Article

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Reference

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REVIEW

A Multidimensional Approach to Apathy after Traumatic Brain Injury

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Abstract Apathy is commonly described following traumatic brain injury (TBI) and is associated with serious consequences, notably for patients' participation in rehabilitation, family life and later social reintegration. There is strong evidence in the literature of the multidimensional nature of apathy (behavioural, cognitive and emotional), but the processes underlying each dimension are still unclear. The purpose of this article is first, to provide a critical review of the current definitions and instruments used to measure apathy in neurological and psychiatric disorders, and second, to review the prevalence, characteristics, neuroanatomical correlates, relationships with other neurobehavioural disorders and mechanisms of apathy in the TBI population. In this context, we propose a new multidimensional framework that takes into account the various mechanisms at play in the facets of apathy, including not only cognitive factors, especially executive, but also affective factors (e.g., negative mood), motivational variables (e.g., anticipatory pleasure) and aspects related to personal identity (e.g., self-esteem). Future investigations that consider these various factors will help improve the understanding of apathy. This theoretical framework opens up relevant prospects for better clinical assessment and rehabilitation of these frequently described motivational disorders in patients with brain injury.

Keywords Apathy · Traumatic brain injury · Motivation · Depression · Executive functions · Self-esteem

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Introduction

Apathetic manifestations are common across a wide variety of neurological and psychiatric conditions, such as traumatic brain injury (TBI; Lane-Brown and Tate 2009), disorders involving the basal ganglia (Stuss et al. 2000; Pluck and Brown 2002), Alzheimer's disease (Fernandez Martinez et al. 2008) and cerebrovascular accident (Andersson et al. 1999b; Jorge et al. 2010). Apathy not only appears to be common, but it has also been related to a wide range of negative consequences for the patients and their caregivers. Indeed, apathy has been associated with a poor recovery and rehabilitation outcome (Gray et al. 1994; Hama et al. 2007), loss of social autonomy (Prigatano 1992; Mazaux et al. 1997), financial and vocational loss (Lane-Brown and Tate 2009), cognitive decline (Dujardin et al. 2007; Robert et al. 2002; 2006) and caregiver distress (Marsh et al. 1998; Willer et al. 2001). Despite the important prevalence of this problematic manifestation and its social and economic costs, apathy is defined in a number of different ways (Lane-Brown and Tate 2009) and its underlying psychological processes are poorly understood. The lack of clarity surrounding the construct of apathy has the unfortunate consequence that apathy is often neglected in clinical practice and rehabilitation programmes are not targeted. In this context, the main objective of this

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article is to open up relevant prospects for better clinical assessment and rehabilitation of these frequently described apathetic manifestations after a TBI. In this perspective, the first part of the present article reviews the current definitions of apathy and its assessment tools, and proposes a discussion of their limitations. The second part of the article presents the existing findings related to apathy in the TBI population (the prevalence, neuroanatomical correlates, relationships with other neurobehavioural disorders, and the psychological processes involved). Based on all these data, a new approach to apathy after TBI is proposed that assume four dimensions of apathy (cognitive, affective, motivational, and related to personal identity), which are defined by a precise identification of the various mechanisms potentially involved.

Current Conceptions of Apathy

Definitions of Apathy

The concept of apathy is found in various sections of the *Diagnostic and Statistical Manual of Mental Disorders* (4th ed., text rev.; *DSM-IV-TR*; American Psychiatric Association 2000) and in the definitions of many types of disorders (Clarke et al. 2011). By contrast, the *International Classification of Diseases* (*ICD-10*; World Health Organization 1993) makes no reference to apathy. Marin (1991) differentiated between apathy as a symptom of other problems (such as emotional distress or an altered level of consciousness) and apathy as a syndrome. In the latter, the key feature, according to Marin, is a lack of motivation, characterised by diminished goal-directed cognition (as manifested by decreased interests, a lack of plans and goals, and a lack of concern about one's own health or functional status), diminished goal-directed behaviour (as manifested by a lack of effort, initiative and productivity) and reduced emotional concomitants of goal-directed behaviours (as manifested by flat affect, emotional indifference and restricted responses to important life events). Goal-directed behaviour is defined as a set of related processes (motivational, emotional, cognitive and motor) by which an internal state is translated, through action, into the attainment of a goal (Schultz 1999; Brown and Pluck 2000). More specifically, the "goal" can be immediate and physical, such as relieving thirst, or long-term and abstract, such as being successful in one's job or pursuing happiness. The term "directed" means that the action is mediated by knowledge of the contingency between the action and the outcome (Dickinson and Balleine 1994). The diversity of psychological mechanisms involved in goal-directed behaviours, which are central in Marin's formulation of apathy, argue in favour of a multifactorial model of apathy. Marin's concept was later put into practice by Starkstein et al. (2001) in a set of diagnostic criteria for apathy, as follows: (A) lack of motivation relative to the patient's previous level of functioning or the standards of his or her age and culture as indicated by subjective account or observation by others; (B) presence for at least 4 weeks during most of the day, of at least one symptom belonging to each of the following three domains: (i) diminished goal-directed behaviour, (ii) diminished goal-directed cognition and (iii) diminished concomitants of goal-directed behaviour; (C) the symptoms cause clinically significant distress or impairment in social, occupational or other important areas of functioning; and (D) the symptoms are not due to diminished level of consciousness or the direct physiological effects of a substance.

Stuss et al. (2000) argued that apathy cannot be clinically defined as a lack of motivation, as did Marin (1991), notably because the assessment of motivation is problematic and usually requires inferences based on observations of affect or behaviour. The authors suggested that apathy should be defined as "an absence of responsiveness to stimuli—internal or external—as demonstrated by a lack of self-initiated action". Consequently, the construct of initiation is central to Stuss et al.'s definition. Stuss et al. (2000) also postulated that apathy may represent a number of related but separable states, depending on the neural substrates involved and the functional disturbances (cognitive, behavioural, affective) underlying the clinical presentation. According to the authors, the involvement of the dorsolateral prefrontal cortex circuit may result in an absence of initiated behaviour due to an executive dysfunction disorder affecting cognitive flexibility, planning and novel responsiveness, among other things. Involvement of the lateral orbitofrontal cortex would lead to apathy associated with a lack of limbic affective input, including processes such as reward sensitivity and an interest in new learning, while involvement of the anterior cingulate may result in apathy due to a reduction in direct motivational response to external and internal stimuli. The authors also posited a type of apathy termed "social apathy", which takes the form of impaired self and social awareness due to lesions in anterior frontal regions.

Other investigators emphasised that the lack of spontaneity observed among apathetic patients can be reverted by external cues. In this sense, constructs such as the auto-activation deficit (AAD; Levy and Dubois 2006) and athymhormia (Habib 2004) describe patients who are incapable of self-activating thoughts or self-initiating actions but who have a relatively spared ability to generate externally driven behaviour. In line with Stuss et al. (2000), Levy and Dubois (2006) divided apathetic syndrome into three subtypes (emotional, cognitive and behavioural) but replaced the behavioural subtype with the concept of auto-activation. The disruption of auto-activation processing may result in the most severe form of apathy, in most cases the lesions bilaterally affecting the associative and limbic areas of the internal portion of the globus pallidus. Consequently, Levy and Dubois (2006) defined apathy as "the quantitative reduction of self-generated,

voluntary and purposeful behaviours" and suggested the following subtypes of apathy: emotional-affective (inability to associate affective/emotional signals, such as interest, with ongoing and forthcoming behaviours), cognitive (inability to elaborate a plan of action due to impairments in several executive functions, including planning, flexibility and rule finding) and auto-activation (inability to activate thoughts or initiate action).

Recently, a new set of diagnostic criteria for apathy has been proposed (Robert et al. 2009; Mulin et al. 2011) in the context of a consensus meeting that included members of the European Psychiatric Association, the European Alzheimer's Disease Consortium and the Association Française de Psychiatrie Biologique (see Table 1). The criteria follow the same general structure as those proposed by Starkstein et al. (2001), with changes specifically to the B criterion. According to Mulin et al. (2011), a diagnosis of apathy can be made in the presence of diminished motivation in comparison to the patient's previous level of functioning (criterion A), and two of the three following domains of apathy, which must be present for at least 4 weeks (criterion B): (1) loss of or diminished goal-directed behaviour, (2) loss of or diminished goal-directed cognitive activity and (3) loss of or diminished emotions. In addition, symptoms should cause clinically significant impairment in various functional domains (criterion C). Exclusion criteria specify symptoms that mimic apathy, such as the direct physiological effects of a substance (criterion D). More specifically, criterion B is based on the premise that changes in motivation can be observed (and measured) by examining a patient's responsiveness to internal and external stimuli. Consequently, each of the three proposed domains of apathy (behaviour, cognition and emotion) includes two symptoms: the first symptom pertains to self-initiated or "internal" actions, cognitions and emotions ("initiation symptom"), and the second symptom to the patient's responsiveness to "external" stimuli ("responsiveness symptom").

Apathy Assessment Scales

Several instruments are currently used to measure the severity of apathy in neurological and psychiatric disorders (see Table 2) (see Clarke et al. 2011 for a review). Marin et al. (1991) developed the *Apathy Evaluation Scale* (AES), an 18-item scale that assesses apathy within the month prior to the time of assessment and covers the behavioural, emotional and cognitive aspects of apathy. Each item is rated on a 4-point Likert scale, from 0 (not at all true) to 4 (very true). This scale may be administered in a self-report version (AES-S), or may be completed by relatives or caregivers (AES-I) or by a clinician after a semi-structured interview with the participant (AES-C). Its psychometric properties have been found to be acceptable in some studies (Marin et al. 1994; Ramirez et al.

Table 1 Diagnostic criteria for apathy proposed by Mulin et al. (2011, p.159)

For a diagnosis of Apathy the patient should fulfill the criteria A, B, C and D.

A—Loss of or diminished motivation in comparison to the patient's previous level of functioning and which is not consistent with his age or culture. These changes in motivation may be reported by the patient himself or by the observations of others.

B—Presence of at least one symptom in at least two of the three following domains for a period of at least four weeks and present most of the time.

