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REVIEW

Apathy Through History: A Disorder at the Crossroads of Psychiatry and Neurology

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Abstract
Apathy is a multidimensional neuropsychiatric condition characterized by a reduction in goal-directed behavior, cognition, emotion, or social interaction, with significant clinical implications in both neurological and psychiatric disorders. Despite its prevalence, apathy has traditionally been viewed as a secondary symptom of other pathologies. This article offers a historical and conceptual review of apathy, tracing its evolution from early philosophical ideas of emotional detachment to its current status as a transdiagnostic construct. The review examines the gradual emergence of apathy as a distinct syndrome, emphasizing contributions from psychiatry and neurology. Key concepts such as abulia, anhedonia, akinetic mutism, athymhormia, and avolition are discussed to illustrate the shifting boundaries between these terms. Drawing from historical and modern perspectives, the article explores the semantic evolution of apathy-related terms and their convergence in current diagnostic frameworks. It also addresses ongoing clinical challenges in detecting and differentiating apathy from overlapping syndromes. Ultimately, this review advocates for recognizing apathy as an independent syndrome with distinct neurobiological foundations, emphasizing its importance in behavioral neuroscience and clinical neuropsychiatry.

Keywords Apathy · Anhedonia · History · Psychiatry · Neurology · Transdiagnostic

Introduction

Apathy is a relatively common behavioral and emotional disturbance in neurological and psychiatric disorders. It is currently defined as "*a quantitative reduction in goal-directed activity in behavioral, cognitive, emotional, or social dimensions compared to the individual's previous functioning in these areas, causing clinically significant impact on the patient, and not solely attributable to a reduced level of consciousness, the physiological effects of a substance, or major environmental changes*" (Robert et al., 2018).

A patient with apathy may exhibit diminished interest in hobbies or topics of conversation that previously motivated them. They often require external prompting to initiate activities, even for routine tasks, which are frequently abandoned prematurely if they demand sustained effort. Patients with apathy commonly present as placidly indifferent to their surroundings, to the needs and feelings of their loved ones, and even to their own illness (Robert et al., 2018). Indeed, it may be inappropriate to state that a patient "suffers" from apathy, as their emotional world—including negative emotions—is typically impoverished and blunted (Le Heron et al., 2019; Valentino et al., 2018).

In recent years, research has identified apathy in approximately one-third of individuals with stroke (Caeiro et al., 2013), between 20% and 70% of patients with traumatic brain injury (Starkstein & Pahissa, 2014), and in common

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neurodegenerative disorders such as Alzheimer's disease, with a prevalence of 49% (Zhao et al., 2016), Parkinson's disease (prevalence of 40%) (den Brok et al., 2015), or vascular dementia (prevalence of 65%) (Staekenborg et al., 2010). Apathy is also found in less common conditions, such as frontotemporal dementia, affecting 90% of patients with the behavioral variant (Diehl-Schmid et al., 2006), amyotrophic lateral sclerosis, with a prevalence of 29% (Kutlubaev et al., 2023), and Huntington's disease, with prevalence estimates ranging from 32% to 85% (Matmati et al., 2022). Additionally, it has been reported in psychiatric disorders, such as schizophrenia (Yazbek et al., 2014) and major depressive disorder (Yuen et al., 2015). Beyond clinical categories, apathy has also been observed as a trait or subclinical expression in the normotypic population, particularly among older adults (Brodaty et al., 2010; Pagonabarraga et al., 2015), as well as in association with treatment using selective serotonin reuptake inhibitors (SSRIs) (Masdrakis et al., 2023).

Apathy is characterized by a wide range of negative consequences for both patients and their caregivers. It has been associated with reduced rehabilitation benefits and poor recovery, an increased risk of cognitive decline and dementia, loss of physical, social, and financial autonomy, caregiver burden, poor social reintegration, a higher likelihood of depressive symptoms, increased incidence of cardiovascular diseases, and higher mortality rates (Arnould et al., 2015; Clarke et al., 2010; Dujardin et al., 2007; Hama et al., 2007; Starkstein et al., 2006). In a longitudinal study involving more than 3,000 older adults with cognitive impairment, apathy was found to be the most stable behavioral symptom over time and the one with the greatest impact on mortality, with a 3:1 ratio compared to other disturbances (van der Linde et al., 2016). In this context, the course of apathy tends to be insidious, persisting or even worsening over time (Brodaty et al., 2013; Mayo et al., 2009). This, combined with the limited efficacy of current pharmacological and non-pharmacological interventions, underscores the severity of this condition (Fahed & Steffens, 2021).

Despite its prevalence and significance, apathy has historically been regarded as merely a symptom within various neurological conditions, such as Parkinson's disease (Brissaud, 1895; Pagonabarraga et al., 2015), and psychiatric disorders, such as schizophrenia (Strauss & Cohen, 2017). Over time, various terms emerged to describe behavioral manifestations related to apathy, including abulia, anhedonia, athymhormia, and avolition, which have overlapped to varying degrees with the current concept of apathy. These terms aimed to capture the deficits in motivation, volition, or affect observed in patients, although many have fallen out of use or been redefined (Husain & Roiser, 2018; Prange et al., 2018).

It was not until the seminal work of Robert Marin in the early 1990s that apathy began to consolidate as an independent nosological entity, present in both mental and neurological disorders (Marin, 1990). Marin proposed that apathy could not only be a secondary symptom of other disorders, such as depression or altered levels of consciousness, but could also constitute its own syndrome, characterized by a significant reduction in motivation across the behavioral, emotional, and cognitive domains (Marin, 1991). Since then, numerous attempts have been made to systematize diagnostic criteria and group typical apathy-related disturbances into various models (Levy & Dubois, 2006; Robert et al., 2009, 2018; Starkstein et al., 2006).

