INTERNATIONAL JOURNAL OF MULTIDISCIPLINARY RESEARCH AND ANALYSIS

ISSN(print): 2643-9840, ISSN(online): 2643-9875

Volume 08 Issue 02 February 2025

DOI: 10.47191/ijmra/v8-i02-38, Impact Factor: 8.266

Page No. 799-805

Nutritive Approach: A Natural Gold Treatment for Alzheimer's disease

Dhanashree S Dorge[1], Siya S Veer[2], Laxman R Bandgar[3], Madhuri S Nalawade[4], Kumudini R Pawar[5]

[1,2,3] Students Abhinav Education Society's College of Pharmacy (B.Pharm), NArhe, Pune-41

[4,5]Associate Professor Abhinav Education Society's College of Pharmacy (Narhe) Pune-41

ABSTRACT: A nutritive strategy aimed at preventing, slowing, or halting the pathogenesis of disease represents a hopeful avenue that has garnered extensive research attention. A significant body of epidemiological evidence indicates that dietetic intake may play a pivotal part in the beginning and advancement of Alzheimer Disease or Alzheimer Dementia (AD). Modifiable environmental factors contributing to AD include potential metabolic disturbances arising from dietetic deficiencies or excesses, which may be addressed through nutritive subjoining or dietetic adjustments. Numerous nutritive subjoining encompass a variety of fitness advancing components, such as free-radical scavengers, vitamins, trace minerals, carbohydrates, and lipids, which may possess unique MOA that influence cytological well-being and rebirth, declining or especially target infectious routes involved in the progression of AD. The implementation of nutritive modifications is advantageous due to their cost-effectiveness, ease of application, social acceptability, and general safety, with minimal adverse effects in most instances. A wide range of nutritive interferences has been explored and continues assessed in the quest to identify effective additives, combinations of additives, or dietetic changes that can aid in the precaution and therapy of AD. This evaluation emphasizes some important nutritive complexes and dietetic transformations that have been examined in human populations, while also discussing the rationale for their potential benefits in the prohibition and management of AD.

KEYWORDS: Alzheimer Dementia (AD), Nutrition, Dietary supplements, Omega 3 fatty acids, Proteins.

INTRODUCTION

Alzheimer Disease or Alzheimer Dementia (AD) is a complex neurodegenerative condition influenced by multiple factors. AD is the most common type of dementia. AD is commonly found in elder who causes cognitive dysfunction in them. AD is named in honor of Alois Alzheimer, a German psychiatrist in 1906. He had a patient named Auguste D, her age was fifty, suffered from mental illness. After her death, the autopsy of her brain showed dense deposits, now known as amylaceous plaques and twisted strands of fiber known as neurofibrillary tangles (NFTs) [1]. AD is characterized by various biochemical changes, which encompass alterations in the metabolism of amylaceous precursor protein, tau protein phosphorylation, free radical damage, energy impairment, chondriosome dysfunction, and swelling, dysfunction of cell membrane fats and disturbance of chemical transmitter routes. [2] Medications such as antidepressants, antipsychotics, mood stabilizers, anxiolytics, and hypnotics are employed in the management of behavioural disturbances. Additionally, nutritive subjoining and regular physical activity be of great importance in both the prohibition and therapy of AD. [3]

AD is categorized into two distinct types- 1) Early-onset Alzheimer- This type of AD is normally found in people aged under 65. Disease diagnosed in their 40s or 50s age. [3] 2) Late-onset AD is the most prevalent form of this condition that is found in people having age 65 and older. Disease process was flaming for many years. [4] The symptoms of AD are forgot about recent conversation, things, increasing confusion, weight loss, difficulty in changing position, problems with short and long term memory. [5] Diagnosis of AD is done by various tests that are- Medical history assessment, evaluation of mental status, neuropsychological assessments, blood analysis, cerebrospinal fluid examinations, and brain imaging studies and ECG. [6] AD develops from multiple causes such as genetics, lifestyle, environment, age, family history, head injury vascular disease and infection. It is explained by two methods- Cholinergic hypothesis [7] and amylaceous hypothesis. [8] Risk factors of AD are age and gender, genetic factor, environmental factor, infection, cardiovascular disease, obesity and diabetes. [9] Complications associated with AD include

restlessness and agitation, issues related to bladder and bowel control, depression, an increased risk of falls, infections, wandering behaviour, as well as malnutrition and dehydration. [10]

