A Brief Review of Alzheimer's Disease

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ABSTRACT

Alzheimer's disease is one of the fastest growing risk factors in middle age, which increases the risk of Alzheimer's disease in coronary weddings and higher levels of homocysteine, which can lead to withdrawal due to lifestyle changes. Cigarettes prevent Alzheimer's disease by recalling memories. Also, despite the fact that there is no definitive cure for Alzheimer's, the ability to increase acetylcholine levels and reduce ameloid beta deposition and your medications such as Donepezil has proven to be very effective when treating Alzheimer's.

Abbreviations: AGES: Advanced Glycation End Products; CABG: Coronary Artery Bypass Surgery; BMI: Body Mass Index; MMSE: Mini-cog & MiniMental State Examination; BChE: Butyryl-Choline Esterase; NSAIDs: Nonsteroidal Anti-Inflammatory; DM: Diabetes Mellitus; HTN: Hypertension; AChE: Acetylcholinesterase; AB: Amyloid Beta; NMDA: N-Methyl-D-Aspartate; cAMP: Cyclic Adenosine Monophosphate; PDE4: Phosphodiesterase-4; DBS: Deep Brain Stimulation; LSS: Lanosterol Synthase; OSA: Obstructive Sleep Apnea; CPAP: Continuos Positive Airway Pressre; HTR2A: 5-Hydroxytryptamine Receptor 2A; HTR2C: 5-Hydroxytryptamine Receptor 2C

Introduction

Alzheimer's Disease (AD) constitutes progressive brain dysfunction and typically occurs in old age. In this disease, that is becoming increasingly prevalent around the world, memory and thinking power are slowly lost. Even the ability to do simple things is taken away from the person. One major cause of AD is the deposition of amyloid-beta peptides and tau proteins in the brain, which lead to the destruction of brain nerve cells and cognitive dysfunction. A high consumption of saturated fatty acids, unlike unsaturated fatty acids, play a role in the development of AD through the destruction and damage of brain neurons [1-3]. Type 2 diabetes also has been seen to increase the risk of AD [4]. Genetic

factors through mutations in Presenilin, APP and ApoE genes can be effective in the risk of AD [5]. Down syndrome is another condition that increases the risk of AD [6]. One of the diagnostic criteria for AD is an increase in beta amyloid levels, the control of which can be effective in the prevention and treatment of AD [7]. Prevention of diabetes, hypertension and obesity also prevented AD [8]. In addition to the above, proper nutrition with nutrients such as olive oil, nuts, plants and omega-3 rich substances is very effective in preventing AD by reducing amloid deposition [9]. Oxidative stress, which includes beta amyloid deposition, is involved in AD [10]. Thus, antioxidants such as vitamins C and E

can reduce stress and prevent AD [11]. NMDA receptor antagonists, as well as cholinesterase inhibitors, can be used to treat AD [12,13]. BACE1 receptors cause synaptic disruption of neuronal function. The use of drugs such as verubecestat and elenbecestat, which inhibit BACE1 receptors, has an effective role in the treatment of AD [14]. People can also increase the life of neurons and prevent AD to some extent by consuming vitamin E, which plays a vital role in reducing stress [15].

Diabetes, Coronary Artery Disease, and High Levels of Homocysteine Increase the Risk of Alzheimer's Disease by Increasing the Deposition of Amyloid Plaques and Tau Protein

Factors such as cardiovascular disease, hypertension, and coronary artery gene play a part in the development of AD. Heart failure, atrial fibrillation, dysfunction of the heart and structure reduce the blood flow to the brain and damage nerve cells in the brain [16]. One of the most important risk factors are cerebrovascular accidents (strokes), which increases the risk of Alzheimer's disease through cerebrovascular injury and cerebral ischemia [17]. Advanced glycation end products cause the formation of amyloid plaque deposits and tau proteins in the brain (AGES) and also increase beta-amyloid production and phosphorylation of tau proteins in the brain by disrupting insulin signals [18]. Cardiac surgeries, such as CABG and coronary artery bypass grafting increase cerebral hypoperfusion due to cerebral hypoperfusion [16]. The coronary artery gene is also a risk factor for Alzheimer's. Excessive expression of amyloid precursor proteins leads to the deposition of beta amyloids and tau protein and the formation of nerve nodes in the brain [19]. Strokes lead to dysfunction of the blood-brain barrier and ischemia in the brain, which is accompanied by an increase in beta amyloid in the brain and hyperphosphorylation of tau proteins in the brain. Strokes are the most important risk factors for Alzheimer's and 20% of strokes Are caused by heart disorders [20]. Hypercholesterolemia and hyperhomocysteine increase the risk of Alzheimer's disease [21]. Hypercholesterolemia increases cholesterol and its role in crossing the blood-brain barrier plays a role in increasing beta amyloid deposition. By affecting the brain, it increases the incidence of Alzheimer's. High cholesterol levels also contribute to cardiovascular disease. High cholesterol levels in middle age are also an important risk factor for late-onset Alzheimer's disease. High levels of homocysteine lead to cerebrovascular disease and stroke and vascular endothelium.

