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REVIEW ARTICLE

Effects and Mechanisms of Amino Acids in Alzheimer's Disease: a Narrative Review

Sapto Yuliani,[1] Rahmah Annura,[1] Lolita Lolita,[1] Dwi Utami,[1] Danang Prasetyaning Amukti[2]

1. Faculty of Pharmacy, Universitas Ahmad Dahlan, Yogyakarta, Indonesia.
2. Department of Pharmacy, Faculty of Health Sciences, Alma Ata University, Yogyakarta, Indonesia.

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Corresponding author:
SAPTO YULIANI
E: sapto.yuliani@pharm.uad.ac.id
T: +62 81228950580

Abstract

Alzheimer's disease is the most common form of dementia, a complex and progressive neurodegenerative disorder that slowly decreases memory, thinking skills and the ability to perform the simplest tasks. It is characterised by the formation of extracellular amyloid-beta (Aβ) plaques and intracellular neurofibrillary tangles (NFTs). In the human body, amino acids are a source of metabolites and neurotransmitters. Various studies have also proven their association with Alzheimer's disease. Therefore, the research was conducted to review the effects of amino acids and their mechanisms on Alzheimer's disease. This descriptive study is a narrative review of articles on *in vivo* and *in vitro* amino acid-induced Alzheimer's disease in the *PubMed* and *ScienceDirect* databases. The selected papers were in English, topic-relevant and published from 2011 to 2021 in journals of Q1, Q2 or Q3 category according to the *Scimago Journal & Country Rank*. The search yielded 27 relevant articles, but only 22 with 12 types of amino acids were included. Amino acids with positive effects were glutamine, d-serine, selenomethionine, s-adenosylmethionine, d-ribose-l-cysteine, s-allyl-cysteine, N-acetylcysteine, Se-methyl-selenocysteine and l-theanine, whereas some negative results come from homocysteine and N-methylamino-L-alanine. While taurine generally has a positive effect, there is a mechanism that negatively influences Alzheimer's disease. These amino acids are involved in all parts of the pathophysiology mechanism of Alzheimer's disease differently. The mechanisms include preventing (positive impact) or inducing (negative impact) mitochondrial dysfunction, inflammation, oxidative stress, formation of oligomers/plaque Aβ, tau hyperphosphorylation and neuronal/synaptic damage. Thus, not all amino acids have the activity of preventing/treating Alzheimer's disease.

Key words: Alzheimer's disease; Amino acids; Mechanism; Review, narrative.

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Introduction

Alzheimer's disease is a progressive, complex neurodegenerative disorder that slowly decreases memory, thinking abilities and the ability to complete the simplest daily tasks.[1] It begins with difficulty recalling recently learned information, which can then progress to more severe symptoms.[2] The molecular mechanisms of Alzheimer's disease are very complex, but the most dominant sign is the formation of extracellular amyloid plaques consisting of intracellular amyloid-beta (Aβ) and neurofibrillary tangles (NFTs).[3] Alzheimer's is the most common form of dementia, making up 60–70 % of the cases. Although there are no specific data on its global prevalence, it

is apparent that the incidence of dementia is increasing. An estimated 35.6 million people were living with dementia in 2010, which is expected to have increased rapidly to 115.4 or 152 million by 2050.[4, 5] In addition, some shortcomings of the pharmacological therapies generally used in patients with Alzheimer's persist. For instance, oral monotherapies with donepezil, rivastigmine and galantamine cause side effects like gastrointestinal disturbances. Accordingly, there is a dire need to discover alternative therapies or agents to prevent Alzheimer's disease, one of which is amino acids.[6]

Amino acids are organic substances that compose proteins. Amino acids also play an essential role in cellular metabolism, although at differing degrees depending on variations in their side chains.[7] Free amino acids determine neurotransmission and receptor signalling pathways and are involved in neurotoxicity, meaning that a change in their levels may be an early indicator of neurodegeneration.[8, 9] Glutamine supplementation in two mouse models of Alzheimer's can increase the levels of two synaptic proteins, ie vesicle-associated membrane protein 2 (VAMP2) and synaptophysin, while reducing inflammation-induced neuronal cell cycle activation, tau phosphorylation and ataxia-telangiectasia-mutated (ATM) activation.[10, 11] reported that I-serine supplementation can prevent behavioural and synaptic deficits in mice with Alzheimer's.[12] Also, a prospective study demonstrated that low levels of branched-chain amino acids/BCAAs like leucine, isoleucine and valine are associated with an increased risk of developing dementia, apart from other conventional risks.[13, 14] The mechanism involved is mammalian target of rapamycin (mTOR) activation, mTOR proves to have two contrasting effects on the pathophysiology of Alzheimer's, ie inducing a decline in Aβ pathology while increasing tau phosphorylation.[15, 16]

Based on the studies mentioned above, the findings are inconsistent in that some amino acids can prevent Alzheimer's disease while others act as stimulants. Therefore, to overcome the opposite roles, a narrative review of how and by which mechanisms amino acids affect Alzheimer's disease based on reports from several studies was provided.

