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EDITORIAL

Various Methods of Laser Photobiomodulation Therapy for Alzheimer's Disease

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ABSTRACT

Alzheimer's disease is the most common neurodegenerative disease. It is believed that the number of people suffering from Alzheimer's disease worldwide is about 32 million, and the number of people with the preclinical stage of the disease can be up to 300 million.

For a long time, it was believed that Alzheimer's disease arises as a result of disorders in the metabolism of amyloid beta and tau-protein in cerebral tissue. According to numerous recent studies, it has been established that the disease is accompanied by dyscirculatory angiopathy of Alzheimer's type. This is an Alzheimer's disease-specific complex lesion of the cerebral vascular system with arterial, microcirculatory, and venous bed disorders.

One of the most promising directions in the field of brain revascularization, as well as the regeneration of cerebral tissue in Alzheimer's disease, is the use of laser with low output power. This direction was named laser "photobiomodulation therapy".

Currently, laser photobiomodulation therapy is divided into transcranial, intranasal, intravascular (intravenous) and transcatheter intracerebral methods of treatment.

Laser energy has a complex effect on cerebral tissues. Photobiomodulation therapy stimulates angiogenesis, causes collateral and capillary revascularization, restores the exchange of adenosine triphosphate in neuronal mitochondria, improves cellular and tissue metabolism, stimulates neurogenesis, and causes regeneration of tissue structures.

Various types of Photobiomodulation therapy are non-traumatic, physiological, pathogenetically substantiated, effective methods for the treatment of cerebral microcirculatory disorders in Alzheimer's disease. The choice of one or another method of laser photobiomodulation therapy is purely individual and depends on the specific clinical case.

Keywords: Alzheimer's Disease; AD; CSVD; Dyscirculatory angiopathy of Alzheimer's type; PBMT; Transcatheter Intracerebral Laser Photobiomodulation Therapy; Angiogenesis; Neurogenesis.

Introduction

Alzheimer's disease (AD) was first described by the German psychiatrist Alois Alzheimer in 1907, and as a result, the disease was named after him. AD is the most common neurodegenerative disease, and the number of patients is increasing every year. Only in the United States in 2023, 6.7 million people aged 65 and over are suffering from the disease. It is expected that by 2060 the number of patients will have exceeded 13 million people. It should be noted that patients at a younger age were not taken into account; therefore, the real number of patients is even higher. From 2000 to 2019, reported deaths from AD increased by 145% in the United States [1]. It is believed that worldwide the number of patients suffering from AD is about 32 million people, and the number of patients with the preclinical stage of the disease TDR-0 can be up to 300 million people [2].

Despite many years of research, the etiology of AD is not fully understood. For a long time, it was believed that AD arises solely as a result of disorders in the metabolism of amyloid beta (Aβ) and tau-protein in cerebral tissue. At the same time, there are still no ways to treat these pathological disorders and, consequently, to treat AD. The development of drugs that cause a decrease in the level of amyloid beta (Aβ) and tau-protein is carried out in many countries, but a specific drug ready for wide clinical use has not yet been created[2]. The presence of other reasons contributing to or causing the development of this disease was not widely considered for many years.

For the first time, pathological changes in intracerebral vessels in AD were described by the Swiss psychiatrist Ferdinand Morel in 1932. The identified cerebrovascular changes were named dysoric or drusoid angiopathy [3].

A detailed description of all changes in cerebral angioarchitectonics, which include arterial, capillary and venous disorders in AD, was made in 2004. These pathological changes were named "Dyscirculatory angiopathy of Alzheimer's type" (DAAT) [4-6].

Dyscirculatory angiopathy of Alzheimer's type refers to cerebral small vessel disease (CSVD); however, this disorder is a complex lesion of the cerebral vascular system, in which not only the microcirculatory, but also the arterial as well as the venous bed suffer [7, 8]. These specific cerebrovascular changes appear decades before the first clinical symptoms of the disease. These changes lead to involutive disorders of the temporal lobes and characterize the preclinical stage of AD TDR-0 [6, 7, 9]. The same changes in angioarchitectonics are present in all patients with later clinical stages of AD, TDR-1 - TDR-3 [6, 7]. Similar changes are detected in childhood in the direct descendants of patients suffering from AD, which indicates the genetically determined, hereditary nature of these changes [7, 10]. Studies conducted in recent years have shown that 30 of the 45 major genes associated with the risk of developing Alzheimer's disease are expressed in the cerebral vasculature, which confirms the genetic nature of vascular lesions in AD [11].

