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REVIEW

FMRI contributions to addressing autobiographical memory impairment in temporal lobe pathology

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Abstract

Episodic autobiographical memory (AM) allows one, through the recollection of sensory-perceptual details, thoughts and feelings, to become aware of an event as belonging to one's own past as well as being able to project into one's future. Because AM provides a sense of self-continuity, contributes to the integrity of the self, and helps predicting future experiences, any deficit of AM may have debilitating consequences for everyday life functioning. Understanding AM failure and the underlying neural mechanisms has the potential to shed light on brain reorganization mechanisms and engagement of compensatory processes. Functional magnetic resonance imaging (fMRI) provides the most promising imaging method to tackle these issues. We reviewed evidence from the few studies that used fMRI to investigate the functionality of the residual tissue, the neural reorganization and compensatory mechanisms in patients with neurological conditions due to impaired medial temporal lobe. Overall, these studies highlight the importance of the left hippocampus, which when atrophied and not functional leads to AM deficits but its residual functionality may support relatively normal AM recollection. When damaged hippocampal tissue is not functional, other brain regions (*e.g.*, the medial prefrontal cortex) may be involved to compensate impairment, but they appear generally ineffective to support detailed episodic recollection.

Key words: Functional magnetic resonance imaging; Autobiographical memory; Amnesia; Medial temporal lobe; Memory deficit; Reorganization

Core tip: Functional magnetic resonance imaging investigations of patients with impaired autobiographical memory (AM) can greatly contribute to further our understanding of brain reorganization mechanisms and engagement of compensatory processes after damage to the medial temporal lobe. These investigations are reviewed here. Overall, they highlight the importance of the left hippocampus, which when atrophied and not functional leads to deficits in AM but its residual functionality may support relatively normal AM recollection. When damaged hippocampal tissue is not functional, other brain regions (*e.g.*, the medial prefrontal cortex) may be involved to compensate impairment, but they appear generally ineffective to support detailed recollection.

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INTRODUCTION

Remembering autobiographical memories (AM) involves recollection of contextual information (time and place)

and sensory-perceptual and affective details of personal experiences with a sense of self-awareness[1-3]. AM, not surprisingly, contributes to both one's sense of personal identity (who we are) and sense of self-continuity. Over the last two decades, there has been a growing interest in understanding the neural correlates of normal AM and, more recently, impaired AM. The reasons of the remarkable increase in the number of studies in this topic is, very likely, the mentioned AM contribution to the construction of the sense of self across time, but also the important social role AM plays in the development of new relationships and the nurturing of existing ones, and most particularly, its role as a directive function, where the past serves as a basis for guiding present and future behaviors[4,5]. In the context of the broader issue under consideration in this special topic [with focus on functional magnetic resonance imaging (fMRI)], the present review aims to discuss emerging research that highlights the usefulness of fMRI in the examination of AM in patients with damage to the core memory structures in the medial temporal lobe (MTL). The emphasis of this review is, therefore, on fMRI investigations of AM impairment due to neurological conditions affecting the MTL. The decision to focus on the MTL is driven by evidence that MTL plays a pivotal role in normal AM functioning and its damage typically leads to amnesia for past events[6-9] (but see also[10]) and that fMRI examinations of AM in neurological patients, which to date are limited in number, have been more often reported in the case of patients with damage to the MTL. The underlying question we would like to tackle is to what extent these investigations shed light on the functionality of the residual tissue, the neural reorganization and compensatory mechanisms (either efficient or not) in the case of damage to the MTL. Before discussing the fMRI studies of AM in patients with MTL damage, we introduce AM and summarize the highlights of neuroimaging evidence in healthy participants to provide the context to discuss functional neuroimaging findings in pathology.

THEORETICAL CONSIDERATIONS OF AM

Episodic AM allows one to become aware of an event as belonging to one's own past as well as being able to project into one's future. This sense of self-continuity across time[2,3,11] is grounded in the recollection of sensory-perceptual details, thoughts and feelings. Typically, episodic memory has been distinguished from semantic memory, which refers to general knowledge, knowledge about public facts and people, as well as personal knowledge (*e.g.*, date of birth, the name of our parents and friends). However, these systems are highly interdependent[12] in relation to the self. Turning back to episodic AM, some authors consider that it is a uniquely human system[13] (but see[14] for a different standpoint). Moreover, Tulving's Serial Parallel Independent model places this memory system at the apex of a pyramid, which implies the highest memory achievement in evolution[15]. Tulving *et al*[2] defined AM as consisting of three major constructs: sense of self, autonoetic consciousness and subjectively sensed time. But even more closely related to the self, is Conway's Self Memory System[1,12]. Very briefly, Conway views episodic memories and conceptual autobiographical knowledge as discrete systems that both operate with the "working self" in a bidirectional manner. The working self is conceived as a mechanism that controls access to memories according to the individual's present goals. Importantly, the working self is constrained by the memories and knowledge within the autobiographical knowledge base.

