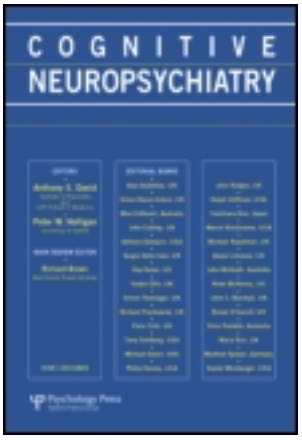

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The affective neuropsychology of confabulation and delusion

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The affective neuropsychology of confabulation and delusion

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The paper reviews the history of the scientific understanding of the role of emotion in confabulation and delusion. I argue that the significance of emotion in the pathogenesis of these symptoms was obscured by academic polarisation between psychodynamic and neurocognitive traditions and was also often obfuscated by rigid distinctions between psychogenic and neurogenic explanations. This tradition of epistemic dualism was implicitly maintained in the fields of cognitive neuropsychology and cognitive neuropsychiatry. This paper focuses on memory-related confabulation following ventromedial frontal lobe lesions, awareness-related confabulation following right perisylvian lesions, and delusions of various aetiologies. Ambiguity regarding the definition and taxonomy of symptoms renders direct comparison difficult, but certain overriding principles are becoming discernible. Recent findings suggest that emotion and motivation influence both confabulation and delusion. These influences may be instigated directly by neural dysfunction or indirectly by life changes and altered social circumstances, or by a combination of these. Importantly, the rejection of epistemic dualism in the conceptualisation of both symptoms can allow us to study them in parallel and draw conclusions about the relation between cognition and emotion. Specifically, confabulation and delusion can be described as faulty attempts to balance the conflicting demands of accurate and self-serving reality representation.

Keywords: Confabulation; Delusion; Emotion; Motivation; Reality monitoring.

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INTRODUCTION

Until recently, research on emotion was limited in both cognitive science and neuroscience. Emotion was considered too elusive to be studied in the laboratory, despite the pivotal role ascribed to it by great nineteenth-century scholars such as William James, Sigmund Freud, and John Hughlings Jackson. This neglect penetrated neurology, psychiatry, and their relationship during most of the twentieth century. In this paper, I will focus on the role of emotion in confabulation and delusion, as this role was conceptualised in neurology and psychiatry. I will argue that the influence of affective processes in false memories and beliefs was deemphasised due to rigid distinctions between psychogenic and neurogenic explanations. I will further discuss how this tradition of epistemic dualism was implicitly maintained in the fields of cognitive neuropsychology and cognitive neuropsychiatry. Subsequently, I will present recent approaches to confabulation and delusion that take emotions into account and I will review supportive empirical evidence. These perspectives can be heuristically labelled as *the ''affective neuropsychology'' of confabulation and delusion*.

Defining delusions, confabulations, and their relation

Delusions are considered a critical clinical manifestation of psychosis and are of determining importance in the diagnosis of schizophrenia. Notwithstanding their diagnostic significance, defining delusions has proven a persistent, rarefied challenge. Delusions are typically described by diagnostic manuals as firmly sustained false beliefs, which are resistant to correction and are not shared by members of the same sociocultural group. Yet the utility of these characteristics is repeatedly questioned. Characteristically, even the notion that delusions represent beliefs has incited considerable objections (see Bayne & Pacherie, 2005, for review). Some scholars have concluded that delusions are context-dependent, multidimensional, and impossible to adequately define (David, 1999). Others have proposed heuristic definitions. For example, Oltmanns (1988) suggested that the presence of delusions can be verified by checking the presence of a number of different characteristics, without assuming any of them is necessary or sufficient for defining delusions.

The term ''confabulation'' was first used at the turn of the twentieth century, substituting Korsakoff's term ''pseudoreminiscences'' (Korsakoff, 1889/1996), to describe the false recollections of amnesic patients. Amnesic confabulatory behaviour is nowadays considered pathognomonic of Wernicke-Korsakoff's syndrome, and is reported in many other neuropathologies (see Johnson, Hayes, D'Esposito, & Raye, 2000, for a meta-analysis). In

this memory-related use of the term, confabulations are understood as false memories produced without conscious knowledge of their falsehood. Relative consensus surrounds this definition (Dalla Barba, 1993; Johnson et al., 2000; Moscovitch, 1989). Less agreement exists regarding the subtypes and characteristics of memory-related confabulation, but some evidence suggests that spontaneous confabulations may be distinct phenomena from provoked memory errors (see, for review, Kopelman, 2010 this issue; Schnider, 2003). Typically, confabulatory content is operationally described as disorganised, polythematic, and fleeting (Baddeley & Wilson, 1986). However, organised, content-specific, and persistent confabulations have also been reported (Baddeley & Wilson, 1986; Burgess & McNeil, 1999).

Furthermore, the term confabulation is also used to describe the false statements of patients in many other memory-independent neurological syndromes, such as Anton's syndrome (unawareness of blindness), unawareness of hemiplegia, and cerebral disconnection syndromes (DeLuca, 2000; Feinberg & Roane, 1997; Hirstein, 2005). In these syndromes, patients' general memory abilities are not typically compromised. However, their ability to perceive and draw correct inferences about themselves and their environment is defective. Thus, they can unintentionally produce erroneous statements about their condition, or their abilities ("awareness-related confabulation"). For example, cortically blind patients with Anton's syndrome may deny any subjective experience of visual loss. However, it should be noted that the behaviour which constitutes confabulation in these patients is not identical across studies of even the same syndrome. For example, in anosognosia for hemiplegia one may designate as a confabulation patients' general erroneous assessment of their bodily state (e.g., "There is nothing wrong with my arm, I can move it"; Davies, Davies, & Coltheart, 2005), or one can refer only to instances of "illusory limb movements" (e.g., when the patient claimed he moved his arm as instructed by the examiner at that particular moment; Feinberg, Roane, & Ali, 2000), or just to associated somatoparaphrenic beliefs (e.g., this arm belongs to my niece) and false memories (e.g., false events and excuses, e.g., I went walking yesterday and I am tired now).

