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Review Article

Autobiographical memory dysfunctions in depressive disorders

Monika Talarowska, PhD,[1]* Michael Berk, MD,[2,3] Michael Maes, MD[2,4,5] and Piotr Gałecki, MD[1]

[1]*Department of Adult Psychiatry, Medical University of Lodz, Lodz, Poland,* [2]*IMPACT Strategic Research Center, Barwon Health, Deakin University, Geelong,* [3]*Orygen, The National Centre of Excellence in Youth Mental Health, Department of Psychiatry and The Florey Institute of Neuroscience and Mental Health, The University of Melbourne, Melbourne, Australia,* [4]*Department of Psychiatry, Faculty of Medicine, Chulalongkorn University, Bangkok, Thailand, and* [5]*Health Sciences Graduate Program, Health Sciences Center, State University of Londrina, Londrina, Brazil*

Autobiographical memory (AM) is a ubiquitous human experience that belongs to long-term declarative memory. It plays interpersonal and intrapsychic functions. The main aim of this study is to present results of contemporary research on AM in recurrent depressive disorders. The available research literature suggests that AM dysfunctions are a precursor and risk factor for recurrent depressive disorders and that they also appear to be a consequence of depressive symptoms in a bidirectional and interacting manner. These data suggest that AM might be a viable therapeutic target for cognitive remediation strategies, given the impact of cognition on diverse clinical outcomes.

Key words: autobiographical memory, depression, diagnosis, etiology, symptoms.

RUMINATIONS OF A negative emotional origin are one of the characteristic features of ongoing depressive states. As early as in 1991, Nolen-Hoeksema and Morrow[1] considered them both a possible cause as well as a consequence (symptom) of depression. Ruminations in patients suffering from recurrent depressive disorders (rDD) most often concern recollections from autobiographical memory (AM).[2,3]

AM is a common feature for every human. It belongs to long-term declarative memory. It includes episodic elements (knowledge such as 'I remember that ') and semantic elements (knowledge such as 'I know that '), and hence enables the storage of information about our lives for a long time. Episodic elements dominate over semantic elements in AM. The first refer to events that took place in a specific time and spatial context. They include conscious and specific memories concerning one's own past, sensory and visual events as well as thoughts and feelings accompanying them. The semantic component of AM does not depend on time and is connected with the so-called noetic consciousness. Owing to that, we are able to recreate general facts about the world and personal events without the necessity of experiencing again the specific context, as opposed to the case with the episodic element.[4,5]

AM plays both interpersonal (establishing and maintaining relationships with others) and intrapsychic roles.[6] Efficient operation of AM provides the feeling of stability with respect to ourselves and the world that surrounds us. Moreover, it enables us to devise more or less detailed plans for the future, make them come true and efficiently solve problems that appear on the way (thanks to the memories of similar events from the past stored in memory).[7]

The main aim of this study is to present results of contemporary research on AM in rDD. We will also make an attempt to answer the question included in

*Correspondence: Monika Talarowska, PhD, Department of Adult Psychiatry, Medical University of Lodz, Aleksandrowska 159, 91-229, Lodz, Poland. Email: talarowskamonika@wp.pl
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the title of this article: Are AM dysfunctions a cause or perhaps a consequence of depression symptoms?

SEARCH STRATEGY

For the sake of this narrative review, we carried out a comprehensive search in the PubMed/MEDLINE electronic databases from the very beginning to 1 July 2015. The terms we looked for included: 'autobiographical memory,' 'amygdala,' 'hippocampus' and 'HPA-axis' cross-referenced with 'depress*.' We only took into consideration articles written and published in English. Articles were taken into account and included in our study based on their overall quality of methodology. Moreover, relevant meta-analyses were included.