The brain is damaged, thus depositing amyloid peptides in the brain and doubling the risk of Alzheimer's disease. Smoking is an important risk factor for cardiovascular disease and its effects on cerebrovascular damage and destruction. It linked to nearly 14% of Alzheimer's cases worldwide [22]. Coagulation abnormalities such as increased coagulation, increased thrombin production and low anticoagulant response, cause venous thrombosis and thus increase the risk of stroke as well [23]. Inflammatory factors cause vascular atherosclerosis and atherosclerosis. Due to the passage of inflammatory factors through the blood-brain barrier, they cause blood-brain barrier disorders and activate microglia, which increase the accumulation of beta-amyloid in the brain and tau protein, resulting in neuronal destruction [24]. Additionally, age is known to be the strongest risk factor for Alzheimer's disease [25]. According to recent studies, the prevalence of Alzheimer's is higher in women than men due to less cognitive differences in women, differences in specific sex hormones, and estrogen levels versus testosterone levels [26].

Lifestyle has a significant effect on the risk of Alzheimer's disease. Decreased physical and mental activity is associated with an increased risk of dementia. Physical activity has neuroprotective effects and reduces atrophy of the brain in the hip area. Mental activities, including education, higher education, job complexity, activity, and leisure such as brain games increase cognitive reserve [27]. A diet high in saturated fatty acids increases the risk of AD, while unsaturated fatty acids such as omega-3s reduce the risk of heart disease, thus reducing the risk of AD. Eating fruits, vegetables, legumes, grains, and unsaturated fatty acids reduce the risk of AD due to the antioxidant properties of vitamins A, B, and C due to their protective effects on brain neurons against oxidative stress [28]. Obesity accelerates ageing in the brain and increases vascular pathways and nerve damage in the brain [29]. High alcohol consumption causes cerebrovascular disease and vascular dementia and increases cholesterol levels while reducing fibrinogen and thrombotic factors [30].

Factors Such as Quitting Smoking, Walking and Taking Antioxidants to Reduce Oxidative Stress are Very Effective in Preventing Alzheimer's

AD is a common form of dementia [31], a progressive and persistent neurological disease and degenerative disorder that affects large areas of the brain, like the cerebral cortex and the hippocampus. Dementia has an average disease duration of 8 to 10 years and an average age of 80 years, with experts predicting that the disease prevalence is likely to triple by 2050 [32]. Physiological symptoms begin 20 years before clinical signs and the criterion for diagnosing the disease is an increase in extracellular amyloid-beta (AB) [31]. Women are more susceptible than men, with experts having confirmed the possibility of prevention [32,33]. One of the preventive interventions is non-steroidal anti-inflammatory drugs. Such efficacy of low-dose naproxen has been investigated to prevent disease progression [34]. Exacerbating risk factors include HTN, genetics, DM obesity, hyperlipidemia, depression, smoking and low education [35,36]. The main factor is old age and prevention of AD can help increase prevention by 10 to 20%. There is also a need for lifestyle changes and planning for long-term care and screening in

order to improve the condition or prevent it [37]. In contrast, anti-amyloid drug therapies have failed to produce significant results combatting AD or dementia [38].