PATHOPHYSIOLOGY

AD is marked by deterioration in brain function, leading to impairments in cognitive abilities and behavioural changes in individuals. It has been observed at the microscopic level that the evolution of amylaceous plaques, also known as senile plaques, consists of amorphous aggregates of Amyloid β (Aβ). Additionally, there is an accumulation of hyperphosphorylated Tau protein, which leads to the formation of neurofibrillary tangles, accompanied by significant neuronal loss. These formed plaques are abnormal clusters of protein fragments and degenerative nerve cells consist of twisted strands of proteins called tangles. Pathophysiology encompasses several factors, including choline-based disease, amylaceous and tauopathies, as well as free radical damage and chondriosome dysfunctions. [11] The pathological characteristic involves the evolution of neuritic plaque, which arises from the accumulation of Aβ. Aβ consists of small soluble polypeptides that are produced through the separation of the APP by the enzymes α-secretase, β-secretase, and γ-secretase. A disruption in the synthesis and elimination of Aβ results in the formation of various toxic oligomeric structures, including protofibrillar, filaments, and plaques, based on the degree of multimerization. In free radical damage supposition big oxygen intake of the brain, means that the brain has a greater risk of free radical damage. Free radical damage resulting from beta accumulation plays an central role in the growth and advancement of dementia. Taking into account the causes and effects of inflammatory processes, this condition is classified as a neurodegenerative disease. The neuron is characterized by a significant presence of polyunsaturated FA. These FA can engage with reacton forming free radicals, developing in lipid oxidation and subsequent cellular apoptosis. Additionally, a reduced level of glutathione within nerve cell contributes to free radical damage-related destruction. [12] The cholinergic hypothesis examines the diminished influence of apolipoprotein (APOE) in relation to the "Cholinergic Hypothesis." It has been observed that the interactions with cholinergic receptors is diminished in certain areas of the brain, particularly in cases of slight to medium impairment, and this depletion is linked to various neurological signs. [13] Metal homeostasis is included in the progression of these diseases. The ionosphere and metal chelators serve as regulators of transition metal homeostasis. Redox-active transition metals, primarily including copper, iron, and various trace metals, be of great importance in this process are found to be high . Neurodegenerative disorders also include copper, manganese, aluminium and zinc [14].

NUTRINTIONAL MANAGEMENT

Nutrition encompasses a variety of nourishing components, both in terms of quantity and quality that are present in food. Without adequate knowledge, individuals may consume inappropriate types and amounts of food, which can adversely affect their brain well-being. Proper nutrition empowers patients, enabling them to make informed dietetic choices. Furthermore, nutrition is of great importance in the evolution of the human immune organization and aids in the prohibition of severe complications associated with AD.

Objectives

1 To encourage and enhance a well-being dietetic pattern.
2 To sustain the enjoyment of eating by conveying affirmative messages regarding food selections.
3 To postpone or avert the severe consequences associated with AD.
4 To equip individuals with practical resources for daily meal planning, rather than concentrating on particular micronutrients or individual foods.
5 To enhance overall well-being and facilitate daily activities.
6 To prevent the consumption of detrimental substances such as tobacco and alcohol.

Classification of nutrients:

Vital nutrients

Vital nutrients are pivotal for the normal functioning of the body and cannot be produced internally. As these nutrients cannot be synthesized by the body, it is imperative that they are incorporated into the diet. They are fundamental to metabolic processes and the proper functioning of tissues and organs. e.g. Vitamin B12 B6, Amino acid, Fatty acid, and minerals (selenium, zinc, iron, copper).

Non- vital nutrients

Non-vital nutrients are those that the body can produce independently and can also be obtained through dietetic sources. These nutrients can either be synthesized internally or incorporated into one's diet. They have a significant effect on well-being. E.g.- dietetic fibres, Vitamin K, cholesterol, Amino acid. [15]

Nutritive Approach: A Natural Gold Treatment for Alzheimer's disease

A. Micronutrient:

Minerals are vital trace elements commonly present in various food sources, including plants, fruits, meat, and fish. Numerous minerals are pivotal for the nutritive management of AD. Recent research has been highlighted the free-radical scavenger and anti-swelling properties of certain minerals, which may be significant for brain well-being and the progression of AD. [16]

NUTRITIVE ELEMENTS WITH THEIR CORRESPONDING WELL-BEING ADVANTAGES AND SOURCES:

Sr. No.	Vital substances	Advantages for well-being	Source
1)	Fat-soluble Vitamins Vitamin A	evolution of CNS, effective lowering cognitive decline and AD pathology [17]	Beef liver, cod liver oil, sweet potatoes, carrots, spinach, broccoli, mango, tomato juice, and pumpkin pie. [18]
2)	Vitamin D	Physiological function and protection of the CNS and regulation of bone metabolism, cognitive decline [19], improves cognitive performance in people having senile dementia [20]	Salmon, herring, sardines, cod liver oil, canned tuna, egg yolks, mushrooms, and foods fortified with vitamin D. [21]
3)	Vitamin E	free-radical scavengrs, anti-inflammatory properties, protect against neurodegeneration [22]	Sunflower seeds, almonds, hazelnut oil, pine nuts, goose meat, avocado, Brazil nuts, mango, and turnip greens. [23]
4)	Vitamin K	Anti-aging, AD fighting properties, nerve cell death reduced, protects nerves from harmful sub in the brain [17]	Broccoli, Brussels sprouts, cabbage, grains, eggs, seafood, meat, spinach, and vegetable oil. [24]
5)	Water-soluble Vitamins Vitamin C	free-radical scavengrs, delays the amylaceous plaque formation [18]	Blackcurrants, guavas, acerola cherries, mustard spinach, kiwis, broccoli, lemons, papayas, strawberries, oranges. [25]
6)	Vitamin B12	Maintaining neuronal well-being and haematopoiesis, [26] beneficial effects on cognition and inflammatory status, [17] reduce the risk of memory loss [27]	Beef, fortified cereal, salmon, milk and dairy products, eggs. [28]
7)	Vitamin B6	Prevents shrinkage of main regions in the brain, decrease the level of homocysteine [29]	Dairy products, eggs, poultry liver, beef, carrots, spinach, sweet potatoes, green peas, bananas, and avocados. [30]
8)	Vitamin B3	Niacin converts food into cytological energy, repairing DNA and free-radical scavengrs properties, Nicotinamide restores cognitive function, [17] repairing brain cells, treating symptoms like brain fog, depression, etc. [31]	Beef, chicken, seafood, whole grain rice, enriched cereals and breads, nuts, seeds, legumes, and bananas. [32]
9)	Vitamin B1	Thiamine shows neuroprotective effects, benfotiamine improves MMSE based cognitive scores and delays cognitive decline. [33]	Pork, fish, beans, lentils, green peas, sunflower seeds, yogurt. [34]
10)	Minerals Iron		

		In CNS iron synthesis myelin, neurotransmitter, metabolism and maintaining high capacity of neuron, synthesis of ATP, ADP and enzyme functions. [16]	Red meat, poultry, fish, lentils, vegetables, fortified food. [35]
11)	Copper	CNS evolution, neuromodulation, angiogenesis and hypoxia. [15]	Shelfish, legumes, red meat, vegetables, nuts. [15]
12)	Manganese	Maintains protein, nucleic acid, lipid membranes, vital for cytological and chemical messenger signalling, pivotal role for neurological transmissions. [15]	Shellfish, nuts, brown rice, oatmeal, legumes, black tea, black pepper, spinach, pineapple. [15]
13)	Selenium	Maintenance of human well-being, acts on immune defence, thyroid gland, cardiovascular functions and cancer prohibition, biological oxidation, cytological differentiation, protein synthesis, gene transcription, depletion of Aβ aggregation, hyperphosphorylation of tau protein, and the prohibition of neuronal death. [15]	Bread, cereals, seafood, cruciform, vegetables, Brazil nut. [36]
14)	Zinc	Maintains human well-being, an element involved in various physical and chemical signalling, biochemical pathways, cell organization, immune system, the processes of maturation and evolution, wound healing, immune system regulation, catalytic reactions, and substance synthesis, as well as the regulation of neurotransmitters, are all interconnected. [15]	Dark chocolate, some vegetables, whole grains, eggs, dairy products, nuts, seeds, legumes, shellfish meat. [37]

B. Macronutrients:

Macronutrient is one of the important factors for the human body, it gives energy to human body.