Domain B1—Behaviour:

Loss of, or diminished, goal-directed behaviour as evidenced by at least one of the following:

Initiation symptom: loss of self-initiated behaviour (for example: starting conversation, doing basic tasks of day-to-day living, seeking social activities, communicating choices).

Responsiveness symptom: loss of environment-stimulated behaviour (for example: responding to conversation, participating in social activities).

Domain B2—Cognition:

Loss of, or diminished, goal-directed cognitive activity as evidenced by at least one of the following:

Initiation symptom: loss of spontaneous ideas and curiosity for routine and new events (i.e., challenging tasks, recent news, social opportunities, personal/family and social affairs).

Responsiveness symptom: loss of environment-stimulated ideas and curiosity for routine and new events (i.e., in the person's residence, neighbourhood or community).

Domain B3—Emotion:

Loss of, or diminished, emotion as evidenced by at least one of the following:

Initiation symptom: loss of spontaneous emotion, observed or self-reported (for example, subjective feeling of weak or absent emotions, or observation by others of a blunted affect).

Responsiveness symptom: loss of emotional responsiveness to positive or negative stimuli or events (for example, observer-reports of unchanging affect, or of little emotional reaction to exciting events, personal loss, serious illness, emotional-laden news).

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

D—The symptom (A–B) are not exclusively explained or due to physical disabilities (e.g. blindness and loss of hearing), to motor disabilities, to diminished level of consciousness or to the direct physiological effects of a substance (e.g. drug of abuse, a medication).

Table 2 Summary of the characteristics of the most commonly apathy measures

Apathy assessment scales	Characterisitcs
Apathy Evaluation Scale (AES) Marin et al. (1991)	The AES is a 18-item scale that measures apathy over the past 4 weeks and covers the behavioural, emotional and cognitive aspects of apathy. There are three versions of the scale: self (AES-S), informant (AES-I), caregiver (AES-C). The validity of the scale has been examined in several neurological and psychiatric disorders. The psychometric properties are not acceptable in all validation studies, notably in the TBI population.
Apathy Scale (AS) Starkstein et al. (1992)	The 14-item AS is an abridged and slightly modified version of Marin's instrument. Validation studies of the AES have been conducted in small sample sizes of patients with Parkinson's disease, patients with Alzheimer's disease as well as patients who have had a stroke. Some studies indicated that item 3 is ambiguous and should be removed from the scale to increase its internal consistency. No CFE of the AS has been conducted.
Neuropsychiatric Inventory (NPI) Cummings et al. (1994)	The apathy subscale of the NPI is an informant-based interview that assesses the presence, frequency and severity of apathetic symptoms. The evaluation is exclusively dependent on the opinion of a caregiver. The NPI-apathy subscale has been mainly validated across a range of different types of neurodegenerative disease.
Apathy Inventory (AI) Robert et al. (2002)	The AI provides a separate assessment of three dimensions of apathy (emotional blunting, lack of initiative, lack of interest). The evaluation is based on changes that have occurred since the onset of the disease. There are two versions of the scale: caregiver (AI-caregiver), patient (AI-patient). Validation of the AI has been demonstrated only by the scale's developer in a mixed sample consisting of patients with mild cognitive impairment, Alzheimer's disease, Parkinson's disease as well as healthy controls. Given the brevity of the scale, the AI seems to constitute a first clinical exploration.
Lille Apathy Rating Scale (LARS) Sockeel et al. (2006)	The LARS is an extensive 33-item questionnaire that assesses apathy in the month prior to the evaluation. The items are divided into nine domains and four subscales (intellectual curiosity, emotion, action initiation, self-awareness). Patient and caregiver versions of the LARS exist. Most responses are coded on a dichotomous scale. Validation studies of the LARS have been conducted in small sample sizes of patients with Parkinson's disease.
Frontal Systems Behavior Scale (FrSBe) Grace et al. (1999)	The FrSBe-apathy subscale consists of 14 items. Rating is based on behaviours prior to the onset of the disease and current behaviours. The scale has different versions: self, informant and clinician-rated. High internal consistency and good test-retest reliability of the family version is reported. However, several factor analysis of the FrSBe did not confirm the factor solution retained in the initial construction (i.e., apathy, disinhibition, executive dysfunction).

2001; Lampe et al. 2001; Thompson et al. 2002; McPherson et al. 2002), but not in others (Glenn et al. 2002). Indeed, Glenn et al. (2002) reported they were unable to find a cut-off score on the AES-S and AES-I with sufficiently high sensitivity and specificity with respect to a clinician's judgement of the presence of apathy in patients with TBI. In terms of factor structure, Marin et al. (1991) identified a three-factor structure using a principal component analysis of the AES on each of the rater sources (AES-S, AES-I, AES-C) in a study comprising 123 subjects with right or left hemisphere stroke, probable Alzheimer's disease, major depression as well as healthy elderly controls. Subsequent factor analytic studies of the AES have reported somewhat different factor structures (Sagen et al. 2010). For instance, Ahearn et al. (2012) reported a two-factor solution of the AES-C version in a group of 99 patients with Parkinson's disease. Taken together, further factor analyses, especially confirmatory factor analyses (CFA), are needed to more specifically examine the factor structure of the scale in different patient sample.

The 14-item *Apathy Scale* (AS) was developed by Starkstein et al. (1992) as a simplified version of Marin's instrument, and therefore has six items in common with it. The AS was validated for use in small-sample-sized studies, in patients with Parkinson's disease (Starkstein et al. 1992) and Alzheimer's disease (Starkstein et al. 2001), and in patients who have had a stroke (Starkstein et al. 1993). A recent internal consistency reliability analysis of the items from the AS revealed that they all positively correlated with the total apathy score between .4 and .7, except for item 3 ("Are you concerned about your condition?"; Kirsch-Darrow et al. 2011). This item had a negative correlation with the total score (r=−.14), and the analyses showed that deleting this item improved internal consistency reliability (i.e., Cronbach's alpha) from .831 to .855. Accordingly, Pedersen et al. (2012) showed in a group of 194 non-demented patients with early untreated PD that item 3 of the AS had a negative item-total correlation, and that removal this item raised the Cronbach's α but did not substantially alter the others psychometric properties. Besides, the authors conducted an exploratory factor analysis (EFA) that identified two factors. To the best of our knowledge, the 16 items of the AS were not submitted to a CFA.

Apathy is also assessed as a specific component of the *Neuropsychiatric Inventory* (NPI), a multidimensional instrument that was developed to quantify neurobehavioural disturbances in patients with dementia and caregiver distress caused by such behaviours (Cummings et al. 1994). The NPI is based on a structured interview with a caregiver who is familiar with the patient. A screening question is asked first, followed by subquestions if the response to the screening question suggests the presence of abnormalities involving the neuropsychiatric domain. The frequency and the severity are then explored, as well as the caregiver distress associated with the neuropsychiatric alteration. The subscale used to investigate

apathy includes questions related to a loss of interest; lack of motivation; decreased spontaneity, affection and enthusiasm; loss of emotions; and disinterest in carrying out new activities. Good internal consistency, test-retest, interrater reliabilities and convergent validity of the NPI-apathy subscale have been demonstrated in different samples, such as ambulatory patients with dementia, outpatients with Alzheimer's disease and nursing home residents (Clarke et al. 2011). Recently, EFA and CFA of the NPI scores were conducted on data collected from 491 caregivers of patients with Alzheimer's disease (Garre-Olmo et al. 2010). The CFA model that satisfactorily grouped the NPI scores into three factors included a psychotic syndrome, an affective syndrome and a behaviour syndrome. Despite the demonstrated validity of the scale, the evaluation depends only on the opinion of the caregiver, who may not always be available or reliable (Sockeel et al. 2006). Indeed, Lane-Brown and Tate (2009) emphasised that the use of informants to rate apathy has some limitations in that dimensions of apathy relate to the inherently subjective experience of internal drive and motivation. Likewise, some studies showed that apathetic patients reported more severe apathy than that reported by the patients' relatives (Muller et al. 2006; McKinlay et al. 2008). Given these issues, obtaining reports from multiple informants, both patients and significant others, is recommended (Lane-Brown and Tate 2009).

Robert et al. (2002) designed the *Apathy Inventory* (AI) as a scale for the global assessment of apathy, with separate assessments for emotional blunting, lack of initiative and lack of interest. The AI consists of two sets of clinician-led interviews, one for the caregiver and one for the patient. Based on the format of the NPI, frequency and severity are assessed during the caregiver interview, while the patient is asked to estimate intensity using a Likert-style scale (1–12). The questions deal with any behavioural changes that have occurred since the onset of the disease. Validation of the AI was carried out in a mixed sample consisting of healthy controls, patients with mild cognitive impairment, patients with Alzheimer's disease as well as patients with Parkinson's disease (Robert et al. 2002). Good internal consistency, test-retest and interrater reliabilities have been demonstrated by the scale's developer, but replication studies are needed (Clarke et al. 2011). Given the brevity of the scale, the AI seems to constitute a first clinical exploration rather than a scale that allows access to the complex aspects of apathy.

Based on the main conceptual principles proposed by Marin et al. (1991), the *Lille Apathy Rating Scale* (LARS) was developed to assess the multiple dimensions of apathetic syndrome (Sockeel et al. 2006). The scale consists of a structured interview that assesses apathy within the month prior to the time of the assessment. It includes 33 items that are divided into nine different domains. Eight of these concern the main clinical manifestations (reduction in everyday productivity, lack of interest, lack of initiative, extinction of novelty seeking and motivation, blunting of emotional responses, lack of concern and poor social life), while the ninth refers to the loss of self-awareness. With the exception of the first three questions (which are coded on a 5-point Likert-type scale), responses are coded by the clinician on a dichotomous (yes/no) scale, with an additional "NA" (not available) condition for non-classifiable answers or non-applicable items. However, the yes-no answer format may not be sensitive to change insofar as neither the premorbid condition, nor the frequency of the disturbance in everyday life nor its impact on others are taken into account. The LARS total score ranges from −36 to +36 points, with higher scores indicating greater apathy. Sockeel et al. (2006) conducted a factor analysis in a sample of 159 patients with probable Parkinson's disease that revealed a four-factor structure, labelled intellectual curiosity, emotion, action initiation and self-awareness. Internal consistency of the LARS was found to be good (α=.80) and the four-month test–retest reliability was very good (r=.95). To the best of our knowledge, the 33 items of the LARS were not submitted to a confirmatory factor analysis. An informant-rated version of the scale (LARS-i) was also developed (Dujardin et al. 2008). So far, the validity of the LARS has been assessed only in patients with Parkinson's disease (Sockeel et al. 2006; Dujardin et al. 2007; Zahodne et al. 2009) and in small-sample-sized studies, particularly for the caregiver-rated version whose validation was conducted in a sample of 60 patients with probable Parkinson's disease and their respective caregivers (Dujardin et al. 2008).