Recently, some studies show that the contribution of AM to the sense of self is not crucial. Klein[16,17] and Klein *et al*[18] presented a series of patients both studied by him and reported in the literature, who have lost the entire fund of episodic memory and who are unable to simulate future personal events, but retain the sense of self. However, a distinction is made between the sense of self, which is preserved in amnesic patients[19] and the sense of self-continuity across time, which depends on AM (Klein SB, personal communication to LM, December, 2013).

Importantly, recollection of past personal experience is considered to be a reconstructive process with memories recreated from their constituent elements. Particularly, autobiographical memories are not static records of the past; rather they are considered as mental reconstructions, which are constrained by two simultaneous, even contradictory, demands: correspondence with the real event and coherence, as time goes by, with the individual's self-image. More precisely, memory reconstruction must reflect reality by providing sensory-perceptive and eventually affective details that represent, as closely as possible, the experience and also be in accordance with the rememberer's current self-image and goals[12]. By making available memories that match current self-beliefs and goals, the main function of AM would be to maintain the integrity of the self[12].

Bearing in mind that AM provides a sense of self-continuity, contributes to the integrity of the self, and helps predicting future experiences, it is not surprising that AM impairment may have debilitating consequences for everyday life functioning. Consequently, understanding AM failure and the underlying neural mechanisms has the potential not only to strengthen the progress of memory research, but importantly, to shed light on brain reorganization mechanisms and eventually to help in planning treatment and in monitoring the effects of therapeutical interventions with the final aim to achieve better management of patients with AM deficits.

FMRI EXAMINATION OF NORMAL AM FUNCTIONING

The advent of fMRI made the examination *in vivo* of different human abilities, in general, possible. More particularly, fMRI (in contrast to earlier neuroimaging techniques, such as positron emission tomography) provides

the most promising new imaging method and offers a number of important advantages in the study of neural correlates of human memory. Among the main fMRI strengths are the improved spatial resolution, the fast speed of data acquisition which allows more flexible experimental designs and the unrestricted number of observations due to the absence of radiation exposure. Moreover, in the last decade, fMRI has undergone a rapid development and provided new ways to design experiments (*e.g.*, event-related, self-paced designs) and to analyze data (*e.g.*, independent component analysis, spatiotemporal partial least squares analysis, psychophysiological interactions analysis, dynamic causal modelling, multi-voxel pattern analysis) allowing segregation of the time-course of memory retrieval processes, examination of the connectivity among brain regions and investigation of memory representation in specific brain regions. This continuous progress has led to improved and refined testing of hypotheses about the neural correlates of both normal and impaired AM.

Evidence from fMRI studies in healthy participants allowed the establishing of a brain network of AM retrieval comprising the MTL, prefrontal cortex (PFC) and posterior cortices[20-22]. Despite the proliferation of functional neuroimaging studies of AM over the last 10-15 years, many of the critical issues (*e.g.*, MTL involvement according to the remoteness of memories, lateralization of the AM networks) continue to be debated, leading nevertheless to greater refinement of the theories derived from the lesion research. For instance, there is a debate regarding the involvement of MTL, especially the hippocampus, in retrieval of personal events according to the age of memories. This debate originated from the lesion studies and is reflected in fMRI studies in healthy subjects. On the one hand, the Standard Consolidation Theory states that memories (without making a distinction between episodic and semantic memories) are initially dependant on the MTL but over time, they undergo consolidation in the neocortical structures and eventually become independent of the MTL[23]. On the other hand, the Multiple Trace Theory postulates a lifelong involvement of the MTL for retrieval of episodic and context-specific memories[6,24]. Recently, the latter has been updated to explicitly include a transformation account of memory, which considers the dynamic nature of memories and suggests that episodic memories may transform to semantic or gist-like versions represented in neocortical areas outside of the hippocampus, but those that continue to contain rich episodic/contextual details remain dependant on the hippocampus[25,26].