In an attempt to clarify the use of the term confabulation across domains, some authors distinguish between its various applications (e.g., DeLuca, 2000; Feinberg & Roane, 1997; Fotopoulou & Conway, 2004). For instance, Feinberg and Roane (1997) distinguish between "neutral" and "personal" confabulation. Neutral confabulation relates to an underlying deficit (e.g., cortical blindness) in any sensory domain and it is usually confined to that domain. By contrast, personal confabulation includes self-referential content that is not restricted to any sensory domain, is held with delusional conviction, and is refractory to correction. Unfortunately, these distinctions are not consistently used in the literature. In the remainder of the paper,

I will use the term confabulation to refer to two syndromes: namely "memory-related confabulation" following damage to the ventromedial frontal lobe (VFL) and "awareness-related confabulation" as it occurs following perisylvian lesions to the right hemisphere and in relation to anosognosia for hemiplegia (unawareness of paralysis). Although one may observe that at the level of individual symptoms, VFL patients may also produce some confabulations that relate to unawareness of deficit and patients with right perisylvian lesions (RPL) may also produce some confabulations about past events, the terms "awareness-related confabulation" and "memory-related confabulation" will be used here to refer to these two different neuropathological syndromes, which are marked primarily by one or other type of confabulation and which are associated with RPL versus VFL damage, respectively.

Given the aforementioned ambiguity about the defining characteristics and subtypes of both delusion and confabulation, the relation between the two phenomena is not clearly delineated. Thus far, no single criterion has been proposed as sufficient or necessary to distinguish between them (Gilleen & David, 2005; Kopelman, 1999). Although confabulations have diagnostic use mainly within neurology and delusions within psychiatry, delusions also occur following acquired brain injury or disease (e.g., Feinberg & Roane, 1997). For example, some authors describe anosognosia for hemiplegia as a delusion (e.g., Davies et al., 2005). Similarly, memory-related confabulation is reported in the context of schizophrenia (e.g., Nathaniel-James & Frith, 1996). Some studies distinguish delusions from confabulations in the same patient population (Baddeley, Thornton, Chua, & McKenna, 1996; Lee et al., 2007; Mattioli, Miozzo, & Vignolo, 1999), but the criteria for such distinctions seem arbitrary and are rarely used in more than a handful of studies. Others claim that they represent different facets of the same underlying aetiology. For example, Hirstein (2005) argues that confabulations, delusions, and the false memories of healthy individuals can be defined along a continuum of self-deception. Attempting to disentangle and to clarify the overall relation between the two concepts goes beyond the scope of this paper. Here I will consider each symptom as it has been addressed within its respective field and I will focus on the role of emotion in both phenomena.

EMOTION IN CONFABULATION

Classic views: Memory-related confabulation as deficit, compensation, or defence

Memory-related confabulation was first described by Korsakoff (1889/1996) as a symptom of a specific memory disorder caused by "multiple neuritis,

which is an extensive inflammation and degeneration of nerves throughout the body'' (p. 2). Given this neurological background, various early authors attempted to differentiate ''organic'' confabulations from other forms of false memories and beliefs (e.g., ''delusional memories'') that were encountered in disorders without a known ''organic'' aetiology (for review, see Markova & Berrios, 2000). Similar distinctions between organic and nonorganic explanations of symptoms (e.g., motor paralysis) were more widely established. Progressively, the divide between psychiatry and neurology grew deeper. Memory-related confabulation fell on the neurological side of the division and thus it was considered a ''neurogenic'' memory failure. Not surprisingly, the primary focus of studies on memory-related confabulation in the first half of the twentieth century was on specifying its neurological aetiology and its relation to amnesia.

By contrast, describing memory-related confabulation as motivated was synonymous with negating its neurogenic nature and indeed some authors did (see next section). Emotion and motivation were seen as the mark of psychogenic processes and thus their role in organic disease could only be conceptualised as secondary. Thus, for those neurologists and psychiatrists who were convinced by the clear neurological onset of memory-related confabulation, potential observations of motivational factors had to be deemphasised. For instance, in one of Talland's (1961) case descriptions he noted that ''motivational influences undoubtedly played their part in producing her glib evasive answers as well as her obstinate adherence to a past image of herself, but the mechanism by which such influences interacted with the amnesic derangement could not be determined'' (p. 376). Several other authors recognised motivational factors in confabulating patients. Given however the more general assumption of a rigid distinction between cognitive deficits (as impairments to functions directly subserved by brain processes) and emotional consequences (as secondary reactions to a new psychological reality, essentially unrelated to the damaged brain structures and functions) it was hard to assume a direct interaction between a memory dysfunction and emotion. Hence, Talland concluded his influential review with this frequently quoted statement: ''Confabulation serves no other purpose, is motivated in no other way than factual information based on genuine data'' (p. 393).

At the other extreme, some authors proposed that memory-related confabulation is not ''neurogenic'', but is instead caused by some psychogenic compensatory mechanisms. The ''embarrassment hypothesis'' proposed that memory-related confabulation occurs as a purposive act contrived by the patient to spare him from the embarrassment of not being able to remember the events of his life (see Talland, 1961, for review). This perspective explained confabulation as the result of some compensatory motivational mechanism, which is essentially unrelated to normal memory

processes and the neurocognitive deficits leading to their impairment. This view was criticised as patients seemed genuinely unaware of their memory errors (anosognosic) and many confabulating patients did not show a tendency to increase their confabulation rate when they were confronted with questions to which they did not know the answer (see Schnider, 2008, for review).

The incoherent and at times wishful quality of memory-related confabulation was frequently compared to the recollective quality of dreams and this led some psychodynamic authors to suggest that the individual's wishes guided memory-related confabulation in the same way as they controlled dream fantasy (reviewed by Berlyne, 1972). More generally, several early authors attributed the occurrence of memory-related confabulation, at least in part, to tendencies inherent in the patient's premorbid personality structure, such as increased suggestibility, or denial coping strategies (see Berlyne, 1972; Fotopoulou, 2009; Schnider, 2008, for reviews). Notwithstanding these descriptions, psychodynamic interpretations of memory-related confabulation were relatively scarce. The hypothesis that confabulation is a form of "psychological denial", i.e., a compensatory coping mechanism instigated by excessive anxiety, was mostly advanced in the work of Weinstein and colleagues (for review, see Weinstein, 1996). They observed that the patients most likely to show confabulation and denial of deficit were the ones characterised by their relatives as premorbidly stubborn, introverted with regard to their feelings, and prestige- and power-seeking. In addition, these patients had premorbidly been using coping mechanisms such as denial and minimisation when confronted with other health issues. Their theory emphasised that although confabulations frequently involve references to past events, they are to some degree, symbolic representations, or dramatisations of some current personal preoccupation or disability. These symbolic functions are based on kernels of awareness of deficit, i.e., confabulations implicitly express illness preoccupations and anxieties, which the patient is not capable of fully appreciating (see also Feinberg & Roane, 1997). Finally, Weinstein (1996) observed that during the narration of confabulations, patients become engrossed in detail, lose their previous irritability, and then appear utterly relaxed. He thus argued that memory-related confabulation seems to have an ultimate adaptive role.