The level of amyloid-beta deposits is completely regular [39] and any changes in the prevention and prevention of symptoms advanced AD is effective [40]. One of the effective factors in preventing type 2 AD is antioxidant treatments such as substances like manganese and selenium [41] that block the penetration of mitochondria [42]. Some studies have examined estrogen as a potential factor in prevention in women [43]. Risk of disease per year of education is reduced by 17% [44]. There is an effective link between smoking and AD. More specifically, smoking two packs increases the risk of cognitive impairment during the day compared to half a pack.[39] Also, quitting smoking reduces oxidative stress and reduces the risk of AD [45]. Sleep disorders such as reduced nighttime sleep, nap daily awakening and sleep cycle inversion have a direct impact on AD, and improved sleep conditions reduce the likelihood AD [46]. Depression may be one of the early signs of Alzheimer's pathology and antidepressant treatments may be non-existent. Directly reduces the risk of AD [38]. A promising non-pharmacological approach to control and the formation of AD musical symptoms therapy is a kind of sensory stimulation that creates a response as well as the accompaniment of music with the recollection of memories and feelings of impact.

It is a safe and effective method [47]. Physical exercise reduces dementia and improves cognitive function and reduces the incidence of AD by around 50% [48]. Research has also shown that 85 minutes of walking per week reduces the risk of AD by 40% [49]. Hypertension is also a known cause of AD and treatment with antihypertensive drugs reduce the risk of AD by 38%. Oxidative stress is the first feature of the AD brain and In fact, it is the raw material of the disease and is a gradual and long process that causes the deposition of amyloid B and the formation of fibrous nerve nodes. Beta-carotene, retinol, and urate are other nutritional oxidants, but high doses are ineffective. There are some dietary strategies for prevention that are considered due to their high effectiveness and low side effects [33]. Also, diet modification and the use of vitamins B12, E and omega-3 [50] and minerals such as iron and Aluminum [51] can reduce the risk of AD factors. Some supplements, such as fatty acid supplements, have effects in preventing symptoms [52]. One of the best diets is the Mediterranean diet [53] with the consumption of vegetables and fruits [54]. Research has shown that some probiotics and prebiotics are effective in delaying the onset of the disease [55].

Is there an Effective and Reliable Treatment for Alzheimer's?

There are three categories of Alzheimer's disease, including mild, moderate and severe, based on clinical criteria, patient Age, MiniMental State Examination (MMSE) or (Mini-cog) and functional behavioral testing [56]. With age, genetic factors play the most important role in disease progression [57]. Accordingly, with the ipscs (Induced Pluripotent Stem Cell) method, the neurons of the infected person were connected to their neural ancestors, and thus the pattern of disease progression is determined, which is very effective in initial treatment and diagnosis [58]. If diagnosed before the onset of clinical symptoms, the effect of the drug is much greater [59]. Although there is no definitive strategy for the treatment of Alzheimer's Disease (AD) [60] cholinesterase inhibitors in combination with an NMDA (Memantine) inhibitor are among the first line of treatment [61]. Serotonin receptors are involved in the cognitive process and protection of nerve cells by increasing the secretion of amyloid precursor (APP) and stimulating the cholinergic process, so serotonin-receptor agonists may also have a potentiating effect.

The best time to treat Alzheimer's is before symptoms occur. The treatment process uses different therapies, most of which require more testing and research. In the pathogenesis of Alzheimer's disease, amyloid plaques have the greatest impact and the first factor in the pathogenesis of AD is Ab-42, which is most associated with the pathogenesis of AD, which causes Memory loss and activation of microglia, synaptic degradation, and oxidative damage. Production of Ab and intoxication through vascular effects is effective and vitamin B intake leads to a decrease in homocysteine. RAGE immunoglobulin is regulated in AD by affecting microglia and hippocampal cells and the amyloid binds to this receptor and may be active. The formation of the amyloid cascade eventually leads to neuronal death. Cholesterol-lowering factors, which inhibit the formation of beta-amyloid, are a factor in the treatment and prevention of AD. The use of estrogen is not effective in improving the disease and is not recommended in postmenopausal women. Anti-inflammatory drugs are also mainly involved in primary and secondary prevention trials [62] and may reduce the risk of AD. The importance of NSAIDs or non-steroidal anti-inflammatory drugs in their anti-inflammatory properties [63].