To date, the only comprehensive historical and conceptual review on apathy is that by Prange et al. (2018), which primarily focuses on Parkinson's disease and other movement disorders. Their analysis elegantly traces how apathy, as observed in parkinsonism, emerged as a behavioural syndrome linked to basal ganglia pathology and dopaminergic treatments, and how it gradually came to be considered a neuropsychiatric disorder within this specific neurological context.

The present review complements and expands this work in three main ways. First, it broadens the historical lens beyond movement disorders to include psychiatric traditions, particularly the study of negative symptoms, anhedonia, and loss of will in schizophrenia and related affective and psychotic conditions. Second, it systematically compares apathy with related constructs such as abulia, anhedonia, athymhormia, and avolition, highlighting where these concepts overlap and where they diverge across neurological and psychiatric etiologies. Third, by integrating historical contributions from both psychiatric and neurological traditions—including movement disorders, focal lesions, and fronto-subcortical syndromes—the review situates the evolution of apathy within a broader neuropsychiatric framework, clarifying how different conceptual strands have converged to shape the contemporary understanding of the construct (Figs. 1 and 2).

The present paper provides a detailed historical account of apathy from its earliest descriptions to its modern formulation in the 1990s. In doing so, it reviews related terms—such as abulia, anhedonia and affective indifference—to clarify how these constructs have shifted, overlapped or diverged across different periods and clinical traditions. While apathy is now understood as a specific and transdiagnostic construct (Husain & Roiser, 2018), the focus here is on its conceptual evolution and on the historical relationships among these terms (Figs. 3 and 4).

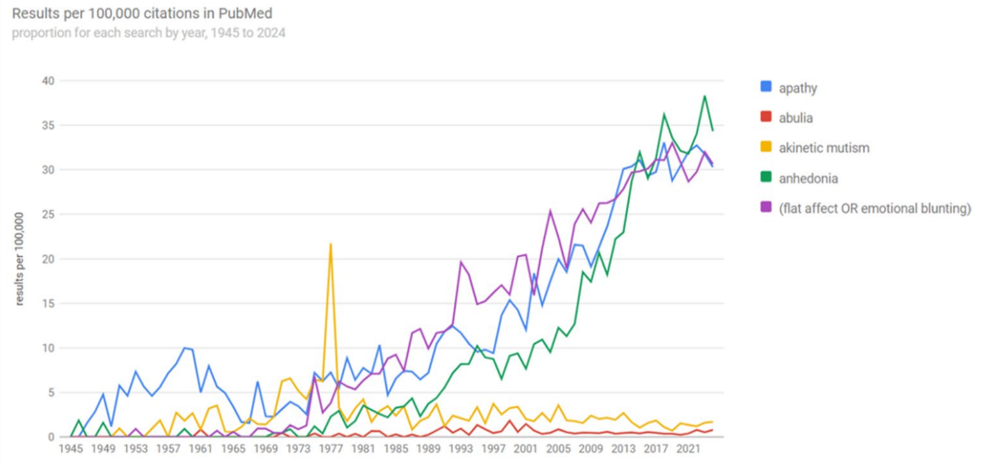

Fig. 1 Publications over time in PubMed database containing the term 'apathy' as well as other frequently related terms. Note: The search, conducted on 26/06/2024 in PubMed by Year, used the term 'apathy' along with other concepts or disorders commonly associated with it, such as abulia, akinetic mutism, anhedonia, and flat affect/emotional blunting (Marin & Wilkosz, 2005). Notably, there was a sharp increase in publications on akinetic mutism in 1977, as well as an exponential rise in publications on apathy, anhedonia, and emotional blunting starting in the 2000s

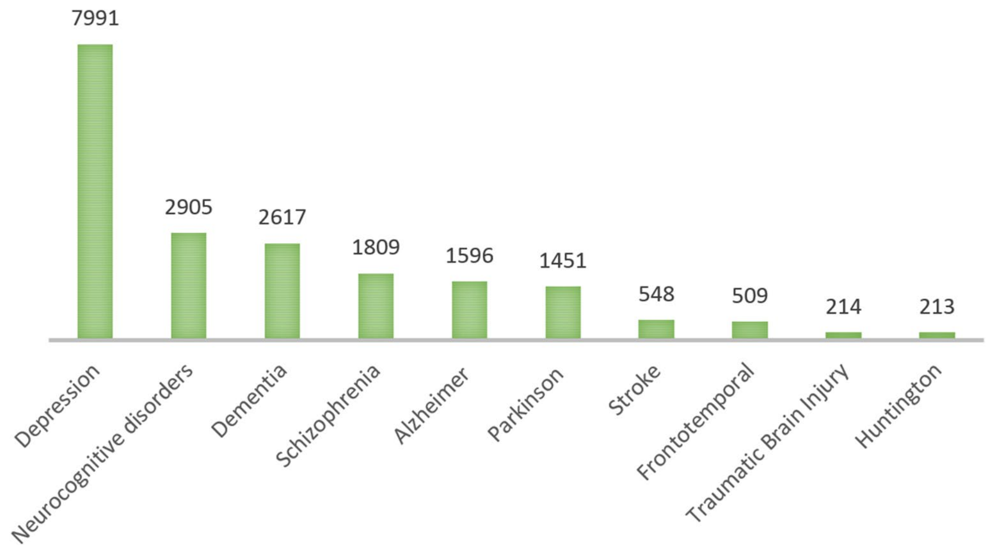

Fig. 2 Number of publications indexed in the PubMed database linking apathy and associated conditions with various neurological and psychiatric disorders. *Note*: The search, conducted on June 26, 2024, used the three most cited terms from the previous figure: apathy, anhedonia, and affective flattening/blunting, which were linked to various neurological and psychiatric pathologies, as well as to two general nosological categories (neurocognitive disorders and dementia)