Although these views highlighted potential positive aspects of memory-related confabulation, there was little experimental evidence in their support. In a study on senile dementia, Gainotti (1975) found that 75% of the tested confabulating patients were described by their relatives as having a strong tendency to seek prestige and domination and to deny, ignore, or rationalise illness prior to their brain disease. However, these coping mechanisms were considered independent of the specific neuropathological features of the syndromes they aimed to explain. Thus, their explanatory power with

regards to the syndromes themselves was limited. Indeed, Weinstein (1996) acknowledged that these descriptions were less likely to be relevant to spontaneous confabulation with vivid imagery, as reported in patients with anterior communicative artery aneurysms and anterior cingulectomies. Nevertheless, not all psychodynamic authors considered emotional mechanisms in isolation. Instead, some regarded the emotional manifestations of confabulation as the direct consequence of cognitive dysfunctions. For instance, Betlheim and Hartman (1924/1951) argued that the characteristic amnesic derangement of the Korsakoff syndrome caused a lack of cognitive restraint. This, in turn, allowed the normally implicit effect of primitive, emotion-based forms of cognition to become more explicit and colour recollection even under controlled experimental conditions. Thus, memory-related confabulation is regarded as the direct result of an organically caused dysfunction of the normal mechanisms of remembering.

Yet in the following decades this perspective received little experimental attention. Instead, the more simple "embarrassment" or "defence" explanations were established as the motivational hypotheses, only to be deemphasised and largely dismissed as more sophisticated neurocognitive models of memory-related confabulation came to the foreground (see next section). This dominance of "cooler" cognitive explanations coincided with a more general and at times rather passionate academic rejection of the once prevalent field of psychoanalysis. Although the criticisms were often validated and substantiated by solid empirical data, it has more recently become apparent that some psychodynamic ideas may be worth investigating and possibly integrating with current neuroscientific knowledge (e.g., Kaplan-Solms & Solms, 2000; McKay, Langdon, & Coltheart 2005).

Modern views: The cognitive neuropsychology of memory-related confabulation

Consistent with the tradition of cognitive neuropsychology, research on memory-related confabulation in the last decades has predominately approached confabulation as a useful symptom from which one can infer neurocognitive models of normal memory function (e.g., Dalla Barba, 1993; Gilboa et al., 2006). In this tradition, two main classes of theories have been put forward: explanations that focus on impaired temporality or reality monitoring (Dalla Barba, 1993; Johnson et al., 2000; Schnider, 2003) and explanations that emphasise deficits in the control of memory retrieval (Burgess & Shallice, 1996; Gilboa et al., 2006; Moscovitch, 1989). With regard to the first main class of theories, confabulating patients are conceived to misattribute experiences of a given time to events that occurred at another time, or to confuse the order of experienced events (Dalla Barba, 1993;

Talland, 1961; Schnider, 2003). In a similar vein, it has been suggested that confabulating patients are unable to distinguish between real and imagined events (Johnson et al., 2000), or alternatively that they are unable to distinguish between events that are currently relevant and those that are not (Schnider, 2003; but see Gilboa et al., 2006).

With regard to the second main class of theories, memory-related confabulation has been explained as a deficit in the control of memory retrieval. Memory-related confabulation can concern both experiences encoded and stored before the onset of brain damage as well as experiences that occur subsequent to the brain damage. Thus, memory-related confabulation seems to be associated more with retrieval rather than encoding or storage difficulties. In the "strategic" or "generative" retrieval accounts, confabulation is explained as a deficit in the strategic processes that are required during the organised and accurate retrieval of memories (Burgess & Shallice, 1996; Conway & Tacchi, 1996; Moscovitch, 1989). According to these models, when memories are not elicited directly or automatically by a cue, a number of control processes, including memory search and monitoring processes, are called for to guide recollection. Memory-related confabulation represents a failure of one or more of these processes.

These models have undoubtedly increased our understanding of memory-related confabulation. Given the recent technological sophistication of neuroimaging, these studies have also been able to specify the sites necessary for these higher order memory functions, such as the orbitofrontal cortex and the ventromedial prefrontal cortex (e.g., Schnider, 2003; Turner, Cipolotti, Yousry, & Shallice, 2008). However, the emotional facets of memory-related confabulation seem to have been neglected by cognitive neuropsychologists even more than they were previously deemphasised by neurologists and psychiatrists. With the marked exception of a case study by Conway and Tacchi (1996), no study on memory-related confabulation published in the 1980s or 1990s focuses on the emotional content of confabulation (although see Feinberg & Roane, 1997, and Kopelman, 1999, for clinical and theoretical considerations). It is of note that the role of emotion in memory-related confabulation was not empirically tested and rejected on the grounds of negative findings. Instead, until the twenty-first century the emotional content of memory-related confabulation was for the most part left outside the scope of empirical investigations of confabulation.

The affective neuropsychology of memory-related confabulation: The return of the emotional

Fotopoulou and colleagues (reviewed by Fotopoulou, 2008), motivated by the clinical descriptions of Conway and Tacchi (1996) and Solms (2000),

aimed to systematically assess the role of emotion in memory-related confabulation. The main hypothesis put forward by these investigators was that the false recollections of confabulating patients should show a self-serving bias that is greater than that typically encountered in the memory distortions of healthy volunteers (Walker, Skowronski, & Thompson, 2000). Of course, the vicissitudes of human motivation extend far beyond self-serving biases. However, this operationalisation was considered a useful first step to the challenge of systematically studying the role of emotion in memory-related confabulation. Moreover, this exaggeration of self-serving biases in memory was not seen as an exaggeration of motivation per se (a psychogenic explanation). Instead, it was conceptualised as the direct outcome of reduced executive control over memory. There seems to be a trade-off between the influence of cognitive inhibition and motivational influences on memory (Conway, 2005). For example, although emotion typically enhances memory, trying to suppress the emotional impact of an event may lead to worse memory for the event (Gross, 2002). Consequently, impairment in one aspect (retrieval control and inhibition) may generate exaggeration in the other (excessive influence of emotion on memory). Specifically, when irrelevant memory representations are not inhibited and memories are not retrieved in an appropriate manner (as the various neurocognitive theories support; see earlier), motivational factors may acquire a greater role in determining which memories are selected for retrieval and accepted as true. This lack of affective regulation in memory was expected due to the typically reported damage to the ventromedial frontal cortex in memory-related confabulation, which is more generally thought to be responsible for affective regulation (e.g., Bechara, Damasio, & Damasio, 2000) and particularly the inhibition and extinction of previously rewarded responses (Elliott, Friston, & Dolan, 2000). Thus, damage to these areas may also lead to a failure of extinction or inhibition of rewarding representations in the domain of personal memory. Therefore, personally "rewarding" representations may be preferentially selected over other, less positive candidate thoughts and memories, irrespective of their low pertinence to reality.