The *Frontal Systems Behaviour Scale* (FrSBe) is a 46-item scale that was designed to assess and quantify behavioural disturbances associated with damage to the frontal-subcortical brain circuits (Grace et al. 1999). The FrSBe is composed of three subscales (related to three frontal lobe behavioural syndromes, as proposed by Cummings in 1993): disinhibition, apathy and executive dysfunction. The apathy subscale consists of 14 items, each rated on a 5-point scale. Three different versions are available (self, informant and clinician). For each statement, ratings are given to indicate how often an individual engaged in a particular behaviour, both before and after the onset of an illness or injury. An EFA performed by the test developers (Stout et al. 2003) on data from a sample of 324 caregivers of patients with a "variety of neurological syndromes" (about 63 % of whom were diagnosed with a neurodegenerative disease) led to the extraction of three factors. These three factors were related to the three subscales proposed on the basis of frontal systems behavioural theory. A number of studies have reported fair to high internal consistency for the FrSBe-apathy subscales, with Cronbach's alpha coefficients ranging from 0.72 to 0.88 (Grace and Malloy 2001; Velligan et al. 2002). The exploratory analysis suggests that some revision or elimination of specific items may be warranted to refine the scale and enhance the validity of the subscales (Stout et al. 2003; Malloy and Grace 2005; Barrash

et al. 2011). Indeed, Stout et al. (2003) found significant correlations between the three factor scores indicating that the subscales are not completely independent, which is not surprising, because disinhibition, apathy and executive dysfunction partly involve the same psychological processes (e.g., executive impairments). Recently, Niemeier et al. (2013) conducted several CFA and EFA of the FrSBe in acute TBI population, across both self-report and family-report administration. The analysis failed to produce factors solutions consistent with each or that resembled the factor solution retained by Stout et al. (2003). Besides, the scale had sufficiently high internal consistency, and good test–retest reliability of the family version. The authors suggested that the FrSBe is an appropriate measure for use in an inpatient TBI population when using only the total score and the informants' ratings, although its subscales and patient administration require additional psychometric research.

In summary, apathy is still defined in a number of different ways and there exist many inconsistencies in the literature regarding the assessment of this manifestation. Indeed, several scales are currently available to assess apathy but, as seen previously, these instruments have some methodological limitations and their psychometric properties remain questionable. As highlighted by Wood et al. (2008) in a recent review that discusses issues surrounding the assessment and comprehension of behavioural disorders after acquired brain injury, a clear theoretical or conceptual basis is required to develop reliable and valid measures of neurobehavioural disability. This drives item generation beyond the level of generalities of observable behaviour and enables informed views on the origins of these manifestations. Thus, it appears that various theoretical limitations of the current approach to apathy could compromise the establishment of a valid tool to assess apathetic changes. In the next section, we discuss some of these conceptual limits.

Limitations to the Current Approach to Apathy

As reviewed earlier, most definitions acknowledge that apathy refers to a set of behavioural, cognitive and affective features. There is some agreement within the literature that disorders of interest, action initiation and emotional reactivity are all dimensions of apathy and that diminished goal-directed behaviour is at the core of the disorder. However, the exploration of apathy remains mostly descriptive. Few studies have tried to examine the mechanisms underlying each dimension of apathy, notably for the emotional and motivational dimensions (Starkstein and Leentjens 2008). Indeed, current conceptions attempt to develop diagnostic criteria for apathy that "will be easily applied in practice and research settings" (Robert et al. 2009), without taking into account the variety of mechanisms at play in the facets of apathy. These models are congruent with the descriptive approach adopted in the DSM-III (American Psychiatric Association 1980) and its successors, where disorders are made up of diagnostic categories defined descriptively in terms of symptoms that have been observed to co-vary in individuals. The phenomenon of symptom co-variation was initially expected to indicate a common aetiology, but the goal of clarifying these underlying factors has remained elusive (Kendler and First 2010). Considerable other evidence shows that the current *DSM* approach has significant limitations in validity and clinical utility (Van Praag 2000; Kendell 2002; Widiger and Trull 2007; Banzato 2008; Krueger and Bezdjian 2009; Jewell et al. 2009; Dalal and Sivakumar 2009), for example, the fact that diagnostic categories do not provide enough treatment specificity (Kupfer et al. 2002; Skodol and Bender 2009). Precise identification of the underlying mechanisms is much needed to ensure targeted and effective rehabilitation insofar as some apathy traits result in similar overt behaviours but their aetiologies may be heterogeneous.

Based on a categorical approach, the recent diagnostic criteria for apathy proposed by Mulin et al. (2011) seem arguable in some aspects. As indicated previously, the core feature of these criteria is a loss of motivation that persists over time (4 weeks or more), with the presence of at least one symptom in at least two of the three following domains being required: (1) loss of, or diminished, goal-directed behaviour; (2) loss of, or diminished, goal-directed cognitive activity; and (3) loss of, or diminished, emotions. Although initiative and interest can theoretically be dissociated, it is quite uncommon to find this distinction in clinical practice. Indeed, a lack of interest is closely related to a lack of initiative and, on the other hand, a lack of initiated actions may gradually lead to a lack of interest. For instance, people with a loss of interest in their leisure activities will in most cases end up initiating few actions directed to these hobbies; conversely, people with difficulties in initiating actions directed to their leisure activities are likely to become detached and disinterested from these activities. Accordingly, Sockeel et al. (2006) showed a significant correlation (r=.56, p<.01) between the dimensions "intellectual curiosity" and "action initiation" in the LARS. In this context, it would be interesting to examine the relationships between a lack of initiative and a lack of interest, using the IA that evaluates separately these dimensions. Consequently, the establishment of diagnostic criteria based on these dimensions of apathy seems largely debatable insofar as a loss of goal-directed behaviour and a loss of goal-directed cognitive activity are strongly interrelated. Furthermore, Mulin et al. (2011) postulated that change in motivation can be measured by examining a patient's responsiveness to internal and external stimuli. For example, a "loss of spontaneous emotion" is dissociated from a "loss of emotional responsiveness to positive and negative stimuli or events". This distinction, particularly the notion of "spontaneous emotion", is contrary to most

theories of emotion (Frijda 1986; Lazarus 1999; Scherer 2001; Ellsworth and Scherer 2003) that define emotions as an affective state triggered by or focused on an event or a situation. More precisely, appraisal theories of emotion support the view that the elicitation and the differentiation of emotions are mainly determined by cognitive appraisals of an event such as its relevance or implications for the person (Scherer 2001; Sander et al. 2005). Consequently, it seems unlikely and theoretically restricted to consider emotions as "spontaneous".

A typical example of the ambiguities related to the categorical approach of apathy concerns the relationships between apathy and depression. Some studies have reported that apathy can occur independently from depression in neurological disease (Marin et al. 1993; Kant et al. 1998; Levy et al. 1998; Kirsch-Darrow et al. 2006). Although apathy can occur in the absence of depression, however, most studies show that a considerable proportion of patients exhibit both apathy and depression (Andersson et al. 1999b; Starkstein et al. 2006). This is not surprising, since apathy and depression share many common features, as evidenced by their definition criteria. On the one hand, the diagnosis of a major depressive episode requires either a depressed mood or a loss of interest or pleasure (according to the *DSM-IV-TR*, 2000), and on the other hand, the diagnosis of apathy requires a loss of motivation in at least two of the following three dimensions (according to Mulin's criteria for apathy, 2011): reduced goal-directed behaviour, goal-directed cognitive activity and emotions. Thus, a loss of interest or pleasure and a loss of goal-directed cognitive activity or emotions undoubtedly overlap. In this sense, the recent confirmatory factor analysis of individual items from the AS (Starkstein et al. 1992) and the *Beck Depression Inventory-II* (BDI-II, Beck et al. 1996) conducted by Kirsch-Darrow et al. (2011) in patients with Parkinson's disease supports the evidence that a loss of interest and of pleasure are common features to both apathy and depression. More precisely, the analysis revealed a four-factor structure: (1) an apathy factor representing a loss of motivation, (2) a dysphoric mood factor representing sadness and negativity, (3) a loss of interest/pleasure factor representing the features common to both apathy and depression and (4) a somatic factor representing bodily complaints. Furthermore, as we will see later, specific criteria for depression such as a negative mood (Ready et al. 2003) could contribute to the presence of apathetic manifestations. Indeed, replicated evidence shows that moods have congruent effects on motivation and behaviour in that people who face a challenge judge task difficulty higher in a negative mood than in a positive mood, which results in disengagement when the task is objectively difficult (for reviews, see Gendolla and Brinkmann 2005; Richter et al. 2006). More generally, it would be necessary to examine precisely the overlaps and differences between apathy and depression rather than considering these entities as separate.