AM IN RECURRENT DEPRESSIVE DISORDERS

AM dysfunctions are observed in a variety of mental disorders, such as post-traumatic stress disorders,[8] schizophrenia[9] and rDD.[8]

The information stored in AM concerns ourselves and has a strong impact on our well-being; additionally, it determines – to a large extent – the types of actions we undertake in the future.[10] When we recall unpleasant events, we update in our memory other unpleasant events and unpleasant emotions associated with them. A similar phenomenon is observed in the case of memories with a positive emotional charge. Therefore, the executive mechanisms present in AM steer not only the operation of this very memory but also take part in the regulation of emotional processes.[11] We may make attempts to reduce or increase the level of emotional tension by 'blocking' specific recollections in our memory,[12] yet finding an answer to the question concerning effectiveness of such actions is another issue.

Excessive generation of negative recollections may be one of the predictive factors for depressive disorders.[13] This dependency may also have a different character – examined individuals who had undergone at least one episode of depression in the past related to significantly fewer positive recollections from their lives as compared to healthy subjects.[14]

The expression of overgeneral AM (OAM) was introduced in recent years with the aim of underscoring the distinct nature of recollections stored in AM of people affected by rDD.[15] They are less detailed and specific autobiographical memories, which are characterized by excessive generalization.

Specific autobiographical memories refer to events that happened in a specific place and in a specific time, and did not last longer than 24 h (e.g. 'I spent the last Saturday of June in Krakow with my friends'). Overgeneral memories refer to fairly often repeated situations ('I go to church every Sunday') or to situations that last longer than 1 day ('I was in second grade of middle school last year').[16] In response to an emerging stimulus, patients with symptoms of depression recollect overgeneral memories more often than the specific ones.[3] This concerns mainly negative memories. In response to positive and neutral stimuli, the affected find significantly fewer specific memories than healthy people.[17] Moreover, they have difficulties in reconstructing details of events, even the ones that are significant to them.[18]

OAM is considered both a risk factor for the occurrence of rDD symptoms throughout life and a factor that makes a prognosis of the disease worsen.[19,20] Based on the experiments conducted by Hermans *et al.*,[21] the frequency of recollecting OAM from declarative memory is a better predictor for the course of the disease than the severity of the first episode, the patient's self-assessment or the presence of negative convictions about oneself and the world. People with depression also tend to refer to personal memories from the perspective of an observer, not a participant of events (actor).[22] This phenomenon mainly concerns memories of a positive emotional charge.[18] According to Libby and Eibach,[23] a change of perspective (observer–actor) takes place when memories are not in conformity with our self-assessment at the time of recollecting them from the memory (recollections have a positive emotional charge but self-assessment of the affected is reduced). A negative image of oneself is a feature of psychological functioning, which has been confirmed many times and described in patients suffering from depression.[24] Difficulties in formulating personal objectives, which could be significant for them in the future[25] and could undoubtedly support the process of treatment, are consequences of OAM in people affected by depression. Therefore, the presence of OAM reduces chances of the applied therapy to be successful and effective.

OAM were also confirmed in patients with rDD who – at the time of examining – were in the stage of disease remission.[26–28] Young *et al.*[28] proposed that dysfunctions of AM should be treated as a constant

feature of cognitive functioning in the people treated due to diagnosed depression, observed both before the occurrence of the first symptoms and during remission.

It turns out that OAM are observed in the people suffering from rDD regardless of age[29] and are probably independent of culture.[30] According to Woody *et al.*,[31] the OAM are also found in the children of mothers who are treated due to depression (in response to verbal stimuli of a negative emotional charge). The presence of OAM in adolescence is also a predictor of depression development in adulthood.[13,32] What is more, the examined women recollected from AM much more negative memories than positive as compared to a group of men selected carefully in terms of age and education.[17] This fact is one of the explanations for the predominance of women over men in the incidence of depression symptoms.

Additionally, it is worth mentioning the experiments carried out by Söderlund *et al.*[33] According to the authors, deterioration of autobiographical episodic memory (facts from life), but not of semantic memory (common knowledge), is observed in patients suffering from depression. The dysfunctions concerned the time interval from 2 weeks to even 10 years before the experiment! The patients recollected events from public life to a smaller extent than the comparative group, which is presumably caused by the fact that they paid much less attention to them than healthy subjects.