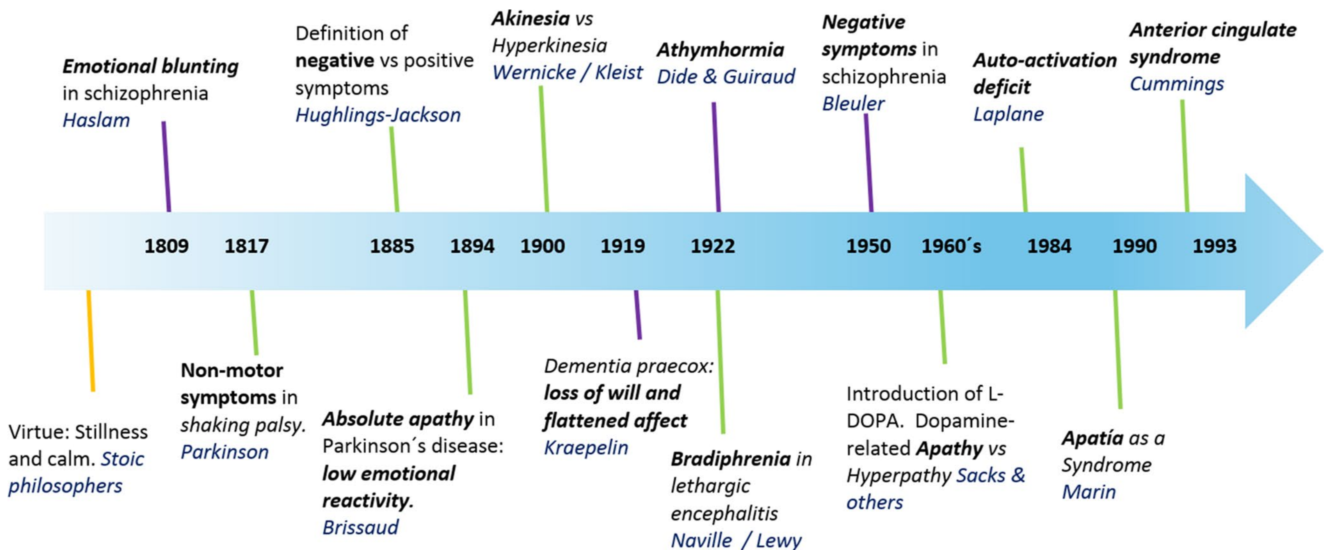

Fig. 3 Timeline of the evolution of concepts associated with apathy, highlighting key milestones and authors. Note: The vertical purple bars represent contributions or concepts derived from the study of patients with mental illness, while the green bars are from the field of neurological disorders

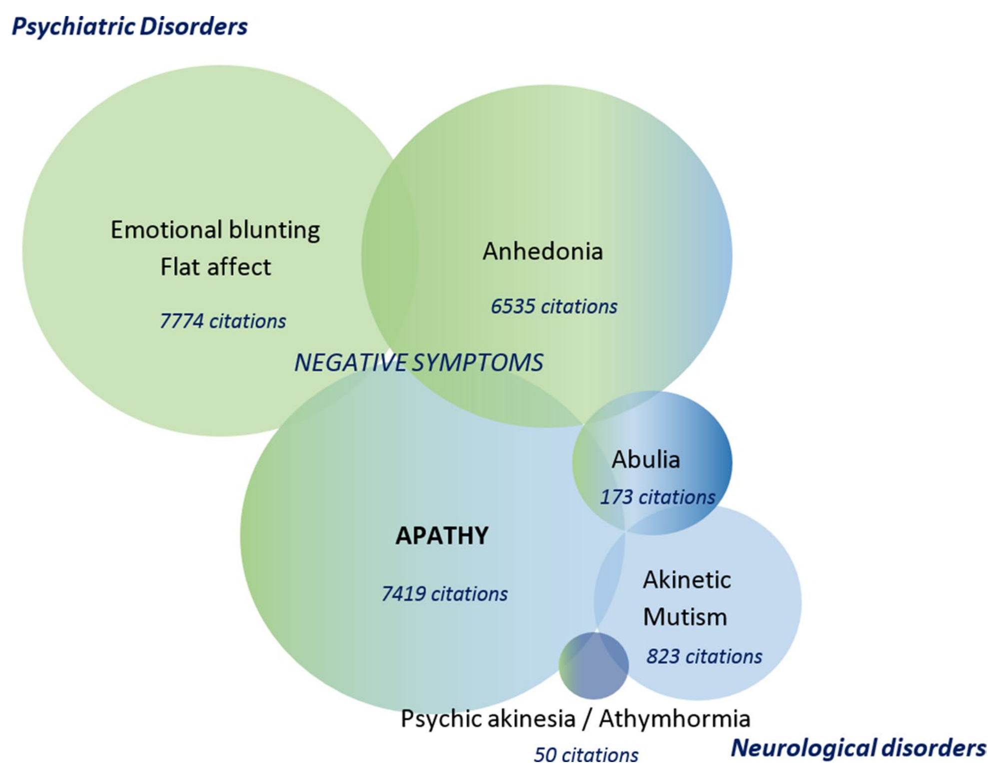

Fig. 4 Relationship of apathy with other concepts in the medical literature. Note: Figure adapted from PubVenn. The size of the circle corresponds to the relative importance of the term based on the number of publications containing it in the Title or Abstract. The relative overlapping areas correspond to publications that contain both terms. The green color represents concepts primarily associated with the affective dimension, while blue represents the motor and motivational dimensions. The search was conducted on 22/1/2025 in https://pubvenn.appspot.com/

This work adopts an integrative historical approach, drawing on contributions from both psychiatry and neurology to illustrate how different conceptualizations of diminished motivation have shaped the modern understanding of apathy. By tracing the evolution and scientific reception of the various terms associated with this phenomenon, the review aims to clarify the foundations upon which the contemporary concept of apathy has been built, thereby setting the stage for the historical analysis that follows.