Further, apathy is better viewed as a continuum or spectrum ranging from "mild" to "severe" impairments, rather than a simple categorical disorder that places people into two categories, those who are "apathetic" and those who are "not apathetic". The categorical approach seems unable to capture the diversity and complexity of the apathetic manifestations. Indeed, in a recent comprehensive review of published taxometric research that included 177 articles, Haslam et al. (2012) showed that most psychological variation is dimensional, with a few potentially important exceptions. More specifically, the data strongly suggested that taxa are unlikely to be found in the domains of normal personality and personality disorders within the internalizing spectrum, such as mood, anxiety and eating disorders, or in the general externalizing domain. The only domains in which taxonic findings were at least somewhat prevalent were schizotypy and substance use disorders. Thus, the analysis of the taxometric literature supports the view that most psychological variation is dimensional and that many influential taxonic findings of early taxometric research are likely to be spurious. Furthermore, it is increasingly recognised that behavioural disturbances are determined by several factors (Riggio and Wong 2009). In particular, Kendler et al. (2010) argue that psychiatric disorders—depression, anxiety and other psychopathological states—are not defined in terms of essences, but in terms of complex, mutually reinforcing networks of causal mechanisms. From this view, Cramer et al. (2012) investigated the prediction of a common cause framework by comparing the impact of four stressful life events on disaggregated depressive symptoms in twins with a dysphoric episode. The results showed that stressful life events influence the correlations between depressive symptoms in marked ways; these differences were significant and could not be explained by underlying differences in acute liability to develop a dysphoric episode. Instead, the results were better explained by a network perspective, in which there is no common cause and where the symptoms and direct (causal) relations between them are the causes of psychiatric disorders. These results are consistent with the strong inter-relationship discussed previously between apathetic symptoms, particularly between a lack of initiated actions (or lack of goal-directed behaviours) and a lack of interest (or lack of goal-directed thoughts). In the same way, it could be argue that emotional blunting results in a lack of interest and inversely, a lack of interest could lead to a lack of emotional reactivity. Thus, direct relationships at the level of the symptoms themselves could be involved in the occurrence of apathetic manifestations. Interestingly, Kendler et al. (2010) have specified that psychiatric disorders result from more or less stable patterns of complex interaction between neural systems, psychological states, environmental inputs and socio-cultural variables. In the same way, apathy seems associated with various psychological dysfunctions, some directly related to brain damage, and others more related to psychological reactions (e.g.,

negative mood) or to personal identity (e.g., self-esteem). More specifically, biological factors, social factors and life events could contribute to apathetic manifestations through their joint effects on various psychological processes such as cognitive, affective, motivational and relational processes. Thus, it appears obvious that a multifactorial and integrated approach, which incorporates mechanisms at multiple levels, is clearly required to gain a better understanding of apathy.

Apathy after TBI

TBI is characterised by a wide range of neurobehavioural changes, representing a major source of disability and handicap (Levin 1995; Kendall and Terry 1996). These disturbances are often perceived as most disabling for patients in the long term as they attempt to reintegrate into family and community life (Ownsworth and McKenna 2004; Kelly et al. 2008). Neurobehavioural disturbances have an important impact on social integration and often lead to more frequent and intensive consultations with health care centres. Recently, Ciurli et al. (2011) sought to characterise neurobehavioural changes among a group of 120 individuals with severe TBI. Using the NPI (Cummings et al. 1994), the authors found that family caregivers reported a wide range of neuropsychiatric symptoms, such as apathy (42 %), irritability (37 %), dysphoria/depressed mood (29 %), disinhibition (28 %), eating disturbances (27 %) and agitation/aggression (24 %). Apathy was commonly described among the TBI population (Andersson and Bergedalen 2002; Lane-Brown and Tate 2009) with serious consequences, notably for patients' participation in rehabilitation (Gray et al. 1994; Kant et al. 1998), family life (Marsh et al. 1998) and later social reintegration (Mazaux et al. 1997). Despite its frequent occurrence and its negative impact on patients' functioning, apathy is commonly noted but rarely investigated among the TBI population.

Prevalence

Apathy was studied in the TBI population in nine studies that used different scales such as the NPI, the FrSBe and, in particular, the AES (see Table 3). Only those studies were selected whose main objective was to measure the prevalence of apathy or to characterize neuropsychiatric disorders following TBI and in which the scores or percentages of apathy were specified. Therefore, a total of 554 patients were assessed and 265 described as apathetic, with an average prevalence rate of 47.83 % (265 of 554). The lowest prevalence rate found in the studies was 20 % (Al-Adawi et al. 2004) and the highest was 72 % (Lane-Brown and Tate 2009). These findings indicate that apathy is a frequent symptom following TBI, but also highlight the significant variation in prevalence rates, probably related to differences in the definition of apathy and the assessment tools. Indeed, apathy is still described and assessed in a number of different ways, with no instrument specially developed or thoroughly validated for the TBI population (Lane-Brown and Tate 2009).

Although one might expect to find greater apathy among those with severe TBI, most studies do not report any correlation between apathy and the severity of the brain injury, as assessed by coma length, duration of post-traumatic amnesia or the Glasgow Coma Scale (Van Zomeren and Van den Burg 1985; Andersson et al. 1999a; Glenn et al. 2002; Andersson and Bergedalen 2002). Age and education were also found to have no significant association with apathy among the TBI population (Van Reekum et al. 2005; Andersson and Bergedalen 2002). Some studies indicate that apathy is more frequent or visible in the chronic phase than in the subacute stage (Thomsen 1984; Van Zomeren and Van den Burg 1985; Kelly et al. 2008). However, other studies found no significant correlation between time since injury and apathy score (Andersson et al. 1999a; Andersson and Bergedalen 2002; Lane-Brown and Tate 2009). Kant et al. (1998) reported some contrasting data from these studies, namely, that younger patients were more likely to be apathetic than older patients who were often both depressed and apathetic, but that patients with severe injury were more likely to exhibit apathy alone.

Neuroanatomical Correlates of Apathy and TBI

Recently, Knuston et al. (2013) investigated the neural basis of apathy in patients from the Vietnam Head Injury Study (VHIS), an ongoing inpatient follow-up study of the long-term sequelae of penetrating TBI (Raymont et al. 2011). The results revealed that increased apathy symptoms were associated with brain damage to specific areas including the left middle, superior and inferior frontal regions, insula, supplementary motor area, anterior cingulate cortex (ACC) as well as lesions to the white matter tracts in the corona radiata and the corpus callosum. Because of its various findings, this study will be used as a common thread for the following presentation on the neuroanatomical correlates of apathy in the TBI population. It is of note that some data from other neurological disorders (e.g., neurodegenerative diseases, stroke or HIV) will be included in order to allow a broader and more comprehensive view of the brain networks involved in apathy.

Thus, as reported above, Knuston et al. (2013) showed that apathy is associated with brain damage to frontal areas, in particular the left middle, superior and inferior frontal regions (including the operculum and pars triangularis). Other studies conducted in patients with TBI found a specific association between apathy and different prefrontal cortex regions. For instance, Sarazin et al. (2003) reported a significant relationship between apathy and decreased regional glucose metabolism in the right medial area 10 among a group of patients with

Table 3 Apathy prevalence rates in studies involving subjects with TBI

Author	Sample size	Injury severity	Time since injury (months)	Assessment instrument	Percentage of subjects with apathy
Van Zomeren and Van den Burg (1985)	57	Severe	24	Personal	23 %
Kant et al. (1998)	83	62 mild, 8 moderate, 9 severe	?	AES-S & AES-I (cut-off >34)	71.08 % (AES-S)
Marsh et al. (1998)	69	Severe	12.9 (±1.1)	Head Injury Behaviour Rating Scale (relative version)	Lack of motivation: 54 %, lack of initiative: 42 %
Andersson et al. (1999a)	30	Severe	10.5 (±1.68)	AES-C (cut-off >34)	66.7 %
Andersson et al. (1999b)	28	?	12.6 (±10.99)	AES-C (cut-off >34)	46.4 %
Andersson and Bergedalen (2002)	53	Severe	12.2 (±10.06)	AES-C (cut-off >34)	62.3 %
Al-Adawi et al. (2004)	80	6 mild, 2 moderate, 36 severe	8.35 (±4.50)	AES-S Arabic language version (cut-off >34)	20 %
Lane-Brown and Tate (2009)	34	Severe	80.58 (±71.64)	AES-I (cut-off >37) and FrSBe-A	AES-I: 69 % FrSBe-A: 72 %
Ciurli et al. (2011)	120	Severe	10.6 (±15.1)	Neuropsychiatric Inventory	42 %

AES-C Apathy Evaluation Scale-Clinician version, *AES-I* Apathy Evaluation Scale-Informant version, *AES-S* Apathy Evaluation Scale-Self report version, *FrSBe-A* Frontal Systems Behaviour Scale-Apathy sub-scale, *TBI* traumatic brain injury.

frontal lobe damage including traumatic lesions. These findings are consistent with subsequent group studies of patients with damage to the ventromedial prefrontal cortex (vmPFC) (including the orbitofrontal) that have identified typical behavioural changes such as impaired goal-directed behaviour and blunted affect (Barrash et al. 2000). More specifically, several studies have shown that ventral and medial sectors of the prefrontal cortex have been mainly associated with valuation, reward learning, emotion regulation and decision-making (Bechara et al. 1994; Fellows and Farah 2005a; Glascher et al. 2012). Due to its crucial role in these psychological processes, and specifically in the ability to use emotion to guide behaviour (see the role of the vmPFC in decision-making and goal-directed activity, Damasio 1994), the vmPFC could have a role to play in the occurrence of apathetic manifestations in patients with TBI.

Furthermore, several studies have reported a specific association between apathy and other prefrontal cortex regions such as the lateral prefrontal cortex or the ACC. Indeed, Paradiso et al. (1999) showed that patients with lateral prefrontal damage had a greater reduction of motivation than patients with medial prefrontal damage in a mixed population of patients with frontal lobe pathology (both TBI and acute stroke). In this sense, another research conducted in patients with stroke showed a significant decreased blood flow in the right dorsal lateral frontal and left fronto-temporal cortices in patients with apathy compared to non-apathetic stroke controls (Okada et al. 1997). These results point in the same direction as accumulating evidence that demonstrates a key role of the lateral prefrontal cortex in the executive control, allowing in particular people to coordinate their thoughts and actions with their intentions to support goal-directed social behaviours (Raymont et al. 2011). Besides, several studies reported that damage to the ACC is associated with apathy both after a TBI (Knuston et al. 2013), or in the course of a neurodegenerative disease (Benoit et al. 1999; Migneco et al. 2001; Zamboni et al. 2008; Massimo et al. 2009). More specifically, it is argued that the ACC supports the selection and maintenance of goal-directed behaviours, through its contribution to cognitive control and reinforcement (Holroyd and Yeung 2012). In addition, the ACC is thought to determine the level of effort required to achieve the goal.

