# Autobiographical memory dysfunctions in depressive disorders

Autobiographical memory in depression

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**Abstract**

Autobiographical memory is a ubiquitous human experience which 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 autobiographical memory in recurrent depressive disorders (rDD). The available research literature suggests that autobiographical memory dysfunctions are a precursor and risk factor for rDD and that they also appear to be a consequence of depressive symptoms in a bidirectional and interacting manner. These data suggest that autobiographical memory 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

## Introduction

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]

Autobiographical memory 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...*"), hence enables to store information about our lives for a long time. Episodic elements dominate over semantic elements in autobiographical memory. 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 autobiographical memory 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 autobiographical memory 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 autobiographical memory in recurrent depressive disorders (rDD). We will also make an attempt to answer the question included in the title of this article. Are autobiographical memory 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 the 1st of 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.

**Autobiographical memory in recurrent depressive disorders**

Autobiographical memory dysfunctions are observed in a variety of mental disorders, like Post-Traumatic Stress Disorders (PTSD)[8], schizophrenia[9] and rDD[8].

The information stored in autobiographical memory 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 autobiographical memory 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 underwent at least one episode of depression in the past, relate to significantly fewer positive recollections from their lives as compared to healthy subjects.[14]

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

Specific autobiographical memories refer to events which happened in a specific place and in a specific time, and did not last longer than 24 hours (e.g. *I spent the last Saturday of June in Krakow with my friends*). Overgeneral memories refer to fairly often repeated situations (e.g. *I go to church every Sunday*) or to situations that last longer than one 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 which makes a prognosis of the disease worsen.[19,20] Based on the experiments conducted by Hermans et al.[21], the frequency of recollecting overgeneral autobiographical memories from declarative memory is a better predictor for the course of the disease than the

severity of the first episode, patient's self-assessment or the presence of negative convictions about oneself and the world. The 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.

OAMs were also confirmed in patients with rDD who – at the time of examining – were in the stage of disease remission.[26,27,28] Young et al.[28] proposed that dysfunctions of autobiographical memory 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 OAMs 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 OAMs 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 autobiographical memory 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.

What is interesting, OAMs are characteristic not only for people affected by depression, but they were also confirmed in patients with PTSD, 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 autobiographical memory has been continuously expanding in recent years.

**Neuroanatomical foundation of autobiographical memory**

A series of structures of the human brain are engaged in the processes of autobiographical memory 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] What is interesting, dysfunctions or neuroanatomical changes in each of those regions are considered characteristic also 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 parahipocampal cortex (measured with the use of blood oxygen level dependent fMRI, BOLD) during recollection of memories from autobiographical memory 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] OAMs 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 both in the case of recollecting positive and negative memories from autobiographical memory 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 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 (BDI).

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, behaviour 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 behaviour patterns and physiological reactions, which enable to take 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 (realised emotions) (Table 1).[53]

Table 1.

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 Drvetes[55], an excessive activity of the left amygdala nucleus in response to negative stimuli is observed in the patients with rDD. The activity of the right amygdala nucleus increases with the observed positive reaction to antidepressant treatment.[56]

**Function of the HPA 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 control secretion of glucocorticosteroids from the adrenal cortex to blood.[57]

Dysregulation of the hypothalamic-pituitary-adrenal 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% to 75% patients with diagnosed depression.[59] The following symptoms are diagnosed: increase concentration of glucocorticoids in plasma, urine and cerebrospinal fluid, changes in the daily profile of glucocorticoids secretion with more frequent and longer periods of secretion, increased secretion of glucocorticoids in response to ACTH, 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 the disease.[60]

The hippocampus is a structure of the brain which 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

memorising information.[63] The relationships between dysregulation within the HPA axis and deterioration of autobiographical memory in patients with rDD are described, among others, by Schlosser et al.[64] OAMs are observed also in people who went 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] or 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 autobiographical memory.

**Neurotransmission disorders and further directions of studies**

Various works dedicated to the links between particular neurotransmitter system and autobiographical memory deterioration 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 5-HTTLPR genotype and a lifetime history of 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 autobiographical memory was not 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]

Table 2.