Evolution of the Concept of Apathy and Related Disorders

The Philosophical Origin of the Term

Long before it was described in medical nosography, the term "apathy" existed as a philosophical concept whose meaning gradually evolved. The word apathy originates from the Greek *ἀπάθεια* (*apátheia*), which can be broken down into the prefix "a" (without) and the root "pathos" (feeling, emotion, illness). Originally, it referred to the absence of feeling or, alternatively, the absence of suffering, something considered a virtue by Stoic philosophers. Indeed, the *Royal Spanish Academy (RAE)* defines apathy as "impassivity of the mind," a definition close to the Stoic one, but also as "slackness, indolence, lack of vigor or energy." On the other hand, the *Oxford English Dictionary* defines apathy as "lack of interest or enthusiasm," which captures a cognitive aspect of apathy (lack of interest) but also an emotional dimension (enthusiasm). Finally, the *French Larousse Dictionary* defines apathy as "the indolence or indifference of someone taken to the point of total insensitivity," adopting the Stoic perspective but also adding the meaning of "slowness to act or react, passivity, or inertia," which particularly emphasizes its motivational aspect.

Thus, apathy as a philosophical concept is viewed as the absence of emotions or passions that could disturb the individual's mind. It is, therefore, an intentional attitude closely linked to will. This concept has partially persisted into modern times, incorporated into some definitions of apathy. However, the positive connotations that Stoic philosophers associated with this state have not been carried over.

Apathy in Medical Science of the 19th and 20th Centuries

The adoption of this concept by science in general, and medical science in particular, did not occur until the 19th century, when numerous experts in medicine and psychology detected, reported, and described these alterations in their patients, giving rise to numerous concepts that today partially or completely overlap with the concept of apathy (Prange et al., 2018).

Apathy in Psychiatry

In parallel with research on patients with neurological diseases, much of the classical descriptions and reflections on apathy and associated alterations were made in the context of mental illnesses, particularly schizophrenia. While in the context of neurocognitive disorders, apathy is the most frequently used term, in the domain of psychiatric and neuropsychiatric disorders, the term "negative symptoms" was historically coined to refer to one of the core components of psychotic disorders, especially schizophrenia (Bleuler, 1950; Kraepelin, 1919). Negative symptoms involved a significant reduction in goal-directed activities, pleasure, emotional expression, and speech (Strauss & Cohen, 2017).

As Marder and Galderisi (2017) note in their review of the origins of the study of *negative symptoms* in schizophrenia, the first descriptions of these symptoms date back to the early 19th century. In 1809, John Haslam described a mental illness in young people characterized by blunting of sensitivity and emotional indifference (Haslam, 1809), which are now considered core features of the negative symptoms in schizophrenia and other neuropsychiatric disorders. Almost simultaneously, Philippe Pinel described "idiocy" in his treatise on mental illness as "*a more or less absolute destruction of the functions involved in understanding*" but also in "*heart disorders*," highlighting the affective dimension of mental illness (in Dollfus & Lyne, 2017). During the 19th century and the first half of the 20th century, not only were the behaviors of apathetic patients or those with negative symptoms described, but attempts were also made to offer explanations for the possible underlying mechanisms. This is the case of the eminent neurologist John Hughlings Jackson, who regarded negative symptoms as "*losses or reductions in aspects of higher cognitive and emotional functions*," while *positive symptoms* (hallucinations and delusions) were seen as a "phenomenon of release," that is, distortions or episodic exaggerations of normal functioning (Jackson, 1885).

Building on the idea that negative symptoms reflect impairments in cognitive and emotional functions, Kraepelin (1919) described the negative symptoms of *dementia praecox* as "*a weakening of the emotional activities that are the primary drivers of will*, *emotional blunting*, *failure of mental activities*, *and a loss of control over willpower*, *effort*, *and the ability for independent action*." As early as 1906, Kraepelin emphasized the importance of analyzing the affective domain rather than the intellectual one, highlighting that emotional blunting, and negative symptoms in general, were the predominant features of this illness (Dollfus & Lyne, 2017).

Just a few years later, in 1922, psychiatrists Dide and Guiraud (1922) proposed describing *dementia precox* as "juvenile atimormia." The neologism *atimormia*, coined by the authors themselves, was derived from the Greek words *thimos* (feeling or mood) and *horme* (impulse), preceded by the privative "a." Thus, it referred to a lack of feelings and impulses in the absence of physical alterations. This term was later revived to describe the symptoms exhibited by patients with lesions in the striatum (Habib & Poncet, 1988).

Bleuler (1950), in line with previous ideas, considered *emotional blunting* to be central to *schizophrenia*, a term he introduced, while defining hallucinations, delusions, and catatonia as acute exacerbations (Bleuler, 1950). One of his descriptions of patients with schizophrenia highlights some of the most striking features of severe apathy: "*Many schizophrenics… sitting… with expressionless faces*, *hunched over*, *are the very image of indifference. They allow themselves to be dressed and undressed like automatons*, *led from their usual place of inactivity to the dining room and back again without showing any signs of satisfaction or dissatisfaction*."