MTL

Neuroimaging evidence suggests that the MTL is a crucial node in the AM retrieval network[21,27] involved in binding together the multimodal representations of an episode. Specifically, studies directly comparing autobiographical to semantic memory retrieval revealed greater engagement of the MTL, particularly on the left side[28-30]. Of note, MTL activation observed by some semantic memory studies involving famous people recognition[31,32] could be explained by the association of this semantic information with autobiographical memories[33], as suggested by lesion research[34,35]. As for the MTL's involvement according to age of memories, an increasing number of fMRI studies provided evidence that, when phenomenological qualities and especially vividness are considered, MTL activations are observed for retrieval of rich and vivid and both recent and remote autobiographical memories[36-41], which resonates with the Multiple Trace Theory positing long-life involvement of MTL for vivid context-specific recollections.

Prefrontal cortex

Available evidence also highlights the role of different PFC sub-regions in AM retrieval[22,27]. Specifically, among the PFC sub-regions, the ventrolateral PFC and the medial PFC appear to be systematically linked to retrieval of personal events[27,38].

Lateral PFC: Systematic activation of the ventrolateral PFC during recollection of autobiographical memories is associated with successful memory retrieval, involving initial strategic search and selection of appropriate information[27,42,43]. Specifically, activity in the lateral PFC has been observed early during retrieval[28,29,44], supporting therefore its role in strategic search operations and initial recovery processes consistent with current models of AM[1] that emphasize the reconstructive retrieval of memories. Additionally, the more ventral (orbital) portion of the lateral PFC, part of the frontotemporal junction interconnected through the ventral branch of the uncinate fascicle, has been attributed a crucial role in ecphory (triggering) of memory retrieval[45,46] and synchronisation of emotional and factual components of the personal memories during conscious self re-experiencing[47-49]. The latter is also supported by recent evidence suggesting the involvement of parts of the ventrolateral PFC in enhanced re-experiencing of emotional autobiographical memories[50].

Medial PFC: Activation of the medial PFC is also systematically reported during recollection of autobiographical memories[27] and linked to the role of medial PFC in self-referential processes[51], of which AM is an essential part. Indeed, there is evidence that increased activity in medial PFC distinguished real life AM from laboratory-based episodic memory imaging studies[38,52,53] and its more ventral portions are associated with real self-relevant events[54] and self-perspective[55]. Recently, ventromedial PFC has been found to contain more information about remote memories (although both recent and remote memories are represented there[37]). It should be noted that overall in the neuroimaging research, the medial PFC is linked to a variety of functions[56], such as self-referential[51] and emotional[57] processing, mentalizing[58,59], intuitive assessment of "felt rightness"[60] as well as in some regulatory

mechanisms[61]. Therefore, it can be suggested that medial PFC might in general be supporting processes related to self-awareness and self-regulation.

Posterior cortices

Retrieval of autobiographical memories in healthy participants also leads to activations in the posterior cortices (*e.g.*, precuneus, visual cortices), which are considered to support the multimodal and visual representations associated with the event and visual imagery (and visualized re-experiencing)[20,62]. Specifically, fMRI studies provided evidence that posterior cortices are later involved during (re)construction of autobiographical memories[28,29] to support the retrieval of specific details. For instance, precuneus because of its role in egocentric (view-dependant, relative to the observer) representation of a place, has been thought critical for autonoetic awareness in remembering events from a first person perspective[63]. This is by comparison to MTL, which is involved in allocentric (view-independent) representations[64], but only those that are rich instead of schematic[7].

In summary, fMRI investigations of AM in normal conditions have been very informative in establishing what is called the typical AM brain network, which can be used as a framework for investigation and better understanding of neural correlates of AM impairment.

