ISSN(O):2582-693X

Original Article

# Interconnectedness between Homoeopathic Therapeutics and their Pathophysiological Changes in Dementia

**Tanuj Rajvanshy[1], Resham Rana[2]**

[1]Vice-Principal, Dept. of Materia Medica, Swasthya Kalyan Homoeopathic Medical College & Research Centre, Jaipur, Rajasthan

[2]M.D. (PGR), Dept. of Materia Medica, Swasthya Kalyan Homoeopathic Medical College & Research Centre, Jaipur, Rajasthan

**Corresponding Author:** Prof. Dr. Tanuj Rajvanshy,Vice-Principal, Dept. of Materia Medica, Swasthya Kalyan Homoeopathic Medical College & Research Centre, Jaipur, Rajasthan

E-mail: drtanujrajvanshy@gmail.com



How to cite this article: : Rajvanshy T, Rana R. Interconnectedness Between Homoeopathic Therapeutics and Their Pathophysiological Changes in Dementia. GFNPSS-IJMR 2026; 7:3: 3328-3335

**Submitted:** 10 March 2026: **Accepted:** 18 March 2026: **Published:** 31 March 2026

## Abstract

**Background:** Dementia is a form of chronic and progressive neurological disorder of the nervous system that deranges everyday functioning, most noticeable through impaired memory, cognition, and language. It is biologically linked to factors including oxidative damage, disturbances in neurotransmitter activity, abnormal protein deposits, deterioration of nerve cells, and impaired vascular health. The present scenario and current medical approaches largely focus on alleviating symptoms rather than reversing the course of the illness.

**Materials and Methods:** A review-based approach was undertaken, focusing on the pathophysiological changes in dementia and their correlation with the action of selected homoeopathic medicines. Literature was analysed to highlight the role of individualized prescribing in modulating metabolic and neurofunctional processes.

**Results:** Findings suggest that homoeopathy, being a holistic and individualized system, may complement existing therapies by stimulating innate healing responses. Remedies correlated with dementia pathology demonstrate potential in addressing oxidative stress, neuro inflammation, and cognitive decline, thereby extending therapeutic options beyond conventional symptomatic management.

**Conclusion:** Integrating homoeopathic principles with conventional understanding of dementia pathology could provide a more comprehensive management strategy. Such an approach not only supports symptom relief but also targets deeper neurofunctional and metabolic imbalances, potentially improving quality of life and expanding treatment alternatives for dementia patients.

**Keywords:** Dementia, Homoeopathy, Neurodegeneration, Oxidative Stress

## Introduction

Dementia is a form of chronic or progressive neurological syndrome characterized by a decline in cognitive functions such as memory, thinking, language, reasoning, and judgment that is severe enough to interfere and disrupt the daily life and activities [1].

From a biological point of view, neurodegeneration, abnormal protein aggregation, imbalance in neurotransmitter activity, oxidative stress, and vascular impairment are among the pathophysiological alterations which is directly associated with dementia. Indigenous medicine mostly treats symptoms and has little power to stop the progression of disease. As a holistic medical approach, homoeopathy places a strong emphasis on individualization and aims to activate the natural healing process of body. This article explores the relationship between homoeopathic medicines and the pathogenesis of dementia to yield out the important information for integrative care.



https://doi.org/10.46376/ijmr/07.03.2026:3328-3335

## Pathophysiological Changes in Dementia [2]

| | |
|---|---|
| Degeneration of neurons | • Progressive neuronal loss specifically in the brain and hippocampus which resulting into cognitive deterioration, disorientation and memory impairement |
| Neurofibrillary tangles with amyloid plaques | • Accumulation of Beta amyloid protein outside of neurons leads to entanglements of Neuronal tau protein which disrupts synaptic communication. |
| Unbalanced Neurotransmitters | • Alzheimer's dementia is a result of insufficient acetylcholine.<br>• Altered dopaminergic and serotonergic levels alters the Behavioral symptoms. |
| Stress from oxidation and mitochondrial failure | • Death of neurons is caused and accelerated by damage from free radicals causing weariness, indifference and further gradual decline in cognitive function. |
| Insufficiency of cerebrovascular Function | • Decreased cerebral blood flow causes vascular dementia resulting into mood fluctuation, impaired cognition and abrupt memory loss. |
| The Inflammatory Mechanism | • In later stages neurons are damaged by prolonged neuroinflammation and microglial activation. |