Methods

This narrative review was prepared using an inductive approach, ie a literature study of articles in reputable journals, followed by a qualitative analysis to conclude the published findings. These data were selected based on, among others, their relevance to the set topic. The PICO framework was used to guide the article selection process, with P = patients with Alzheimer's disease, I = intervention with amino acids, C = not applicable (as no direct comparator was used) and O = the underlying pathophysiological mechanisms. This ensured that the selected articles were relevant to the research objective and that no similar narrative reviews were available. Furthermore, Boolean Operator with keywords adjusted to the journal databases, *PubMed* and *ScienceDirect*, were used for the literature search, as shown in Table 1.

The search results were checked for duplicates with the *EndNote* application and satisfaction of requirements, including the suitability of the title and abstract with the researched topic and other inclusion and exclusion criteria. The title or abstract should contain the words "Alzheimer's disease", "AD", or "Alzheimer's dementia" and "amino acid" or the name of the amino acid. To be included, the articles were written in En-

Table 1: Search keywords applied to the literature databases

Database	Key words
ScienceDirect	Alzheimer's disease AND amino acid AND (consume OR drug) AND mechanism
PubMed	(((((Alzheimer's disease[MeSH Terms]) OR (Alzheimer's disease[Title/Abstract])) AND ((amino acid) OR (amino acid[MeSH Terms]))) AND ((consume) OR (drug))) AND (mechanism)) NOT ((review) OR (review[Publication Type]))

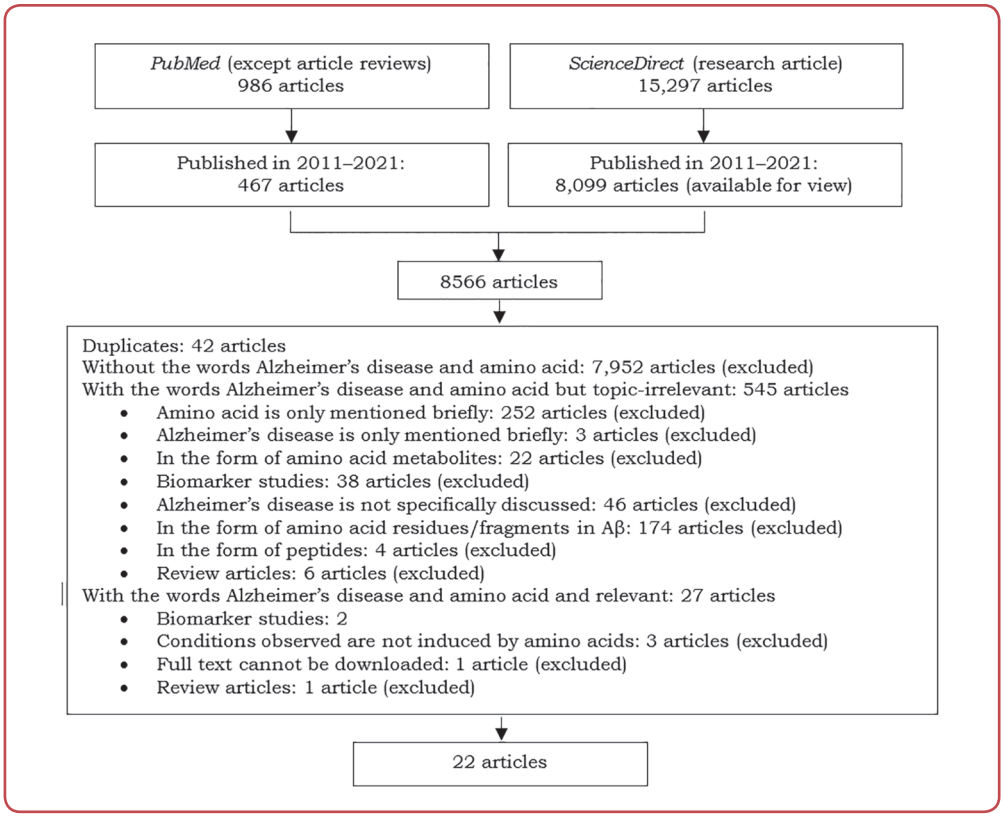

Figure 1: Literature search chart

glish and published between 2011 and 2021. On the contrary, the exclusion criteria were as follows: the observed objects or neurodegenerative disorders were not given or induced by amino acids, amino acid metabolites or peptides, amino acid residues, or amino acid fragments in Aβ; the studies did not specifically discuss Alzheimer's or off-topic; biomarkers studies; review articles; non-downloadable full text. The article selection also factored in the journal's quality, ie Q1, Q2, or Q3 category based on the *Scimago Journal & Country Rank* (Figure 1).