Consistent with this hypothesis, a number of single-case and group studies on memory-related confabulation (e.g., Fotopoulou, Conway, Tyrer, et al., 2008; Fotopoulou, Solms, & Turnbull, 2004) found that the content of spontaneous confabulations contains mostly positive and wishful descriptions (to a degree greater than that found in the memory distortions of healthy volunteers). A positive bias has also been observed in the provoked confabulations, elicited during an autobiographical memory interview, in another patient with memory-related confabulation (Fotopoulou, Conway, Griffiths, Birchall, & Tyrer, 2007). In the latter study, the patient's confabulations portrayed a self-representation that was more

positive than that depicted in his real memories and those depicted in the memories of matched healthy controls. A further case series ($N = 4$) showed that patients with memory-related confabulation were more likely to make reality monitoring errors, accepting false or temporally irrelevant information as part of their recent past, when the information was pleasant rather than unpleasant (Fotopoulou, Conway, & Solms, 2007). For example, a patient was significantly more likely to falsely claim that he actually remembered winning the lottery recently than to falsely claim that he remembered losing his job. Finally, in a recent prose recall study, patients with memory-related confabulation ($N = 15$) showed a strong selective bias in recalling negative self-referent stories, in that they recalled such information in a manner which portrayed a more positive image of themselves (Fotopoulou, Conway, Solms, Tyrer, & Kopelman, 2008). This positive bias was not present in stories that were not encoded in a self-referent manner, and was significantly greater than that observed in the recall of healthy volunteers and amnesic nonconfabulating patients. This study shows that patients with memory-related confabulation do not have a difficulty in processing negative emotions in general. Instead, they show a specific self-related motivational bias in their memory.

In the studies reviewed here, patients frequently described themselves as being in familiar surroundings, and as performing professional or leisure activities, instead of being at the hospital (see also Turnbull, Berry, & Evans, 2004). Patients also often minimised their current disabilities and attributed them to premorbid traits and attitudes (e.g., "I could never remember names"). Moreover, some patients persistently denied the death of close relatives and other unpleasant events of the remote or recent past and they were noted to inflate their abilities, exaggerate their previous professional skills, and overstate their social and financial position (Fotopoulou, Conway, Griffiths, et al., 2007). Given the close link between autobiographical memory and personal identity (Conway, 2005; McAdams, 2001), we have conceptualised these tendencies in the content of memory-related confabulation as serving at least two functions: self-enhancement (embellishment of one's self-image) and self-coherence (adherence to one's premorbid self-image) (Fotopoulou, 2008). In the first case, patients' confabulations point to a tendency to inflate one's current or past self-identity; in the second case patients' confabulations seem to establish a sense of self-continuity with one's past (premorbid) identity. Both tendencies have been noted in the autobiographical distortions of healthy volunteers (see Conway, 2005, for review).

More generally, social psychology has provided considerable evidence that people are motivated to view their current self favourably and engage in considerable memory distortion in order to maintain such a view (e.g., Walker et al., 2000). This is particularly evident in older adults who show a strong self-serving positivity bias in their autobiographical recollections

and this seems to have positive effects on their mood and well-being (for review, see Mather & Carstensen, 2005).

Normally aging adults and patients with memory-related confabulation show deterioration and dysfunction respectively in prefrontal brain regions (Hedden & Gabrieli, 2004). We therefore have claimed that the resulting deterioration of executive memory processes may be causing the observed exaggeration of self-enhancement and self-coherence in the memory of both normally aging adults and patients with memory-related confabulation (Fotopoulou, Conway, Solms, et al., 2008). This proposal is consistent with recently emerging models of memory-related confabulation that include personal goals and wishful ideation among the critical factors that may determine the retrieval of false memories in confabulating patients (Gilboa et al., 2006; Johnson et al., 2000; Kopelman, 1999; Metcalf, Langdon, & Coltheart, 2007). Acknowledging the role of emotion in memory-related confabulation has also pivotal clinical consequences (see Fotopoulou, 2008, for review). It thus seems that at least some authors have recognised that emotion may have a determining role in memory-related confabulation and that a total psychological picture of the syndrome requires consideration of the interaction between neurocognitive factors and emotion.

I have thus far emphasised that memory-related confabulation may not result from the exaggeration of psychological motivation per se, but rather from an exaggeration (due to neurocognitive impairment) of the organically predetermined influence of motivation on memory formation. It is, however, plausible that given the self-threatening situation patients find themselves in after brain damage, the psychological need to avoid recalling negative self-referent information may be exaggerated (a secondary, indirect effect of brain damage). Furthermore, certain patients may be premorbidly more inclined than others to hold a positive self-regard and distort memories in self-enhancing ways, depending on their previous coping strategies, social role, and other psychosocial characteristics. Thus, potential individual differences should not be excluded from the understanding of memory-related confabulation without further study.

Awareness-Related Confabulation and Negative Emotions

The Dalla Barba confabulation battery (1993) was used to measure the propensity to confabulate about personal facts, personal past episodes, knowledge of famous facts and famous people, and orientation in time and place in a consecutive series of 15 patients with right perisylvian lesions (RPL) resulting in anosognosia for hemiplegia (AHP) (Fotopoulou, 2005; Fotopoulou & Conway, 2004). We found an increased propensity to

confabulate on the battery in five of these individuals (Fotopoulou, 2005; Fotopoulou, unpublished data). Specifically, although these five patients with RPL confabulated less on average than 15 patients with VFL lesions and memory-related confabulation recruited in the same period (Fotopoulou, Conway, Tyrer, et al., 2008), they confabulated more than amnesic nonconfabulatory patients, nonconfabulating controls with RPL, and healthy controls. Four of these patients with RPL and awareness-related confabulation were also clearly attempting to act upon their confabulations, e.g., one patient "secretly" called her lawyer to say that she was healthy but nevertheless held in hospital against her will. There was no obvious neuroanatomical difference between the lesions of patients with RPL and AHP who showed awareness-related confabulation and those who did not.