Interestingly, OAM are characteristic not only for people affected by depression, but they were also confirmed in patients with post-traumatic stress disorder, among people considering suicide attempts,[34] in patients with nutrition disorders,[35,36] in people suffering from chronic pain[37] and in people diagnosed with borderline personality disorder.[38] The area of research over AM has been continuously expanding in recent years.

NEUROANATOMICAL FOUNDATION OF AM

A series of structures of the human brain are engaged in the processes of AM as a type of declarative memory. They are mainly medial parts of the temporal lobes, the diencephalon and the frontal lobes (the hippocampus, the thalamus, the amygdala nucleus and the prefrontal area).[39] Interestingly, dysfunctions or neuroanatomical changes in each of those regions are also considered characteristic for the course of depression.[40] Each of the aforementioned structures is also involved in the organism's response to stressful stimuli.[41]

The hippocampus

The hippocampus formation plays a significant role in memory and learning processes as well as in fast and adequate-to-a-given-situation reactivity to changes of the environment.[42] The main objective of medial parts of the temporal lobes is integration of a series of information (regarding objects and their mutual spatial relationships), which guarantees that our memory is consistent.[43]

The activity of the hippocampus and the parahippocampal cortex (measured with the use of blood-oxygen-level dependent functional magnetic resonance imaging) during recollection of memories from AM is significantly lower in patients with symptoms of depression as compared to a control group of healthy subjects.[44] The quoted results were confirmed several years later by a team of scientists from New Zealand.[2]

In subsequent studies conducted by Young *et al.*,[45] the volume of the left hippocampus correlated positively with the amount of specific autobiographical memories in a group of patients with symptoms of depression. An inversely proportional relationship was observed for the volume of the left hippocampus and the amount of overgeneral memories. This phenomenon may also be observed among healthy subjects with the familial risk of rDD occurrence.[45]

The frontal lobes

Deterioration of executive functions is considered one of the possible causes of intrusive ruminations and OAM in the course of depression.[12] Frontal dysfunctions in patients with rDD have been described for many years. Their characteristic features include: deterioration of the ability to inhibit unpleasant contents, reduced capacity of working memory, reduced ability to update and keep memories significant for action in working memory, as well as deterioration of verbal fluency and cognitive elasticity.[19] We observed the aforementioned deficits in many research studies and experiments conducted by us.[46] OAM are probably a consequence of frontal functions deterioration, which is linked with difficulties in inhibiting overgeneral memories.[12]

Theoretical assumptions were confirmed in neuroimaging examinations. Young *et al*.[16] indicated that in the case of recollecting both positive and negative memories from AM in a group of people with symptoms of depression, reduced activity of the dorsomedial prefrontal cortex (DMPFC) and increased activity of the dorsolateral prefrontal cortex (DLPFC) were observed. Similar results to the ones presented above were obtained by Sperduti *et al*.[6] Moreover, alongside OAM, the effectiveness of frontal functions, mainly verbal fluency, was reduced.[47] Interesting research findings were presented by Stoessel *et al*.[48] They evaluated the activity of the cingulate gyrus when recollecting autobiographical memories in 12 people in happy relationships and in 12 people who were unhappily in love (the experiment was conducted shortly after their relationships had ended). When recollecting autobiographical memories, a reduced activity in the cingulate gyrus was observed in the people who ended their relationships in comparison to the control group. The unhappily in love subjects also recorded increased results using the Beck Depression Inventory.

It is important to remember that one of the objectives of DMPFC is to control emotions, while DLPFC is responsible for the verification of conformity of memories of events that actually took place. The area of the prefrontal cortex and the callosal gyrus take part not only in the regulation of cognitive processes, but also mood, behavior and social relations, which may serve as a confirmation for the assumptions of the so-called mind theory.[49,50] Therefore, in the case of patients with rDD, working memory deterioration is connected not only with difficulties in planning of actions, undertaking a purposeful activity or with reduction of mental plasticity, but also affects increased sensitivity of the affected to negative feedback and difficulties in inhibiting and avoiding such type of data.[51]