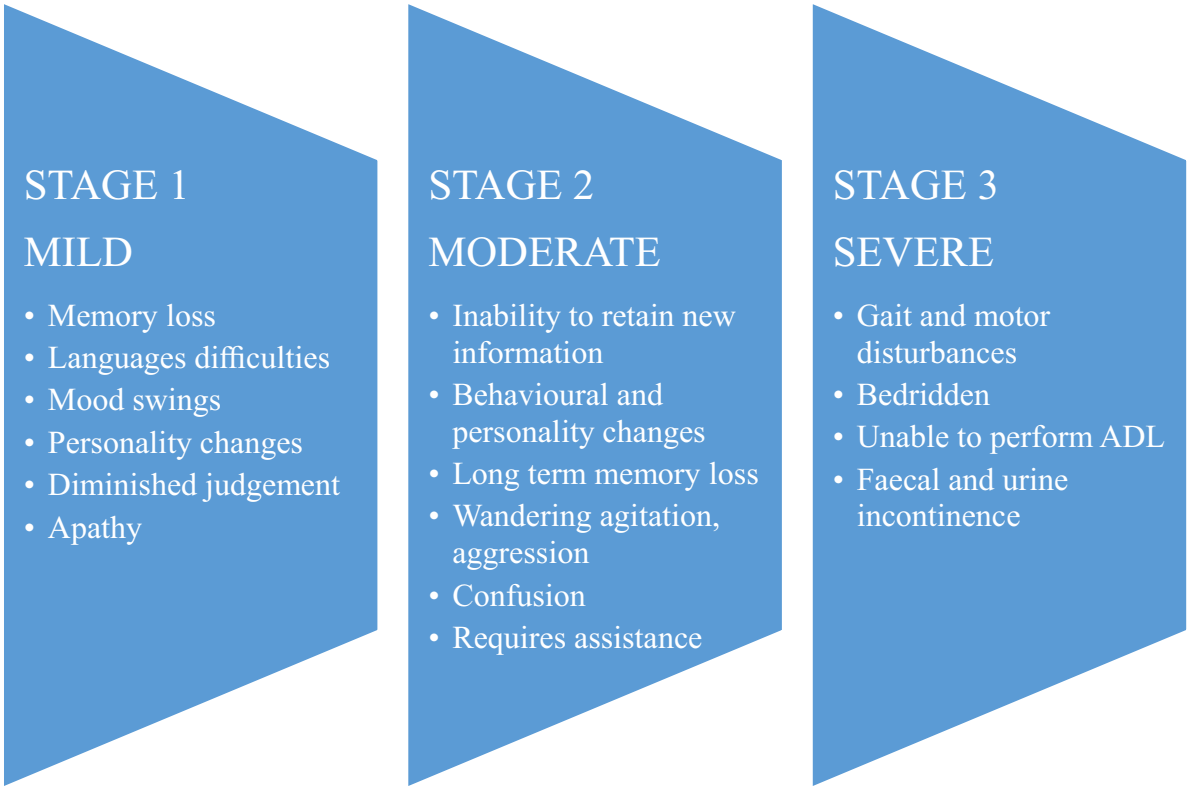
## Types of Dementia [2]:

| CORTICAL DEMENTIA: | SUBCORTICAL DEMENTIA: | PROGRESSIVE DEMENTIA: | PRIMARY DEMENTIA : | SECONDARY DEMENTIA: |
|---|---|---|---|---|
| •Predominantly affecting brain's cortex or outer layer .<br>•Cortical dementia tend to cause problems with memory, language, thinking and social behaviour. | •Dementia that affects parts of brain below the cortex.<br>•Subcortical dementia tends to cause changes in emotions and movement in addition to problems with memory. | •Worsens over time and interfere more and more with cognitive abilities. | •Dementia such as Alzheimer's disease is itself the main disease and not a result of any other medical disorder | •Dementia that occur as a result of a physical damage to brain. |



https://doi.org/10.46376/ijmr/07.03.2026:3328-3335

## Stages of Dementia[2]




## Homoeopathic Remedies:

### 1. Abrotanum

Dull and feeble mind with no capacity for thinking, as if all mental and bodily power were gone easily tired out by conversation or mental effort [3].

| Active Principle (Abrotanum) | Pathophysiological Effect | Correlation with Dementia |
|---|---|---|
| Thujone, cineole (volatile oils) | CNS irritability, confusion, memory lapses | Decline in cognition and disorientation[4] |
| Bitter lactones (absinthin) | Poor assimilation, malnutrition | atrophy of neurons, cortical degeneration[5] |
| Flavanoids, tannins | Altered vascular tone, weak circulation | Vascular dementia contribution[6] |
| General action (marasmus) | Progressive wasting & nervous exhaustion | Gradual weakness of cortical and progress in dementia |


https://doi.org/10.46376/ijmr/07.03.2026:3328-3335

## 2. Agaricus

Dementia due to mental palsy, crypto mania, makes verses, sings, talks but does not answer questions.