Results

In the initial search, the PICO questions (PICO: P: population, I: intervention; C: comparison; O: outcome) were applied to the *PubMed* database and the results included no narrative reviews that summarised the mechanisms of amino acids in Alzheimer's disease. The subsequent search in two databases returned a total of 16,281 articles, consisting of 986 from *PubMed* and 15,297 from *ScienceDirect*. Based on the year and type of publication, 467 articles in *PubMed* and 8,967 in *ScienceDirect* were published in 2011 to 2021 and were research (not review) articles. However, only 8,099 of the 8,697 articles in *ScienceDirect* were available for viewing because only a maximum of 1,000 articles could be displayed in one year. Therefore, 8,566 articles were collected at this stage.

After checking for duplicate data, 42 articles were omitted. Then, 7,952 articles were excluded because their title or abstract did not contain the words "Alzheimer's disease", "AD", or "Alzheimer's dementia" and "amino acids" or the name of amino acids. In addition, 545 articles containing both terms were sorted out because they were biomarker studies, review articles, or articles that were off-topic as they only briefly mentioned amino acids or Alzheimer's or discussed amino acids in the form of metabolites, residues/fragments in Aβ, or peptides. The process obtained 27 articles that contained the two terms and matched the research topic. However, five of them met the exclusion criteria, namely, biomarker studies (2 articles), no disease induction by amino acids (1), non-downloadable full text (1) and review articles (1). In the end, 22 articles were obtained. Afterward, the quality of the journal was checked and it was found that each article was published in at least Q3 journals based on the *Scimago Journal & Country Rank*.

Table 2: Effects of amino acids on Alzheimer's disease

No	PMID	Authors (year)	Amino acids	Research methods	Effects
1	31119171	Wang et al (2019)	Glutamine	*in vivo/in vitro*	Prevention
2	32781134	Liu et al (2020)	D-serine	*in vivo*	Prevention
3	29974078	Zhang et al (2018)	Seleno-methionine	*in vivo*	Prevention
4	30527255	Park et al (2018)	Luteolin, l-theanine	*in vivo*	Prevention
5	28137967	Zhang et al (2017)	Seleno-methionine	*in vivo*	Prevention
6	22221883	Fuso et al (2012)	SAM	*in vivo*	Prevention
7	25502280	Kim et al (2015)	Taurine	*in vivo*	Prevention
8	28109879	Zheng et al (2017)	Seleno-methionine	*in vivo/in vitro*	Prevention
9	28453493	Li et al (2017)	SAM	*in vivo/in vitro*	Prevention
10	33217524	Ogunlade et al (2021)	D-ribose-l-cysteine	*in vivo*	Prevention
11	32130882	Le Douce et al (2020)	L-serine/d-serine	*in vivo*	Prevention
12	29944861	Zhu et al (2018)	L-theanine	*in vivo*	Prevention
13	28284975	Reeta et al (2017)	Taurine	*in vivo*	Prevention
14	21376020	Javed et al (2011)	S-allyl cysteine	*in vivo*	Prevention
15	28411131	Shahidi et al (2017)	NAC	*in vivo*	Prevention
16	33689275	Du et al (2021)	Se-methyl-selenocysteine	*in vivo*	Prevention
17	30003389	Kovalska et al (2018)	Homocysteine	*in vivo*	Induction
18	27896923	Li et al (2017)	Homocysteine	*in vivo/in vitro*	Induction
19	22001762	Fujiki et al (2012)	Homocysteine	*in vitro*	Induction
20	23139767	Wang et al (2012)	Homocysteine	*in vitro*	Induction
21	22841708	Goto et al (2012)	BMAA	*in vivo*	Induction
22	33153477	Silva et al (2020)	BMAA	*in vitro*	Induction

BMAA: β-N-methylamino-L-alanine (BMAA); SAM: S-adenosylmethionine;

The twenty-two articles obtained from the literature search were reviewed. From them, 12 amino acids were studied for their effects and mechanisms in Alzheimer's disease. Sixteen articles described a positive result, ie amino acids can inhibit Alzheimer's disease, whereas six others indicated a negative effect, ie amino acids can trigger Alzheimer's disease. The effects of amino acids on Alzheimer's disease are described in Table 2.

The articles from the search results showed experiments on various amino acids. Although several articles discussed the same amino acid, they studied different mechanisms and could complement each other. In further detail, the mechanisms of amino acids in Alzheimer's were divided into three major categories. These mechanisms are summarised in Tables 3, 4 and 5.

Table 3: Mechanisms of amino acids related to Aβ plaques

No	Authors (year)	Designs		Mechanisms						
		In vivo	*In vitro*	Amino acids	Glucose/energy metabolism	GPx and TrxR activity	Anti-oxidants	Oxidative stress	Aβ metabolism/ expression	Others
1	Wang et al (2019)	y	y	Glutamine	–	–	↑	↓	-	–
2	Liu et al (2020)	y	-	D-serine	↑	–	–	–	↓	–
3	Zhang et al (2018)	y	-	Seleno-methionine	–	↑	↑	–	↓	–
4	Park et al (2018)	y	y	Luteolin, l-theanine	–	–	–	–	–	–
5	Zhang et al (2017)	y	y	Seleno-methionine	–	–	–	–	–	Autophagy, cathepsin D levels & p62 degradation ↑
6	Fuso et al (2012)	y	-	SAM	–	–	–	–	–	PSEN1 and BACE1 expression ↓, global RNA modulation is restored