FMRI INVESTIGATION OF AM IMPAIRMENT IN PATHOLOGIES AFFECTING MTL

Over the past decade, the rapid development of functional neuroimaging techniques and experimental designs (more flexible event-related, self-paced designs, shortened repetition times, new analyzing tools) has made the use of functional neuroimaging protocols in patients possible, which, besides clinical issues, advances our understanding of the neural networks of memory[65,66] and its reorganization in case of damage. The use of functional neuroimaging techniques in brain damaged patients can help to better understand not only how damage alters the neural network supporting AM retrieval, but also potential reorganization of this network through compensatory mechanisms (efficient or not) solicited to cope with memory impairment. It is important to note, however, that the combined use of neuropsychological and neuroimaging methods has advantages over the use of either approach alone[65,66]. The purpose of the review is not to provide an exhaustive literature review of memory impairment due to brain damage and its assessment with neuroimaging in general (for more general reviews on human memory disorders and the application of neuroimaging, we refer the readers to[65,67,68]). Rather the aim is to present and discuss examples of studies that have used fMRI in particular (as the most advanced neuroimaging method and targeted by the scope of the present special topic) to investigate AM impairment in patients with damage to the MTL regions, because of their critical importance for retrieval of specific autobiographical events as highlighted by functional neuroimaging research in normal subjects and lesion research (although the latter still continues to debate the long-life involvement of the hippocampus). Therefore, we will discuss studies that use fMRI protocols to investigate AM in patients with different neurological conditions affecting the MTL and hence, shed light on the functionality of damaged MTL and the potential reorganization of the AM network. We selected the studies based on that they examine AM through fMRI in pathologies with overt damage to the MTL (Table 1). The neurological conditions presented below differ in terms of the age at which they occur, their focal or diffuse nature, and progression.

Developmental amnesia

Developmental amnesia is a memory disorder associated with selective hippocampal damage resulting from hypoxic/ischemic episodes that occur perinatally or early in childhood[69]. Typically, developmental amnesia is characterized by severely impaired episodic AM and relatively preserved semantic memory[70,71], which makes it possible to investigate developmental deficits selectively and to shed light on the neural reorganisation of the AM network due to early life damage. In fact, among the first neuroimaging studies of AM in amnesic patients is the study carried out by Maguire *et al*[72], who used fMRI in the case of a developmental amnesic patient, Jon (initially reported by Vargha-Khadem *et al*[71]). Jon presented with impaired AM (but he was able to recall some personal memories) and had a relatively preserved semantic memory[72]. Jon showed a similar pattern of brain activations to control subjects during memory retrieval but the activations and the interactions among them were different from those observed in controls. Of particular interest, Jon's retrieval of autobiographical events was associated with increased bilateral activity of the hippocampus, in spite of the 50% volume loss bilaterally. Moreover, hippocampus and medial PFC were significantly more activated during retrieval of events for which Jon had clear and conscious recollection (autonoetic consciousness) compared to those he knew but could not remember experiencing. Overall, the findings suggest that the residual hippocampal tissue was functional and contributed to retrieval of the few preserved autobiographical "islands". Moreover, they point out the crucial role of autonoetic awareness during AM retrieval mediated, very likely, by the medial PFC. Therefore, these findings provide insights to mechanisms of brain plasticity[73].

Hypoxia (in adulthood)

Deprivation of oxygen supply (hypoxia) in adulthood also leads to damage to the MTL, specifically the hippocampus, and severe deficit in memory of past events[74-76]. By comparison to patients with developmental amnesia, patients with hypoxic MTL damage in adulthood showed a much more severe pattern of memory impairment. In

Table 1 Summaries of functional magnetic resonance imaging studies investigating autobiographical memory in patients with medial temporal lobe damage

Ref.	Pathology	Patients	Lesion side	Remote memory profile	Compensatory Activations
Addis *et al*[97]	Temporal lobe epilepsy	11 patients	Left	Mild impairment episodic AM	mPFC, posterior medial structures
				Relative preservation semantic AM	mPFC-PHG connectivity
Berry *et al*[124]	Limbic encephalitis	Single-case, Mrs B	Bilateral	Impaired AM (recent events)	Left vlPFC, posterior cortices
Maguire *et al*[72]	Developmental amnesia	Single-case, patient Jon	Bilateral	Impaired AM (few preserved events) relatively normal semantic memory	Functional residual hippocampi mPFC
Maguire *et al*[77]	Hypoxia in late age	Single-case, patient VC	Bilateral	Severe impairment AM	Lateral temporal areas (for personal facts)
Maguire *et al*[110]	Semantic dementia	Single-case, patient AM	Initially left later bilateral	Initially relatively intact AM followed by gradual deterioration	Year 1: Initially functional right and residual left hippocampi, Year2: mPFC, vlPFC, precuneus, Year 3: few occipito-temporal areas
Manning *et al*[98]	Temporal lobe epilepsy	Single-case, patient JR	Left	Preserved AM, impaired public memory	Contro-lesional right MTL, mPFC, posterior cortices
Meulenbroek *et al*[113]	Alzheimer's disease	21 patients	Bilateral	Episodic-to-semantic shift	mPFC, left vlPFC, posterior cortices
Viard *et al*[111]	Semantic dementia	Patients JPL and EP	JPL: bilateral	JPL: impaired AM	JPL: right hippocampus, vlPFC, occipital areas
			EP: bilateral but sparing hippocampi	EP: initially relatively preserved AM	EP: both hippocampi