Constant talking and laughing and thinks himself as immensely wealthy and happy (2nd stage of dementia). Confusion of head, cannot find the right word and wants to be alone, frequently caused by over excited mind and worry.

| Active principle ( Agaricus) | Pathopysiological Effects | Correlation with Dementia |
|---|---|---|
| Muscimol (isoxazole derivative) | Potent GABA-A receptor agonist- sedative , alters neurotransmission | Impaired memory, confusion,drowsiness- resembles cognitive decline in dementia[7] |
| Ibotenic acid | Excitotoxicity via glutamate receptor overstimulation results into neuronal death | Loss of neurons and cortical atrophy similar to neurodegeneration in Alzheimer's[8] |
| Muscarine(alkaloid) | Parasympathomimetic: excessive cholinergic activity leads to delirium , confusion. | Disturbance in cholinergic pathways, which are central in pathophysiology of dementia[9] |
| Bufotenine( trace indole derivative) | Serotonin receptor interaction leads to hallucinations, altered perception. | In dementia there is behavioural changes (hallucination , delusions)[10] |

## 3. Anacardium Orientale

Dementia of old people with rapid memory loss and mental vigor, mental fatigue and brain-fag from overexertion.

| Active principle (Anacardium) | Pathopysiological Effects | Correlation with Dementia |
|---|---|---|

## 4. Calcarea Phosphorica

Dementia in young persons or in tendency of masturbation, total loss of memory, writes

| | | |
|---|---|---|
| Cardol & Cardanol (alkyl phenols) | Neurotoxic at higher doses causes mitochondrial dysfunction , impairement of neuronal metabolism | Loss of energy in neurons contributing to memory loss and cortical atrophy[12] |
| Anacardic acids (phenolic lipids) | Strong activity of pro-oxidant leads to lipid peroxidation,oxidative stress resulting into neuronal membrane damage | Oxidative stress is a major cause of neurodegeneration in Alzheimer's and other dementias[11] |
| Tannins & Flavonoids | Antioxidant and neuroprotective effect- balance oxidative stress | Protective role against memory impairement[14] |
| Anacardium resin ( irritant principle) | Altered neurotransmission induces mental dullness, confusion , irritability | Resembles the clinical state of dementia i.e., forgetfulness, loss of confidence, "two will" phenomenon[13] |



https://doi.org/10.46376/ijmr/07.03.2026:3328-3335

wrong words, wishes to be at home, and when wants to go out from place to place, does not want to do what he has to do, easily frightened and depressed.

| Active principle (Calcarea Phos) | Pathopysiological Effects | Correlation with Dementia |
|---|---|---|
| Calcium ions($Ca^{2+}$) | Calcium is essential for neurotransmission, synaptic plasticity &memory formation | Calcium dysregulation in aging- excitotoxicity-neuronal loss seen in Alzheimer's pathology[15] |
| Phosphate ions($PO4^{3-}$) | Major role in ATP production , phospholipid membrane integrity & phosphoprotein signaling in neurons | Loss of energy & defective phosphorylation impair cognition & neuronal survival in dementia[16] |
| Calcium Phosphate balance | Balance neuronal structure, myelination & conduction of signals | Imbalance results into neurodegeneration, poor synaptic repair - cognitive decline[17] |
| Bone brain metabolic link | Hypophosphatemia & hypocalcemia leads to irritability, poor concentration, cognitive weakness | Seen in elderly age people with osteoporosis and neurodegeneration - dementia[18] |

### 5. Conium Maculatum

Senile dementia ailments of old maids and widows from ungratified sexua1 desire, alternate excitement and depression, excessive difficulty to recollect things, especially dates.