Dyscirculatory angiopathy of Alzheimer's type affects arteries, arterioles, capillaries, venules and veins and is a hallmark of Alzheimer's disease (AD) [6,7,12].

In natural aging, as well as in patients suffering from other neurodegenerative and ischemic diseases, the combination of such changes in cerebral angioachitectonics does not occur [4, 6, 7].

Dyscirculatory angiopathy of Alzheimer's type is of a complex, multicomponent nature. Increased tortuosity develops in the intracerebral arterial branches. In the temporal regions and the hippocampus, there is a reduction and a decrease in the number of capillaries, they become thinner, their branching decreases, which leads to the development of hypovascular zones in these regions. Further, a similar process occurs in the frontoparietal regions, where hypovascular zones also develop. In the brain, hemodynamics is disturbed, the blood flowing in through the arterial branches cannot pass through the reduced arterioles and capillaries, which causes a decrease in the distal arterial blood flow. This leads to a natural protective reaction to the disorders in capillary blood flow. In the temporal and frontoparietal regions, arteriovenous shunts open, through which the blood coming through the arteries, bypassing the capillaries, is discharged into the venous bed. Increased flow of arterial blood into the venous bed leads to its overflow, stagnation and impaired venous outflow. Therefore, large, pathologically dilated venous trunks develop. As a result, there is a complete restructuring of cerebral hemodynamics.

Developed changes in the cerebral arterial, microcirculatory and venous bed lead to AD-specific hypoperfusion and hypoxia. As a result, the death of mitochondria in the cells of the smooth endoplasmic reticulum and the Golgi apparatus

occurs in the brain, the metabolism of adenosine triphosphate (ATP) is disturbed, a gradual loss of synapses, degeneration and death of neurons develop [13-17]. The combination of such cerebral arterial, microcirculatory, venous and hemodynamic changes leads to tissue structures damage. Thus, neurovascular unit (NVU) damage occurs [14, 15].

Developed cerebral microvascular and hemodynamic disorders affect the metabolism of amyloid beta (Aβ), which leads to a decrease in its natural excretion and an increase in its accumulation[18,19,20]. These pathological changes cause the deposition of amyloid beta in the cerebral tissue and vascular wall. In turn, this causes a decrease in the elasticity of microvessels, narrows their lumen, thereby further contributing to a decrease in intracerebral blood flow [20]. At the same time, natural, physiological, intracerebral angiogenesis decreases and dysfunction of the blood-brain barrier (BBB) develops [21, 22]. In the pathogenesis of AD, these pathological changes are inextricably linked, gradually developing; they exacerbate each other and lead to cerebral dysfunction and neurodegeneration [6, 7, 10].

The more pronounced cerebrovascular changes are, the more actively cerebral hypoperfusion and hypoxia is aggravated and developed, the faster amyloid beta accumulates in the cerebral tissue and AD develops [23, 24]. As a result, over the years, operate the processes that lead to the gradual development of the disease and the growing of its clinical symptoms [6, 7, 18, 19]. The speed of development of these changes is important, since it determines the time of the patient's transition from the preclinical stage of TDR-0 to the clinical stages of AD TDR-1, TDR-2, TDR-3 [6, 7, 13].

The data on circulatory and hemodynamic disorders in AD obtained in recent decades have led to the fact that an increasing number of researchers point to the need to develop new methods of treating this disease. These methods should be aimed at restoring and normalizing cerebral blood supply and microcirculation, as well as restoring the normal metabolism of amyloid beta, reducing its level and developing regenerative processes in cerebral tissue [6, 9,23,24].

Treatment Methods

Conservative treatment methods for such complex cerebrovascular lesions are of little effect and do not give the desired result [6, 7, 9, 23]. One of the most promising directions in the field of brain revascularization and cerebral tissue regeneration in various neurodegenerative and ischemic lesions is the use of laser with low output power [7, 24, 26-29].