The patients with RPL and awareness-related confabulation appeared better oriented in time and place than the patients with VFL damage and memory-related confabulation and they also performed better on tests of executive functions, and episodic and autobiographical memory (Fotopoulou, 2005; Fotopoulou & Conway, 2004). Despite these relatively preserved abilities, patients with awareness-related confabulation produced false statements about their bodily and mental integrity and their direct everyday-living implications. Unlike the memory-related confabulations of patients with VPF lesions, awareness-related confabulations rarely extended to the remote past. Emotional differences between the two confabulation syndromes were also noted. The valence of anosognosic false statements in patients with RPL was mostly positive (e.g., "I am not paralysed. There is nothing wrong with me"). However, the majority of the associated false memories and beliefs (indirectly linked to patient's disabilities) were predominately negative, often "paranoid" or self-scolding in content. For example, one patient frequently believed that certain members of staff were trying to kill her. At times she described specific murder attempts. We used the same method we had used previously to measure the valence of memory-related confabulation in patients with VFL lesions (Fotopoulou et al., 2004; Fotopoulou, Conway, Tyrer, et al., 2008) to measure the emotional valence of awareness-related confabulation in the first three of the confabulating patients with RPL. Indeed, we confirmed that the emotional content of awareness-related confabulation was negative on average across the three patients and individually (Fotopoulou, 2005).

We have interpreted these unpleasant false statements in awareness-related confabulation as misattributions (either internalisations or, externalisations) of unpleasant emotions that relate to "implicit" or occasional awareness of one's disability (Fotopoulou & Conway, 2004; see also Feinberg & Roane, 1997). For example, a patient with an RPL and AHP expressed intense anger towards the hospital staff because they had "amputated" her left arm and "put it into a mincemeat maker, remoulded it and put it back

on''. These bizarre beliefs and the related accusations of staff may have been influenced by paralysis-related negative feelings (implicit emotional awareness), despite the patient's explicit unawareness of her paralysis. The presence of this "implicit'' awareness of deficits in AHP has been experimentally documented (Nardone, Ward, Fotopoulou, & Turnbull, 2007); however, the issue of whether these misattributions are motivated is the subject of ongoing debate. At the one extreme, a psychogenic hypothesis is that awareness-related confabulation reflects defence mechanisms against anxiety and depression, not much different to memory-related confabulation (Weinstein, 1996). At the other extreme, some authors have suggested that all features of AHP can be explained on the basis of specific neurocognitive deficits (e.g., deficits in forward motor planning; Heilman, Barret, & Adair, 1998) and thus awareness-related confabulation is unrelated to other syndromes of confabulation. Challenging the rigidity of the distinction between psychogenic and neurogenic factors in these explanations, a number of authors have recently proposed intermediate, more parsimonious explanations.

Some have remarked that AHP is the consequence of abnormal affective regulation (Turnbull, Owen, & Evans, 2005), or a deficient affective drive to respond to uncertainties about current bodily states (Vuilleumier, 2004). Damage to right hemisphere areas responsible for affective evaluation and novelty detection might play a determining role in such deficits (Ramachandran, 1995). Alternatively, damage to subcortical circuits (e.g., basal ganglia) that are involved both in motivation and in detection of "errors'' might lead to an inability to revise beliefs based on novel perceptual experience (Vuilleumier, 2004). Marcel et al. (2004) recently argued that right brain damage may alter emotional and attitudinal processes implicated in self-attribution (versus external attribution) of perceptual experiences (Marcel, Tegnér, & Nimmo-Smith, 2004). Interestingly, we assessed three patients with RPL and awareness-related confabulation on the prose recall experiment outlined earlier (Fotopoulou, Conway, Solms, et al., 2008). We found that their performance was similar to that of patients with VPL lesions and memory-related confabulation; they remembered the self-referent unpleasant stories as significantly less negative (i.e., in more self-serving ways) than other-referent unpleasant stories (Fotopoulou, 2005).

Thus, although the emotional valence of awareness-related confabulation is predominately negative, this syndrome may still reveal the exaggeration of self-serving emotional mechanisms similar to those discussed in relation to memory-related confabulation. Moreover, as in the case of memory-related confabulation, it is plausible to assume that neurocognitive deficits disrupt the existing neural mechanisms by which cognition controls and inhibits emotion. For example, some people may find it difficult to initially accept negative news, such as the diagnosis of a serious illness, or the dangers of smoking. They may instead misattribute these unpleasant facts to the

incompetence of doctors, or the intentions of health campaigners, respectively. Progressively, however, they may manage to appreciate the situation in more realistic terms. In the event of stroke to the critical right-hemisphere areas, these denial copying strategies may be exaggerated due to the dysfunction of those basic cognitive mechanisms (required for body awareness) that allow one to appreciate the situation in realistic terms and control the related emotions. AHP and awareness-related confabulation may therefore not represent "defences" in the psychodynamic sense. Rather they seem to be *neurological equivalents* of these defences, i.e., the premorbid tendencies of an individual are exaggerated due to a specific neurocognitive deficit. In Ramachandran's terms, "what one is really seeing in these patients is an amplified version of Freudian defense mechanisms caught in *flagrante delicto*; mechanisms of precisely the same sort that we all use in our daily lives. However, since the defenses are grotesquely exaggerated, studying them might give us, for the first time, an *experimental handle* on defense mechanisms" (Ramachandran, 1994, p. 26, original emphasis).

In summary, both neurocognitive and emotional factors seem to play a role in awareness-related confabulation. However, it appears that different cognitive deficits, combined with different direct and indirect emotional influences, lead to the two confabulation syndromes discussed here, namely awareness-related and memory-related confabulation.