Another cortical structure, namely the insula, has been associated with apathy in the TBI population (Knuston et al. 2013). Damage to insula could lead to reduced motivated behaviour, due to a lack of awareness of emotional and motivational states. Indeed, several findings in the literature have demonstrated the role of the insula in emotion, attention, cognitive control, intentions, awareness of body states and time perception (Damasio 1994; Craig 2009). A specific association between insula and apathy was also reported in patients with stroke or neurodegenerative disorders (Manes et al. 1999; Robert et al. 2012). For instance, Manes et al. (1999) showed that patients with right insular infarction had a significantly higher frequency of subjective anergia, underactivity and tiredness than patients with either left insular lesions or non-insular lesions. In this context, it has been proposed that the anterior insula, through its intimate connections with amygdala, hypothalamus, ACC and orbitofrontal cortex, serves to compute high-order meta-representation of the primary interoceptive activity, which is related to the feeling of pain and its emotional awareness (Craig 2007; Decety 2011). These representations play an important role in the learning and adaptation of prosocial behaviours (see the role of the insula in empathy feeling for other's emotions, Adolphs 2009), and they may guide decision-making and

homeostatic regulation (Singer et al. 2009). It is of note that several studies have reported that the insula is vulnerable to TBI (Bendlin et al. 2008; Dal Monte et al. 2012).

Some studies conducted in patients with TBI have highlighted the role of subcortical areas, particularly the basal ganglia, in the mechanism of apathy (Grunsfeld and Login 2006; Spalletta et al. 2012). It is of note that hemorrhagic lesions occurring in the basal ganglia are frequently reported in patients who have sustained a TBI (Xu et al. 2007; Shah et al. 2012). A study by Finset and Andersson (2000) showed in a mixed population of patients with acquired brain damage including TBI that apathetic manifestations were most severely present in patients with subcortical lesions. In addition, Grunsfeld and Login (2006) reported an interesting case study of abulia in a patient who suffered penetrating brain injury during endoscopic sinus surgery causing right basal ganglia injury. Apathetic manifestations, lack of motivation and initiative were the changes that dramatically stood out after the surgery. According to the authors, the patient illustrates the consequence of disruption of fronto-subcortical circuits at the level of the basal ganglia. Specifically, the anterior cingulate circuit is reported to be essential for the initiation of behaviour, motivation and goal-directed orientation. Moreover, apathy is commonly experienced by patients with neurodegenerative diseases that affect the basal ganglia, such as progressive supranuclear palsy (Aarsland et al. 2001), Huntington's disease (Hamilton et al. 2003) and Parkinson's disease (Pluck and Brown 2002). Basal ganglia probably constitute a key network for motivation processes that enables expected rewards to energize behaviour without the need for the participants' awareness (Pessiglione et al. 2007; Schmidt et al. 2008). More specifically, several contributors hypothesized that apathy may at least partially result from fronto-basal ganglia dysfunction, a network involved in the generation and control of self-generated purposeful behaviours (Levy and Dubois 2006).

Another aspect to consider in the neural basis of apathy is the white matter tracts, which might induce disconnections between cortical brain regions. Indeed, Knuston et al. (2013) showed that apathy in penetrating TBI was significantly associated with several areas of white matter, that is, the superior and anterior corona radiata and the genus and body of the corpus callosum. Several studies conducted in patient with HIV or with Alzheimer's disease have reported a significant relationship between apathy and white matter damage, particularly in the corpus callosum (Hoare et al. 2010; Hahn et al. 2013). Given that damage to the white matter tracts is common following TBI, it would be interesting to explore the relationships between white matter regions and the various dimensions of apathy.

Interestingly, emerging findings conducted in patients with chronic traumatic encephalopathy (CTE) reported that apathetic manifestations are one of the most common neurobehavioural changes following repetitive mild TBI (Baugh et al. 2012). Recently, McKee et al. (2013) analyzed the brains of 85 individuals with a history of repetitive mild TBI and found evidence of CTE in 68 subjects. Apathetic manifestations were reported in patients with CTE, especially in those with a moderate to severe p-tau pathology. To be more precise, six of the 13 (46 %) patients with severe p-tau pathology, involving widespread regions of the neuraxis including white matter with prominent neuronal loss, gliosis of the cerebral cortex and hippocampal sclerosis, were reported as apathetic by the family. Thus, it seems that repetitive TBI and axonal injury might trigger molecular pathways that result in the overproduction and aggregation of other proteins which could be involved in the development of apathy following repetitive TBI. In this sense, multiple epidemiological studies have shown that trauma is a risk for dementia (Shively et al. 2012), especially Alzheimer's disease, which is frequently associated with apathetic manifestations (Fernandez Martinez et al. 2008). Nevertheless, very few studies have to date specifically investigated the apathetic manifestations as well as their neural basis in the CTE population. Consequently, the existing findings must be interpreted with caution and further studies are needed to examine the neurodegenerative changes and their relationships with apathetic manifestations in patients with repetitive TBI.

As reviewed above, cortical regions involved in the limbic system such as the insular cortex or the orbitofrontal cortex have been related to apathetic manifestations in patients with TBI. Furthermore, several studies in the literature have clearly pointed to the role of subcortical regions of the limbic system, particularly the amygdala, in the pathophysiology of apathy. Indeed, diminished amygdala activity is reported to cause abnormal emotional responses resulting in apathy, fearlessness and/or aggressiveness (Mesulam 2000). More specifically, the amygdala is considered as a system for relevance detection, i.e., necessary for detecting the extent to which stimulus events are relevant for the current needs, goals and values of the individual (Sander et al. 2003; Ousdal et al. 2008). In addition, numerous studies showed that patients with amygdala damage were impaired on decision-making tasks and failed to generate emotional responses when they received a reward or a punishment (Bechara et al. 1999; Weller et al. 2007). In this context, Adolphs (2003) specified that amygdala is one of a set of structures that mediate the association of perceptual representations of social signals with emotional response, cognitive processing and behavioural motivation. Thus, damage to the amygdala can have an impact on the outcomes of goal-directed behaviours due to a disrupted ability to assess the significance of social and emotional signals. Besides, some studies have showed that the amygdala, as well as the vmPFC, is activated when people imagine positive future goals and hopes (Johnson et al. 2006; Sharot et al. 2007). Diminished activity in these regions may

underpin the long-term planning and foreshortened future time perspective seen in apathetic individuals (Fellows and Farah 2005b; Lawrence et al. 2011). Taken together, lesions to the amygdala could lead to apathetic symptoms in patients with TBI due a disruption in emotion, social behaviour and reward processes. It is of note that damage to the amygdala is reported in the TBI population as illustrated by the study of Warner et al. (2010) which showed in patients with traumatic axonal injury that the highest rates of atrophy among subcortical structures were noted bilaterally in the amygdala.

In summary, the findings reviewed above indicate that brain damage in cortical and subcortical areas, including the lateral prefrontal cortex, the vmPFC, the ACC, the basal ganglia, the insula as well as the white matter tracts connecting these regions, are significantly related to apathetic manifestations following TBI (see Fig. 1). These data suggest that an extensive brain network is involved in apathetic manifestations, which supports the hypothesis that apathy is not a single entity but rather multiple (Stuss et al. 2000; Levy and Dubois 2006). In this context, it is essential to better understand the contribution of the various components of this cerebral network in the occurrence of apathetic manifestations as well as the complex interactions between these components. More specifically, additional studies are needed to identify the diverse vulnerable cerebral structures to TBI that could have an impact on the integrity or functioning of this complex network and to examine their links with apathetic manifestations. It will be particularly interesting to conduct these studies in patients with closed TBI that may have white matter shearing and pathology, which is more extensive than lesions in penetrating TBI. Promising techniques for quantifying diffuse injury such as diffusion tensor imaging may be useful for characterizing microstructural brain injury contributing to regional white matter loss in the TBI population.

Apathy and Other Neurobehavioural Disorders

Since there is considerable overlap in the clinical presentation of apathy and depression, some studies have investigated the co-occurrence of these disorders in patients with TBI (Kant et al. 1998; Andersson et al. 1999b; Finset and Andersson 2000; Glenn et al. 2002; Lane-Brown and Tate 2009; Ciurli et al. 2011). For instance, Kant et al. (1998) reported that 10.84 % of TBI patients seen consecutively in a neuropsychiatric clinic were apathetic but not depressed, while 60 % were both apathetic and depressed. In most studies, apathy is more common in TBI patients diagnosed with depression than in those without depression (Andersson et al. 1999b), which underlines, as seen previously, the important overlaps between apathy and depression. In addition, Glenn et al. (2002) found a moderate correlation between apathy (AES) and depression (BDI) scores, arguing that it may not be possible to distinguish between neurologically based apathy and apathy caused by depression within the TBI population.

Furthermore, apathy is frequently reported with other neurobehavioural syndromes in persons with TBI, such as impulsive behaviours (Ciurli et al. 2011; McAllister 2008). In 1990, Marin stressed the apparent contradiction of diagnosing apathy and disinhibition in the same patient. Some studies have demonstrated a significant correlation between apathy and disinhibition, but only in patients with dementia (Levy et al. 1998; Starkstein et al. 2004). Indeed, no studies in the literature have examined the relationships between apathy and impulsive behaviours in patients with TBI. Recently, Rochat et al. (2010) explored the nature of impulsivity changes after moderate to severe TBI by using a multidimensional approach (Whiteside and Lynam 2001). Urgency (tendency to experience strong reactions, frequently under conditions of negative affects), lack of perseverance (inability to

