International Journal of Pharmaceutical Science Invention
ISSN (Online): 2319-6718, ISSN (Print): 2319- 670X
www.ijpsi.org Volume 11 Issue 4 || Jul. –Aug. 2022 || PP. 24-30

A Micro Analysis of Alzheimer's disease by using bioinformatics tools

Rabindra Kumar Mishra[1]*, Arya Apriyam Kumar Swain[2], Ankita Behura[3], Ganugula Naveen Kumar[4]

[1]*Department of Basic Science and humanities, GIET University, Gunupur, Rayagada, Odisha, India, 765022*
[234]*Department of Biotechnology, GIET University,Gunupur, Rayagada,Odisha,India,765022*

Abstract:
This article gives an overview of neurodegenerative illnessesand a genetic defect that causes Alzheimer's disease. This article also provides the concept of various forms of amyloid deposition in the brain characterizes chronic, progressive, and sometimes familial central nervous system illnesses. In this study, we administered medications that just regulated Alzheimer's symptoms. There have been no medications discovered to successfully inhibit the growth of Alzheimer's disease. This review provides information on how genetic variables can cause or alter AD pathogenesis. Therefore, developing animal models to mimic late-onset AD pathogenesis could help researchers compare early and late-stage forms of the disease. This could offer new pharmacological targets for treatment as well as processes particular to late-onset Alzheimer's disease.
Keywords*: Alzheimer's disease, cognitiveimpairment, Allelic, Neurofibrillary, APPs*

Date of Submission: 14-07-2022 Date of acceptance: 29-07-2022

I. INTRODUCTION:

AD is a brain disease that slowly destroys reasoning abilities. The majority of people who have this condition have early problems that show in their mid-60s. This disease has a long and illustrious history. Alzheimer's is the name of the disease. He observed that one of the women had died of a strange mental ailment. Memory loss, linguistic difficulties, and erratic behavior are among her symptoms. He scanned her brain after she died and discovered numerous abnormalities, including amyloid plaques and tangled bundles of fibers known as neurofibrillary or tau. [3] The breakdown of connections between nerve cells in the brain is another hallmark. Many other brain changes are thought to have a role in Alzheimer's disease as well.

Clinical and pathological investigations imply that the disease has a long preclinical period, with the first signs of AD pathology appearing 10–15 years before symptomatic symptoms appear. The defining clinical profile of Alzheimer's disease is a persistent and cumulative decrease in the cognitive domain. Current diagnostic criteria can reliably diagnose Alzheimer's disease in the great majority of cases. ([3] As drugs are discovered, there is a growing interest in finding people who are in the early symptomatic and pre-symptomatic stages, because it is among this group that such treatments have the greatest chance of success. The most effective strategy to detect people in the very early stages of cognitive impairment is to employ informant-based approaches to determine a person's cognitive and performance level.

The abbreviation list is as follows:

Abbreviation	**Explanation**
FDA	Food Drug Administration
CNS	Central Nervous System
AD	Alzheimer's disease
LDL	Low-Density Lipoprotein
MW	Molecular Weight
PHF	Paired Helical Filament
APPS	Amyloid Precursor Protein

Symptoms:
Alzheimer's disease is characterized by cognitive impairment.

Table 1: The first symptoms are as follows

Depression, hallucinations, or paranoia
Irritability, anger, and aggression Repetition of one's own words is useless
Attitude morphs into Restlessness, roaming and becoming disoriented Anger, apathy, loneliness Losing objects
Forgetting Obsessive or inappropriate social behavior
Unable to recognize faces or objects
Difficulty handling money, hiding things, or pacing
Communication problems
dependent on others for his/her care
Unable to leave the bed most of the time,

Characteristics of Alzheimer's disease:

AD is a degenerative brain disease that damages memory cells, thinking abilities, and the ability to perform even basic tasks. The early stage of this disease begins with mild forgetfulness.Logical thinking will be destructed. A few individuals with this disease may possiblychange their capacity and stick to it.Alzheimer's disease patients typically have a difficult time performing daily tasks. Symptoms and behaviors change as the disease gets worse. They may begin to worry about the future and feel stress and anxiety. They will begin to need more help from family or co-workers.Cursing, kicking, striking, and biting are all examples of inappropriate behavior. Swearing, sobbing, shouting, loud demands for attention, self-talk, loneliness, depression, cold or heat, loud noises, and pain are all behaviors that occur in people with severe Alzheimer's People can contract various diseases and infections at this point.[4] Agitation, delirium, psychosis, restlessness, and depression are all possible symptoms.