7	Kim et al (2015)	y	y	Taurine	–	–	–	–	↓		–
8	Zheng et al (2017)	y	y	Seleno-methionine	–	–	–	–	–		–
9	Li et al (2017)	y	y	SAM	–	–	↑	↓	–		–
10	Ogunlade et al (2021)	y	-	D-ribose-l-cysteine	–	–	↑	↓	↓		–
11	Le Douce et al (2020)	y	-	L-serine/d-serine	–	–	–	–	–		–
12	Zhu et al (2018)	y	y	L-theanine	–	–	–	–	–		–
13	Reeta et al (2017)	y	-	Taurine	–	–	↑	↓	–	ROCK-II expression ↑	
14	Javed et al (2011)	y	-	S-allyl-cysteine	–	–	↑	↓	–		–
15	Shahidi et al (2017)	y	-	NAC	–	–	–	–	–		–
16	Du et al (2021)	y	-	Se-methyl selenocysteine	↑	–	↑	–	–	Cortical ATP ↑	
17	Kovalska et al (2018)	y	-	Homocysteine	–	–	–	↑	↑		–
18	Li et al (2017)	-	y	Homocysteine	–	–	–	–	↑	5-LO levels ↑	
19	Fujiki et al (2012)	-	y	Homocysteine	–	–	–	↑	–	mitochondrial membrane potential is eliminated	
20	Wang et al (2012)	-	y	Homocysteine	–	–	–	–	–		–
21	Goto et al (2012)	y	-	BMAA	–	–	–	–	–		–
22	Silva et al (2020)	-	y	BMAA	↓	–	–	–	↑	Metabolism is disrupted and mitochondrial fragmentation is induced	

BMAA: β-N-methylamino-L-alanine (BMAA); GPx: glutathione peroxidase; TrxR: Thioredoxin reductase; BACE1: Beta-site amiloride precursor protein cleaving enzyme 1; PSEN1: γ-secretase Presenilin 1; ROCK-II: Rho kinase-II;

Table 4: Mechanisms of amino acids related to tau proteins

No	Authors (year)	Designs		Mechanisms								
		In vivo	*In vitro*	Amino acids	Anti-oxidants	GPx and TrxR activity	Oxidative stress	Positive GFAP expression	Inflammation marker	Tau protein expression	GSK3 and Akt	Others
1	Wang et al (2019)	y	y	Glutamine	↑	↓	–	–	–	–	–	–
2	Liu et al (2020)	y	-	D-serine	–	–	–	↓	↓	–	–	NMDAR1 ↓
3	Zhang et al (2018)	y	-	Seleno-methionine	↑	↑	–	–	–	↓	–	–
4	Park et al (2018)	y	-	Luteolin, l-theanine	–	–	–	–	↓	↓	↓	FOXO-1 phosphorylation and CREB ↑
5	Zhang et al (2017)	y	y	Seleno-methionine	–	–	–	–	–	↓	↓	Autophagy, cathepsin D Levels and p62 degradation ↑
6	Fuso et al (2012)	y	-	SAM	–	–	–	–	–	↓	–	–
7	Kim et al (2015)	y	-	Taurine	–	–	–	↑	–	–	–	–
8	Zheng et al (2017)	y	y	Seleno-methionine	–	–	–	–	–	–	↓	–
9	Li et al (2017)	y	y	SAM	↑	–	↓	↓	↓	–	–	–
10	Ogunlade et al (2021)	y	-	D-ribose-l-cysteine	↑	–	↓	–	–	–	–	–

11	Le Douce et al (2020)	y	-	L-serine/d-serine	–	–	–	–	–	–	–	–
12	Zhu et al (2018)	y	y	L-theanine	–	–	–	–	–	–	–	–
13	Reeta et al (2017)	y	-	Taurine	↑	–	↓	–	↓	–	–	ChAT expression ↑
14	Javed et al (2011)	y	-	S-allyl-cysteine	↑	–	↓	–	–	–	–	–
15	Shahidi et al (2017)	y	-	NAC	–	–	–	–	–	–	–	–
16	Du et al (2021)	y	-	Se-methyl selenocysteine	↑	–	–	–	–	–	–	–
17	Kovalska et al (2018)	y	-	Homocysteine	–	–	↑	↑	–	↑	–	–
18	Li et al (2017)	y	y	Homocysteine	–	–	–	–	–	–	–	–
19	Fujiki et al (2012)	-	y	Homocysteine	–	–	↑	–	–	–	–	–
20	Wang et al (2012)	-	y	Homocysteine	–	–	–	–	–	–	–	–
21	Goto et al (2012)	y	-	BMAA	–	–	–	–	–	–	–	–
22	Silva et al (2020)	-	y	BMAA	–	–	–	–	–	–	–	Cytosolic IκBα levels ↓, p62 flux is disturbed