Classification of Macronutrients

1) Carbohydrates:

Carbohydrates are a food nutrient, an important source of energy. Glucose is significantly implicated in the progression of AD. Elevated glucose levels have been associated with the deterioration of particular brain regions. Furthermore, these heightened glucose levels have been linked to an increase in various proteins within neurons. [38]

2) Fatty Acids:

Dietetic fatty acid is one of the macronutrients in the human body. Omega-3 FA be of great importance in the context of AD. [39] They participate in managing BP, blood consistency, vasocontraction and protected system functions. Furthermore, omega-3 FA are pivotal for the growth and evolution of the brain. [40]

3) Proteins:

The protein is mostly present in muscle, bone, skin, hair and other body parts as well as tissues. AD results from the irregular accumulation of proteins within brain cells, with the beta amylaceous protein being a key contributor to the condition. [41] They come in different molecular forms that collect in neurons. The amylaceous protein and tau protein are responsible for AD. The amylaceous protein accumulation creates plaques surrounding brain cells, while the tau protein accumulation leads to the formation of tangles within these cells. [42]

C. Polynutritionl additive

Nutritive subjoining is of great importance in influencing the progression and incidence of AD and represent a contemporary strategy being explored by researchers. Particularly formulated medical dietetic foods, available in various combinations, are utilized as targeted interventions for AD. This strategy is predicated on the understanding that the disease induces complex biochemical changes in metabolic pathways, which are influenced by multiple nutritive co-factors. Addressing these intricate pathways may necessitate combination therapies to achieve optimal results, as the utilization of sole-operative enhancement is often insufficient, except in rare cases where a particular nutritive deficit is present. Recent inventive advancement focus on delivering these nutrient combinations in a controlled environment to magnify neuronal well-being and ensure synaptic steadiness.

1) Souvenaid :

Souvenaid is the pioneering medical nutrition product formulated to enhance synapse formation and functionality in the early stages of AD. It has been part of a continuous improvement program spanning over 12 years. The recommended dosage is a 125-ml drink, providing 125 k cal, to be consumed once daily as a nutritive additive tailored to meet the particular nutrient needs associated with the disease. Souvenaid includes ω-3 PUFA (EPA and DHA), uridine (in the form of uridine monophosphate), and choline, along with phospholipids and various other co-factors. Clinical trials involving sick persons with slight AD receiver the native drug indicated a notable improvement in memory function and enhanced brain functional connectivity, a finding that was also corroborated by an animal study. Additionally, there was a preservation of the organization of brain networks, which may hypothetically mitigate the progressive disruption of these networks over time. Nevertheless, the intervention did not yield significant changes in the Modified Disease Assessment Scale Cognitive Sub-scale or other outcome measures. Despite the observed benefits in patients receiving the native drug, Souvenaid did not demonstrate ability to slow cognitive decline in individuals undergoing therapy for mild-to-moderate AD. [43]

2) Chyawanprash :

Chyawanprash has served as a potent herbal nutritive additive since ancient times. This formulation is recognized for its healing properties, aimed at enhancing overall immunity and promoting longevity. Enriched with beneficial nutriments, which includes free-radical scavengers such as Vit C, amino acids, dietetic fibers, Na and a significant concentration of alkaloids and steroidal glycoalkaloids, it is an excellent additive for cardiovascular well-being and managing elevated cholesterol levels. Additionally, it contains a blend of rejuvenating flora, flavours, ores, and Vit C. This remarkable herbal additive is particularly advantageous for the prohibition of various brain-related conditions, owing to its immunomodulatory and rejuvenating effects. [44]

Cognitive degeneration confusion, such as mental illness and AD, are believed to be partially linked to depletion in cholinergic neurons, a particular category of brain cells. Chyawanprash may enhance cognitive function by stimulating the activity of these neurons. This formulation is composed of 12 vital ingredients, including Amla, Nimtree, Pipal, Kanaje, Basil, Arjuna, Ghee, and Honey. Both Indian gooseberry and vitamin C have been demonstrated to improve memory, which may explain the cognitive benefits associated with chyawanprash. Research involving mice has indicated that daily enhancement with chyawanprash can lead to improved memory. Additionally, its free-radical scavengers properties contribute to enhanced brain function, further supporting the role of cholinergic neurons in cognitive well-being. [44]

3) Axona :