AM: Autobiographical memory; mPFC: Medial prefrontal cortex; vlPFC: Ventro-lateral prefrontal cortex; MTL: Medial temporal lobe; PHG: Parahippocampal gyrus.

an fMRI examination, Maguire *et al*[77] investigated memory in a patient, VC (initially reported by Cipolotti *et al*[75]), who had MTL damage due to hypoxia in late adulthood. Given that VC did not have reliable memory of personal past events to be investigated in a functional neuroimaging procedure[78,79], only his memory for personal facts and general knowledge were examined. In the context of broadly comparable to control subjects' memory network, VC exhibited increased activity in lateral temporal regions compared to controls and did not show any activity in the residual hippocampi, while hippocampal activations were revealed in controls as well as in developmental amnesic patient Jon for personal facts. These findings suggest that in the case of hypoxic MTL damage in adulthood, deficits of AM are much more severe and could be due to the absence of residual functionality in lesioned hippocampi. Overall, combined together findings from developmental and adult-acquired amnesia due to hypoxia point to the importance of age at which damage occurs, which is of great importance for reorganization and compensatory brain mechanisms. This issue clearly needs further investigation by systematic fMRI examination of patients with damage occurring at different periods of life.

Temporal lobe epilepsy

Temporal lobe epilepsy (TLE) is a chronic neurological condition characterized by partial epileptic seizures originating in the temporal lobe, accompanied usually by hippocampus sclerosis[80] and associated with memory deficit[81]. Overall, memory for the past has been much less investigated than anterograde memory (*i.e.*, acquisition of new information) in patients with TLE. However, in the last decade an increasing number of studies also explored remote memory in TLE patients[82-88]. They revealed that TLE affects remote memory, particularly AM, with left TLE leading to severe AM deficit. Similarly to clinical neuropsychological studies, the majority of the functional neuroimaging studies focused on testing anterograde memory[89,90] and on pre-surgical evaluation to predict post-surgical memory changes[91-96], while only a handful of functional neuroimaging studies examined the neural correlates of AM in patients with TLE[97,98] (see also[99] for patients with transient epileptic amnesia). The studies by Manning *et al*[98] and Addis *et al*[97] presented left TLE patients with different AM profiles, which illustrates the fact that the same disease can lead to different patterns of memory performance and brain reorganization.

In a single-case report, Manning *et al*[98] investigated the interaction between AM and semantic memory in a patient, JR (initially reported by Manning *et al*[100]), who underwent surgical resection of the left MTL for treatment of long-standing TLE with teenage onset of seizures. JR presented a very rare pattern of remote memory dissociation, such as preserved AM and selectively impaired semantic memory for public events and famous people. During retrieval of autobiographical episodes associated with famous people, JR showed increased activations in the intact right MTL (parahippocampal gyrus), several posterior cortices (posterior cingulate cortex, precuneus, temporo-occipital junction) and medial PFC. These findings suggest that contralesional right MTL may be sufficient to adaptively take charge of AM in case of left MTL damage according to the age at which epilepsy

occurred and the developmental course of AM ability, which typically emerges gradually across the preschool years.

Addis *et al*[97] investigated the AM cerebral network in a group of patients with left TLE with significant left hippocampal atrophy and mild AM impairment (reflected in reduction of the episodic details of memories). The authors found that in the absence of significant activation and connections of the residual left hippocampal tissue, retrieval of personal memories in left TLE patients was associated with increased activations in the posterior cortices, including posterior cingulate/retrosplenial and precuneus, right hippocampus (albeit sub-threshold) as well as strong direct connections between the left medial posterior cortices (posterior cingulate/retrosplenial) and left medial PFC, and between left parahippocampal gyrus and left medial PFC. These findings suggest that the AM impairment in left TLE could be due to reduced engagement and connections of the lesioned left hippocampus, compensated to some degree by pathways involving medial PFC and medial posterior cortices, which were insufficient to support detailed episodic-specific recollections.