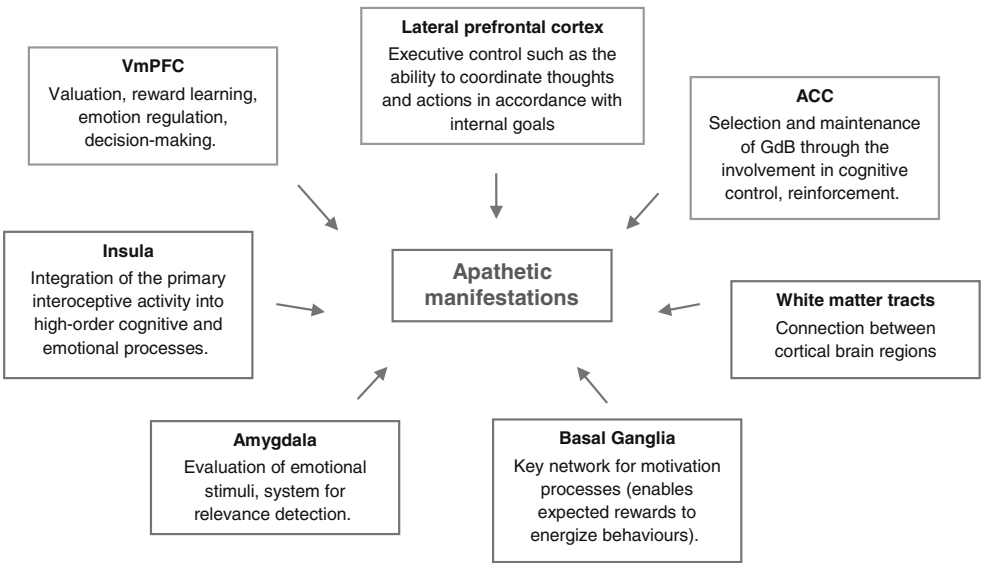

Fig. 1 The main neuroanatomical correlates of apathy (and their related psychological processes) in individuals with TBI

remain focused on a task that may be boring or difficult) and lack of premeditation (inability to anticipate the future consequences of actions) significantly increased after TBI, whereas sensation seeking (tendency to enjoy and pursue activities that are exciting and openness to trying new experiences) decreased. According to the authors, the decrease observed in the sensation seeking dimension could be due to apathy and the associated motivational changes. Similarly, the lack of perseverance reported in patients with TBI could depend on motivational processes in that a decreased interest in activities and/or goals may lead to poor mobilization of cognitive resources required to complete complex tasks and/or goals.

Mechanisms Associated with Apathy after TBI

A few studies have sought to investigate the mechanisms underlying apathy in patients with TBI. Andersson and Bergedalen (2002) showed that apathy in the TBI population was strongly linked to specific cognitive deficits, but relatively unrelated to global intellectual efficiency. The authors emphasised that patients are often perceived as more cognitively impaired than they actually are, probably because lack of motivation and reduced self-initiated actions tend to camouflage cognitive resources. In line with these findings, a recent study reported that subjects with TBI demonstrated significantly reduced goal-directed behaviours, diminished motivation and a general lack of dynamism in comparison with participants with mild mental retardation, despite having consistently greater intellectual capacities (Cattelani et al. 2008). Andersson and Bergedalen (2002) showed that specific cognitive deficits related to frontal lobe dysfunction were associated with apathy; these included memory in terms of acquisition and recall (as assessed by the California Verbal Learning Test), executive functions (as assessed by the Wisconsin Card Sorting Test) and psychomotor speed (as assessed by the Trail Making Test). Several authors argued that ecological tests are more sensitive to apathy than traditional executive measures that essentially focus on isolated executive aspects without considering the demands of the real world that involve multiple processes and self-initiated behaviours (Reid-Arndt et al. 2007; Esposito et al. 2010). To be more precise, Muller et al. (2006) showed that apathetic TBI patients had significantly reduced performance on the Behavioural Assessment of Dysexecutive Syndrome battery in comparison with non-apathetic TBI patients. In addition, these results are in accordance with the findings of Esposito et al. (2010) in patients with Alzheimer disease. The authors reported that the number of rule breaks on the Modified Six Elements Task (Wilson et al. 1996) was a significant predictor of apathy, as assessed by the AI (Robert et al. 2009) and of lack of initiative in particular.

Furthermore, apathy has been associated with some emotional and motivational changes in TBI patients, in particular those assessed with cardiovascular measures. Andersson et al. (1999b) showed a significant relationship between apathy and heart rate hyporeactivity, during a performance of the Raven's Progressive Matrices task and an arithmetic task, in 72 persons with a TBI, stroke or hypoxic brain injury. In another study, in contrast to non-apathetic TBI patients, apathetic TBI patients did not display autonomic reactivity when communicating their traumatic event, illustrating the emotional blunting and lack of emotional responsivity associated with apathy (Andersson et al. 1999a). This is further supported by the subjective ratings of emotional discomfort, as apathetic TBI patients find therapeutic interaction significantly less emotionally disturbing than TBI patients without apathy. According to Andersson et al. (1999a), the correlation between heart rate hyporeactivity and the level of apathy should be understood in the context of dysfunctional motivational and rewards systems. Indeed, enhancing motivation by adding, for example, monetary incentives, has been shown to increase cardiovascular reactivity in response to both social and mental stress situations. In this context, apathy is also associated with altered responses to novel stimuli in the TBI population, as it correlates strongly with the reduction in hit scores for novel stimuli on a unimanual two-choice response time test (Godefroy and Rousseaux 1997).

In summary, few attempts have been made to examine the mechanisms underlying apathy in patients with TBI. However, the existing data support the argument that a variety of mechanisms are at play in various facets of apathy after TBI or more generally in persons with brain damage. In the following section, we propose a multidimensional framework that takes into account the mechanisms at play in the various dimensions of apathy.

Towards a Multidimensional Approach to Apathy

As noted previously, the exploration of apathy is to date mostly descriptive, i.e. only based on the identification of the various symptoms of apathy. However, most definitions acknowledge that these symptoms are related to cognitive, affective or behavioural dimensions but the precise identification of the psychological processes underlying each dimension has remained relatively unexplored. Such an approach could contribute to a better understanding of both socio-emotional disorders following brain damage and the cerebral and psychological components of apathy. Moreover, such an approach is needed to ensure targeted and effective rehabilitation insofar as the same apathetic symptom could have different etiologies. For instance, a lack of initiative could be due to a dysfunction in executive processes such as in multitasking as showed by Esposito et al. (2010) in patients with Alzheimer disease or to a negative mood which is reported to have a congruent effect on motivation through its effect on

effort mobilization. In this context, we propose a new multidimensional framework that assume four dimensions of apathy (cognitive, affective, motivational and related to personal identity), which are defined by a precise identification of the various mechanisms potentially involved.

Cognitive Dimension

Apathy is commonly related to disruption of cognitive processing, but the specific processes involved are still unclear. According to Levy and Dubois (2006), the quantitative reduction of goal-directed behaviour could be due to impairments in several executive functions that are needed to elaborate a plan of action, such as impairments in planning, set shifting and rule finding. The basic executive functions, inhibition, shifting, updating, as defined in the model by Miyake et al. (2000), also seem to be involved. More precisely, the inability to inhibit prepotent responses could compromise the elaboration of action plans that are suited to environmental requirements. Individuals with set-shifting impairments might have difficulties in finding alternative ways to resolve a complex situation, which might decrease initiation towards goal attainment. Furthermore, a deficit in updating and monitoring the contents of working memory could affect the achievement of goal-directed behaviours insofar as the relevant information would not be updated and thus not taken into account in the action plans. Various neuropsychological tests, that are sensitive to cognitive impairments following TBI, could be used to evaluate these basic executive functions such as the Hayling sentence completion test (Burgess and Shallice 1997), the Trail Making Test (Reitan and Wolfson 1988) or the n-back task (Miyake et al. 2000; Owen et al. 2005). Another cognitive process that might be relevant for better comprehension of apathy is sustained attention. Some studies suggest that the variability of reaction times results from difficulties in maintaining or sustaining attention on task goals across time due to executive dysfunction (West et al. 2002; Duchek et al. 2009). Rochat et al. (2013) have demonstrated that larger variability in reaction times constitutes a significant predictor of lack of perseverance (inability to remain focused on a task that may be boring or difficult) in older adults with or without a diagnosis of Alzheimer's disease. In this study, sustained attention was evaluated using the Sustained Attention to Response Task (Robertson et al. 1997), in which the participants must withhold a response to infrequent and unpredictable stimuli (targets) during a period of rapid and rhythmic responding to frequent stimuli (nontargets). Several studies have demonstrated that this task provides a valid measure of sustained attention (Dockree et al. 2004; Manly et al. 2003) and is sensitive to deficits after TBI (McAvinue et al. 2005). Taken together, attentional difficulties could contribute to apathy, especially to loss of interest, due to progressive attentional disengagement that compromises the achievement of a task.

Furthermore, the recent findings of Esposito et al. (2010) in patients diagnosed with Alzheimer's disease suggested that mechanisms underlying multitasking, such as coordination between internal and external information, planning and prospective memory, constitute a key component of goal-directed behaviours. Indeed, Burgess et al. (2005) hypothesised that a control system, "the supervisory attentional gateway system", should play a key role in the ability to flexibly allocate attention towards either internal representations (stimulus-independent thought, i.e., goals, action plans, emotion, etc.) or external information (stimulus-oriented thought, i.e., information provided by the environment). This cognitive control mechanism, which relies mainly on the activity of the rostral prefrontal cortex (Brodmann's area, BA 10), may support a wide range of situations critical to competent human behaviour in everyday life, such as multitasking or remembering to carry out intended actions after a delay (Burgess et al. 2005). Thus, a disturbance that specifically affects the flexible allocation of attention towards internal representations and external information could contribute to the presence of apathetic manifestations, and particularly to lack of initiative, by making the person incapable of coordinating intentions and environmental stimuli. The ability to flexibly allocate attention between internal and external information could be assessed with a task recently developed by Gilbert et al. (2005), in which participants have to process stimuli in two alternating experimental phases: a stimulus-oriented thought phase (cognitive processes provoked by incoming sensory information) and a stimulus independent thought phase (cognitive processes that are not related to any information in the immediate sensory environment). Thus, future studies should explore the integrity of the flexible allocation of attention between internal and external information in the TBI population and their relationships with apathetic manifestations.

Moreover, prospective memory also appears to be linked to self-initiated behaviours insofar as it requires the occurrence of the appropriate event or time to activate the memory of the intention, without external solicitations, and to prompt the person to initiate an action (Einstein et al. 1995). In terms of assessment, two broad classes of prospective memory tasks are distinguished (McDaniel and Einstein 2007): (1) the time-based task requires that the person perform a specific task or action at a certain time; (2) the event-based task requires that the person perform an action when some external event occurs. Time-based task is reported to involve a high degree of controlled and self-initiated processes (Einstein and McDaniel 1990). As impairment in multitasking is frequently reported in patients with TBI (Rochat et al. 2009), it would be interesting to explore multitasking components and their relationships with the different facets of apathy within this population. Multitasking could be assessed with the Modified Six Elements Test (SET), which is a subtest of the Behavioural Assessment of the Dysexecutive Syndrome (Wilson et al.