The problem of the disease:

As in Alzheimer's disease, people experience many difficulties. The person may suffer from many problems [5]. These include-

- Wandering and getting lost.
- It takes a long time to conduct a routine daily task.
- Having problems managing your money and paying your bills.
- Always repeating the question.
- Changes in personality.
- Changes in behavior

The number of Americans living with Alzheimer's is growing. It is thefastest-growing disease. More than 6 million Americans have Alzheimer's. [6]

1. When it comes to mild, it includes-
- mood deviation
- Lingual problems

2. When it comes to moderate, it includes –
- dementia
- aggression
- learning problem
- Dependent on others for daily activities.

3. When it comes to severe, it includes –
- Bedridden
- Motor impairment

The Cause of Alzheimer's disease:

1. Alzheimer's is a reversible disease that affects the cells of the brain that cause the impairment of intellectual functioning. [7]

2. It is a disease that generally destroys the ability to remember, imagine, and learn.

3. The actual reason for this disease is not known, but several factors and aspects are implicated in it. [8]

ALZHEIMER DISEASE PROTEIN SEQUENCE:

MALRLVADFDLGKDVLPWLRAQRAVSEASGAGSGGADVLENDYESLHVLN
VERNGNIIYT YKDDKGNVVF GLYDCQTRQN ELLYTFEKDL QVFSCSVNSE
RTLLAASLVQ STKEGKRNEL QPGSKCLTLL VEIHPVNNVK VLKAVDSYIW
VQFLYPHIES HPLPENHLLL ISEEKYIEQF RIHVAQEDGN RVVIKNSGHL
PRDRIAEDFV WAQWDMSEQR LYYIDLKKSR SILKCIQFYA DESYNLMFEV
PLDISLSNSG FKLVNFGCDY HQYRDKFSKH LTLCVFTNHT GSLCVCYSPK
CASWGQITYS VFYIHKGHSK TFTTSLENVG SHMTKGITFL NLDYYVAVYL

PGHFFHLLNV QHPDLICHNL FLTGNNEMID MLPHCPLQSL SGSLVLDCCS
GKLYRALLSQ SSLLQLLQNT CLDCEKMAAL HCALYCGQGA QFLEAQIIQW
ISENVSACHS FDLIQEFIIA SSYWSVYSET SNMDKLLPHS SVLTWNTEIP
GITLVTEDIA LPLMKVLSFK GYWEKLNSNL EYVKYAKPHF HYNNSVVRRE
WHNLISEEKT GKRRSAAYVR NILDNAVKVI SNLEARNLGP RLTPLLQEED
SHQRLLMGLM VSELKDHFLR HLQGVEKKKI EQMVLDYISK LLDLICHIVE
TNWRKHNLHS WVLHFNSRGS AAEFAVFHIM TRILEATNSL FLPLPPGFHT
LHTILGVQCL PLHNLLHCID SGVLLLTETA VIRLMKDLDN TEKNEKLKFS
IIVRLPPLIG QKICRLWDHP MSSNIISRNH VTRLLQNYKK QPRNSMINKS
SFSVEFLPLN YFIEILTDIE SSNQALYPFE GHDNVDAEFV EEAALKHTAM
LLGL

Table 2: Function and Contributing Factors

intellectual functioning	**factor**
Neurochemical factors	Somatostatin, Acetylcholine, Substance p
Environmental factors	Cigarette smoking, metals, toxins, infections
Genetic and biological factors	Oxidized LDL receptor 1 and angiotensin 1 are bound and tied to the way the brain cells bind to the Apolipoprotein4
Risk factors	Injuries to the head, smoking, drinking, family history

Medical sign investigation of the disease:

Researchers are beginning to find out what causes the complex brain changes that lead to Alzheimer's disease's onset and development. Memory and other cognitive problems appear to begin a decade or more before brain damage occurs. (Table 3) During the preclinical phase of AD, people may have seemed to be side effects, but detrimental changes in the brain are occurring. AD causes damage to the nervous system, which manifests as initial clinical features. Different people's ADD manifests itself in different ways. [9]. There are also some people who have been diagnosed with minor cognitive impairment. People experience reduced memory loss and other cognitive impairments as the complaint progresses. [10]

Table 3. Psychological behaviour and functions:

Onset	In your immediate post-twenties,
Progression	7-13 years
Signs	Schizophrenia
Sporadic	significant majority
Transmissible	There is no evidence of this.
Familial	5-10%o
Chromosomal loci	21, 14, 19
Gene with mutation	APP
Cells that are sensitive	Cortical, hippocampal, cholinergic basal forebrain neuron
Disease of the Extracellular matrix System	Neurofibrillary tangles, neuropil threads
Amyloid	Aβ
Cell death	Significant
Models	Aged nonhuman primates; transgenic mice β

Mapping:

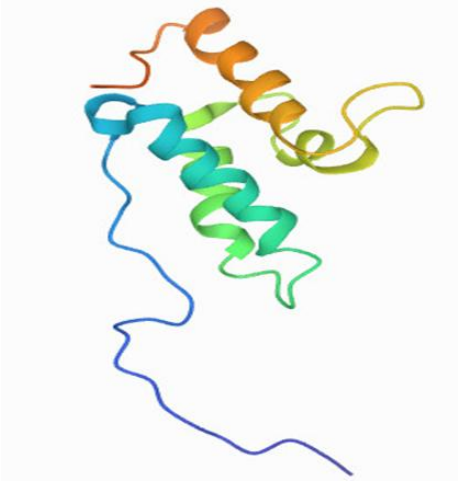
In research on the genetic code in Alzheimer's disease patients, D10S1423, situated at 10p13, was found as a possible susceptibility locus in a research study. [8] Allelic relationships were discovered in this study (Boston and Pittsburgh) [8–11]. In a community of 397 German AD issues, [8], [12], and [13], researchers found a connection between the D10S1423 234-bp gene codes and AD. Over 12 years of meticulous monitoring found that 325 undiagnosed 1st-degree family members of AD probands who had the D10S1423 234-bp mutation, the APOE E4,genetic code, or both underwent a prospective, continuous, dual evaluation of the time of hazard to one's lifewith AD [14]. According to the best-fitting model, only people who had both risk alleles had a risk percentage that deviated considerably from one. Females were also strongly connected with an increased chance of acquiring Alzheimer's disease after adjusting for these genes.

Molecular Genetic:

AD is the typical form of advanced illness, begins with a gradual loss of memory and progresses to higher cognitive processes and behavior (Table 1).Anomalies in brain cells in a number of neural circuits cause receptor impulses to destinations in the amygdala, hippocampus, and neocortex to be lost. (Table2) Neurofibrillary tangles form in the affected brain cell. [15, 16] PHFs are primarily made up of improperly phosphorylated isoforms, a microtubule-associated protein with a low molecular weight. Goedert et al. demonstrate that τ is phosphorylated during formation, that embryonic brain τ is a set of pre-defined relative to

the 6 adult τ isoforms, and that the 6 routinely expressed τ-isoforms are phosphorylated incorrectly in Alzheimer's patients' brains.Serine-202, [8, 17] which is usually phosphorylated in fetal τ is phosphorylated in the brains of Alzheimer's disease people, showing that the illness-related phosphorylation mimics a developmental pattern. The abnormal phosphorylation of τ is expected to affect intracellular transport, cellular geometry, and neuronal survival via altering microtubule-related processes.[18] 3-pleated amyloid deposits, notably in the hippocampus and neocortex, are a feature of AD.

Amyloid can be found surrounding blood vessels and is made up of a 4-kDa peptide amyloid generated by chromosome 21 genes that code for larger APPs. APPs can be endoproteolytically cleaved at position 16 inside Af3 by APP a-secretase to make the APP ectodomain; similarly, a few APPs can be endocytosed and destroyed via endosomal pathways. [19] Furthermore, soluble A 3 (4 kDa) is usually released by cultivated cells and is seen in the cerebral fluid. Investigations utilizing synthetic A and B showed that the 3-peptide generates fibrils and that disease-related mutations increase fibrillogenic potential. [19] Most neurons express APP transcripts and proteins; APPs are found in cell bodies, proximal dendrites, and axons and are transported to axons and terminals via the rapid anterograde axonal transport mechanism. [20] Although the actual activities of APPs in neurons are unknown at this time, they may play a role in synaptic connections. Furthermore, cholinergic agonist-activated muscarinic receptor subtypes Ml and M3 activation increases the production of APP derivatives that are accessible. According to Nitsch et al., the membrane potential of the electric field super fused hippocampus pieces enhances the discharge of neurochemical and soluble APP. Early-onset, autosomal dominant familial AD has been linked to at least ten pedigrees (at codons 717 (of APP-770) and 693 (which have been linked to early-onset, autosomal dominant familial AD in at least ten pedigrees). Significantly, cells transfected with cDNA expressing APP release higher quantities of AP-related peptides, despite the double amino acid change. Furthermore, new research has shown a key early-onset locus on chromosome 14 (14q24.3) and a susceptibility locus on chromosome 19 linked to late-onset illness. The latter is assumed to code for apolipoprotein E, the major cholesterol transporter. There is a link between the apolipoprotein E type e4 allele (the brain-enriched isoform) and the prevalence of AD in these late-onset relatives. The mechanisms that cause A and B to be deposited in vivo are still unknown.