Overall, the above-mentioned studies provided complementary evidence that depending on the onset of the epilepsy, damage to the left hippocampus can differently affect AM and a different pattern of reorganization of the AM network can be observed, despite several apparent differences between the two studies (single case *vs* group study, after *vs* before surgical treatment). Specifically, in a case of late childhood/teenage occurring epilepsy, right MTL could be sufficient to successfully mediate AM. Otherwise, regions outside MTL could be solicited to compensate left MTL damage, such as medial PFC and medial posterior cortices, which support residual AM (less detailed memories) but appear inefficient to maintain an overall normal level of detailed, episodic-specific AM recollections.

Neurodegenerative diseases

Neurodegenerative diseases are neurological conditions characterized by progressive degeneration and/or death of neuronal cells. Of particular interest in the study of memory are semantic dementia, a form of frontotemporal dementia, and Alzheimer's disease since both involve neurodegenerative processes in the temporal lobes[101,102]. Usually in the memory literature, semantic dementia and Alzheimer's disease present a dissociable neuropsychological memory profile at an initial stage of the disease[103,104]. While semantic dementia is characterized by a profound and amodal loss of semantic memory in the context of relatively preserved episodic AM[105-107], Alzheimer's disease is typically characterized by severe impairment of episodic AM in the context of relative sparing of semantic memory[108,109]. Despite the interesting dissociation within remote memory observed in these two neurodegenerative diseases, there are only a handful of fMRI studies examining AM in patients with semantic dementia and Alzheimer's disease.

Semantic dementia: Only two studies, to our knowledge to date, have investigated the neural correlates of AM in semantic dementia using fMRI[110,111], one of them presenting a longitudinal fMRI follow-up of a semantic dementia patient[110]. The fMRI studies provided evidence of efficient and inefficient compensatory mechanisms, which led to relative initial maintenance of normal level of AM performance and to impaired, namely lacking episodic-specificity AM, performance, respectively.

In a single-case report, Maguire *et al*[110] used fMRI to investigate neural correlates of AM in a semantic dementia patient, AM, as a function of the progression of the dementia at three separate occasions (years 1, 2 and 3). Initially, the patient showed relatively normal AM scores, but with the progression of the disease his AM gradually deteriorated. To begin with, the patient had volume loss in the left hippocampus and left anterior lateral temporal cortex. However, at year 3, the atrophy encompassed the temporal lobes bilaterally, including both hippocampi. As for changes through time of the AM retrieval network, initially at year 1, the patient exhibited increased activations in regions of the consensual AM network, including the intact right hippocampus, and importantly increased activation of the remnant left hippocampus, which was not further observed during the following fMRI examinations (years 2 and 3). Moreover, at year 2, the patient showed increased activations of ventromedial PFC and precuneus, among other brain regions, to finally end up at year 3 with disengagement of the AM network, except for the occipitotemporal cortices. These findings reveal how the progression of dementia and MTL atrophy could affect AM retrieval and the associated neural correlates. Initially, despite the volume loss in the left hippocampus, the residual hippocampal tissue was still functional and therefore could support relatively preserved recollection of personal events. Over the course of the dementia process, the hippocampus became inactive and it seemed to be compensated by increased activity in the medial PFC and precuneus, which, with the progression of the dementia, appeared in turn to become non-operational.

Viard *et al*[111] used fMRI to investigate AM according to the remoteness of memories in two semantic dementia patients, JPL and EP, with different patterns of hippocampal atrophy and AM profiles. While JPL presented with impaired AM recollections (reflected in reduction of specific episodic details) and severe atrophy of both hippocampi, EP presented with initially preserved AM recollections with greater reliance on visual imagery than healthy controls and relative preservation of both hippocampi, despite atrophy in adjacent temporal cortices. In terms of brain activations and interactions, while JPL exhibited less activity in the left anterior hippocampus (remote memories) and increased activity in the right posterior hippocampus, functionally connected with the posterior occipital cortices, EP exhibited increased activity in both left and right anterior hippocampi (for both recent and remote memories), which were functionally connected to each other. These findings suggest that

atrophy together with absence of functionality in the residual hippocampal tissue might explain impaired AM, suggesting that activation in right posterior hippocampus and interactions with occipital cortices may have been recruited to compensate left hippocampal deficit, but that this compensatory mechanism was insufficient to support a normal level of rich episodic-specific recollections.