EMOTION IN DELUSIONS

Classic views

The conceptualisation of delusions is closely linked to the history of the study of psychosis. Initially psychosis was considered a subcategory of the wider class of neuroses, but it eventually came to be regarded as distinct from neuroses. The meaning of this distinction took many different forms during the twentieth century and was marked by the development of the equally variable distinction between functional and organic explanations (see Beer, 1996, for review). At the end of the nineteenth century the neurologist Freud argued that all mental phenomena, including neurosis and psychosis, have psychological determination (Freud, 1924/1961). He further envisioned that his psychological observations would be linked to neurochemical findings in the future (as opposed to the neuroanatomical explanations of his time). Nevertheless, most psychiatrists at the time viewed psychosis as unrelated to psychological mechanisms. This view was crystallised in the writings of Karl Jaspers (1963), who distinguished "affective illness from madness proper" on the basis that the first is meaningful to the healthy and allows empathy, whereas the second is not understandable, i.e., it is not psychologically meaningful. This distinction between neurosis and psychosis took many other

forms and varied among countries; however, it gradually became accepted that neurotic and psychotic behaviours are of different aetiology (Roth, 1963). These clinical observations were systematised by others, notably Kurt Schneider, and became embedded in psychiatric classification systems.

In this epistemological context, delusions, as one of the hallmarks of schizophrenia, were separated from the study of "psychological factors", such as mood and motivation. Characteristically, Jaspers (1963) distinguished between primary delusions (delusions proper), which were uniquely bizarre and nonunderstandable, and secondary delusions (delusion-like ideas), which stem understandably from normal emotional states (see Eilan, 2000, for a critical review). This distinction seems to have informed the currently prevailing distinction between mood-incongruent delusions that have been mostly associated with schizophrenia and mood-congruent delusions that are mostly related to affective psychosis. Yet there has been little and inconclusive empirical investigation of the relationship between affect and delusion across diagnostic categories (e.g., Oulis et al., 2000). Crucially, delusions have been mostly studied in the context of schizophrenia and the possible emotions conveyed have been considered as the mere noise of the pathological mechanism that produced them. As a consequence, the possible psychosocial and motivational aspects of delusions were, until recently, neglected (for critical reviews see Freeman & Garety, 2003; McKay et al., 2005).

In brief, the mainstream study of delusions at the end of the twentieth century inherited two epistemological principles from the sharp distinction between neurosis (as functional disorders) and psychosis (as organic with known or unknown causes): (1) delusions should be studied as pathological phenomena, i.e., essentially unrelated to "normal" psychological states; and (2) delusions should be studied as emotionally meaningless phenomena, i.e., essentially unrelated to normal emotional and motivational processes (see Freeman & Garety, 2003; McKay et al., 2005, for reviews).

Modern views: The cognitive neuropsychiatry of delusions

In the last few decades, there has been a change in the conceptualisation of delusion with relation to the above two principles. Clinical psychologists, encouraged by evidence that psychological treatment can reduce delusions and hallucinations (e.g., Drury, Birchwood, Cochrane, & Macmillan, 1996), endeavoured to understand the symptoms of psychosis in psychological terms (see later). In parallel, the study of delusions has been significantly influenced by the tradition of cognitive neuropsychiatry. This tradition, using the methods of cognitive neuropsychology, addresses psychiatric symptoms as resulting from impairments to processes implicated in normal models of neurocognitive function (David, 1993).

Cognitive neuropsychiatric theories can be divided into two main categories: "perceptual" and "cognitive" theories (see Davies, Coltheart, Langdon, & Breen, 2001, for review). Perceptual models, as outlined by Maher (1974), assume that delusions are explanations of abnormal perceptual experience derived via normal reasoning processes, while cognitive accounts propose that delusions reflect cognitive and reasoning biases or abnormalities. A number of candidate biases have been put forward, including probabilistic reasoning bias, attributional biases, and attentional biases (see Gilleen & David, 2005, for review). Other cognitive theories postulate that patients with delusions have reality monitoring deficits (Johnson et al., 2000), i.e., there is impairment of the higher order cognitive ability to distinguish between internal and external representations. Alternatively, delusions have been considered as resulting from impairments of "theory of mind", the cognitive processes that enable individuals to reflect upon and take appropriate account of the mental states of others (Frith, 1992).

The so-called "two-factor" theories of delusions consider both perceptual and cognitive factors and were developed mainly to explain monothematic (vs. polythematic) and circumscribed (vs. elaborated) delusions (Davies et al., 2001; Stone & Young, 1997). Whereas Maher's perceptual theory suggests that abnormal perception is sufficient to cause delusions (one-factor theory), two-factor theories postulate that perceptual aberrations may be necessary to explain how delusions, in particular bizarre delusions, are generated but are not sufficient to explain why the delusions are adopted and maintained in the face of implausibility and contrary evidence (Davies et al., 2001; Langdon & Coltheart, 2000). A second factor, such as some of the cognitive biases and deficits described by cognitive theories, is required which leads to the adoption of the delusional belief and prevents the individuals from revising their beliefs in light of strong evidence against them. This "two-factor" approach to delusions has more recently been applied to all monothematic delusion other positive symptoms (Coltheart, Langdon, & McKay, 2007). Thus, this approach claims that although bizarre, monothematic delusions are aetiologically heterogeneous (they are caused by different "first-factor" pathologies), they share a neurocognitive homogeneity (they are caused by a combination of two neurocognitive factors).

The psychoanalytic position: Delusion as defence

As in the case of confabulation, the first motivational theories of delusion stemmed from the psychodynamic tradition. Contrary to mainstream psychiatry, psychoanalytic theories proposed that the content of delusions, although it may appear incomprehensible to the observer, is of great personal meaning to the patient. In Freudian thought, delusions are formed

as a second, derivative step in the pathogenesis of psychosis (Freud, 1924/1961). According to Freud, psychosis involves a pathological disruption of normal perception, memory and judgement (cognition) due to the overwhelming conflict between the person's motivation and the demands of external reality. Delusions are formed as subsequent substitutions of the appropriate contents of cognition by representations that are more compatible to the subject's wishes and drives. In this sense, delusions are regarded as motivated phenomena serving "defensive", self-protecting functions; delusions are formed and maintained as attempts to relieve or protect individuals from the psychological pain or distress accompanying psychological conflict. Nevertheless, it is important to emphasise that Freud regarded delusions as mostly unsuccessful attempts to protect the individual from anxiety. He noted that the initial cognitive disruption necessarily leads to illness. The representation of reality "cannot be remolded in satisfying forms" and thus leaves the individual with false memories, beliefs, and perceptions that "are of a most distressing character and [are] bound up with a generation of anxiety" (1924/1961 p. 186).