In the Vivo study, low-density lipoprotein oxidation (Cu2+-mediated) or direct induction of neuronal death by copper-metallated APP is possible. Can control neurite microcapsules by attaching to extracellular matrix substances like heparin and collagen I and IV.Protease inhibitory activity is present in the BPTI-containing splice isoforms. Induces the internalization of amyloid-beta peptide and the onset of mitochondrial dysfunction in cultured cortical neurons through an AGER-dependent pathway that involves activation of p38 MAPK.Gives GPC1 the Cu2+ ions it needs to release nitric oxide (NO), which then causes the heparansulfate chains on GPC1 to break down.Lipophilic metal chelators with metal-reducing activity are amyloid-beta peptides. Bind metals that are brittle, like copper, zinc, and iron. Cu2+ and Fe3+ can be converted to Cu+ and Fe2+ in vitro, respectively.. In comparison to amyloid-beta protein 40, protein 42 is a more potent reductant. Inhibiting metal-catalyzed lipoprotein oxidation, amyloid-beta peptides bind to lipoproteins and apolipoproteins E and J in the CSF and to HDL particles in plasma.APP42-beta may trigger mononuclear phagocyte activation and inflammatory reactions in the brain. Encourages TPK II-mediated phosphorylation as well as tau aggregation. Oxidative stress and neurotoxicity are caused by interactions with overexpressed HADH2. in lipid rafts, which also bind GPC1.

Table 4: DRUG DATA AVAILABLE FOR ALZHEIMER DISEASE

DRUG NAME	GROUP	BRAND NAME	CHEMICAL FORMULA	DRUG BANK ACCESSION NUMBER	DRUG BANK LINK
Aluminumsulfate	Approved	Domeboro	$Al_2O_{12}S_3$	DB11239	https://go.drugbank.com/drugs/DB11239
Risperidone	Approved, Investigational	Perseris, Risperdal	$C_{23}H_{27}FN_4O_2$	DB00734	https://go.drugbank.com/drugs/DB00734
Dihydroergocristine	Approved, Experimental	--	$C_{35}H_{41}N_5O_5$	DB13345	https://go.drugbank.com/drugs/DB13345
Aducanumab	Approved, Investigational	Aduhelm	--	DB12274	https://go.drugbank.com/drugs/DB12274
Donanemab	Investigational	--	--	DB16647	https://go.drugbank.com/drugs/DB16647
Vanutidecridificar	Investigational	--	--	DB12132	https://go.drugbank.com/drugs/DB12132
Avagacestat	Investigational	--	$C_{20}H_{17}ClF_4N_4O_4S$	DB11893	https://go.drugbank.com/drugs/DB11893
GSI-136	Investigational	--	$C_{11}H_{18}ClNO_3S_2$	DB12819	https://go.drugbank.com/drugs/DB12819
Latrepirdine	Investigational	--	$C_{21}H_{25}N_3$	DB11725	https://go.drugbank.com/drugs/DB11725
Begacestat	Investigational	--	$C_9H_8ClF_6NO_3S_2$	DB12263	https://go.drugbank.com/drugs/DB12263
Aleplasinin	Investigational	--	$C_{28}H_{27}NO_3$	DB12635	https://go.drugbank.com/drugs/DB12635
Cerlapirdine	Investigational	--	$C_{22}H_{23}N_3O_3S$	DB12229	https://go.drugbank.com/drugs/DB12229
Talsaclidine	Investigational	--	$C_{10}H_{15}NO$	DB12287	https://go.drugbank.com/drugs/DB12287
Semagacestat	Investigational	--	$C_{19}H_{27}N_3O_4$	DB12463	https://go.drugbank.com/drugs/DB12463
Lecozotan	Investigational	--	$C_{28}H_{29}N_5O_3$	DB12540	https://go.drugbank.com/drugs/DB12540
Crenezumab	Investigational	--	--	DB11959	https://go.drugbank.com/drugs/DB11959

Clinical Features:

All the emotions, actions, ideas, and sentiments are the outcomes that travel via the brain's millions of nerves and synapses. In a person with Alzheimer's disease [21, 22], and 23], the chemical alterations in the brain completely destabilise the healthy equilibrium. This process includes the harmful proteins, β-amyloid and τ protein. The aberrant τ protein accumulates within the nerve knots. Theβ-amyloid binds together to form plaques, which gradually accumulate in the cells. Other changes in the brain also have a part in how we relate to age.