Axona is the nutritive additive available for nutritive management of mild-to-moderate stage AD. Sick persons with AD experience a gradual and region-particular depletion in the cerebral metabolic rate of blood glucose, particularly affecting the posterior cingulate, parietal, temporal, and prefrontal cortices, with this decline manifesting early in the disease progression. Typically, glucose serves as the primary energy source for neuronal function; however, during certain conditions, such as starving, acetone bodies produced by the liver can act as other energy originators. Research utilizing mouse models has indicated that inducing ketosis may offer therapeutic benefits for Alzheimer's. Axona is an established product of octanoic acid designed particularally to address the deficiency diseases linked to AD. This medium-chain triglyceride product, made up of glycerin and octanoic acid, is break down in the liver to produce the acetone body BHB, giving neurons with an alternative power originator. Axona is administered following meals. [45]

CONCLUSION

A thorough approach to nutrition must be considered a fundamental aspect of managing AD in all patients. However, challenges arise due to insufficient resources and a general lack of awareness among patients, well-being care providers, policymakers, and society at large regarding the provision of nutritive subjoining for individuals with Alzheimer's. It is vital for policymakers to acknowledge the significance of nutritive therapy and allocate resources towards the training and evolution of registered

dieticians. This would enable the integration of nutritive support into lifestyle modification therapies for patients, ultimately helping to alleviate the complications associated with AD.

REFERENCES

1) Maurer, K., Volk, S., & Gerbaldo, H. (1997). Auguste D and Alzheimer's disease. Lancet (London, England), 349(9064), 1546–1549.
2) Cai, H., Cong, W. N., Ji, S., Rothman, S., Maudsley, S., & Martin, B. (2012). Metabolic dysfunction in Alzheimer's disease and related neurodegenerative disorders. Current Alzheimer research, 9(1), 5–17.
3) https://www.medicalnewstoday.com/articles/159442
4) World Health Organization. (2016). ICD-10 International statistical classification of diseases and related health problems 10th revision Volume 2 Instruction manual Fifth edition 2016.
5) https://www.mayoclinic.org/diseases-conditions/alzheimers-disease/symptoms-causes/syc-20350447
6) de Leon, M. J., Convit, A., DeSanti, S., Bobinski, M., George, A. E., Wisniewski, H. M., ... & Saint Louis, L. A. (1997). Contribution of structural neuroimaging to the early diagnosis of Alzheimer's disease. International Psychogeriatrics, 9(S1), 183-190.
7) Breijyeh, Z., & Karaman, R. (2020). Comprehensive Review on Alzheimer's Disease: Causes and Treatment. Molecules (Basel, Switzerland), 25(24), 5789.
8) Farlow, M. R., Lahiri, D. K., Poirier, J., Davignon, J., Schneider, L., & Hui, S. L. (1998). Treatment outcome of tacrine therapy depends on apolipoprotein genotype and gender of the subjects with Alzheimer's disease. Neurology, 50(3), 669–677.
9) https://www.alzint.org/about/risk-factors-risk-reduction/
10) https://www.healthline.com/health/alzheimers-disease-complications
11) Kamboj, Parul & Thakur, Dr. Ajit. (2018). Pathophysiology and management of Alzheimer's disease: an overview. Journal of Analytical & Pharmaceutical Research. 7.10.15406/japlr.2018.07.00230
12) Liu, J., Liu, Z., Zhang, Y., & Yin, F. (2015). A novel antagonistic role of natural compound icariin on neurotoxicity of amyloid β peptide. Indian Journal of Medical Research, 142(2), 190-195.
13) Kumar, A., Singh, A., & Ekavali (2015). A review on Alzheimer's disease pathophysiology and its management: an update. Pharmacological reports : PR, 67(2), 195–203.
14) Barage, S. H., & Sonawane, K. D. (2015). Amyloid cascade hypothesis: Pathogenesis and therapeutic strategies in Alzheimer's disease. Neuropeptides, 52, 1–18.
15) Fei, H. X., Qian, C. F., Wu, X. M., Wei, Y. H., Huang, J. Y., & Wei, L. H. (2022). Role of micronutrients in Alzheimer's disease: Review of available evidence. World Journal of Clinical Cases, 10(22), 7631.
16) Liu, J. L., Fan, Y. G., Yang, Z. S., Wang, Z. Y., & Guo, C. (2018). Iron and Alzheimer's Disease: From Pathogenesis to Therapeutic Implications. Frontiers in neuroscience, 12, 632.
17) Ono, K., & Yamada, M. (2012). Vitamin A and Alzheimer's disease. Geriatrics & gerontology international, 12(2), 180-188.
18) Bhatti, A. B., Usman, M., Ali, F., & Satti, S. A. (2016). Vitamin Supplementation as an Adjuvant Treatment for Alzheimer's Disease. Journal of clinical and diagnostic research : JCDR, 10(8), OE07–OE11.
19) Gangwar, A. K., Rawat, A., Tiwari, S., Tiwari, S. C., Narayan, J., & Tiwari, S. (2015). Role of Vitamin-D in the prevention and treatment of Alzheimer's disease. Indian journal of physiology and pharmacology, 59(1), 94–99.
20) https://www.healthline.com/nutrition/9-foods-high-in-vitamin-d
21) Grimm, M. O., Mett, J., & Hartmann, T. (2016). The Impact of Vitamin E and Other Fat-Soluble Vitamins on Alzheimer´s Disease. International journal of molecular sciences, 17(11), 1785.
22) https://www.ncbi.nlm.nih.gov/pmc/articles/PMC5751107/
23) https://www.healthline.com/nutrition/foods-high-in-vitamin-e
24) https://www.healthline.com/nutrition/foods-high-in-vitamin-k
25) https://www.healthline.com/nutrition/vitamin-c-foods
26) Lauer, A. A., Grimm, H. S., Apel, B., Golobrodska, N., Kruse, L., Ratanski, E., Schulten, N., Schwarze, L., Slawik, T., Sperlich, S., Vohla, A., & Grimm, M. O. W. (2022). Mechanistic Link between Vitamin B12 and Alzheimer's Disease. Biomolecules, 12(1), 129.
27) https://www.aan.com/PressRoom/Home/PressRelease/872
28) https://www.healthline.com/nutrition/vitamin-b12-foods
29) https://www.hsph.harvard.edu/nutritionsource/vitamin-b6/
30) https://www.healthline.com/health/vitamin-b6-foods