GSK3: Glycogen synthase kinase 3; GPx: glutathione peroxidase; GFAP: Glial fibrillary acidic protein; BMAA: β-N-methylamino-L-alanine; Akt: Protein kinase B; TrxR: Thioredoxin reductase; FOXO-1: Forkhead box protein O1; ChAT: choline acetyltransferase; IκBα: Nuclear factor kappa B inhibitor alpha;

Table 5: Mechanisms of amino acids related to synapses

No	Authors (year)	Designs		Mechanisms						
		In vivo	*In vitro*	Amino acids	Wnt3a/ β-catenin signalling	Apoptosis	Learning abilities and memory	Motor abilities	Cholinesterase activity	Others
1	Wang et al (2019)	y	y	Glutamine	↑	↓	↑	–	–	–
2	Liu et al (2020)	y	-	D-serine	–	–	↑	↑	–	p-JNK, p-c-Jun and ATF2 expressions ↓, NMDAR1 ↓
3	Zhang et al (2018)	y	-	Seleno-methionine	–	–	↑	–	–	–
4	Park et al (2018)	y	-	Luteolin, l-theanine	–	–	↑	–	–	–
5	Zhang et al (2017)	y	y	Seleno-methionine	–	–	↑	–	–	–
6	Fuso et al (2012)	y	-	SAM	–	–	↑	–	–	–
7	Kim et al (2015)	y	-	Taurine	–	–	↑	–	–	–
8	Zheng et al (2017)	y	y	Seleno-methionine	↑	–	–	–	–	Neural stem cell differentiation and proliferation ↑
9	Li et al (2017)	y	y	SAM	–	↓	–	–	–	–
10	Ogunlade et al (2021)	y	-	D-ribose-l-cysteine	–	–	↑	–	↓	Improved hippocampal histo-morphology, neurotransmitter levels ↑
11	Le Douce et al (2020)	y	-	L-serine/d-serine	–	–	↑	–	–	Hippocampal LTP and LTD are restored, fEPSP NMDAR ↑
12	Zhu et al (2018)	y	y	L-theanine	–	–	↑	–	–	Hippocampal LTP ↑, dopamine/ D1 receptor or 5-PKA pathway receptor is activated

13	Reeta et al (2017)	y	-	Taurine	–	–	↑	–	↓	–
14	Javed et al (2011)	y	-	S-allyl-cysteine	–	↓	↑	–	–	Improved hippocampal histomorphology, DNA fragmentation↓, bcl2↑
15	Shahidi et al (2017)	y	-	NAC	–	–	↑	–	–	PS and EPSP ↑
16	Du et al (2021)	y	-	Se-methyl selenocysteine	–	–	↑	–	–	Proteins related to signalling pathways ↑
17	Kovalska et al (2018)	y	-	Homocysteine	–	↑	–	–	–	Neuronal damage ↑
18	Li et al (2017)	y	y	Homocysteine	–	–	–	–	–	–
19	Fujiki et al (2012)	-	y	Homocysteine	–	↑	–	–	–	–
20	Wang et al (2012)	-	y	Homocysteine	–	↑	–	–	–	Expression of 14-3-3e mRNA↓, calcineurin ↑
21	Goto et al (2012)	y	-	BMAA	–	–	–	↓	–	Abnormal NMDA responses
22	Silva et al (2020)	-	y	BMAA	–	–	–	–	–	–

BMAA: β-N-methylamino-L-alanine (BMAA); SAM: S-adenosylmethionine; NAC: N-acetylcysteine; NMDA: N-methyl-d-aspartate; Wnt: Wingless/Int-1; JNK: c-Jun N-terminal kinase; ATF: Activating transcription factor 2; LTP: Long-term potentiation; LTD: Long-term depression; fEPSP: field excitatory postsynaptic potential; PKA: Protein kinase A;

Discussion

Amino acids that prevent Alzheimer's disease

Glutamine is the most abundant free amino acid in the human body and plays a critical role in central nervous system function, particularly in neurotransmitter synthesis and cellular energy metabolism.[17] In cognitive studies, glutamine supplementation has been shown to reduce error time and enhance reaction time in memory tasks using a passive avoidance apparatus, suggesting a positive effect on learning and memory processes. Beyond its cognitive effects, glutamine also exhibits neuroprotective properties.[18] It has been reported to reduce oxidative stress, limit neuronal damage and enhance antioxidant defence mechanisms. One important pathway involved in these effects is the Wingless/Int-1 (Wnt)3a/β-catenin signalling pathway, which glutamine activates.[19] This pathway plays a vital role in supporting neuronal survival, regulating synaptic plasticity and preserving the integrity of the blood-brain barrier. Since these functions are often impaired in neurodegenerative disorders, especially Alzheimer's disease, glutamine's ability to modulate these pathways suggests its potential as a protective agent against cognitive decline.[20, 21]