Thus, in addition to providing insightful descriptions of behavioral deficits that we now recognize as manifestations of apathy, they proposed several mechanisms that might be associated with this alteration. Specifically, they focused their explanations on the emotional dimension of apathy (flattening, blunting, lack of reactivity) and suggested that, unlike positive symptoms such as delusions or hallucinations, negative symptoms resulted from a structural deficit in higher cognitive and emotional processes. That is, apathy and other defect-related symptoms in psychiatric disorders are always accompanied by cognitive deficits.

Apathy in Neurology

Research on negative symptoms in mental illness and apathy in neurological studies has traditionally progressed in parallel, with only recent transdiagnostic formulations of this disorder fostering a closer alignment between these two perspectives (Husain & Roiser, 2018). In this regard, the current conceptualization of apathy stems from studies conducted with neurological patients in the 19th and 20th centuries. Prange et al. (2018) outlined a conceptual journey of the term 'apathy' and other related terms, based on the study of neurological patients with movement disorders. In his 1817 publication *An Essay on the Shaking Palsy*, James Parkinson was the first to describe the psychiatric symptoms of the disease that bears his name, among which apathy was later included by other authors. It was not until Edouard Brissaud, one of Jean-Martin Charcot's disciples, that greater attention was paid to the non-motor symptoms of Parkinson's disease, specifically apathy. Brissaud, who took charge of the Tuesday lectures at the Salpêtrière hospital between 1893 and 1895, devoted two consecutive lectures,

the 22nd and 23rd, to Parkinson's disease, where he reported psychic signs of the disease, including apathy. As Prange et al. (2018) note, for the first time in history, apathy was recognized as an independent pathological condition. Brissaud described the *absolute apathy* of some Parkinson's patients as "*a state of mixed indifference and absence of reaction to external and internal events… the patients do not speak any more than they move… they seem disconnected from the world around them, indifferent to everything… they remain inert in the absence of impulse, as if they needed to be provoked to react*" (Brissaud, 1895).

In his description, Brissaud highlighted the lack of emotional reactivity to environmental stimuli in apathetic patients, emphasizing the affective dimension of the disorder. Moreover, with the phrase "*patients do not speak more than they move…"* he skillfully pointed to the association between the reduced motor behaviors of these patients (*hypokinesia*), which is a hallmark of Parkinson's disease, and the reduction in their language and mental life (Prange et al., 2018). Brissaud explicitly separated apathy from depression, anxiety, and cognitive impairments, attributing it to a process of "intellectual welding" (*soudure intellectuelle*) that reflected the patients' motor disability. He argued that apathetic patients remained inert in the absence of external impulses, as if they needed to be provoked to react. This similarity between motor and psychic manifestations was also reflected in *amimia* (lack of facial expressiveness), which mirrored the patients' mental state. Despite the innovative nature of this perspective, Brissaud's description of apathy in Parkinson's disease was not internationally recognized at the time (Prange et al., 2018).

An important contribution to the conceptual framework of apathy at the beginning of the 20th century came from Karl Wernicke's classification of behavioral syndromes in mental illness in Berlin in 1900 (Ajuriaguerra, 1971). Wernicke coined the term *akinesia* to describe behaviors similar to catatonia, contrasting them with opposite manifestations, such as *hyperkinetic* behaviors like mania. In his classification, he hypothesized that motor signs represented only the "tip of the iceberg" of internal psychic movements, which he also referred to as a "psychosis of mobility" (in Prange et al., 2018). His disciple, Karl Kleist, defined *akinesia* as a syndrome characterized by a lack of initiative and drive in frontal behavioral syndromes following traumatic brain injury (Ajuriaguerra, 1971), a view strikingly similar to Brissaud's "absolute apathy." In 1923, Friedrich Heinrich Lewy redefined *akinesia* in the way it is understood today, as a delay in the initiation of movement in parkinsonism (Ajuriaguerra, 1971). This was probably one of the first approaches where the reduction of initiative was directly associated with slowness in initiation (Prange et al., 2018).

Although the term *apathy* eventually gave rise to various other terms, its original meaning was preserved by François Naville, who, in 1922, introduced a new term: *bradyphrenia.* While studying the mental complications of the epidemic known as encephalitis lethargica, Naville described the syndrome of apathy, distinguishing between its motor, cognitive, and emotional components. He characterized it as: "*A very frequent and peculiar state of mental fatigue and fading […] characterized by a reduction in voluntary attention, spontaneous interest, initiative, effort, and work capacity, accompanied by objective fatigue as well as a slight decline in memory*" (Naville, 1922). Naville successfully differentiated the cognitive and motivational aspects of apathy in patients with Parkinson-like symptoms caused by encephalitis lethargica. He also described the symptoms associated with *bradyphrenia*: a slowing of thought distinct from motor slowness, contributing to an overall deceleration in action. Naville noted deficits in initiation and novelty-seeking behaviors and observed that these patients struggled more with problem-solving tasks requiring external stimulation than with tests independent of such stimulation. Furthermore, he remarked that external repeated stimulation could improve their performance. Naville explained, "*It is not only the peripheral execution of movements that is slow, nor solely the time lost between the given command and the beginning of execution, but primarily and fundamentally, the distribution of commands and movements itself is slowed or, at times, suspended—something that can disappear under external stimulation.*" Based on this observation, Naville drew a parallel between alterations in motor tone and the loss of "psychic tone" in post-encephalitic patients. He hypothesized that, at least in diseases affecting the basal ganglia, mental slowing was not solely due to the slow execution of movements but also to a loss of initiative and drive.