31) https://www.netmeds.com/health-library/post/vitamin-b3-functions-food-sources-deficiencies-and-toxicity
32) https://www.hsph.harvard.edu/nutritionsource/niacin-vitamin-b3/
33) https://www.mdpi.com/20763921/12/2/415#:~:text=A%20limited%20number%20of%20human,brain%20amyloid%20accumulation%20%5B44%5D.
34) https://www.hsph.harvard.edu/nutritionsource/vitamin-b1/
35) https://www.nhs.uk/conditions/vitamins-and-minerals/iron/
36) https://ods.od.nih.gov/factsheets/SeleniumHealthProfessional/#:~:text=Brazil%20nuts%2C%20seafood%2C%20meat%2C,regions%20%5B3%2C13%5D.
37) https://www.healthline.com/nutrition/best-foods-high-in-zinc
38) Caamaño, D., de la Garza, A., Beltrán-Ayala, P., & Chisanuano, A. M. (2016). Nutrition and neurodegenerative diseases: the role of carbohydrates and gluten. Int J Nutr Sci, 1(2), 1-6.
39) Gustafson, D. R., Bäckman, K., Scarmeas, N., Stern, Y., Manly, J. J., Mayeux, R., & Gu, Y. (2020). Dietary fatty acids and risk of Alzheimer's disease and related dementias: observations from the Washington Heights-Hamilton Heights-Inwood Columbia Aging Project (WHICAP). Alzheimer's & Dementia, 16(12), 1638-1649.
40) Derbyshire, E. (2018). Brain health across the lifespan: a systematic review on the role of omega-3 fatty acid supplements. Nutrients, 10(8), 1094.
41) Rahman, M. M., & Lendel, C. (2021). Extracellular protein components of amyloid plaques and their roles in Alzheimer's disease pathology. Molecular Neurodegeneration, 16(1), 59.
42) Sajjad, R., Arif, R., Shah, A. A., Manzoor, I., & Mustafa, G. (2018). Pathogenesis of Alzheimer's disease: role of amyloid-beta and hyperphosphorylated tau protein. Indian J. Pharm. Sci, 80(4), 581-591.
43) Burckhardt, M., Watzke, S., Wienke, A., Langer, G., & Fink, A. (2020). Souvenaid for Alzheimer's disease. Cochrane Database of Systematic Reviews, (12).
44) https://www.netmeds.com/health-library/post/chyawanprash-benefits-uses-ingredients-method-dosage-and-side-effects
45) Roman, M. W. (2010). Axona®(Accera, Inc): a new medical food therapy for persons with Alzheimer's disease. Issues in mental health nursing, 31(6), 435-436.