| Active principle ( Conium) | Pathopysiological Effects | Correlation with Dementia |
|---|---|---|
| Conhydrine & Pseudoconhydrine | Weaker alkaloids; affect motor control, cause giddiness, mental dullness | In Senile dementia patients often shows apathy, slowed cognition, vertigo-like symptoms [21] |
| Coniine (piperidine alkaloid) | Blocks nicotinic acetylcholine receptors at neuromuscular junctions results into neuromuscular weakness | Malfunction Cholinergic , loss of acetylcholine neurons in hippocampus & cortex-resembles dementia pathology[19] |
| ɣ- Coniceine (precursor alkaloid) | Neurotoxic, causes tremors, confusion, memory imparement in poisoning | Reflects cortical & Subcortical neuronal dysfunction seen in dementia[20] |



https://doi.org/10.46376/ijmr/07.03.2026:3328-3335

## 6. CROTALUS HORRIDUS:

Incipient stage of senile dementia, mental delusions such as mistakes in keeping accounts or writing letters, forgetfulness in figures, names, places, thinks he is the prey of enemies or of hideous animals, dislike to members of his own family.

| Active principle (Crotalus venom) | Pathopysiological Effects | Correlation with Dementia |
|---|---|---|
| Crotoxin (Phospholipase A2+ crotapotin complex) | Strong neurotoxin; inhibits synaptic transmission, causes neuronal apoptosis and oxidative stress | Neurodegeneration & synaptic failure is cause for Alzheimer's & other dmentia[22] |
| Phospholipase A2 (PLA2) | Induces lipid peroxidation, damages neuronal membranes which triggers inflammation in brain tissue | Resembeles to neuronal membrane breakdown & neuroinflammation in dementia[23] |
| Metalloproteinases ( Snake vemon proteases) | Degrade extracellular matrix -vascular fragility- microhemorrhages in brain | Cerebral microbleeds & vascular dementia are strongly correlated[24] |
| Disintegrins & hemorrhagins | Disrupt platelet aggregation & endothelial function - microthrombosis / ischemia | Vascular dementia pathology: ichemia, microinfarcts, impaired perfusion[25] |

## 7. Glononium

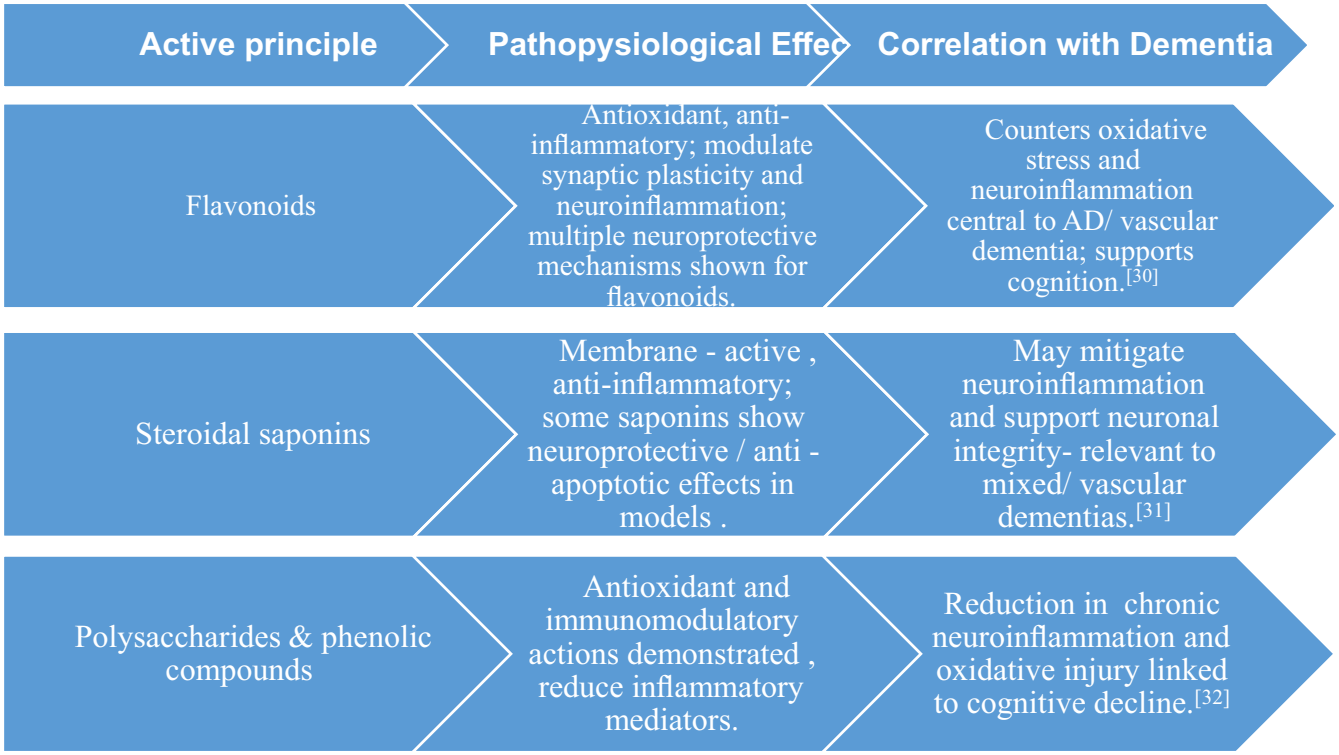
Acute dementia, religious mania, well known street seem strange, forgets where she lives, attempts to run away, fear of death, being poisoned, disinclined to speak, would hardly answer. Bad effects from mental excitement, fright, fear, mechanical contusion.