Nevertheless, the descriptions and observations made during this period could be regarded as somewhat unsystematic from today's perspective. Various authors characterized this phenomenon in markedly different ways and generally confined their studies to mental illness. The advances in neurology, cognitive neuroscience, and neuropsychology that emerged in the second half of the 20th century significantly contributed to shaping the modern concept of apathy, which has a history of little more than thirty years.

In this regard, another turning point in the conceptualization of apathy through the study of neurological diseases occurred between the 1970s and 1990s. During these decades, research on neuropsychiatric syndromes associated with basal ganglia dysfunction, including focal lesions, led to the proliferation of various terms such as *athymhormia*, *psychic auto-activation deficit*, and *pure psychic akinesia*. These terms were later replaced by others, such as

abulia or *anhedonia* (Prange et al., 2018), all of which share features with apathy. For instance, in 1982, Laplane et al. described a severe form of apathy, which they termed the *syndrome of loss of psychic auto-activation*, in a patient who had sustained lesions in the lenticular nuclei. Subsequently, the same research group reported three additional cases, all of which involved patients who had experienced toxic encephalopathy (Laplane et al., 1984).

The introduction of levodopa (L-DOPA) during the transition from the 1960s to the 1970s not only revolutionized the treatment of Parkinson's disease and significantly improved patients' quality of life but also opened a new line of research linking dopamine deficits or excesses to behavioral alterations such as apathy or behavioral disinhibition, respectively (Prange et al., 2018). Oliver Sacks was among the first to systematically describe the side effects of L-DOPA, juxtaposing motor, cognitive, and emotional symptoms with their counterparts induced by L-DOPA therapy in patients with encephalitic parkinsonism. According to Sacks, the apathy accompanied by emotional flattening observed in parkinsonism transformed into its opposite following L-DOPA therapy, leading him to coin the term **"***hyperpathy*.**"** This state was characterized by increased appetite, heightened sexual arousal, irritability, aggression, and impatience (Prange et al., 2018).

Thus, a continuum of behavioral alterations was proposed, linked to insufficient or excessive dopamine levels in the fronto-subcortical circuits (Pagonabarraga et al., 2015). Among these disorders, apathy emerged as one of the non-motor manifestations associated with dopaminergic deficits. Lastly, another advancement in understanding behavioral symptoms linked to altered dopamine levels arose from the implementation of deep brain stimulation (DBS) of the subthalamic nucleus in the 1990s (Limousin et al., 1995). One of the unexpected side effects was the appearance of post-surgical apathy, which was attributed to a withdrawal syndrome from dopaminergic treatment. This syndrome, in addition to apathy, could include anxiety and depression (Thobois et al., 2010). Post-surgical apathy was associated with mesolimbic dopaminergic denervation and appeared to respond favorably to dopaminergic agonists (Pagonabarraga et al., 2015), further emphasizing the relationship between dopamine-rich structures, such as the basal ganglia and prefrontal cortex, and apathy.

Contributions such as those mentioned above highlight the intriguing parallelism between the motor and psychic domains, an idea elegantly pointed out by authors like Brissaud in movement disorders (Brissaud, 1895) and Dide and Guiraud in patients with schizophrenia (Dide & Guiraud, 1922). Just as motor movements are impossible without muscle tone, these authors implicitly suggest the necessity of an optimal "psychic tone" to facilitate the initiation of emotions and cognitions.

Beyond movement disorders, apathy was also described in the late 20th century in other focal neurological conditions, such as stroke. One notable example is patients with the rare bilateral thalamic infarction, also known as Percheron syndrome or paramedian diencephalic syndrome (Laplane, 1984; Meissner et al., 1987; Engelborghs et al., 2000). These patients commonly exhibit vertical gaze palsy, arousal deficits, and severe anterograde amnesia with confabulations (Meissner et al., 1987). A particularly striking feature in these cases is a profound impairment in "psychic self-activation," often accompanied by initial drowsiness (Engelborghs et al., 2000).

Finally, it is worth highlighting the contribution of Jeffrey L. Cummings's review of the different frontal syndromes. In his manual *Frontal-Subcortical Circuits and Human Behavior* (Cummings, 1993), he synthesizes the behavioral effects of lesions in the frontal lobe and related subcortical structures (basal ganglia and thalamus). While apathy is associated with lesions in the dorsolateral prefrontal cortex (and the subcortical regions connected to it), this impairment takes on particular significance in the so-called *anterior cingulate syndrome*. When lesions are bilateral, this syndrome is characterized by severe akinetic mutism: "*Patients are profoundly apathetic. They usually keep their eyes open, do not speak spontaneously, and respond to questions, if at all, with monosyllables. They move very little, are incontinent, and eat and drink only when fed. They exhibit no emotion, even in response to painful stimuli, and are indifferent to their own circumstances*." (Cummings, 1993).

In summary, the medical and scientific study of apathy and related conditions dates back to the early 19th century, with investigations into the behavioral disturbances of patients with schizophrenia on one hand, and those with neurological diseases on the other. Over the past two centuries, but especially since the early 20th century, analogous or at least closely related conditions to the modern conceptualization of apathy were described, and possible etiopathogenic mechanisms of the disorder were even proposed. Table 1 lists some of the clinical concepts most closely associated with apathy.