1996). As seen previously, this open-ended task has been shown to significantly predict apathy (lack of initiative in particular) in patients with Alzheimer's disease (Esposito et al. 2010).

Motivational Dimension

The disruption of motivational processing, such as the inability to associate affective and emotional signals with ongoing and forthcoming behaviours, may also lead to apathy (Levy and Dubois 2006). To be more precise, processes related to positive reinforcement could play an important role in the motivational aspects of apathy, notably by influencing anticipation of pleasure and/or sensation seeking (Billieux et al. 2008). Favrod et al. (2009) showed a significant correlation between anticipatory pleasure (capacity to feel pleasure about future situations) and lack of initiative in persons with schizophrenia. Indeed, we suggest that a deficit in anticipatory pleasure may explain a decline in initiative because of a reward system dysfunction and the inability to project oneself into future pleasant situations.

As regards the reward system, some authors argue that "wanting" (i.e., motivational incentive value of reward) and "liking" (i.e., the hedonic value of reward) are states of mind that can both be present before receipt of a reward and that can concurrently influence reward seeking (Berridge and Robinson 1998; Leknes and Tracey 2010). The important point is that "wanting" and "liking" are needed together for a full reward, but they can be split apart under certain circumstances, especially by certain brain manipulations or damage. For example, dopamine suppression leaves individuals virtually without motivation for any pleasant incentive, but the hedonic impact of the same incentives remains intact (Berridge 2004). In this context, Vijayaraghavan et al. (2008) investigated the mechanisms underlying motivational changes in a patient (PJ) who developed complete bilateral damage to the globus pallidus after a prolonged state of cerebral anoxia. Marked reduction in spontaneous speech, movement, emotional expression and motivation ensued after the lesion. PJ and 30 male controls performed a task designed to parse hedonic evaluation from incentive motivation. In this task, emotionally laden visual stimuli are presented to the subject, who has the option to increase or decrease the time to view the images. The behavioural measure of wanting corresponds to the amount of work done by the subject to change the viewing time. Liking is measured by the subject's evaluation of pleasantness of the stimuli. The results indicated that, compared with healthy controls, PJ showed normal ratings of hedonic appreciation in response to pleasant stimuli, but significantly reduced viewing time of these stimuli. The investigators concluded that active withdrawal from liked stimuli could constitute the core mechanism underlying the motivational disorder associated with globus pallidus damage.

The findings of Schmidt et al. (2008) showed that the process of translating expected rewards into behavioural activation was specifically impaired in patients with an auto-activation deficit (AAD) induced by bilateral striato-pallidal lesions. Indeed, Schmidt et al. (2008) designed an original behavioural paradigm dissociating an instructed (externally driven) task, in which subjects are explicitly instructed about how hard they must squeeze a hand grip, from an incentive (self-driven) task, in which subjects are free to squeeze the hand grip as they wish, but are aware that the higher the force exerted, the larger the monetary payoff. Skin conductance was simultaneously measured to index affective evaluation of monetary incentives. Patients with AAD showed correct grip force response to external instructions and skin conductance response to monetary incentives. However, they failed to modulate their force in accordance to monetary incentives. In other words, AAD patients assigned adapted affective values to potential rewards, but failed to integrate these values into their physical effort. Thus, dysfunction in incentive motivation, the process that translates an expected reward (or goal) into behavioural activation, seems to be involved in apathetic manifestations. Further studies are required to precisely investigate the nature of motivational changes related to dysfunction of the reward system (and their major components) and their links with the different facets of apathy.

Another aspect to consider in the motivational dimension of apathy is the intensity of motivation, which can be defined as the amount of effort mobilized to attain a goal. Effort has been considered as a central aspect of goal-directed behaviour in all comprehensive models of human goal striving (Gollwitzer 1993; Carver and Scheier 1998; Ryan and Deci 2000). According to the motivational intensity theory (Brehm and Self 1989; Wright and Kirby 2001), human behaviour is guided by an energy conservation principle, namely, that people will avoid wasting resources and thus will not mobilize more effort than necessary for goal attainment. Drawing on this assumption, the theory predicts that experienced task difficulty and importance of success interact to determine effort mobilization: resources are mobilized proportionally to the level of experienced task demand as long as success appears possible and worthwhile (Brehm and Self 1989; Gendolla and Wright 2005; Wright and Kirby 2001). These predictions were confirmed in several studies that used physiological measures of effort mobilization. Indeed, these studies reported that the influence of the sympathetic nervous system on the cardiovascular system increases with momentary effort mobilization and that sympathetic nervous activity is reliably reflected in increased systolic blood pressure and heart contractility (Obrist 1981; Wright 1996; Gendolla and Wright 2005; Richter and Gendolla 2009). For instance, Richter et al. (2008) showed an effect of task difficulty on effort mobilization through cardiac measures of healthy participants. Specifically, the experiment consisted of a recognition memory task (adapted from

Sternberg 1966) that required participants to indicate repeatedly whether a probe character was in a preceding string of characters. Difficulty was manipulated by displaying the initial string for 1,000 ms (low difficulty), 550 ms (moderate difficulty), 100 ms (high difficulty) or 15 ms (extreme difficulty). Results showed that contractility and systolic blood pressure responses rose progressively from the low to the moderate to the high difficulty condition, and then dropped. Furthermore, several physiological studies have reported the effects of situational factors affecting the perceived task difficulty and the amount of justified effort (for a review, see Gendolla et al. 2012), such as conscious mood states (discussed below), self-efficacy beliefs (Wright 1998) and fatigue (Wright and Stewart 2012). From these findings, it is possible to hypothesise a relationship between effort mobilization and apathy in brain-damaged persons through the influence of cognitive impairments. Indeed, individuals with cognitive deficits (attentional, executive, memory, etc.) are likely to mobilize more effort in response to easy cognitive challenges (in order to compensate for their cognitive deficits) and to disengage earlier when the tasks become more difficult. Nevertheless, this hypothesis should be examined more directly.

Affective Dimension

Some affective factors could contribute to the presence of apathetic manifestations. Emotional disorders, and particularly depression, are among the most frequently encountered disturbances after TBI (Tate 1999; Kreutzer et al. 2001). Although estimates vary, the prevalence of depression appears to be approximately 18.5 % to 61 % in the TBI population (Kim et al. 2007), with several negative outcomes reported on patients' functioning (Hibbard et al. 2004). For instance, Fann et al. (1995) showed that depressed TBI patients perceive their injury and their cognitive deficits as being more severe than non-depressed TBI patients do, despite the lack of differences in objective measures of severity of injury and cognitive functioning. As mentioned previously, relationships between apathy and depression are characterized by many common features. Given this important overlap, it would be interesting to explore the links between the dimensions of apathy and other aspects of depression that are not characteristic of apathetic manifestations, such as negative mood (sadness, hopelessness). Negative mood is reported to be specific to depression, but not a feature of apathy (Ready et al. 2003). According to various theories and particularly the *Mood-Behavior-Model* (MBM; Gendolla 2000), moods have an impact on motivation and behaviour. Indeed, the MBM posits that mood states can systematically influence the intensity and persistence of behaviour in contexts that call for effort investment because people use their moods as information for behaviour-related judgments and appraisals (Abele and Gendolla 1999; Gendolla and Brinkmann 2005). This assumption is built on the well-established phenomenon that mood states can influence judgments by means of a mood congruency effect (Clore et al. 1994; Wyer et al. 1999; Gendolla and Krüsken 2002), in that people in a positive mood make more optimistic judgments than do people in a negative mood. Consequently, the MBM considers the extent of subjective demand to be higher in a negative mood than in a positive mood when people are confronted with a challenge (Gendolla et al. 2001). Several studies using mood induction procedures in healthy populations have shown that negative affect increases demand appraisals of difficult situations, which improves subsequent effort mobilization, as measured by cardiovascular adjustments (for reviews, Gendolla and Brinkmann 2005; Richter et al. 2006). For instance, Gendolla and Krusken (2001) demonstrated a joint effect of manipulated mood state and task difficulty on cardiovascular and electrodermal reactivity in a group of university students. As regards the assessment, participants were induced into positive versus negative moods by exposure to music and later performed a letter cancellation task, adapted from the d2 Mental Concentration Test (Brickenkamp 1981). The results showed that cardiovascular reactivity was significantly stronger in a negative mood than in a positive mood when the task is easy. Conversely, reactivity was significantly stronger in a positive mood than in a negative mood when the task was difficult. Furthermore, Brinkmann and Gendolla (2008) reported that dysphoric individuals tend to appraise an easy task as difficult, and a difficult task as too difficult and impossible. Consequently, dysphoric individuals mobilized more effort than non-dysphoric individuals for an easy task and less effort for a difficult task, which results in disengagement of the task. Thus, negative mood may contribute to apathy by increasing the subjective difficulty of the tasks or goals. Studies are needed to explore the possible impact of negative mood on goal-directed behaviours.

Other emotional factors such as anhedonia could have a role to play in apathy. Following the criteria of Marin (1991) and their subsequent revisions, anhedonia (defined as the reduction in the ability to experience pleasure) may be part of the apathy's syndrome (Leentjens et al. 2008). Anhedonia has long been recognised as a core symptom of depression, but has regained attention as a dysfunction of the dopaminergic reward pathway (Fujiwara et al. 2011). The inability to experience pleasurable emotions could have an impact on reward seeking and thus, self-initiated behaviours. There are several rating scales to assess anhedonia such as the Snaith-Hamilton Pleasure Scale (Snaith 1993), the Chapman scales for physical and social anhedonia (Chapman et al. 1976) or the SANS Anhedonia-Asociality subscale (Andreasen 1989).