The use of lasers in medicine began immediately after the discovery of the laser effect in the 1960s. Andrew Mester et al. in 1967 were the first to achieve a good clinical effect by using the ruby laser operating in the low output power mode to heal superficial wounds on the skin [31]. In 1968, N. S. Makeeva and V. V. Schur used a helium-neon laser for the same purpose and with a good clinical effect [32]. After numerous studies, it was found that the use of laser with low output power does not have a damaging or negative effect on biological tissues [28, 31, 32]. Currently, this direction in laser medicine is called laser Photobiomodulation therapy (PBMT) [27-29].

With various ischemic, traumatic and neurodegenerative cerebral lesions, carrying out PBMT using laser with low output power of the red or near-infrared spectral region (600-1100 nm) has a complex and multicomponent effect on the brain. The laser with low output energy, when applied directly, penetrates deep enough into cerebral tissues. At a wavelength of 600 to 700 nm, the penetration depth is 20–40 mm [33]. Laser PBMT stimulates angiogenesis, improves blood circulation, restores adenosine triphosphate (ATP) metabolism in neuronal mitochondria, prevents neuronal death, restores general metabolic processes in tissues, and also stimulates cerebral neurogenesis and regeneration of cerebral tissue [25-29].

Currently, laser PBMT in AD and other cerebral lesions is divided into: transcranial [26-29], intranasal (often in combination with transcranial)[34], intravascular (intravenous)[35] and transcatheter intracerebral methods of treatment [4,5, 30,33].

The transcranial method is technically the simplest non-invasive method. During its implementation, laser energy is supplied to the forehead and scalp. For this method, laser with low output power, red or near-infrared spectral region (600-1080 nm), as well as LED matrices, made in the form of specific “helmets” worn on the head, are used. LED matrices are often used with parallel application of LED sources with different wavelengths: 660 nm and 1064 nm or 660 nm and 1080 nm[26–29]. As a result, to reach the brain, laser energy passes through the tissues of the skin and skull bones. These structures have a high degree of absorption and a rather low degree of transmission of light energy. This reduces the level of energy reaching the brain. As a result, a limited amount of laser energy reaches cerebral tissues. The method is

effective, but to obtain a stable clinical effect, it is necessary to conduct a sufficiently large number of sessions [26-29].

The intranasal method is also a simple, minimally invasive method. During its implementation, laser energy, using special light guide instruments, is supplied to the brain through the nasal cavities [34]. As a result, the energy passes through the mucous membranes of the nose and the thinner bones of the skull. These structures have a lower degree of absorption and a greater degree of transmission of laser energy than the tissues of the skin of the head and bones of the cranial vault. With this method, more energy reaches the brain; however, although the method is effective, it also requires a fairly large number of sessions. To increase the amount of laser energy penetrating the brain tissues and to enhance the therapeutic effect, this method is often combined with the transcranial method, which, in fact, is a multimodal method [34].

The intravascular (intravenous) method is also a simple, minimally invasive method. In fact, this method is intravascular laser irradiation of blood (ILIB). A helium-neon laser (wavelength 632.8 nm) is used as a source of laser energy. When it is carried out, blood is exposed to laser through a peripheral intravenous catheter. There is no direct effect of laser energy on brain tissue. According to the authors, the method has a good clinical effect in patients who have had their first ischemic stroke. All treated patients showed faster rehabilitation with recovery after the stroke. The mechanism of the effect of ILIB on the brain is not completely clear; however, the authors indicate that, according to single-photon emission computed tomography (SPECT), the laser exposure increases regional cerebral blood flow, which improves motor, sensory, and cognitive recovery. To obtain a stable clinical effect, at least 10 sessions are required [35]. There are no data on the use of this method in AD.

The transcatheter intracerebral method is a more complex, but also minimally invasive method [4,5,36, 37]. The intervention is performed in catheterization laboratories. Under local anesthesia and fluoroscopic control, a peripheral artery is catheterized, a micro guiding catheter is passed through it and is led through the carotid arteries and intracerebral arterial branches to the hypovascular zones in the brain. A flexible laser fiber optic light guide instrument with a diameter of 25-100 micrometers is coaxially passed through this catheter, connected to a helium-neon laser (wavelength 632.8 nanometers), after which laser exposure is performed. The intervention is performed on the right and left hemispheres. The duration of laser PBMT is 1200-2400 seconds [4, 5]. Due to the sufficient amount of laser energy, a single intervention is enough to obtain a pronounced, lasting, positive effect. While conducting repeated, delayed angiographic examinations (MUGA), scintigraphy (SG) and rheoencephalography (REG), it was revealed that stimulation of angiogenesis leads to the opening of the collateral arterial and capillary bed, closure of arteriovenous shunts and normalization of venous outflow [7,30].