Serine is a non-essential amino acid whose dextro isomer, d-serine, is an endogenous ligand for the glycine site in the N-methyl-d-aspartate (NMDA) receptor. According to d-serine can improve cognitive and motor skills in Kunming mice compared to those given only an $A\beta_{1-42}$ injection.[22] Furthermore, in mice, 100 mg/kg BW d-serine can ameliorate cognitive deficits in the Morris Water Maze (MWM) test, although no effect was observed in the control group. Additionally, the research revealed that D-serine enhances glucose metabolism, reduces Aβ deposition and inflammation and decreases the number of glial fibrillary acidic protein (GFAP)-positive cells. In addition, it affects the c-Jun N-terminal kinase (JNK) signalling pathway, whose activation is associated with apoptosis. Furthermore, a 10 % l-serine enriched diet for two months proves effective in treating d-serine deficiency in triple-transgenic mouse model of Alzheimer's disease (3×Tg-AD) mice, thus restoring the binding of the NMDA receptor (NMDAR) co-agonist by increasing the NMDAR field excitatory postsynaptic potential (fEPSP).[23]

Selenomethionine is a selenium (Se) analogue of methionine in which the sulphur is replaced by selenium.[24] According to, selenomethionine can improve the memory of mice in the MWM test.

It also contributes to reducing total and hyperphosphorylated tau by inhibiting glycogen synthase kinase 3 (GSK3) and protein kinase B (Akt). Another mechanism is autophagy by reducing mTOR levels while increasing adenosine monophosphate-activated protein kinase (AMPK) and by stimulating more clearance of abnormal protein aggregates in cells. also confirmed the reduction in tau and Aβ, although it was more significant after treatment with selenium-yeast instead of selenomethionine. Furthermore, the same research demonstrated that selenomethionine effectively reduces oxidative stress while increasing antioxidants.[25]

Furthermore, in research, 10 M selenomethionine increased the proliferation of neural stem cells. Still, when administered at 50 M and 100 M, the amino acid decreased cell proliferation. In addition, astrocytes and GFAP were lower after the administration of selenomethionine than in the control. A different study found that the amino acid potentiates β-catenin expression and p-PI3K/PI3K ratio and attenuates GSK3β, all of which support neurogenesis and cell proliferation.[26]

The main methyl donor in the brain, S-adenosylmethionine (SAM), is used as an antidepressant in some countries. S-adenosyl-l-methionine is a sulfonium compound that is the S-adenosyl derivative of L-methionine.[27, 28] According to, improved spatial memory in TgCRND8 mice can be associated with SAM. Also, SAM 400 µg can inhibit the excessive production of Aβ and reduce plaque formation by decreasing the expression of beta-site amiloride precursor protein cleaving enzyme 1 (BACE1) and γ-secretase presenilin 1 (PSEN1), although not significantly different from the effect of SAM 800 µg. SAM can also restore the phosphorylation of tau as much as the control group, although no effect was observed on total tau expression.[29, 30] Furthermore, a study showed that treatment with SAM can also reduce oxidative stress while increasing antioxidants. Reduction in the levels of astrocytes and microglia, as well as inflammation, is also associated with the same treatment. Based on the two studies, S-adenosylmethionine can prevent Alzheimer's disease.[29]

D-ribose-l-cysteine is an analogue of cysteine that has been shown to boost cellular antioxidant capacity by increasing intracellular glutathione (GSH) biosynthesis.[31] According to, it can improve memory and learning. D-ribose-l-cysteine can also increase antioxidants and simultaneously reduce oxidative stress. Dopamine and serotonin levels are elevated, while norepinephrine and acetylcholinesterase (AChE) are reduced in concentration, thus inhibiting synaptic damage. Other reported results are improved hippocampal histomorphology and the absence of Aβ deposits. However, under normal conditions (without cuprizone), dietary supplementation of d-ribose-l-cysteine degenerates the hippocampus. For these reasons, d-ribose-l-cysteine can prevent Alzheimer's disease.[32]

S-allyl-cysteine is an s-hydrocarbyl-l-cysteine that is l-cysteine in which the hydrogen attached to the sulphur is replaced by a prop-2-enyl group. stated that s-allyl-cysteine prevents streptozotocin-induced memory impairment in mice.[33] Also, it inhibits an increase in oxidative stress and, at the same time, a decrease in antioxidants in the hippocampus of the mice. Its apoptotic and neuroprotective effects are attributed to two mechanisms: preventing p53 expression from increasing and Bcl2 expression from decreasing. This amino acid also helps prevent DNA damage a cell death. Based on these studies, s-allyl-cysteine can prevent Alzheimer's disease.[34]

The fourth cysteine derivative observed, N-acetylcysteine (NAC), is the acetate type of the amino acid L-cysteine and is widely used as a specific antidote for acetaminophen overdose.[35] Proved that NAC enhances the memory and learning of Wistar rats with Aβ-induced Alzheimer's disease. NAC can also protect and restore synaptic activity in the brain. In addition, the study found that administering NAC 14 days after Aβ injection for two weeks resulted in a more significant effect than only one day after injection. Therefore, N-acetylcysteine can prevent Alzheimer's disease.[36]

The fifth cysteine derivative, se-methyl-selenocysteine, is a non-proteinogenic L-alpha-amino acid compound with methyl-selanyl-methyl as the side chain and is a derivative of L-selenocysteine. As discussed, se-methyl-selenocysteine improves the learning and memory of Alzheimer's disease mice in the MWM test, overcoming the related dysthymic disorder.[37] In addition, selenium-methyl-selenocysteine has been shown to enhance antioxidants and metabolism-related proteins, preventing metabolic disorders and Alzheimer's disease.[38, 39] L-theanine (γ-glutamyl-ethyl amide) is a unique natural non-protein amino acid found in green tea (*Camellia sinensis*).