**Summary**

After analysing 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 – autobiographical memory 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, which requires further analyses. It is important to suggest a necessity for carrying out additional research work to find out the importance of autobiographical memory 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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MT: Conception and design of the study, drafting the work

PG, MB, MM: Drafting the work and revising it critically for important intellectual content

## References


1. Nolen-Hoeksema S, Morrow J. A prospective study of depression and posttraumatic stress symptoms after a natural disaster: the 1989 Loma Prieta Earthquake. *J Pers Soc Psychol.* 1991; **61(1)**: 115-121.
2. Hach S, Tippett LJ, Addis DR. Neural changes associated with the generation of specific past and future events in depression. *Neuropsychologia*. 2014; **65**: 41-55.
3. Hamlat EJ, Connolly SL, Hamilton JL, Stange JP, Abramson LY, Alloy LB. Rumination and overgeneral autobiographical memory in adolescents: an integration of cognitive vulnerabilities to depression. *J Youth Adolesc.* 2015; **44(4)**: 806-818.
4. Gilboa A. Autobiographical and episodic memory – one and the same? Evidence from prefrontal activation in neuroimaging studies. *Neuropsychologia* 2004; **42**: 1336-1349.
5. Piolino P, Desgranges B, Eustache F. Episodic autobiographical memories over the course of time: cognitive, neuropsychological and neuroimaging findings. *Neuropsychologia* 2009; **47**: 2314-2329.
6. Sperduti M, Martinelli P, Kalenzaga S, et al. Don't be too strict with yourself! Rigid negative self-representation in healthy subjects mimics the neurocognitive profile of depression for autobiographical memory. *Front Behav Neurosci.* 2013; **7**: 41.
7. Kyung Y, Yanes-Lukin P, Roberts JE. Specificity and detail in autobiographical memory: Same or different constructs? *Memory.* 2015; **16**:1-13.
8. Ono M, Devilly GJ, Shum DH. A meta-analytic review of overgeneral memory: the role of trauma history, mood, and the presence of posttraumatic stress disorder. *Psychol Trauma.* 2015 May 11. [Epub ahead of print].

9. Ricarte JJ, Hernández JV, Latorre JM, Danion JM, Berna F. Rumination and autobiographical memory impairment in patients with schizophrenia. *Schizophr Res.* 2014; **160(1-3)**: 163-168.
10. Bluck S. Going global: functions of autobiographical remembering world tour. *Memory.* 2015; **23(1)**: 111-118.
11. Denkova E, Dolcos S, Dolcos F. Reliving emotional personal memories: affective biases linked to personality and sex-related differences. *Emotion*. 2012; **12**: 515–528.
12. Williams JMG, Barnhofer T, Crane C, et al. Autobiographical memory specificity and emotional disorder. *Psychological Bulletin.* 2007; **133**: 122-148.
13. Rawal A, Rice F. Examining overgeneral autobiographical memory as a risk factor for adolescent depression. *J Am Acad Child Adolesc Psychiatry.* 2012; **51(5)**: 518-527.
14. Werner-Seidler A, Moulds ML. Autobiographical memory characteristics in depression vulnerability: formerly depressed individuals recall less vivid positive memories. *Cogn Emot.* 2011; **25(6)**: 1087-1103.
15. Anderson RJ, Goddard L, Powell JH. Reduced specificity of autobiographical memory as a moderator of the relationship between daily hassles and depression. *Cogn Emot.* 2009; **24**: 702–709.
16. Young KD, Bellgowan PS, Bodurka J, Drevets WC. Functional neuroimaging correlates of autobiographical memory deficits in subjects at risk for depression. *Brain Sci.* 2015; **5(2)**: 144-164.
17. Young KD, Erickson K, Drevets WC. Match between cue and memory valence during autobiographical memory recall in depression. *Psychol Rep.* 2012; **111(1)**: 129-148.
18. Lemogne C, Piolino P, Friszer S, et al. Episodic autobiographical memory in depression: specificity, autonoetic consciousness and self-perspective. *Conscious. Cogn.* 2006; **15**: 258–268.