By inhibiting amyloid precursor degrading enzymes (APPs), Notch protein is also inhibited. The notch is an integral membrane protein that is involved in the hematopoietic function and embryonic development [64] and in the structure of cell receptors and lymphoid structures. And erythroid is involved [65]. Amyloid precursor protein (APP) is broken down by two enzymes, g-Secretase and b-Secretase, with lengths of 37 to 42 amino acids. The major amyloid plaques are Ab-40 and Ab-42 [66]. Deposition of abnormal proteins in the hippocampal cortex, including Ab and TAU, causes neuronal destruction [67]. Lanabecestat is an oral BACE (Beta-Secretase) inhibitor that prevents the accumulation of beta-amyloid and is used in patients with mild Alzheimer's disease and those who have recently had it but has no effect on improving or reducing the loss of cognition and function in patients. g-Secretase enzyme inhibitors with gastrointestinal symptoms

include diarrhoea and gastrointestinal bleeding due to inhibition and degradation of Notch protein. The b-Secretase enzyme has the first proteolysis pathway on APP [68]. G-Secretase inhibitors with a lower percentage of Notch inhibition can be used to inhibit AG production [69]. With the destruction of acetylcholine neurons by Ab deposition, the level of acetylcholine decreases, so the main focus of AD treatment is to increase the level of acetylcholine by inhibiting its degrading enzyme, which is associated with high-cost treatment. Tacrine was the first drug in the AChE inhibitors. Which is now removed from the treatment protocol due to liver damage and is less effective [70].

In order to increase acetylcholine, acetylcholine hydrolyzing inhibitors are used in mild to moderate advertisements, including Donepezil with ionic nature, Rivastigmine and physostigmine with astringent nature [71], and Galantamine. New beta and gamma-carbonyl derivatives are potent as anti-Alzheimer's drugs that protect neurons and have a greater inhibitory effect on Butyryl-Choline Esterase (BChE) than the AChE enzyme. And can be used in conditions of nerve damage in Alzheimer's disease [72]. With a defect in cholinergic activity, with a decrease in acetylcholine and due to its role in short-term memory, the symptoms of the disease appear. Eventually, neuronal death begins. Treatment with Donepezil at the appropriate stage is associated with positive effects on the modification of cholinergic activity [73]. In long-term treatment with Donepezil ⅔ patients recovered or remained unchanged for at least six months and after one year 6%, and after three years, 3% of patients recovered or remained unchanged [74]. If Donepezil is used in combination with Memantine, better results will be seen than when used alone [75].

Memantine is used in the late stages of the disease in moderate to severe conditions and is well tolerated in the body and produces better behavioural symptoms [76]. Treatment with Galautamine, a cholinesterase inhibitor, reduces the cost of treatment by delaying FTC (full time care) in addition to therapeutic effects [77]. Rivastigmine inhibits both AChE and BChE (the major hydrolyzers of acetylcholine in the brain) but has more side effects than Donepezil [78] and is effective in treating mild to moderate Alzheimer's [79]. GSK-3 enzyme plays a role in the pathogenesis of AD by inhibiting synapse flexibility and disrupting neuronal conduction and production of the amyloid peptide through degradation of amyloid precursor protein (APP) [80]. Therefore, inhibition of this enzyme by lithium and protein kinase B and other kinases can be therapeutic goals of AD [81]. Lithium is useful in improving long-term disease progression by reducing Ab-42 in CSF [82]. Over-activation of glutamate damages nerve cells and activates NMDA receptors at low concentrations. Over-activation of the NMDA receptor causes cell death, and if the NMDA receptor is depleted or overused by Mg2 + ions. Stimulated synaptic flexibility is impaired.

This glutamate binds to both NMDA and AMDA receptors and blocks Mg2 + NMDA receptors to reduce its activity. Continuous activation of NMDA receptors is associated with the influx of Na ions into the cell and reduced membrane potential Ca ions It enters the cell through the NMDA receptor channel and initiates a number of enzymatic processes that are involved in stabilizing and increasing synaptic power, which increases the sensitivity and activity of the receptor. With the continued influx of Ca ions, nerve cell damage occurs. Poisoning is a process of stimulation, which occurs after glutamate overdose and is effective in stroke and AD. Magnesium ion inhibits NMDA receptor, thus with increasing NMDA receptor activity, magnesium ion decreases, which impairs synapse flexibility and requires a suitable replacement such as Memantine is in Memantine Prevents pathological activation of the NMDA receptor. Suppresses synaptic dysfunction and improves learning. Protects neurons and provides flexibility to repair neurons. Creates TAU (hyperphosphorylated nerve-fibre node) by interfering with microtubule accumulation. Pathological AD is TAU with similar effects to plaque Ab and insoluble hyperphosphorylated deposition in neurons causing neural network destruction.