Confirmed that L-theanine can improve memory in rats showing similar symptoms to Alzheimer's after induction by $A\beta_{25-35}$. L-theanine also improves memory and hippocampal long-term potentiation (LTP) in mice when administered at 0.4 mg/mL, but according to, it does not apply to wild-type mice.[40] L-theanine also efficiently reduces tau phosphorylation by inhibiting Akt and GSK3β. This effect is more significant when combined with luteolin. Furthermore, l-theanine can reduce inflammation even more significantly than the l-theanine-luteolin combination. also stated that L-theanine can increase dopamine, noradrenaline and protein kinase A (PKA) phosphorylation, thus facilitating hippocampal synaptic transmission. Based on these two studies, L-theanine has a positive effect and can be used to treat Alzheimer's disease.[40]

Taurine is an amino sulfonic acid that is a 2-amino derivative of ethanesulphonic acid. It is a naturally occurring amino acid produced in the metabolism of methionine and cysteine.[41] Taurine can improve behavioural performance and spatial memory of Alzheimer's mouse models and restore streptozotocin-induced memory impairments.[42] Increased antioxidants and lowered oxidative stress have also been observed after the administration of taurine. Also, proven to attenuate AChE and butyrylcholinesterase (BuChE) activities. In addition, taurine increases choline acetyltransferase (ChAT), which can catalyse the biosynthesis of acetylcholine, a neurotransmitter, thus maintaining synaptic neurons.[43] It also inhibits Rho kinase-II (ROCK-II), leading to Aβ reduction also stated that this amino acid is also responsible for reducing the insoluble fraction $A\beta_{42}$ in the mouse brain cortex, although there is no significant change in the soluble fraction.[44] However, the administration of taurine has a harmful effect, ie it induces an increase in reactive astrocytes that leads to inflammation, as evident in the elevated expression of GFAP in both amyloid precursor protein (APP)/PS1 and wild-type mice.[45] In contrast, the research findings suggest that taurine reduces inflammation by decreasing IL-β and tumour necrosis factor alpha (TNF-α). For these reasons, it is necessary to further research the effects of taurine on astrocytes and microglia and inflammation.[46] The amino acids mentioned above have different and complex mechanisms by which the pathophysiology of Alzheimer's disease can be prevented. The mechanisms in question are summarised in Figure 2.

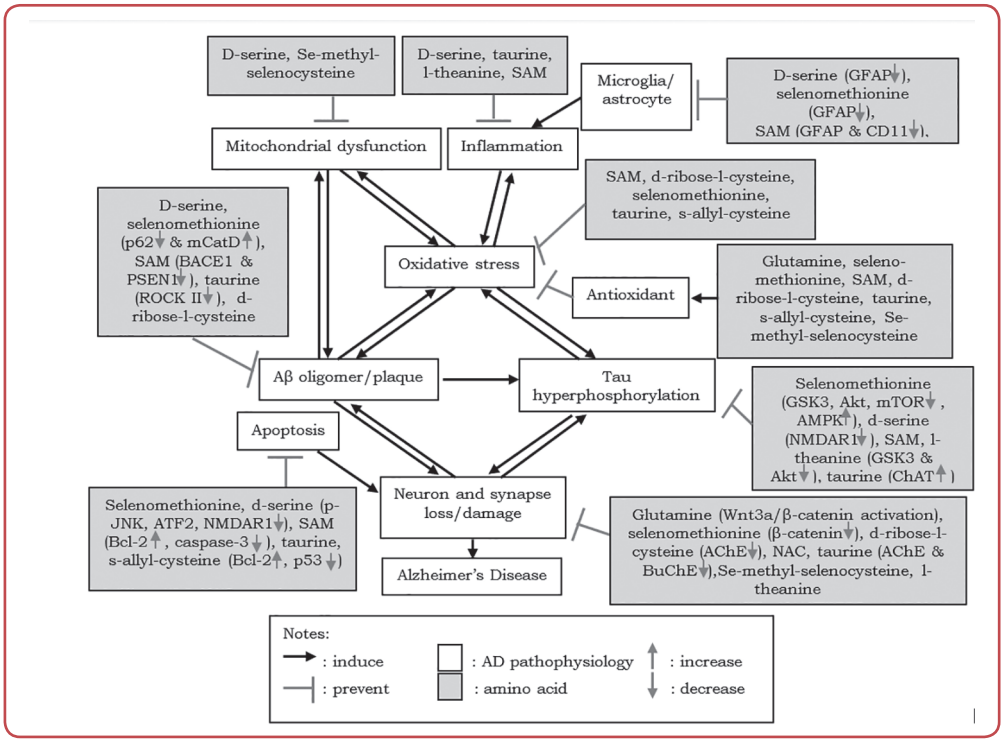

Figure 2: Schematic of the mechanisms of amino acids that prevent Alzheimer's disease