The amygdala nucleus

The amygdala nucleus is a part of the limbic system, which gives stimuli an emotional meaning, depending on previous experiences (emotional memory), in particular the negative ones. It is also involved in detecting a stressor and assigning a proper meaning to it. In response to incoming stimuli, it activates appropriate behavior patterns and physiological reactions, which enable the taking of a proper decision.[52]

Regulation of emotions takes place in three collaborating areas of the brain. The structures of the brain stem are responsible for the most elementary, innate and unconscious impulsive reactions (excitation versus inhibition, autonomous reactions). The limbic system, including mainly the hippocampus and the amygdala nucleus, modifies our emotional reactions depending on the incoming environmental stimuli (unconscious reaction). On the other hand, the prefrontal cortex is responsible for control over emotions and feelings (realized emotions) (Table 1).[53]

In patients with rDD, increased activation of the amygdala nucleus and reduced activity of the frontal lobes are observed in response to negative stimuli.[52] On the other hand, the response of the amygdala nucleus to stimuli with a positive emotional charge is inhibited.[54] An inverse relationship can also be observed in the period of disease remission.[54] The right amygdala nucleus seems to be involved in automatic and fast detection of stimuli with emotional charges, whereas the left amygdala nucleus is responsible for a detailed analysis of such stimuli. According to Drevets,[55] an excessive activity of the left amygdala nucleus in response to negative stimuli is observed in patients with rDD. The activity of the right amygdala nucleus increases with the observed positive reaction to antidepressant treatment.[56]

FUNCTION OF THE HYPOTHALAMIC–PITUITARY–ADRENAL AXIS

The hypothalamic–pituitary–adrenal axis (HPA), referred to as the stress axis, plays a fundamental role in the body's response to stress stimuli – both psychological and physical. Its action has an adaptive character. The main objective of the HPA axis is to restore homoeostasis of the organism. This system coordinates and controls secretion of glucocorticosteroids from the adrenal cortex to blood.[57]

Dysregulation of the HPA axis is also said to be one of the possible causes for the occurrence of symptoms of depression.[58] In terms of pathophysiology, depressive disorders resemble chronic stress. Dysregulation within the HPA axis is observed in 50–75% of patients with diagnosed depression.[59] The following symptoms are diagnosed: increased concentration of glucocorticoids in plasma, urine and cerebrospinal fluid; changes in the daily profile of glucocorticoids

Table 1. Emotion regulation in the brain[†]

Brain stem: Medulla oblongata, pons, mesencephalon	Ascending pathways: Stimulation of cerebral cortex; Sleep versus wake (coma). Descending pathways: Homoeostasis; Regulation of autonomous reactions to stimuli causing fear, anger, sex drive; Motor regulation (escape reaction, sexual behavior, mimics, gestures, behavior aimed at getting food).
Limbic system: Amygdala, thalamus, hippocampus	Receiving, evaluating and reacting to stimuli important for survival. Hippocampus: Regulation of cognitive and emotional processes; Integration of information incoming from the organism and the environment; 'Mapping' of spaces, i.e. encoding the location of stimuli in terms of current needs of the organism.
Cerebral cortex	Two systems for processing of emotional stimuli: 1. Cingulate cortex – commissure – hippocampus: Analysis of meaning of new stimuli; 'Motivational map of space' for collection of emotionally important stimuli. 2. Temporal pole – insular cortex – ventral prefrontal cortex: Coordination of incoming information; Adjusting behavior to requirements of the environment.

[†]Data in this table are from the study by Hallam *et al.*[53]

secretion with more frequent and longer periods of secretion; increased secretion of glucocorticoids in response to adrenocorticotropic hormone; and an increase in the volume of hypophysis and adrenal glands.[57] Dysregulation of the HPA axis is found significantly more often in patients with a greater number of episodes and a longer period of disease.[60]

The hippocampus is a structure of the brain that is particularly susceptible to stress-induced functional changes and dysregulation of the HPA axis. In such cases, the following are observed: a drop in the expression of the brain-derived neurotrophic factor (BDNF), deterioration of long-term potentiation (LTP) and inhibition of neurogenesis in the dentate gyrus (DG).[61] Long-lasting activation of the HPA axis results in an inflow of an excessive amount of calcium ions to hippocampal neurons, which may lead to their necrosis in the process of programmed cell death (the so-called apoptosis). This phenomenon is clearly found among individuals suffering from depressive disorders.[62]