| Active principle | Pathopysiological Effec | Correlation with Dementia |
|---|---|---|
| Nitroglycerine (glyceryltrinitrate,GTN) | Acts as a prodrug of nitric oxide (NO)- vasodilation of cerebral vessels | Enhances cerebral blood flow - addresses the vascular insufficiency which is a major contributor to vascular dementia[26] |
| Nitric Oxide (NO) release | Modulates neurotransmission, synaptic plasticity, memory pathways | Impaired NO signaling is linked with Alzheimer's - related to memory loss; The action of gloninum stimulates correction of vascular hypoperfusion[27] |
| Cerebral vasodilation | Reduces ischemic hypoxia; prevents micro-infarcts | Chronic hypoperfusion is implicated in vascular and mixed dementia- Gloninum mechanism overlaps[28] |
| Homoeopathic pathogenesis | Sudden rush of blood to head, throbbing, confusion, forgetfulness and inability to recognize places | resembeles vascular dementia episodes[29] |



https://doi.org/10.46376/ijmr/07.03.2026:3328-3335

Uterine dementia, doubts her salvation, walks floor day and night aggravation by consolation, weeps much and is very timid, has to keep very busy to suppress sexual desire, curses and uses obscene language.




Financial support and sponsorship: Nil

Conflicts of interests: There is no conflict of interest

References:

1. McCarney, R., Warner, J., Fisher, P., & Van Haselen, R. (2003). Homeopathy for dementia. Cochrane Database of Systematic Reviews, 1, CD003803. https://doi.org/10.1002/14651858.CD003803
2. Shelokar, P. (n.d.). Dementia PRESENTATION. SlideShare. Retrieved August 29, 2025, from https://www.slideshare.net/slideshow/dementia-presentation-57420552/57420552
3. Farrington, E. A., & Bartlett, C. (2010). A Clinical Materia Medica (S. Lilienthal,Ed.). Kessinger Publishing.
4. Tabanca, N., Demirci, B., Blythe, E. K., Bernier, U. R., Ali, A., Wedge, D. E.,Khan, I. A., & Başer, K. H. C. Composition of Artemisia abrotanum and A. pontica Essential Oils and Their Repellent Activity against Aedes aegypti. Planta Medica, 2011;[cited 2025 Sep. 25]p.77(12). https://doi.org/10.1055/s-0031-1282383
5. Minocha, Tarun, Birla, Hareram, Obaid, Ahmad A., Rai, Vipin, Sushma, P., Shivamallu, Chandan, Moustafa, Mahmoud, Al- Shehri, Mohammed, Al-Emam, Ahmed, Tikhonova, Maria A., Yadav, SanjeevKumar, Poeggeler, Burkhard, Singh, Divakar, Singh, Sandeep Kumar, Flavonoids as Promising Neuroprotectants and Their Therapeutic Potential against Alzheimer's Disease, Oxidative Medicine and Cellular Longevity, 2022; [cited 2025 Sep. 25] 6038996, 13 pages. https://doi.org/10.1155/2022/6038996
6. Albaqami, Jawaher & Benny, Tancia & Hamdi, Hamida & Altemimi, Ammar & Kuttithodi, Aswathi & Job, Joice & Sasidharan, Anju & Narayanankutty, Arunaksharan. Phytochemical Composition and In Vitro Antioxidant, Anti-Inflammatory, Anticancer, and Enzyme-Inhibitory Activities of Artemisia nilagirica (C.B. Clarke) Pamp. Molecules.2022; [cited 2025 Sep. 26] 27. 7119. https://doi.org/10.3390/molecules27207119