1. Insufficient blood and nutrients are delivered to the brain by the vascular system.
2. The lack of glucose in the brain that is needed to run the activity

These advances make the development and testing of promising new therapies easier, including:

1. Medicines (Table 4) that inhibit the accumulation of τ- protein andβ-amyloid proteins in the brain.
2. Changes in lifestyles
3. Therapies aimed at the cardiovascular system, inflammation, and glucose metabolism.

II. Discussion

AD is a degenerative brain disease that damages memory cells, thinking abilities, and the ability to perform even basic tasks. The early stage of this disease begins with mild forgetfulness.Logical thinking will be destructed.Agitation, delirium, psychosis, restlessness, and depression are all possible symptoms.During the preclinical phase of AD, people may have seemed to be side effects, but detrimental changes in the brain are occurring. AD causes damage to the nervous system, which manifests as initial clinical features. Different people's ADD manifests itself in different ways. [9]. There are also some people who have been diagnosed with minor cognitive impairment. People experience reduced memory loss and other cognitive impairments as the complaint progresses.In research on the genetic code in Alzheimer's disease patients, D10S1423, situated at 10p13, was found as a possible susceptibility locus in a research study.], researchers found a connection between the D10S1423 234-bp gene codes and AD. Over 12 years of meticulous monitoring found that 325

undiagnosed 1st-degree family members of AD probands who had the D10S1423 234-bp mutation, the APOE E4, genetic code, or both underwent a prospective, continuous, dual evaluation of the time of hazard to one's lifewith AD.. The abnormal phosphorylation of τ is expected to affect intracellular transport, cellular geometry, and neuronal survival via altering microtubule-related processes.[18] 3-pleated amyloid deposits, notably in the hippocampus and neocortex, are a feature of AD.Significantly, cells transfected with cDNA expressing APP release higher quantities of AP-related peptides, despite the double amino acid change. Furthermore, new research has shown a key early-onset locus on chromosome 14 (14q24.3) and a susceptibility locus on chromosome 19 linked to late-onset illness. The latter is assumed to code for apolipoprotein E, the major cholesterol transporter. There is a link between the apolipoprotein E type e4 allele (the brain-enriched isoform) and the prevalence of AD in these late-onset relatives. The mechanisms that cause A and B to be deposited in vivo are still unknown.. In a person with Alzheimer's disease [21, 22], and 23], the chemical alterations in the brain completely destabilize the healthy equilibrium. This process includes the harmful proteins, β -amyloid and τ protein. The aberrant τ protein accumulates within the nerve knots. Theβ -amyloid binds together to form plaques, which gradually accumulate in the cells.

Acknowledgements:

We would like to show our gratitude to the Dr. N.V.J Rao, Registrar, GIET University, Gunupur, Rayagada, Odisha 765022. For sharing their pearls of wisdom with us during the course of this research.

III. Conclusion:

The encephalopathy of AD is a kind of dementia. The fact that there are various forms of amyloid deposits in the brain characterischaracterizesnic, progressive, and sometimes familial central nervous system illnesses. This review looks at neurodegenerative illnesses and the pathogenic contributions of many components, as well as changes in the behaviorof cell types in the CNS. Alzheimer's disease is the most commonly occurring severe neurological condition in the elderly, as well as a primary factor of death anindisability. The onset of symptoms and phases of the disease should be identified since disease-modifying medicines may have the best likelihood of effectiveness in this demographic. Interactions with sources of information that place emphasis on demonstrating a worsening in a person's mental skills function out of the preceding established evaluation criteria can identify those with even the tiniest signs of memory loss.