Neurogenesis begins to develop in the first months after the transcatheter intracerebral laser PBMT. During repeated, delayed CT and MRI examinations, neurogenesis is manifested in an increase in the volume of normal tissue of the temporal and frontoparietal regions of the brain [30].

The clinical effect after such laser interventions is manifested in memory restoration, a persistent decrease in the severity of dementia and an improvement in cognitive functions. The duration of the clinical effect depends on the stage of AD, the size of the affected cerebral tissue, and the severity of the dementia. In preclinical AD stages of TDR-0 and mild TDR-1, treated patients show recovery of cognitive functions and no dementia. The resulting clinical effect lasts for many years. In the moderately severe AD stage of TDR-2, a decrease in dementia and an improvement in cognitive functions are manifested over 4-4.5 years. In the severe AD stage of TDR-3 it was observed for 2-2.5 years [7,30].

Transcatheter intracerebral laser PBMT showed high efficiency not only in the treatment of AD, but also in the treatment of Binswanger's disease (BD), vascular parkinsonism (VP), various ischemic brain lesions, various forms of stroke [30, 33, 36-38]. The results obtained after laser interventions significantly exceed the results of various conservative methods of treating these cerebral lesions.

Conclusions

In natural aging, as well as in patients with various neurodegenerative and ischemic lesions, disorders of cerebral angioachitectonics similar to Dyscirculatory angiopathy of Alzheimer's type (DAAT) do not occur. Specific cerebrovascular changes similar to Dyscirculatory angiopathy of Alzheimer's type (DAAT) are detected in childhood in direct descendants of AD patients. This indicates a genetically determined, hereditary nature of these cerebrovascular changes.

Despite the absence of signs of dementia and overt cognitive impairment, Dyscirculatory angiopathy of Alzheimer's type (DAAT) occurs in all people with preclinical AD stage of TDR-0. Microvascular and hemodynamic changes gradually, over many years contribute to the disruption of amyloid beta (Aβ) metabolism. As a result, amyloid beta (Aβ) slowly accumulates in the cerebral tissue and vascular wall. The person develops dementia and cognitive deficit, as a result, the patient's condition gradually passes first into the mild clinical stage of TDR-1, then to the moderately severe stage of TDR-2 and the severe stage of TDR-3.

Conservative treatment of such a complex cerebrovascular lesion as DAAT has low efficiency and does not give the desired clinical results.

Various methods of PBMT are physiological, non-traumatic, pathogenetically substantiated, effective methods for the treatment of cerebral microcirculatory disorders in AD and other cerebral neurodegenerative and ischemic lesions.

When performing various types of laser PBMT, the energy of the laser with low output has a complex and physiological effect on cerebral tissues. Laser exposure stimulates angiogenesis, causes collateral and capillary revascularization, restores ATP metabolism in neuronal mitochondria, improves cellular and tissue metabolism, stimulates neurogenesis, and causes regeneration of cerebral tissue structures.

The choice of one or another method of laser PBMT is purely individual and depends on the specific clinical case.