TNF disrupts the hemostatic mechanism and synapses, thus playing a role in the pathogenesis of AD, so its inhibition can be targeted by treatment with Etanercept with a different approach (due to high molecular weight and lack of CSF penetration) in the structure or Transfer it [83]. Lecozotan and Xaliproden are serotonin 5-HT1A receptor agonists that are effective in the mild to moderate phases of the disease. Tramiprostate binds to beta-amyloid to prevent plaque formation. Non-steroidal anti-inflammatory drugs (NSAIDs) that prevent the formation of amyloid plaques by an unknown mechanism. Bapineuzumab is a human monoclonal antibody against amyloid-beta that is not widely used. Vitamin E with its antioxidant properties, can be associated with mild to moderate Alzheimer's It is prescribed with AChE I but is contraindicated in people with cardiovascular problems or a history of warfarin use . This vitamin can slow the progression of the disease in the early stages of the disease [84] but is ineffective in the treatment of Alzheimer's [85].

Rolipram is a phosphodiesterase four inhibitor (PDE4 inhibitor) that prevents the breakdown of phosphodiesterase 4 in cAMP and thus controls the inhibitory effects of amyloid-beta on synapse function for a long time [86]. S-adenosylmethionine reduces amyloid plaques and age-related plaques and reduces the damaging effects of vitamin B deficiency in Alzheimer's, but the process that does this directly by inhibiting or reducing amyloid is unknown [87]. Avagacestat is a gamma-secretase inhibitor that is used in patients with mild to moderate Alzheimer's disease and its tolerance depends on the dose of the drug [88]. Gantenerumab is a human anti-amyloid monoclonal antibody that reduces amyloid in the brain. Amyloid plaque is reduced by two main mechanisms:

1.   Phagocytosis via FC receptor mediated by microglia

2.   Direct dissolution, which can lead to a transient increase in amyloid levels in the blood vessels of the brain [89].

Crenezumab is an anti-myeloid monoclonal antibody that delays the onset of cognitive impairment and slows its progression in patients [90]. anti-amyloid plaque antibodies can induce a new therapeutic process in reducing plaque in the treatment of AD by administering Gelsolin (Gm1) [91] peripherally and IA5P intravenously [92] and reducing beta-amyloid. Dantrolene Ryanodine receptor antagonist inhibits the release of calcium from the endoplasmic reticulum and plays a role in the treatment of AD by preventing the accumulation of amyloid and plaque formation in the hippocampus and slowing down the analysis of memory. Intracellular calcium control has an important effect on neuropathology [93]. Pinocembrin is a flavonoid that improves cognition and protection of the nervous and vascular systems and maintains the structure of neuropil and the function of small vessels, reduces the activity of glial cells and the level of inflammatory markers, without changing the myeloid and oxidative stress [94].

Some statins have the ability to cross the blood-brain barrier and reduce the production of beta-amyloid by altering controlled metabolism in the brain [95]. Herbal medicines such as Salvia officials [96], Melisa officials [97] can be used to compensate for cholinergic activity and increase acetylcholine. Withanolides are a group of steroid-C28 lactones that have the great neuroprotective ability [98]. Given the role of insulin in the brain life and the association between low insulin levels and Alzheimer's disease, [99] it is thought to be a link between two factors that have not yet been confirmed in the treatment of AD and require further research. Single-walled carbon nanotubes can deliver Alzheimer's drugs to the target organ, the lysosome. Acetylcholinesterase Due to its acetyl and ammonium structure, it can be easily absorbed by these carbon tubes and into the brain, into the lysosomes of nerve cells. The entry into the lysosome is dose-dependent and enters at low doses and in high doses causes toxic effects on the mitochondrial organ due to the collapse of the mitochondrial membrane potential and overproduction of oxygen-reactive species [100].

The effect of omega-3 fatty acids may be effective in the primary prevention and treatment of the disease by reducing the rate of cognition [101]. Ketones (KBS) are effective in generating extra energy for nerves and modifying metabolism and have proven their role in preventing and preventing the development of the disease by reducing Ab plaques and protecting neurons. Ketones are effective in the disease process depending on other factors such as APOE4 in the presence of this protein. There is a therapeutic property that reduces the level of Ab. Glucose inhibition prevents the production of ketones and increases fat consumption, increases Ab, which neutralizes the ketone treatment process, and glucose

hypometabolism is one of the diagnostic tools in AD. APOE4 is effective in the transfer of beneficial fats and its deficiency increases the plaque Ab. Finally, ketones are effective in reducing blood sugar and fat, increasing APOE4 protein, stabilizing blood sugar and increasing access to important fats, and the importance of a proper diet [102].