The Contemporary Concept of Apathy

One of the most influential conceptual frameworks in recent decades was introduced by Robert Marin in the early 1990s. Based on extensive clinical observation across various neurological and psychiatric disorders, Marin operationalized apathy as a "*loss of motivation, relative to a previous level, that is not attributable to emotional distress, cognitive impairment, or diminished level of consciousness*." In his model, apathy manifested in three partially dissociable components: reduced productivity (behavior), restricted

Table 1 Description of concepts and disorders analogous or associated with apathy

Concept	Authors (examples)	Description	Observations
Abulia	Bhatia & Marsden, 1994 Berrios et al., 1995	"*Loss, absence, or impairment of the will's ability to execute what is in the mind. In this condition, there is no paralysis or disorder of the muscular system, and the desire or the achievement of the intended goal is often not lacking; however, the transition from intention and desire to execution becomes abnormally difficult or impossible*." (Berrios, 1996, as cited in Prange et al., 2018)	The term was used by French alienists since 1830 to refer to certain mental disorders and was later popularized by psychologists Pierre Janet (1859–1947), Alfred Binet (1857–1911), and Théodule Armand Ribot (1839–1916) (Berrios, 1996) It is now regarded as a severe form of apathy characterized by a significant reduction in spontaneous verbal, motor, cognitive, and emotional behaviors (Bhatia & Marsden, 1994)
Akinetic Mutism	Cairns et al., 1941 Miller & Cummings, 2017 Arnts et al., 2020	A rare neurological disorder that affects initiation and motivation. Patients maintain an intact level of alertness, as well as attention, language, and sensorimotor abilities, but exhibit a drastic reduction in goal-directed behavior and emotions (Miller & Cummings, 2017)	First described in 1941 in patients with tumors in the third ventricle, as well as in patients with cerebrovascular disease, hydrocephalus, and conditions that affect fronto-subcortical circuits. It is considered the extreme of amotivational disorders (Arnts et al., 2020)
Anhedonia	Ribot, 1896. Jordan et al., 2013 Strauss & Cohen, 2017	First defined by Ribot as an "*inability to experience pleasure*", anhedonia is now conceptualized as a multidimensional reduction in reward processing, affecting the experience of pleasure ("*liking*"), the motivational value of rewards ("*wanting*"), or the ability to initiate and pursue rewarding activities (Rizvi et al., 2016; Strauss & Cohen, 2017).	Anhedonia is one of the negative symptoms of schizophrenia and also appears in major depressive disorder (Strauss & Cohen, 2017). Clinical and experimental studies indicate that the deficit may involve anticipatory motivation, reward valuation, or engagement in goal-directed reward-seeking, while hedonic reactivity ("*liking*") may remain relatively preserved in some patients, such as those with Parkinson's disease (Jordan et al., 2013).
Athymhormia	Dide & Guiraud, 1922 Habib & Poncet, 1988 Habib, 2004	Lack of feelings and impulses in the absence of physical alterations. It involves: (1) *anhormia*, considered as the loss of an 'instinctive vital dynamism,' (2) *atimia*, corresponding to the subjective feeling of the same phenomenon, and (3) *motor inertia*, which characterizes the external appearance of these patients and is actually the result of the previous factors (Habib, 2004)	First described in patients with schizophrenia by psychiatrists Dide and Guiraud (Dide & Guiraud, 1922). It is the result of pathology associated with the basal ganglia (Habib, 2004; Habib & Poncet, 1988)
Auto-Activation Deficit	Laplane & Dubois, 2001	Inability to spontaneously initiate mental activity, which is reversible with external stimulation (Laplane & Dubois, 2001).	Associated with pathology in the basal ganglia. Different from abulia observed in frontal lesions, as it is not reversible with external stimulation (Laplane & Dubois, 2001).
Avolition	Strauss & Cohen, 2017	Reduction in the initiation and persistence of goal-directed activities, as well as in the desire to perform these activities (Strauss & Cohen, 2017).	Avolition is one of the negative symptoms of schizophrenia and other psychiatric conditions, such as depression (Strauss & Cohen, 2017).
Emotional blunting/ Flat affect	Kraepelin, 1919 Strauss & Cohen, 2017	Reduction in the external expression of emotions in terms of facial expression, vocal tone, or body gestures (Strauss & Cohen, 2017)	Flat affect is one of the negative symptoms of schizophrenia and other psychiatric conditions, such as depression (Strauss & Cohen, 2017).
Psychic Akinesia	Laplane et al., 1984 Bhatia & Marsden, 1994	Loss of 'drive' or apathy with a loss of initiative, as well as spontaneous emotional responses and thought (Bhatia & Marsden, 1994; Laplane et al., 1984).	Result of bilateral lesions in the globus pallidus or caudate (Bhatia & Marsden, 1994)

goal-setting (cognition), and diminished emotional responses to success and failure (emotion) (Marin, 1990, 1991). Although he did not clearly define the organization of the different dimensions or the potential existence of a hierarchy among them,

these three dimensions, with certain refinements and additional aspects—such as the social dimension (Ang et al., 2017)—are now present in nearly all contemporary definitions of apathy, as well as in the scales used to assess it (Miller et al., 2021; Robert et al., 2009, 2018; Starkstein et al. 2000).

A second major contribution by Marin, reflected in his definition of apathy, was the proposal to consider it a distinct nosological entity and, therefore, a primary condition. Rather than viewing apathy merely as a consequence or secondary symptom of disorders such as depression,

schizophrenia, or transient confusional states, he conceptualized it as a motivational syndrome with its own underlying pathophysiological mechanisms (Marin et al., 1990, 1991).