Declaration of Conflicting Interests

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References

1. 2023 Alzheimer's disease facts and figures. *Journal of Alzheimer's & Dementia*. 2023; 19, (4): 1598-1695. https://doi.org/10.1002/alz.13016
2. Van der Flier WM, De Vugt ME, Smets EMA, Blom M, Teunissen ChE. Towards a future where Alzheimer's disease pathology is stopped before the onset of dementia. *Nature Aging*. 2023; (3): 494-505. DOI: 10.1038/s43587-023-00404-2
3. Morel F. An apparently dyshoric and topical angiopathy. *Monatsschr Psychiat Neurol*. 1950; 120, 5-6: 352-357.
4. Maksimovich I V. Method for Endovascular Treatment of Alzheimer's Disease. 2006. Patent, No.2297860 Russian.
5. Maksimovich I V. Method and Device for Endovascular Treatment of Alzheimer's Disease. 2008. Patent No. 7389776.USA.
6. Maksimovich IV. Dyscirculatory Angiopathy of Alzheimer's Type. *Journal of Behavioral and Brain Science*. 2011; 1 (2): 57-68. DOI: 10.4236/jbbs.2011.12008
7. Maksimovich IV. Cerebrovascular Changes and Cerebral Atrophy in the Development of Dementia during Alzheimer's Disease. *Medical Research Archives*. 2023; 11 (5): 1-10. https://doi.org/10.18103/mra.v11i5.3869
8. Cai Z, Wang C, He W, Tu H, Tang Z, Xiao M, Yan L. 2015. Cerebral small vessel disease and Alzheimer's disease. Clin Interv Aging. 2015; 23, (10): 1695-1704. https://doi.org/10.2147/CIA.S90871
9. Rafii MS, Aisen PS Detection and treatment of Alzheimer's disease in its preclinical stage. *Nature Aging*. 2023; 3: 520-531. DOI: 10.1038/s43587-023-00410-4
10. Maksimovich IV and Polyaev YuA. The Importance of Early Diagnosis of Dyscircular Angiopathy of Alzheimer's Type in the Study of Heredity of Alzheimer's Disease. *Journal of Alzheimer's & Dementia*. 2010; 6, 4S, (21): 43-45. https://doi.org/10.1016/j.jalz.2010.08.133
11. Yang AC, Vest RT, Kern F, Lee DP et al. A human brain vascular atlas reveals diverse mediators of Alzheimer's risk. *Nature*. 2022;603,(7903):885-892. DOI: 10.1038/s41586-021-04369-3
12. Sun N, Akay LA, Murdoc MH, Park Y et al. Single-nucleus multiregion transcriptomic analysis of brain vasculature in Alzheimer's disease. *Nature Neuroscience*. 2023; 26: 970-982 DOI: 10.1038/s41593-023-01334-3
13. Baloiannis SJ and Baloiannis IS. The vascular factor in Alzheimer's disease: A study in Golgi technique and electron microscopy. *Journal of the Neurological Sciences* 2012; 322: 117-121. https://doi.org/10.1016/j.jns.2012.07.010

