation is needed. First, clinically comparing characteristics of apathy, such as severity or subtype, can help establish a more accurate diagnosis. Studies have consistently found that bvFTD patients usually have higher emotional apathy, and generally have more severe apathy. Additionally, utilizing atrophy patterns related to different subtypes of apathy and different diseases would also be incredibly useful as another, more "objective" way to diagnose and distinguish FTD. After acknowledging challenges with current diagnoses and establishing the importance of proper diagnosis, this review proposed possible implementations and future directions, and acknowledged any limitations.

Further studies should focus on developing and validating more holistic and reliable clinical measures of apathy, and on testing whether apathy seen through atrophy in certain areas can precede common symptoms such as cognitive decline. In addition, repeating the experiments in this paper with a larger, more ethnically diverse population sample and including both community-dwelling older adults, along with clinical patients, will further establish the results' reliability as well as this review's claims and analysis, therefore encouraging generalizability. Furthermore, comparing and noting any differences in outcomes across the same experiments with slightly different materials, particularly different apathy tests, will help address this limitation. Lastly, studies integrating more objective components, like structural or functional MRI, with more clinical components, such as apathy tests or behavioral tasks, are a crucial step towards creating more reliable and accurate diagnoses.

As research progresses, it is worthwhile to note that, for neurodegenerative diseases such as bvFTD, some patients may actually prefer not to know about their condition and are against or uninterested in diagnoses. Since there is currently no cure or treatment that can slow or stop bvFTD's progression, some believe that it is better not to know about their condition if there is nothing they can do against it. However, creating accurate diagnostic tools will not force people to receive diagnostic information; instead, it serves as a reliable option for those who wish to know, allowing them to receive a more accurate result. Thus, the development of a more accurate diagnostic tool and or factor for bvFTD is crucial, and must be ethically governed by maintaining patients' autonomy along with respecting their right to give or withhold consent.

While apathy may become a core factor in diagnosing bvFTD, it remains just one piece of a greater puzzle: a mul-

tidimensional assessment framework that considers unique characteristics of apathy along with neurobiological patterns of atrophy, as well as existing diagnostic components such as clinical and behavioral symptoms. Viewing this disorder through a more multidimensional lens enables a more holistic view of the patient's condition, ultimately fostering more accurate diagnoses and improved quality of life.

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