19. Sumner JA. The mechanisms underlying overgeneral autobiographical memory: An evaluative review of evidence for the CaR-FA-X model. *Clinical Psychology Review.* 2012; **32(1)**: 34–48.
20. Sumner JA, Mineka S, McAdams DP. Specificity in autobiographical memory narratives correlates with performance on the autobiographical memory test and prospectively predicts depressive symptoms. *Memory.* 2013; **21(6)**: 646-656.
21. Hermans D, Vandromme H, Debeer E, et al. Overgeneral autobiographical memory predicts diagnostic status in depression. *Behav Res Ther.* 2008; **46(5)**: 668-677.
22. Kuyken W, Moulds ML. Remembering as an observer: How is autobiographical memory retrieval vantage perspective linked to depression? *Memory.* 2009; **17**: 624-634.
23. Libby LK, Eibach RP. Looking back in time: self-concept change affects visual perspective in autobiographical memory. *J Pers Soc Psychol.* 2002; **82(2)**: 167-179.
24. Beck AT. Cognition and therapy. *Arch. Gen. Psychiatry.* 1984; **41**: 1112–1114.
25. Belcher J, Kangas M. Reduced goal specificity is associated with reduced memory specificity in depressed adults. *Cogn Emot.* 2014; **28(1)**: 163-171.
26. Mackinger HF, Pachinger MM, Leibetseder MM, Fartacek RR. Autobiographical memories in women remitted from major depression. *J Abnorm Psychol.* 2000; **109**: 331–334.
27. Spinhoven P, Bockting CL, Schene AH, Koeter MW, Wekking EM, Williams JM. Autobiographical memory in the euthymic phase of recurrent depression. J *Abnorm Psychol.* 2006; **115(3)**: 590-600.
28. Young KD, Bellgowan PS, Bodurka J, Drevets WC. Neurophysiological correlates of autobiographical memory deficits in currently and formerly depressed subjects. *Psychol Med.* 2014; **44(14)**: 2951-2963.

29. Ricarte JJ, Latorre JM, Ros L, Navarro B, Aguilar MJ, Serrano JP. Overgeneral autobiographical memory effect in older depressed adults. *Aging Ment Health.* 2011; *15(8)*: 1028-1037.
30. Dritschel B, Kao CM, Astell A, Neufeind J, Lai TJ. How are depression and autobiographical memory retrieval related to culture? *J Abnorm Psychol.* 2011; **120(4)**: 969-974.
31. Woody ML, Burkhouse KL, Gibb BE. Overgeneral autobiographical memory in children of depressed mothers. *Cogn Emot.* 2015; **29(1)**: 130-7.
32. Kuyken W, Dalgleish T. Overgeneral autobiographical memory in adolescents at risk for depression. *Memory.* 2011; **19(3)**: 241-250.
33. Söderlund H, Moscovitch M, Kumar N, et al. Autobiographical episodic memory in major depressive disorder. *J Abnorm Psychol.* 2014; **123(1)**: 51-60.
34. Williams JM, Broadbent K. Autobiographical memory in suicide attempters. *J Abnorm Psychol.* 1986; **95(2)**: 144-149.
35. Dalgleish T, Williams JMG, Golden AMJ, et al. Reduced specificity of autobiographical memory and depression: the role of executive processes. *J. Exp. Psychol. Gen.* 2007; **136**: 23–42.
36. Bomba M, Marfone M, Brivio E, et al. Autobiographical memory in adolescent girls with anorexia nervosa. *Eur Eat Disord Rev.* 2014; **22(6)**: 479-486.
37. Liu X, Liu Y, Li L, Hu Y, Wu S, Yao S. Overgeneral autobiographical memory in patients with chronic pain. *Pain Med.* 2014; **15(3)**: 432-439.
38. Rosenbach C, Renneberg B. Remembering rejection: specificity and linguistic styles of autobiographical memories in borderline personality disorder and depression. *J Behav Ther Exp Psychiatry.* 2015; **46**: 85-92.