References

[1]. Molecular and cellular mechanisms underlying the pathogenesis of Alzheimer's disease
[2]. TiantianGuo, Denghong Zhang, Yuzhe Zeng, Timothy Y. Huang, HuaxiXu & Yingjun Zhao . *Molecular Neurodegeneration* volume **15**, Article number: 40 (2020)
[3]. Alzheimer's A. 2016 Alzheimer's disease facts and figures. Alzheimer's Dement. 2016; 12(4):459–509.
[4]. C., P., World Alzheimer Report 2018. The state of the art of dementia research: new frontiers. London: Alzheimer's disease International; 2018.
[5]. Prince MJ, WA, Guerchet M, Ali G-C, and Wu Y-T, Prina M. World Alzheimer report 2015: the global impact of dementia: an analysis of prevalence, Incidence, Cost and Trends; 2015.
[6]. Tarawneh R, Holtzman DM. The clinical problem of symptomatic Alzheimer disease and mild cognitive impairment. Cold Spring HarbPerspect Med. 2012; 2(5):a006148.
[7]. Zubenko GS, et al. A collaborative study of the emergence and clinical features of the major depressive syndrome of Alzheimer's disease. Am J Psychiatry. 2003; 160(5):857–66.
[8]. Kalia M. Dysphagia and aspiration pneumonia in patients with Alzheimer's disease. Metabolism. 2003; 52(10 Suppl 2):36–8.
[9]. Lauter H. On the clinical study and psychopathology of Alzheimer's disease. Demonstration of 203 pathologically-anatomically verified cases. PsychiatrClin (Basel). 1968; 1(2):85–108.
[10]. Alzheimer disease and the prion disorders amyloid (3-protein and prion protein amyloidoses Donald L. Price*t*§, David R. Borchelt*§, and Sangram S. Sisodia*§ Deparrtmts of *Pathology, tNeurooy, and *Neuroscience and fNeuropathology Laboratory, The Johns Hopkins University School of Medicine, Baltimore, MD 21205-2196
[11]. Terry RD, et al. Physical basis of cognitive alterations in Alzheimer's disease: synapse loss is the major correlate of cognitive impairment. Ann Neurol. 1991; 30(4):572–80.
[12]. Iqbal K, Liu F, Gong CX. Tau and neurodegenerative disease: the story so far. Nat Rev Neurol. 2016; 12(1):15–27.
[13]. Petrella C, et al. Neuropeptides in Alzheimer's disease: an update. Curr Alzheimer Res. 2019;16(6):544–58
[14]. Katsumoto A, et al. Microglia in Alzheimer's disease: risk factors and inflammation. Front Neurol. 2018;9:978
[15]. Matsui T, et al. Expression of APP pathway mRNAs and proteins in Alzheimer's disease. Brain Res. 2007;1161:116–23
[16]. Zhang YW, et al. APP processing in Alzheimer's disease. Mol Brain. 2011; 4:3.
[17]. Bibl M, et al. CSF amyloid-beta-peptides in Alzheimer's disease, dementia with Lewy bodies and Parkinson's disease dementia. Brain. 2006;129(Pt 5):1177–87
[18]. Welge V, et al. Combined CSF tau, p-tau181 and amyloid-beta 38/40/42 for diagnosing Alzheimer's disease. J Neural Transm (Vienna). 2009;116(2):203–12
[19]. Tang W, et al. Assessment of CSF Abeta42 as an aid to discriminating Alzheimer's disease from other dementias and mild cognitive impairment: a meta-analysis of 50 studies. J Neurol Sci. 2014;345(1–2):26–36
[20]. Lanoiselee HM, et al. APP, PSEN1, and PSEN2 mutations in early-onset Alzheimer disease: A genetic screening study of familial and sporadic cases. PLoS Med. 2017; 14(3):e1002270.
[21]. Haass C, et al. The Swedish mutation causes early-onset Alzheimer's disease by beta-secretase cleavage within the secretory pathway. Nat Med. 1995;1(12):1291–6

[22]. Jonsson T, et al. A mutation in APP protects against Alzheimer's disease and age-related cognitive decline. Nature. 2012; 488(7409):96–9
[23]. Eggert S, et al. Trafficking in Alzheimer's disease: modulation of APP transport and processing by the transmembrane proteins LRP1, SorLA, SorCS1c, Sortilin, and Calsyntenin. MolNeurobiol. 2018; 55(7):5809–29.
[24]. Jacob CP, et al. Alterations in expression of glutamatergic transporters and receptors in sporadic Alzheimer's disease. J Alzheimers Dis. 2007; 11(1):97–116.
[25]. Hynd MR, Scott HL, Dodd PR. Glutamate(NMDA) receptor NR1 subunit mRNA expression in Alzheimer's disease. J Neurochem.2001; 78(1):175–82

Rabindra Kumar Mishra, et. al. "A Micro Analysis of Alzheimer's disease by using bioinformatics tools." *International Journal of Pharmaceutical Science Invention*, vol. 11(04), 2022, pp 24-30. Journal DOI- 10.35629/6718