Other authors, such as Levy and Dubois (2006), argue that apathy is not a unitary construct but rather a phenomenon that can arise from different lesion topologies, leading to reduced goal-directed behavior. Building upon Marin's three-dimensional model, they proposed three subtypes of apathy, each associated with distinct neurocognitive mechanisms: (1) affective-emotional apathy, linked to lesions in the ventromedial and orbitofrontal cortex, as well as subcortical structures connected to these regions; (2) cognitive-executive apathy, associated with lesions in the lateral prefrontal cortex and its related subcortical structures; and (3) auto-activation apathy, resulting from extensive lesions affecting anterior circuits or bilateral frontomedial damage, characterized by a marked reduction in the spontaneous generation of thoughts, emotions, and behaviors. This conceptualization of apathy departs from the traditional notion of "motivation," which is considered a psychological construct that is challenging to operationalize and can only be inferred from observable behavior (Levy & Dubois, 2006). Consequently, more recent definitions have replaced "*reduced motivation*" with "*a reduction in goal-directed activity*" (Robert et al., 2018).

Regardless of the theoretical framework adopted, recent literature increasingly recognizes apathy as a transdiagnostic entity that cuts across traditional disease boundaries (Robert et al., 2018; Husain & Roiser, 2018). There is growing evidence suggesting that the neuroanatomical regions and circuits implicated in apathy are, at least in part, shared across different neurological disorders, psychiatric conditions, and even in healthy aging. A meta-analysis by Yan et al. (2023) identified a significant association between apathy and structural and functional alterations in the putamen and caudate across multiple disorders. Furthermore, structural neuroimaging studies revealed atrophy in the bilateral precentral gyrus, bilateral insula, bilateral medial frontal gyrus, bilateral inferior frontal gyrus, left caudate/putamen, and right anterior cingulate cortex. These fronto-striatal regions and circuits, the authors note, are associated with executive functions (e.g., planning and goal generation), reward processing, and the integration of affective and cognitive aspects for behavioral control (Yan et al., 2023).

Thus, there is now broad consensus that apathy should be regarded as a transdiagnostic entity that can manifest either as a symptom or sign of other conditions or as an independent syndrome. It affects multiple dimensions and may present with distinct clinical features depending on the lesion topology and the specific cognitive and emotional mechanisms involved. This perspective highlights the potential value of a comparative approach to studying diseases in which apathy frequently occurs, along with its associated symptoms. Such an approach may provide critical insights into the pathophysiological mechanisms underlying this potentially heterogeneous condition.

Conclusions

From the philosophical origins of the term to the earliest symptom descriptions, apathy has emerged as an independent disorder situated at the crossroads of neurological and psychiatric diseases. Over history, various concepts have highlighted the affective and emotional aspects of the disorder and its social impact, the reduction in mental life, or the motivational and volitional elements inferred from the dramatic decrease in patients' spontaneous behaviors. On the other hand, from an etiopathogenic perspective, there is significant convergence in associating this multidimensional condition with lesions or dysfunctions in frontobasal circuits and neurotransmitters such as dopamine.

The conceptual evolution of apathy also reflects the broader historical convergence of neurology and psychiatry. Disorders of diminished motivation were initially described within psychiatry, particularly in the literature on melancholia, schizophrenia and the negative symptoms tradition, whereas related syndromes such as abulia, athymhormia or the auto-activation deficit emerged from lesion studies in neurology. As fronto-subcortical circuit models developed, these parallel traditions progressively informed one another, contributing to the consolidation of neuropsychiatry as an integrative discipline in which behavioural syndromes are understood as manifestations of network dysfunction rather than exclusively "functional" or "organic" conditions (Cummings, 1993; Habib, 2004; Laplane & Dubois, 2001). Apathy exemplifies this convergence, standing as a genuinely neuropsychiatric construct shaped jointly by clinical phenomenology, lesion evidence and cognitive neuroscience.

Therapeutic studies have also informed contemporary conceptualizations of apathy. In Parkinson's disease, dopamine agonists such as piribedil and rotigotine have been reported to improve apathy in some studies, though the evidence remains modest and not uniformly replicated (Bogdan et al., 2020). In Alzheimer's disease and related dementias, evidence for pharmacological treatments such as cholinesterase inhibitors or memantine is limited and inconsistent, with minimal benefit reported in many reviews (Fahed & Steffens, 2021). Studies generally show that conventional antidepressants do not consistently improve apathetic symptoms beyond effects on mood, supporting the view that apathy is distinct from depressed mood.

Non-pharmacological interventions also reinforce this distinction. Structured activity programmes, behavioural

activation, environmental enrichment and caregiver-mediated strategies show modest but reproducible improvements in apathy, particularly in dementia (Brodaty et al., 2012). These interventions tend to rely on external cueing and scaffolding of goal-directed behaviour, features that map more closely onto deficits in initiation or auto-activation than onto alterations in hedonic capacity. Such patterns align with contemporary models that conceptualise apathy as a disruption of motivated behaviour and action initiation rather than as a primary deficit of pleasure or affective experience (Husain & Roiser, 2018; Le Heron et al., 2018).

In summary, from the early descriptions by John Haslam and Pinel to Robert Marin's proposal, which marked the beginning of the current nosological conception of the disorder, apathy and its associated symptoms have been known by various names and approached from different perspectives. Some of these have been relegated to the domain of medical historiography, while others have significantly contributed to shaping current knowledge about this transdiagnostic entity, which resides at the intersection of neurology and psychiatry.

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Declarations

Competing Interests All authors certify that they have no affiliations with or involvement in any organization or entity with any financial interest or non-financial interest in the subject matter or materials discussed in this manuscript.

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