7. Olsen RW, DeLorey TM. GABA Receptor Physiology and Pharmacology. In: Siegel GJ, Agranoff BW, Albers RW, et al., editors. Basic Neurochemistry: Molecular, Cellular and Medical Aspects. 6th edition. Philadelphia: Lippincott- Raven; 1999. [cited 2025 sep. 26] Available from: https://www.ncbi.nlm.nih.gov/books/NBK28090/
8. Michelot D, Melendez-Howell LM. Amanita muscaria: chemistry, biology, toxicology, and ethnomycology. Mycol Res. 2003 Feb;107(Pt 2):131-46.[cited 2025 sep. 26] doi: 10.1017/s0953756203007305. PMID: 12747324.
9. Davies JE. The pharmacological basis of therapeutics. Occup Environ Med. 2007 Aug;64(8):e2.[cited 2025 sep. 27] doi: 10.1136/oem.2007.033902. PMCID: PMC2078485.
10. Chen ZR, Huang JB, Yang SL, Hong FF. Role of Cholinergic Signaling in Alzheimer's Disease. Molecules. 2022 Mar 10;27(6):1816.[cited 2025 sep. 27] doi:10.3390/molecules27061816. PMID: 35335180; PMCID: PMC8949236.
11. Cui L, Miao J, Furuya T, Fan Q, Li X, Rathod PK, Su XZ, Cui L. Histoneacetyltransferase inhibitor anacardic acid causes changes in global gene expressionduring in vitro Plasmodium falciparum development. Eukaryot Cell. 2008 Jul;7(7):1200-10.[cited 2025 sep. 27] doi: 10.1128/EC.00063-08. Epub 2008 May 16.PMID: 18487348; PMCID: PMC2446667.
12. Trevisan, M.T.S. et al. (2006). Phenolic compounds in cashew nut shell liquid: toxicological aspects. Food Chem. Toxicol.
13. Clarke (1900). Dictionary of Practical Materia Medica; Boericke (1927).
14. Ogunlana, O.O. et al. (2008). Antioxidant and neuroprotective activity of cashew extracts. Phytotherapy Res.
15. Bezprozvanny IB. Calcium signaling and neurodegeneration. Acta Naturae. 2010Apr;2(1):p.72-82.[cited 2025 sep.28] PMID: 22649630; PMCID: PMC3347543.
16. Nicholls DG. Oxidative stress and energy crises in neuronal dysfunction. Ann N YAcad Sci. 2008 Dec;1147:p.53-60.[cited 2025 sep. 28] doi: 10.1196/annals.1427.002. PMID: 19076430.
17. Clarke (1900); Boericke (1927); Guyton & Hall (Textbook of Physiology).
18. Khosla, S. et al. (2008). Calcium and phosphate metabolism and the aging brain. Endocr Rev.
19. Kumar, A. et al. (2013). Pharmacology of Conium maculatum alkaloids. J Ethnopharmacol. Francis, P.T. et al. (1999). The cholinergic hypothesis of Alzheimer's disease. J Neurol Neurosurg Psychiatry.
20. Clarke (1900), Boericke (1927).
21. Bisset, N.G. (1994). Herbal Drugs and Phytopharmaceuticals.
22. Aird, S.D. (2002). Ophidian envenomation strategies and the role of neurotoxins. Toxicon. Huang WJ, Zhang X, Chen WW. Role of oxidative stress in Alzheimer's disease. Biomed Rep. 2016 May;4(5):519-522.[cited 2025 sep. 29] doi:10.3892/br.2016.630. Epub 2016 Mar 15. PMID: 27123241; PMCID: PMC4840676.
23. Burke JE, Dennis EA. Phospholipase A2 structure/function, mechanism, and signaling. J Lipid Res. 2009 Apr;50 Suppl(Suppl):S237-42.[cited 2025 sep. 29] doi:10.1194/jlr.R800033-JLR200. Epub 2008 Nov 14. PMID: 19011112; PMCID: PMC2674709.



https://doi.org/10.46376/ijmr/07.03.2026:3328-3335