Overall, evidence from fMRI studies of AM in semantic dementia patients highlights the importance of the left hippocampus in retrieval of vivid and specific autobiographical memories, which when atrophied and not functional leads to impaired AM. In the case of late age and progressive damage of MTL, initial functionality of the remnant left hippocampus, rather than the right hippocampus, could support a relatively normal level of AM performance at an early stage of the disease. Subsequent increased engagement of medial PFC and precuneus could be seen as a compensatory mechanism reflecting attempts to maintain AM, although it had gradually declined. It could be also speculated that precuneus involvement could reflect retrieval processes that are based on a more egocentric representation and greater reliance on a self-referential perspective during recollection.

Alzheimer's disease: Episodic to semantic shift is usually reported in patients with Alzheimer's disease and deficit in episodic AM recollection is the hallmark of the disease, even being detected at the very preliminary stage of the disease, known as amnesic mild cognitive impairment[112]. The pattern of "semantization" of episodic AM has been observed in a group of 21 patients diagnosed with early stage, probable Alzheimer's disease who were examined using fMRI[113]. Specifically, these patients presented with a decline in episodic recollection of personal experiences, which contained more semantic and repetitive information and also atrophy in both hippocampi. At the level of brain activations, patients with Alzheimer's disease showed increased activity in ventromedial PFC, left ventrolateral PFC and posterior cortices (lingual gyrus and precuneus). Moreover, increased activity in ventromedial and ventrolateral PFC was linked to decreased volume in the hippocampus. These findings suggest that increased engagement of ventromedial and ventrolateral PFC could reflect a compensatory mechanism supporting retrieval of less detailed and more "semantized" autobiographical memories (*i.e.*, episodic-to-semantic shift in the quality of recollection), very likely relying on some kind of self-involvement.

Encephalitis

Encephalitis is a neurological condition characterized by an acute inflammation of the brain, generally caused by a virus or autoimmunity (*e.g.*, herpes encephalitis, limbic encephalitis). There is usually extensive damage to the temporal lobes, including the medial temporal regions[114-116] and extending to the PFC[117,118], although not necessarily[119], and a severe memory impairment[114]. More specifically, neuropsychological research provides evidence of retrograde amnesia, particularly for autobiographical events[116,120-123]. Despite evidence that encephalitis severely affects retrograde memory, especially AM, and may lead to interesting dissociations in relation to the side of damage[120], fMRI investigations of AM in encephalitic patients are very rare[124]. In a single-case fMRI study, Berry *et al*[124] examined the neural correlates underlying "rehearsed" (reviewed) personal episodes in a woman, Mrs B, diagnosed with limbic encephalitis five years before the neuroimaging investigation and presenting with impaired memory for autobiographical events. The patient used a wearable camera, SenseCam (Microsoft Research, Cambridge) to recode images during personal events, and then reviewed the images approximately every two days during three weeks. During scanning, Mrs B viewed rehearsed SenseCam images, together with never reviewed and new images as well as events recorded in a written diary and also rehearsed every two days during three weeks. At the behavioral level, the patient showed better performance for "rehearsed" SenseCam images, which at the neural level was associated with increased activity in the left ventrolateral PFC, lateral temporal, parietal and occipital regions in the absence of MTL activations. This study suggests a potentially effective way of alleviating AM deficit with a rehearsal-based training using visual material and supported by frontal and posterior activations, which very likely reflects a more general recognition of the event rather than detailed specific recollection, especially given that during scanning, events were not remembered in detail but just recognized (as known or familiar). Further investigation involving detailed recollection of personal events would help better understand the effects of training procedures on AM brain network.