Alzheimer's disease has a great impact on the nutritional process of people with it, so the need for counselling is obvious and in case of deficiency of one of the factors, its supplement can be used [103]. Dietary supplements containing a grape-derived polyphenolic drug have been shown to improve cognitive function in Alzheimer's studies, and its monomer-shaped metabolites can accumulate in high concentrations in the brain and enhance cognitive function. Proanthocyanidin, one of the components of this monomer, improves basal synaptic transmission and synapse flexibility and long-term strengthening in hippocampal slices, and long-term treatment with it can improve spatial memory function and restore and reduce nerve function damage [104].

Contrary to the expectation that exercise causes net benefit in the AD process, it may cause some depression and distress in patients and due to the elderly age of patients and their cardiovascular complications, the need for controlled exercise due to its positive trend in patients' performance is evident. Other non-pharmacological treatments include light therapy. The difference in treatment between a person treated with bright light in the morning or in the afternoon and a person exposed to normal light in the home depends on a variety of conditions and requires further research [105]. Stimulation of deep regions of the brain (DBS) can also be effective in reducing the process of hippocampal destruction and cognition [106].

The effect of semantic lexical stimulation (LSS) on the quality of event memory (Episodic) in Alzheimer's patients improves cognitive function, lexical-semantic ability and verbal event memory and working memory, but an effect on executive functions and quality of attention and nonverbal memory and abilities There is no place to show that improving event memory has nothing to do with increasing attention. LSS includes a series of rehabilitation exercises related to meanings and words that, if practised continuously, reorganize semantic memory (general knowledge and real information) into coded declarative memory. This coding of semantic memory contributes to better performance of event memory. Long-term memory is more improved than short-term memory. Neurons are able to respond appropriately to various stimuli due to their flexibility and extensive interconnection [107]. Depression is a common disease with Alzheimer's that should be treated [108]. Depression contributes to the progression of Alzheimer's and the use of sertraline, fluoxetine and clomipramine can alleviate the symptoms of depression [109].

Sertraline is one of the antidepressants in the group of serotonin reuptake inhibitors that did not show improvement in cognitive function in patients compared to the control group [110]. Trazodone is a hypnotic antidepressant that has no effect on cognitive impairment in AD [111]. One of the symptoms that may occur in patients with Alzheimer's is obstructive sleep apnea (OSA). Continuous nasal positive air pressure (CPAP) therapy can have a significant impact on the quality of life of people with mild to moderate Alzheimer's. People with lower degrees of depression have responded better to this treatment [112]. One of the problems of people with dementia is apathy. The use of Methylphenidate in these people has led to improved mood, daily life activities and performance and has reduced the difficulty of caregivers [113]. Psychedelic drugs such as Olanzapine and Risperidone are used to treat behavioural disorders in Alzheimer's patients. In the treatment of psychosis in AD patients, the use of Aripiprazole showed better results in secondary outcomes than the primary outcomes in anxiety, agitation, and depression [114]. Risperidone also did not have acceptable results in the treatment of psychosis [115]. One of the psychological problems in AD patients is agitation. Citalopram is an antidepressant drug that affects the genetic diversity of serotonin 2A and 2C receptors (HTR2A, HTR2C) and leads to an antidepressant response [116].

Ganoderma Lucidum spore powder (a type of basidiomycete) has been studied to treat Alzheimer's disease but has no effect on the treatment of Alzheimer's disease in humans. Yokukansan is one of the oldest herbal medicines in Japan that has been studied for its effect on Alzheimer's. It has no effect on improving cognitive functions and its concomitant use with donepezil indicates active symptoms such as paranoid and delusions, hallucinations, functional disorders and Aggression has not improved, but it has a positive effect on daily rhythm regulation, emotional disorders, anxiety and fear, and to a lesser extent affects emotions. In Alzheimer's patients who are taking Atorvastatin with Donepezil due to high blood fats, lowering LDL has no effect on improving Alzheimer's.

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