Stress affects the course of cognitive processes. Its mild intensification facilitates remembering; however, strong stressors applied during training deteriorate the process of memorizing information.[63] The relations between dysregulation within the HPA axis and deterioration of AM in patients with rDD are described, among others, by Schlosser *et al.*[64] OAM are observed also in people who have gone through traumatic experiences in their lives. Volume reduction of the grey matter in the area of the medial prefrontal cortex and the left temporal gyrus is also observed in this group.[65]

Moreover, according to Anderson *et al.*,[15] a small amount of specific autobiographical memories may be a predictor of symptoms of depression in people who experience stressful life events. These reports and findings are also confirmed by Aglan *et al.*[66] and Hamlat *et al.*[3] On the other hand, Rosenbach and Renneberg[38] claim that high sensitivity to rejection, which is a consequence of numerous traumatic experiences in relationships with the closest family in childhood, predisposes to dysfunctions of AM.

NEUROTRANSMISSION DISORDERS AND FURTHER DIRECTIONS OF STUDIES

Various works dedicated to the links between the particular neurotransmitter system and AM deterio-

Table 2. Main studies on autobiographical memory

Nolen-Hoeksema and Morrow[1]	1991	Ruminations with a negative emotional background as a constant feature of rDD.
Anderson *et al.*[15]	2009	The introduction of the concepts of OAM and specific autobiographical memories.
Young *et al.*[17]	2012	In response to a stimulus (mainly negative), depressive patients more
Hamlat *et al.*[3]	2015	often recall memories in the generalized form rather than the specific one.
Hermans *et al.*[21]	2008, 2012,	OAM is considered a risk factor for the occurrence of rDD symptoms
Sumner[19]	2013	during a lifetime as well as a factor that makes prognosis in the
Sumner *et al.*[20]		course of this disease worse.
Mackinger *et al.*[26]	2000	The presence of OAM in patients with rDD, who at the time of
Spinhoven *et al.*[27]	2006	examination were in the phase of disease remission.
Young *et al.*[28]	2014	
Marroquín and Nolen-Hoeksema[70]	2015	Prospective memory in patients with rDD.

OAM, overgeneral autobiographical memory; rDD, recurrent depressive disorders.

ration in people affected by rDD have been published in recent times.[67]

Sumner *et al.*[68] investigated whether a polymorphism in the promoter region of the serotonin transporter gene (5-HTTLPR) was associated with OAM in interaction with a lifetime history. There was a significant interaction between the 5-HTTLPR genotype and a lifetime history of major depressive disorder (MDD) in predicting OAM. Among S allele homozygotes, MDD history was associated with greater OAM, whereas no significant relationship between MDD history and OAM emerged among L carriers. Furthermore, there was evidence that a greater number of S alleles were associated with greater memory specificity in individuals without a history of depression. However, the impact of the noradrenergic system on processes of AM has not been confirmed.[69]

The issues of prospective memory in patients with rDD, orientation on the future and emotional prognostication of the future (predicting what may happen in the future and how I will feel then) are considered interesting areas of further research studies and experiments (Table 2).[70]

SUMMARY

After analyzing the available literature and results of experiments and studies conducted in recent times, it is possible to answer positively the question presented in the introduction to this paper. Yes, AM dysfunctions are a cause of rDD, and yes, they are also a consequence of depression symptoms. It seems that we are dealing with a circle of mutual relationships that requires further analyses. It is important to suggest the necessity for carrying out additional research work to determine the importance of AM in the development and course of rDD.

The presented findings seem to confirm that rDD should be treated as a systemic condition, instead of concentrating on examining isolated systems or brain regions that are responsible for them.

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DISCLOSURE STATEMENT

There is no a conflict of interest.

AUTHOR CONTRIBUTIONS

M.T. conceived and designed the study and drafted the work. P.G., M.B., M.M. drafted the work and critically revised it for important intellectual content.

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