39. Greenberg DL, Rubin DC. The neuropsychology of autobiographical memory. *Cortex.* 2003; **39**: 687-728.

40. Whalley MG, Rugg MD, Brewin CR. Autobiographical memory in depression: an fMRI study. *Psychiatry Res.* 2012; **201(2)**: 98-106.

41. Dedovic K, D'Aguiar C, Pruessner JC. What stress does to your brain: a review of neuroimaging studies. *Can J Psychiatry.* 2009; **54(1)**: 6-15.

42. Herold CJ, Lässer MM, Schmid LA, et al. Neuropsychology, autobiographical memory, and hippocampal volume in "younger" and "older" patients with chronic schizophrenia. *Front Psychiatry.* 2015; **6**: 53.

43. Sheldon S, Levine B. The medial temporal lobes distinguish between within-item and item-context relations during autobiographical memory retrieval. *Hippocampus.* 2015 Jun 1. doi: 10.1002/hipo.22477. [Epub ahead of print].

44. Young KD, Erickson K, Nugent AC, et al. Functional anatomy of autobiographical memory recall deficits in depression. *Psychol Med.* 2012; **42(2)**: 345-357.

45. Young KD, Bellgowan PS, Bodurka J, Drevets WC. Autobiographical deficits correlate with gray matter volume in depressed and high risk participants. *Soc Cogn Affect Neurosci.* 2015 Apr 28. pii: nsv047. [Epub ahead of print].

46. Gałecki P, Talarowska M, Anderson G, Berk M, Maes M. Mechanisms underlying neurocognitive dysfunctions in recurrent major depression. *Med Sci Monit.* 2015; **21**: 1535-1547.

47. Douglas KM, Porter RJ, Knight RG, Alsop B. The dynamics of word retrieval in major depression. *Aust N Z J Psychiatry.* 2013; **47(3)**: 276-283.

48. Stoessel C, Stiller J, Bleich S, et al. Differences and similarities on neuronal activities of people being happily and unhappily in love: a functional magnetic resonance imaging study. *Neuropsychobiology.* 2011; **64(1)**:52-60.

49. Uekermann J, Abdel-Hamid M, Lehmkämper C, Vollmoeller W, Daum I. Perception of affective prosody in major depression: A link to executive functions? *J Int Neuropsychol Soc.* 2008; **14**: 552–561.
50. Vasic N, Walter H, Sambataro F, Wolf RC. Aberrant functional connectivity of dorsolateral prefrontal and cingulate networks in patients with major depression during working memory processing. *Psychological Medicine.* 2009; **39**: 977–987.
51. Schlosser N, Mensebach C, Rullkötter N, et al. Selective attention in depression: influence of emotionality and personal relevance. *J Nerv Ment Dis.* 2011; **199(9)**: 696-702.
52. Young KD, Zotev V, Phillips R, et al. Real-time FMRI neurofeedback training of amygdala activity in patients with major depressive disorder. *PLoS One.* 2014; **9(2)**: e88785.
53. Hallam GP, Webb TL, Sheeran P, et al. The neural correlates of emotion regulation by implementation intentions. *PLoS One.* 2015; **10(3)**: e0119500.
54. Yuan H, Young KD, Phillips R, Zotev V, Misaki M, Bodurka J. Resting-state functional connectivity modulation and sustained changes after real-time functional magnetic resonance imaging neurofeedback training in depression. *Brain Connect.* 2014; **4(9)**: 690-701.
55. Drevets WC. Neuroimaging abnormalities in the amygdala in mood disorders. *Ann N Y Acad Sci*. 2003; **985**: 420–444.
56. Victor TA, Furey ML, Fromm SJ, Ohman A, Drevets WC. Relationship between amygdala responses to masked faces and mood state and treatment in major depressive disorder. *Arch Gen Psychiatry*. 2010; **67(11)**: 1128-1138.