Although the concept of "defence" is central to psychoanalytic theory, its meaning has not been consistent throughout the years and varies greatly between different psychodynamic theories (Laplance & Pontalis, 1973). As in the case of confabulation, different psychoanalytic hypotheses have been proposed to account for the defensive function of delusions. Initial proposals emphasised the role of inhibited sexual desires and other unconscious wishes, whereas subsequent models seemed to focus more on which self-protective functions delusions served and which "mechanisms of defence" were employed in each case. This heterogeneity, coupled with the lack of empirical investigation, has contributed to the discrediting of the psychoanalytic approach to delusions in the second half of the twentieth century (e.g., see McKay et al., 2005, for review). These criticisms coincided with more general doubts over the scientific validity of psychoanalysis. Nevertheless, as aforementioned, a number of scientists have, more recently, suggested that certain of the insights of psychoanalytic theory may be worth empirically assessing and integrating with current neuroscientific knowledge. Most relevant to the present paper are the recent theories that consider the role of emotion in delusion (e.g., Bentall, Corcoran, Howard, Blackwood, & Kinderman, 2001). These models are reviewed next.

The affective neuropsychiatry of delusion: The return of the emotional

The recent effort to understand the symptoms of psychosis in psychological terms, brought about by the traditions of clinical psychology and cognitive

neuropsychiatry, have also opened the door to the study of the relation between emotion and false beliefs. Consistent with the psychodynamic notion of projection as a mechanism of externalisation of internal unconscious conflict, Bentall et al. (2001) have postulated that patients with paranoid delusions make excessive external attributions of blame in order to protect a vulnerable self-esteem. However, recent experimental and neuroimaging studies of the predictions of this model have produced mixed results (see Bentall, Fernyhough, Morrison, Lewis, & Corcoran, 2007, for review). Moreover, although an association between low self-esteem, depression, and paranoia is well-established (see Freeman, 2007, for a review), the causal direction of this association is debated.

As Freud had supported (see earlier), it is plausible that paranoid thoughts themselves may be affecting one's mood and self-esteem (see also Bentall et al., 2001). Indeed, Freeman and Garety (2003) have argued that persecutory delusions are a direct reflection of emotional concerns, so that the content of delusions shares the main themes of emotions. Delusions are viewed as attempts to explain (not explain away, as in Bentall's view) anomalous experiences by using emotionally laden, preexisting beliefs about the self and the world. Trower and Chadwick (1995) have argued that one function of paranoia is defensive ("Poor Me Paranoia"); another may be a direct reflection of intense negative emotions ("Bad Me Paranoia") (but see Bentall et al., 2007). Evidence that can unequivocally decide between these alternatives (defensive vs. direct role of affect in delusion) is yet to be put forward. It is also possible that emotions have both roles in the formation of delusions. Whichever is the case, there is now sufficient evidence that low mood, low self-esteem, and negative self-beliefs have a role in the development of paranoid thoughts and perhaps even in other delusions (Garety, Bebbington, Fowler, Freeman, & Kuipers, 2007).

More generally, there is now increasing recognition that emotion can contribute to the development of psychotic symptoms (see Garety et al., 2007, for review). Nevertheless, current motivational accounts of delusions differ with respect to the degree with which they can integrate emotional with neurocognitive factors. Indeed, the multifactorial models of delusions, developed mostly based on research on persecutory delusions (e.g., Bentall et al., 2001; Garety et al., 2007), rarely point to direct aetiological interactions between the cognitive and emotional factors they incorporate. By contrast, some other models have endeavoured to draw direct links between cognitive deficits and emotional factors.

McKay et al. (2005) have incorporated motives in the "two-factor" model of Coltheart and colleagues (described earlier) by proposing that motives are important doxastic (relating to belief) forces. Emotions and desires may affect both perceptual input (the first factor) and belief evaluation (the second factor). For example, in AHP covert awareness of one's disability (Nardone

et al., 2007) and the motive to defend one's self-esteem may lead one to avoid perceptual input relating to the facts of the disability (first stage). In addition, one may be motivated to accept as correct the beliefs that satisfy one's desire not to be disabled, thus reducing the significance of contrary evidence (second stage). This hypothesis remains to be tested. McKay and colleagues also used Ramachandran's (1995) view of the right hemisphere, as a "discrepancy detector", to argue that right hemisphere lesions or abnormalities may underlie the faulty belief evaluation in all delusions. I have outlined the benefits of such integrative, nondualistic hypotheses in the previous sections on memory-related confabulation and awareness-related confabulation. However, the consistency-checking functions of the right hemisphere have not been empirically confirmed (Fotopoulou, Tsakiris, et al., 2008), and this explanation cannot account for delusions associated with subcortical or left-hemisphere lesions (see Kunert, Norra, & Hoff, 2007, for review).

Gibbs and David (2003) proposed a theory that locates the formation and maintenance of mood-congruent delusions in emotion-based memory biases. A substantial body of work shows that affect biases encoding, enhances consolidation, and facilitates the retrieval of mood-congruent memories. These processes can lead to the persistence of biased recall of mood-congruent memories and beliefs. Neuroimaging studies have demonstrated limbic abnormalities in patients with schizophrenia and bipolar affective disorder, including the amygdala and the hippocampus (see Kunert et al., 2007, for review). According to this model (Gibbs & David, 2003), memory of imagined events could be enhanced by inappropriate conjunction of affective tone, mediated by limbic structures, leading to delusions by way of adding misleading contextual information. In more detail, the misuse of contextual information may be facilitated by the reduced ability of the prefrontal cortex to appropriately filter out and inhibit inappropriate affective responses. Thus, enhanced emotional and sensory characteristics of imagined events could lead patients to reality monitoring confusion and acceptance of these imagined events as real (see also Fotopoulou, Conway, & Solms, 2007). Further episodes of strong affect may lead to a re-remembering of these ideas, thus further enforcing their abnormal emotional-tagging and granting them persistent reality status.