Summary

Altogether, the above-presented fMRI studies in patients with MTL damage highlight the importance of the left hippocampus, which when atrophied and not functional leads to deficits in AM. Available fMRI evidence suggests that atrophy in the left hippocampus does not necessary lead to alteration in its activation pattern and to severe AM impairment; namely, residual functionality in the damaged hippocampus may underpin relatively normal AM recollection. When residual hippocampal tissue is not functional, other brain mechanisms come into play to compensate its silence. In some cases, engagement of contralesional MTL structures could be sufficient to support AM, but very likely only in some circumstances, dependent on the age at which damage occurred. In other cases, PFC, more often medial PFC, and posterior cortices could support compensatory processes engaged to deal with the AM deficit, but not necessarily always efficient to support rich and detailed recollections (Table 1). Given the evidence that medial PFC has been associated with processes related to the self[51] and contains more information for remote memories[37] in healthy subjects, it could be speculated that involvement of medial PFC in case of damage could reflect retrieval of more stable

gist-like aspects containing less episodic details of the memories (rather than true detailed episodic-specific recollection supported by hippocampus) accompanied by the overall sense of self-involvement.

CONCLUSION

Overall, the present review of fMRI studies in patients with AM impairment due to damage in MTL core memory structures summarizes the importance of fMRI data in providing insights on how brain damage affects the neural network supporting retrieval of autobiographical memories and how the brain appears to cope with damage by engaging compensatory mechanisms, which can either be efficient or not, so to mediate AM recollection. FMRI may supply additional information over that provided by neuropsychological assessment and structural MRI and combining them together during examination of brain damaged patients would lead to a better and more reliable understanding of memory disorders and the underlying brain activations pattern and, ultimately, better management of the patients. The studies discussed in the present review, presenting different pathological entities affecting MTL and associated with different patterns of AM loss, provide relevant theoretical and clinical information that can guide future functional neuroimaging research of memory impairment. Although the present review focused on the use of fMRI in patients with MTL damage, for completeness, it should be noted that fMRI has started to be used in other pathologies characterized by AM impairment, some of which we briefly enumerate below.

Traumatic brain injury

Traumatic brain injury (TBI) has usually been associated with impaired AM[125-127] together with diffuse axonal injury mainly affecting the connection between frontal and temporal regions[126,128]. Given the diffuse nature of damage, TBI presents a challenge in understanding impairment of AM, which might be linked to a more general deficit in executive functions and alteration of the sense of self. A recent single-case fMRI investigation[129] of a TBI patient, ML (initially reported by Levine *et al*[125]), revealed decreased involvement of the medial prefrontal and posterior cortices for recently encoded personal events, of which retrieval is lacking specificity and autonoetic awareness[125]. This finding underscores the link between autonoetic awareness and medial PFC, which could be involved as a compensatory mechanism only when autonoetic awareness is relatively preserved.

Psychogenic amnesia

While all the above-presented findings underscore the importance of considering fMRI examination in patients with AM impairment due to overt brain damage, it should be also mentioned that fMRI can be used in patients without overt brain damage but who present with a specific deficit in AM, such as psychogenic amnesia (known also as dissociative amnesia)[130,131], which affects the ventrolateral PFC[132] associated with retrieval of emotional memories (see above) and is linked to reduced MTL engagement[133].

Affective disorders

Dysfunctions of AM are also widely reported in affective disorders, such as depression and post-traumatic stress disorder (PTSD)[134-137], which are characterized by intrusions of memory of the traumatic event, bias toward negative memories and an overgeneralization (lacking specificity) of retrieval[138,139]. Recently, there are some studies examining the neural correlates of AM in depression[140,141] and PTSD[142,143], which highlight the abnormal involvement of the PFC and/or emotion-related MTL region, *i.e.*, amygdala. Overall, the fMRI investigations in psychogenic amnesia and affective disorders could also be very informative regarding the interplay between memory and emotion, which has long been neglected, while there is no doubt of emotional and motivational influences on AM.

Psychiatric disorders: The case of schizophrenia

Schizophrenia has been also associated with impaired AM[144-146] in the context of deficits in several cognitive operations (perception, memory) and emotional processing, caused very likely by a more general cognition – emotion disintegration[147]. FMRI examination of AM in schizophrenia revealed an abnormal pattern of activation and correlations with memory performance in the PFC and striatum, respectively[148]. Further fMRI investigations are needed to clearly understand AM deficits in schizophrenia and its link to a more general disturbance at the level of emotion-cognition interaction.

Finally, we would like to mention that it would also be of great use for investigating the effects of cognitive based training programs. Specifically, fMRI can be used before and after training programs to establish beneficial changes in neural activations leading to improved AM[149]. Investigations of this sort are remarkably scarce in the AM research, but hopefully they will emerge in the near future and provide new opportunities to understand reorganization of brain network activation and brain plasticity.

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