57. Hansson PB, Murison R, Lund A, Hammar Å. Cognitive functioning and cortisol profiles in first episode major depression. *Scand J Psychol.* 2015 Jun 1. doi: 10.1111/sjop.12230. [Epub ahead of print].

58. Kim YK, Na KS, Myint AM, Leonard BE. The role of pro-inflammatory cytokines in neuroinflammation, neurogenesis and the neuroendocrine system in major depression. *Prog Neuropsychopharmacol Biol Psychiatry*. 2015 Jun 22. pii: S0278-5846(15)00135-9.

59. Vreeburg SA, Hoogendijk WJ, van Pelt J, et al. Major depressive disorder and hypothalamic-pituitary-adrenal axis activity: Results from a large cohort study. *Arch Gen Psychiatry*. 2009; **66(6)**: 617–626.

60. Porter RJ, Gallagher P. Abnormalities of the HPA axis in affective disorders: Clinical subtypes and potential treatments. *Acta Neuropsychiatrica.* 2006; **18**: 193–209.

61. Malykhin NV, Coupland NJ. Hippocampal neuroplasticity in major depressive disorder. *Neuroscience.* 2015 Apr 28. pii: S0306-4522(15)00388-7. doi: 10.1016/j.neuroscience.2015.04.047.

62. Campbell S, Macqueen G. The role of the hippocampus in the pathophysiology of major depression. *J Psychiatry Neurosci.* 2004; **29**: 417–426.

63. Wingenfeld K, Wolf OT. Stress, memory, and the hippocampus. Front Neurol *Neurosci.* 2014; **4**: 109-20.

64. Schlosser N, Wolf OT, Fernando SC, et al. Effects of acute cortisol administration on autobiographical memory in patients with major depression and healthy controls. *Psychoneuroendocrinology.* 2010; **35(2)**: 316-320.

65. De Brito SA, Viding E, Sebastian CL, et al. Reduced orbitofrontal and temporal grey matter in a community sample of maltreated children. *J Child Psychol Psychiatry.* 2013; **54(1)**: 105-112.

66. Aglan A, Williams JM, Pickles A, Hill J. Overgeneral autobiographical memory in women: association with childhood abuse and history of depression in a community sample. *Br J Clin Psychol*. 2010; **49(Pt 3)**: 359-372.
67. Haddad AD, Williams JM, McTavish SF, Harmer CJ. Low-dose tryptophan depletion in recovered depressed women induces impairments in autobiographical memory specificity. *Psychopharmacology (Berl).* 2009; **207(3)**: 499-508.
68. Sumner JA, Vrshek-Schallhorn S, Mineka S, et al. Effects of the serotonin transporter polymorphism and history of major depression on overgeneral autobiographical memory. *Cogn Emot.* 2014; **28(5)**: 947-958.
69. Wingenfeld K, Kuffel A, Uhlmann C, et al. Effects of noradrenergic stimulation on memory in patients with major depressive disorder. *Stress.* 2013; **16(2)**: 191-201.
70. Marroquín B, Nolen-Hoeksema S. Event prediction and affective forecasting in depressive cognition: using emotion as information about the future. *J Soc Clin Psychol.* 2015; **34(2)**: 117-134.

Table 1. Emotion regulation in the brain.[53]

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

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 overgeneral autobiographical memory (OAM) and specific autobiographical memories. |
| Young et al.[17]<br>Hamlat et al.[3] | 2012<br>2015 | In response to a stimulus (mainly negative), depressive patients more often recall memories in the generalised form rather than the specific one. |
| Hermans et al.<br>Sumner[19]<br>Sumner et al.[20] | 2008,<br>2012,<br>2013 | OAM is considered a risk factor for the occurrence of rDD symptoms during lifetime as well as a factor which makes prognosis in the course of this disease worse. |
| Mackinger et al.[26]<br>Spinhoven et al.[27]<br>Young et al.[28] | 2000<br>2006<br>2014 | The presence of OAM in patients with rDD, who at the time of examination were in the phase of disease remission. |
| Marroquín and Nolen-Hoeksema[70] | 2015 | Prospective memory in patients with rDD. |