This hypothesis brings the conceptualisation of mood-congruent delusion closer to that of memory-related confabulation. It is also consistent with the views of Freeman and Garety (2003), who conceive a direct relation between emotion and delusional content. Furthermore, it tallies with recent evidence showing that pharmacological or psychological treatment of comorbid mood disorder reduces delusions (e.g., Drury et al., 1996; Seretti, Lattuada, Zanardi, Franchini, & Smeraldi, 2000) and increases insight (Spalletta, Ripa, Bria, Caltagirone, & Robinson, 2006). However, this model has not as yet been empirically tested and it cannot easily be used to explain mood-incongruent

delusions and monothematic delusions. In addition, although the role of mood in memory is well-documented, personal goals and motives are also known to be important in memory formation and to serve similar memory and reality-monitoring biasing roles (see Conway & Pleydell-Pearce, 2000, for review). Thus, the role of mood in this model can be extended to include motives, preoccupations, and goals of the individual. This kind of extension would be consistent with the explanation of motivated memory-related confabulation outlined earlier, according to which ventromedial frontal lobe damage enhances the influence of motivation on reality monitoring and memory retrieval (Fotopoulou, Conway, & Solms, 2007; Fotopoulou, Conway, Solms, et al., 2008). Moreover, such an extension to include motives, preoccupations, and goals could provide a potential way of using the same framework to explain mood-incongruent delusions. In a recent study on memory-related confabulation (Fotopoulou, Conway, Tyrer, et al., 2008), we found that the more depressed the patients' mood in a self-reported questionnaire, the more positive the content of their confabulations. Although this finding is preliminary, it is consistent with the idea that some delusions may directly reflect one's mood as Gibbs and David (2003) argue, but others may serve a defensive function (e.g., regulating one's mood) as Bentall and colleagues have argued (see also Trower & Chadwick, 1995).

CONFABULATION AND DELUSION: TOWARDS A SYNTHESIS

This paper outlined the history of research on the role of affect and related processes in confabulation and delusion. I have argued that the significance of emotion in the pathogenesis of these symptoms has been obscured by academic polarisation between psychodynamic and neurocognitive traditions and often obfuscated by rigid distinctions between psychogenic and neurogenic explanations. However, recent research on both confabulation and delusion has brought emotion and motivation to the forefront of academic interest. A number of difficulties and ambiguities regarding the definition and taxonomy of symptoms render direct comparison difficult, but certain overriding principles are beginning to be discernible. Crucially, appropriate directions of future cross-disciplinary research can be envisioned.

Confabulation and delusion can both be described as pathologies involving conflicting demands in memory construction and belief formation (see also Fotopoulou, 2008). Neisser (1988) proposed that remembering is both an act of "utility" (using the past in the service of the present) and "verity" (retracing what took place in the past). Conway (2005) suggested that autobiographical memory is faced with two opposing demands. On the one hand, memories may be altered and even fabricated in order to make the past consistent with current

goals and self-images (the demand of "coherence"). On the other hand, memories should, up to a degree, correspond to past experience, irrespective of current concerns (the requirement of "reality correspondence"). Similarly, Stone and Young (1997), following Fodor, have suggested that the belief formation system entails a permanent tension between two principles in potential conflict: Beliefs should tally with the existing web of belief (conservatism); and beliefs should adhere to the deliverances of current perception (observational adequacy). Confabulation and delusion, as well as "normal" false memories and unusual beliefs, can be described as faulty attempts to manage the balance between these conflicting demands of reality representation.

The studies reviewed here provide evidence that emotion and motivation are of determining importance in how this balanced is achieved (see also McKay et al., 2005). Moreover, the current paper has argued that outdated conceptualisations of emotion and motivation as exclusively psychogenic processes should be rejected. The influence of affect and related processes on confabulation and delusion may be instigated by: (1) neural damage or dysfunction to predetermined mechanisms of interaction between emotion and cognition; (2) hard-wired individual differences in these mechanisms; (3) life changes and social circumstances that influence the emotional and cognitive state of individuals; and (4) any combination of these. Importantly, because of this variety, the parallel study of emotional influences and detectable neurocognitive dysfunction can elucidate the nature of the interaction between the two.

For example, in memory-related confabulation poor reality filtering and weak control of recollection due to anterior limbic damage seem to lead to self-serving biases in recall and motivated reality monitoring (Fotopoulou, Conway, & Solms, 2007). It would be of interest to use some of the experimental and neuroimaging paradigms developed in the study of memory-related confabulation (e.g., Schnider, 2003) to verify whether the same reality monitoring deficits and anterior limbic abnormalities underlie delusion formation in affective psychosis, as hypothesised by some authors (Gibbs & David, 2003). Similarly, findings, experimental procedures and hypotheses from the study of awareness-related confabulation may be used in the study of related idiopathic delusions and vice versa. For example, given the typical explicit unawareness and emotional flatness or lability in patients with awareness-related confabulation, it would be interesting to investigate implicit self-esteem and external attribution biases in these patients.

In addition, the role of emotion in clinical confabulation and delusion can be seen in a continuum with the role of emotion in normal memory and reality distortions in healthy and subclinical populations (see Freeman & Garety, 2003, for discussion). For example, memory-related confabulation seems to share some characteristics with the motivated memory distortion of normal autobiographical memory (Conway & Pleydell-Pearce, 2000). Similarly,

awareness-related confabulation may be conceived as an exaggeration of the denial healthy individuals show in circumstances of great anxiety or loss (Moyer & Levine, 1998). These conceptualisations may allow the study of clinical confabulations and delusions to inform models of normal motivated cognition and vice versa. To these "continuity" perspectives, one may also add a neurodevelopmental dimension. For example, it is plausible that the emotional processes implicated in delusions may have their origin in early adverse experiences that create an enduring cognitive vulnerability (Garety et al., 2007). Indeed, a different line of research within mainstream psychiatry suggests that psychosis represents the endpoint of abnormal developmental pathways which may begin early in life (for review, see Bentall et al., 2007).

Approaches to confabulation and delusion that take emotion and motivation into account seem to have direct applicability to clinical practice (Fotopoulou, 2008; Freeman & Garety, 2003). Such applicability not only has pragmatic significance and potential for translational research but it also opens up the possibility of investigating the effects of psychological and pharmacological treatment on the evolution of symptoms.

By highlighting the these similarities between confabulations and delusions, I do not mean to deemphasise their differences. On the contrary, I reviewed both memory-related confabulation and awareness-related confabulation to emphasise that different mechanisms, by which emotion and motivation may influence cognition, seem to be affected in these symptoms. As I have argued here, the differences between these two confabulation syndromes seem to outweigh the similarities and thus are better explained as related, yet separate pathologies. The same may apply to the relation of confabulation and delusion. It thus seems that a potential effort to establish a neurocognitive homogeneity (even if relative) between all types of confabulations and delusions and form a single explanatory formula may not tally with the rich heterogeneity of confabulations and delusions. Instead, separate theories for different categories of false remembering and false believing, incorporating different motivational and neurocognitive factors and interacting principles, may enhance the sophistication, testability, and pragmatic value of theories.

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