Dimension Linked to Personal Identity, Values and Beliefs

Aspects linked to personal identity should be taken into account in the identification of the mechanisms underlying

apathy (Prigatano 1992; Sagen et al. 2010). For instance, low self-esteem could dissuade the individual from undertaking complex tasks. Indeed, some studies showed that low self-esteem is related to an avoidant coping style in individuals with TBI (Anson and Ponsford 2006; Riley et al. 2010). More specifically, Riley et al. (2010) reported in a group of 41 TBI patients that those with low self-esteem and a negative evaluation of their coping resources were significantly more likely to respond to threat appraisals with avoidance. According to the authors, this link may occur in part because low self-esteem leads to an avoidance of further challenges to self-esteem, and this results in withdrawal and restricted participation. Self-esteem was measured using the Rosenberg self-esteem scale (Rosenberg 1965; Wright et al. 1995), which is reported to have satisfactory reliability in several acquired brain injury studies (Cooper-Evans et al. 2008). The findings of Riley et al. (2010) are in accordance with broader evidence that people who are low in self-esteem are more likely to focus on its protection rather than on steps to bolster it (Baumeister et al. 1989; Tice 1991; Heimpel et al. 2006). Thus, low self-esteem could contribute to apathetic manifestations by leading individuals to avoid valued activities in order to protect their self-esteem from threat. Studies are required to explore self-esteem and its relationships with apathy in patients with TBI. Several studies on self-esteem in children with brain injury have reported a significant low level of self-esteem in children with TBI compared with controls and an association between low self-esteem and behavioural problems (Tremblay et al. 1992; Andrews et al. 1998; Hawley 2012).

In the same way, self-efficacy beliefs may be involved in apathy. Self-efficacy has been conceptualised as "the belief in one's capabilities to organize and execute the courses of action required to manage prospective situations" (Bandura 1995, p. 2). According to Bandura, self-efficacy plays a major role in how goals and challenges are approached and can have an impact on motivation. More precisely, self-efficacy beliefs influence motivation by determining the goals people set, how much effort they invest in achieving those goals and their resilience when faced with difficulties or failure. Thus, people with low self-efficacy tend to set relatively low goals for themselves and to give up when problems surface because they believe that they are incapable of performing a difficult task. In this context, several studies have recently shown a relationship between perceived self-efficacy regarding cognitive abilities and participation or life satisfaction in patients with TBI (Dumont et al. 2004; Wood and Rutterford 2006; Cicerone and Azulay 2007). For instance, Dumont et al. (2004) found that a high level of self-efficacy was a predictor of social participation after TBI, suggesting that belief in one's ability to engage in daily activities and valued social roles increases motivation and thereby opportunities for improving social participation. Anecdotally, field experimenters have targeted self-efficacy as an effective way of increasing motivation at work in healthy adults. Thus, in a self-management training program conducted by Frayne and Latham (1987), workers' absenteeism was reduced by raising their self-efficacy beliefs regarding work. Taken together, these findings appear to show that self-efficacy could have a role to play in apathetic manifestations. Further research is needed to investigate the integrity of self-efficacy and its links with the different facets of apathy in patients with TBI. Regarding assessment, general (Dumont et al. 2004) and specific measures of self-efficacy (Cicerone et al. 2008; Malec et al. 2010) were developed. For instance, Cicerone and Azulay (2007) have developed a measure of perceived self-efficacy for management of symptoms following TBI, the TBI Self-Efficacy Questionnaire, that incorporate three subscales: the ability to obtain assistance, the ability to manage cognitive symptoms and the ability to manage emotional symptoms. The scale developer's have reported good internal reliability (Cicerone and Azulay 2007; Cicerone et al. 2008) but replication studies are needed.

Furthermore, interest has clear motivational and goal components, particularly for exploration, information seeking and learning (Schiefele et al. 1992; Krapp 1999; Sansone and Smith 2000). As advanced by Silvia (2005), interest is closely related to cognitive appraisals, which are determinants of emotions (Roseman and Smith 2001). In particular, two specific cognitive appraisal processes seem to be related to interest (Silvia 2006): (1) an evaluation of the event's novelty and complexity, which refers to evaluating an event as new, unexpected, complex, surprising or mysterious; and (2) an evaluation of the event's comprehensibility ("coping potential"), which involves considering whether one has the skills, knowledge and resources to deal with an event (Lazarus 1991). Thus, a deficit in one of two cognitive evaluations could contribute to the presence of apathetic manifestations and particularly to lack of interest. Most of the experiments that assessed interest have used real-world stimuli, such as abstract art or contemporary poetry and asked participants to rate each picture for interest and for appraisals. The results showed that the more novel and more comprehensible people rate a picture, the more they rate it as interesting. In short, events appraised as new and complex, yet potentially comprehensible, are experienced as interesting.

In addition, the ability to envision future events (*"future thinking"*), which refers to the capacity to momentarily disengage from the immediate environment in order to contemplate hypothetical future scenarios (Suddendorf and Corballis 1997; Wheeler et al. 1997), could have a role to play in apathy. Future-oriented thoughts are reported to be pervasive in daily life (Klinger and Cox 1987) and to have a strong adaptive value (Tulving 2005; Suddendorf and Corballis 2007; Boyer 2008). Indeed, several studies have shown that the simulation of future events serves a number of functions, such as decision making (Bechara and Damasio 2005; D'Argembeau et al.

2009), action planning (Gollwitzer 1999), problem solving (Oettingen 1996), emotion regulation (Taylor et al. 1998; D'Argembeau et al. 2009) and goal achievement (Taylor et al. 1998). Thus, a decreased ability to imagine future events could contribute to the presence of apathetic manifestations by making the person incapable of considering potential consequences prior to acting and hence overriding current needs in favour of long-term goals. Further, difficulties in elaborating and maintaining in one's mind a specific and detailed reward representation could alter its internal evaluation and consequently decrease anticipated pleasure expectation, leading to motivation deficits for goal-directed behaviour. Nevertheless, these hypotheses should be examined more directly. Rasmussen and Berntsen (2012) recently examined the ability to imagine possible events in the personal future in a sample of moderate-to-severe TBI patients. To examine these issues, the authors adopted a standard method based on D'Argembeau and Van der Linden (2004), which involved asking participants to imagine and describe a series of specific events from the personal future. TBI patients imagined significantly fewer episodic, event-specific details compared with healthy controls. In this context, Raffard et al. (2013) showed that apathy is specifically associated with difficulties imaging future pleasant events, and particularly poor self-referential information, in patients with schizophrenia. According to the authors, negative self-beliefs associated with reduced optimism about future could lead to apathetic manifestations through its negative impact on the ability to imagine future pleasant events. In addition, D'Argembeau et al. (2010) showed that executive processes involved in the organisation and monitoring of autobiographical retrieval were correlated with several measures of future thinking. Given the frequent impairment in strategic aspects of memory in patients with TBI (Azouvi et al. 2009), it would be interesting to explore the relationships between these executive processes, the ability to imagine future events and the various apathy dimensions.

Conclusions

Apathetic manifestations are commonly reported in the TBI population and have been associated with a wide range of negative consequences for the patients and their caregivers. However, the exploration of apathy is to date mostly descriptive and the psychometric properties of the apathy assessment scales are questionable. For instance, a strong interrelationship is reported between two apathetic dimensions that is, a lack of interest and a lack of initiative. It is of note that a link between other apathetic dimensions such as an emotional blunting and a lack of interest is hypothesized. Given these relationships, it seems inappropriate to define or establish diagnostic criteria for apathy only on the basis of a symptomatic approach. Indeed, the identification of the psychological processes underlying the apathetic manifestations seems also essential to ensure a better understanding of this disabling disturbance.

As reviewed above, there is evidence in the TBI literature that a variety of psychological mechanisms are involved in apathy. In fact, some studies showed that cognitive as well as affective and motivational processes are implicated in apathy. Further, the various neuroanatomical regions involved in apathy support the diversity of the psychological processes related to this behavioural disorder. Specifically, the findings indicate that brain damage in cortical and subcortical areas, including the lateral prefrontal cortex, the vmPFC, the ACC, the basal ganglia, the insula as well as the white matter tracts connecting these regions, are involved in apathy among the TBI population. Nevertheless, the studies focused on the identification of the various factors involved in the occurence of apathetic manifestations remain few in number and have not taken into account the various dimensions of apathy (i.e. only a global score of apathy was considered in these studies).

More generally, findings in the literature increasingly support the need to adopt a multifactorial and integrative approach towards behavioural disorders, focused on the precise identification of the various psychological mechanisms involved in the various dimensions of the disorder. The interactions between psychological processes and other mechanisms, such as neural systems, environmental inputs and sociocultural variables, must also be taken into consideration. Such an approach seems to have the potential to capture the diversity and complexity of the apathetic manifestations occurring after TBI. In this context, we propose a new multidimensional framework that takes into account the various psychological mechanisms at play in the facets of apathy, such as cognitive aspects (e.g., executive processes, sustained attention, mechanisms underlying multitasking), motivational aspects (e.g., positive reinforcement, effort mobilization), affective aspects (e.g., negative mood, anhedonia) and aspects linked to personal identity (e.g., self-esteem or self-efficacy, interest, future thinking). It is of note that most psychological processes, including those described in this model, are frequently reported to be disrupted in patients with TBI.

Research is clearly needed to confirm and develop this model, notably by using longitudinal studies in order to investigate the predictive role of these processes in the occurrence and persistence of apathetic manifestations. Moreover, further studies are also required to explore the interactions between the various psychological processes involved in apathy and more specifically, to identify potential mediator variables. For instance, it would be interesting to examine the links between effort mobilization and apathy and to consider the extent to which these relationships may be influenced by factors such as negative mood or self-efficacy beliefs. Indeed, theoretical models of self-efficacy postulate that the latter determines in part how much effort an individual will

invest to achieve a goal (Bandura 1995). In this perspective, Esposito et al. (2013) have recently showed in elderly people that the subjective demand of a memory task (i.e., perceived difficulty and estimated effort) operated as a mediator between self-efficacy beliefs and apathy.

The precise identification of the underlying mechanisms of apathy is much needed to enable the development of targeted and effective rehabilitation insofar as some apathy traits result in similar overt behaviours although their causes may be heterogeneous. In this regard, it is essential to implement psychological interventions for apathetic manifestations that are tailored to the specific psychological dysfunctions of the person, interventions that do not currently exist. Such a psychological approach, focused on the person, would address the problem of apathy in all its complexity and unique expression. Besides, the specification of the various psychological mechanisms involved in apathy may also contribute to a better identification of others mechanisms such as neuroanatomical correlates insofar as studies should be more guided by specific hypotheses.

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