14. Nelson AR, Sweeney MD, Sagare AP, Zlokovic BV. Neurovascular dysfunction and neurodegeneration in dementia and Alzheimer's disease. *Biochim Biophys Acta*. 2016; 1862, (5): 887-900. https://doi.org/10.1016/j.bbadis.2015.12.016
15. Baloyannis, S.J. Brain capillaries in Alzheimer's disease. *Hell J Nucl Med* 2015; 18, Suppl 1 :152. DOI: 10.15406/jnsk.2015.02.00069
17. Kimbrough, I.F., Robel, S., Roberson, E.D., Sontheimer, H. Vascular amyloidosis impairs the gliovascular unit in a mouse model of Alzheimer's disease. *Brain*. 2015; 138, (12): 3716-3733. https://doi.org/10.1093/brain/awv327
18. Iadecola C. The overlap between neurodegenerative and vascular factors in the pathogenesis of dementia. *Acta Neuropatho* 2010; 120 (3): 287-396. doi: 10.1007/s00401-010-0718-6.
19. Zlokovic BV. Neurodegeneration and the neurovascular unit. *Nat Med* 2010; 16 (12): 1370-1371. doi: 10.1038/nm1210-1370
20. Zlokovic BV. Neurovascular pathways to neurodegeneration in Alzheimer's disease and other disorders. *Nature Reviews. Neuroscience*. 2011; 12, 723-738. doi: 10.1038/nrn3114
21. Brenowitz WD, Hubbard RA, Keene CD, Hawes CE. Mixed neuropathologies and estimated rates of clinical progression in a large autopsysample. Alzheimers & Dementia. 2017;13(6):654-662 doi: 10.1016/j.jalz.2016.09.015
22. Bell RD & Zlokovic BV. Neurovascular mechanisms and blood-brain barrier disorder in Alzheimer's disease. *Acta Neuropathologica* 2009; 118: 103-113. doi: 10.1007/s00401-009-0522-3
23. Montagne A, Barnes SR, Sweeney MD, Halliday MR, Sagare AP, Zhao Z, Toga AW, Jacobs RE, Liu CY, Amezcua L, Harrington MG, Chui HC, Law M, Zlokovic BV,. Blood-brain barrier breakdown in the aging human hippocampus. *Neuron*. 2015; 85, (2): 296-302. http://dx.doi.org/10.1016/j.neuron.2014.12.032
24. Maksimovich IV. Certain new aspects of etiology and pathogenesis of Alzheimer's disease. Advances in Alzheimer's Disease, 2012; 1, (3): 68-76. DOI: 10.4236/aad.2012.13009
25. De la Torre JC. Cerebral Perfusion Enhancing Interventions: A New Strategy for the Prevention of Alzheimer Dementia. Brain Pathology 2016; 26, (5): 618–631 https://doi.org/10.1111/bpa.12405
26. Hashmi JT, Huang YY, Osmani BZ, Sharma SK, Naeser MA and Hamblin MR. Role of low-level laser therapy in neurorehabilitation. *Arch Phys Med Rehab*. 2010; 12 (2): S 292–305. https://doi.org/10.1016/j.pmrj.2010.10.013
27. Naeser MA, Hamblin MR. Potential for transcraniallaser or LED therapy to treatstroke, traumatic brain injury, and neurodegenerative disease. *Photomed Laser Surg*. 2011; 29 (7): 443-446. https://doi.org/10.1089/pho.2011.9908
28. Hamblin MR. Photobiomodulation for Alzheimer's Disease: Has the Light Dawned? *Photonics*. 2019; 6 (3), 77. https://doi.org/10.3390/photonics6030077
29. Hamblin MR. Mechanisms of photobiomodulation in the brain. In *Photobiomodulation in the Brain*, Edited by Michael R. Hamblin, Ying-Ying Huang. Academic Press is an imprint of Elsevier, London. 2019; p-p. 97-110. https://doi.org/10.1016/B978-0-12-815305-5.00008-7
30. Maksimovich IV. Transcatheter intracerebral photobiomodulation in degenerative brain disorders: clinical studies (Part 1). In *Photobiomodulation in the Brain*, Edited by Michael R. Hamblin, Ying-Ying Huang. Academic Press is an imprint of Elsevier, London, 2019; p-p. 515-528. https://doi.org/10.1016/B978-0-12-815305-5.00038-5
31. Mester E, Szende B, Gartner P. The effect of laser beams on the growth of hair in mice. Radiobiol Radiother (Berl) 1968; 9:621–626.
32. Deviatkov ND. Application of electronics in medicine and biology. Electronic Equipment: Microwave Equipment 1993; 1:66-76.
33. Maksimovich I. V. Transluminal laser angioplasty in the treatment of ischemic brain lesions. Doctoral Dissertation, RUDN University: Peoples' Friendship University of Russia, Moscow, 2004.
34. Salehpour F, Hamblin MR, Di Duro JO. Rapid Reversal of Cognitive Decline, Olfactory Dysfunction, and Quality of Life Using Multi-Modality Photobiomodulation Therapy: Case Report. *Photobiomodul Photomed Laser Surg* 2019; 37 (3): 159-167. DOI: 10.1089/photob.2018.4569
35. Ming-Wei Lai, Chia-Hsin Yang, Pi-Yu Sung, and Sen-Wei Tsai. Intravascular Laser Irradiation of Blood Improves Functional Independence in Subacute Post-Stroke

Patients: A Retrospective Observational Study from a Post-Stroke Acute Care Center in Taiwan. *Photobiomodulation, Photomedicine, and Laser Surgery*. 2022; 40, (10): 691–697. https://doi.org/10.1089/photob.2022.0042

36. Maksimovich IV. Intracerebral Transcatheter Laser PBMT in the Treatment of Binswanger's Disease and Vascular Parkinsonism: Research and Clinical Experience. Photobiomodul Photomed and Laser Surg. 2019; 37, 10: 606-614. https://doi.org/10.1089/photob.2019.4649
37. Maksimovich IV. Study of the Impact of Transcatheter Intracerebral Laser Photobiomodulation Therapy Treatment on Patients with Alzheimer's Disease and Binswanger's Disease. *Medical Research Archives* 2022; 10, (12): 1-13. https://doi.org/10.18103/mra.v10i12.3420
38. Maksimovich IV. Transcatheter intracerebral photobiomodulation in ischemic brain disorders: clinical studies (Part 2). *Photobiomodulation in the Brain*. Edited by Michael R. Hamblin, Ying-Ying Huang, Academic Press is an imprint of Elsevier, London 2019; 529-544. https://doi.org/10.1016/B978-0-12-815305-5.00039-7