SAM: S-adenosylmethionine; GFAP: Glial fibrillary acidic protein; AChE: acetylcholinesterase; BuChE: butyrylcholinesterase;

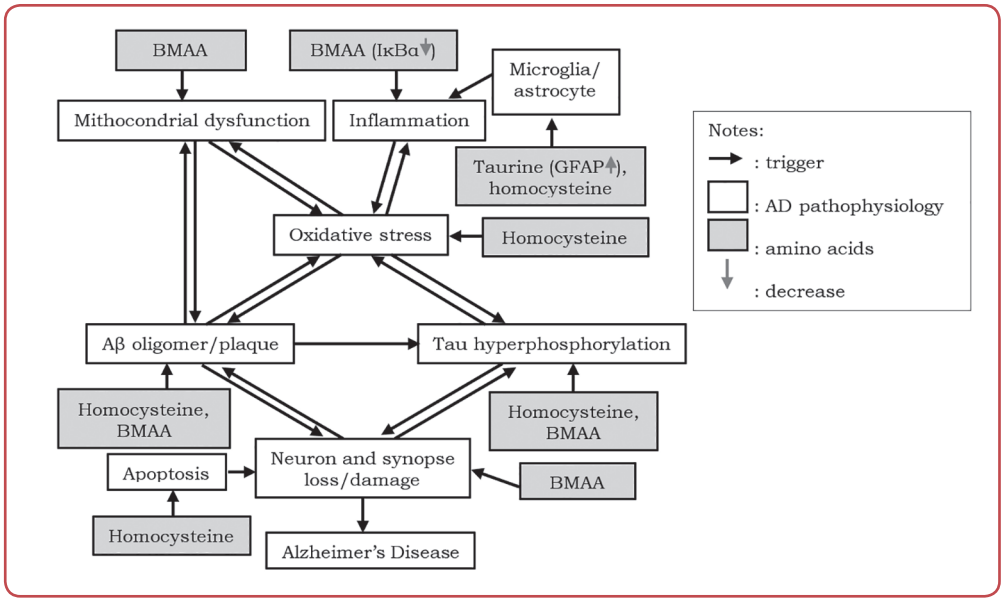

Figure 3: Schematic of the mechanisms of amino acids that trigger Alzheimer's disease
BMAA: β-N-methylamino-L-alanine (BMAA);

Amino acids that induce Alzheimer's disease

A sulfhydryl-containing amino acid, homocysteine is an intermediate product in the normal biosynthesis of two amino acids: methionine and cysteine.[47] Administrating a high homocysteine concentration (hyperhomocysteine) can significantly potentiate lipid peroxidase formation and oxidative stress. In addition, explained that hyperhomocysteine lowers the number of neurons and, at the same time, increases astrocyte reactivity.[48] The number of Aβ plaques and tau proteins also increases after hyperhomocysteine administration, compared with the control and ischaemia-reperfusion groups. Increased $A\beta_{1-40}$ and $A\beta_{1-42}$ levels due to homocysteine are also demonstrated in. In another study, homocysteine effectively reduces cell viability, increases cell death and eliminates mitochondrial membrane potential.[49] Similarly confirmed that increased calcineurin activity contributes to the dephosphorylation and translocation of Bad, thus inducing apoptosis. Overall, based on these studies, homocysteine tends to trigger Alzheimer's disease.[50]

L-β-N-methylamino-L-alanine is a non-proteinogenic l-alpha-amino acid, ie l-alanine in which one of the methyl hydrogens is replaced by a methylamino group.[51] This amino acid is produced by cyanobacteria. The literature search return included two studies discussing β-N-methylamino-L-alanine (BMAA). According to BMAA destabilises the postsynaptic response of tsetse flies (*Drosophila melanogaster*).[52] During the experiment, these flies could not consistently and smoothly land on the side of the cylinder compared with the control and their NMDA response became abnormal with slower rise time, reduced amplitude and longer duration, BMAA has been shown to cause adverse effects. BMAA interferes with mitochondrial metabolism and induces mitochondrial fragmentation.[52] In addition, exposure to BMAA results in decreased inhibition of nuclear factor kappa B (NF-κB), thus increasing astroglia and inflammation. Moreover, it also potentiates intracellular Aβ oligomers and tau phosphorylation.[53] Based on these two studies, BMAA can trigger the onset of Alzheimer's disease. The above amino acids promote the pathophysiology of Alzheimer's disease in complex and different mechanisms. The mechanisms in question are summarised in Figure 3.

Conclusion

Based on the article search results and discussion, various amino acids exhibit distinct influences on the pathogenesis of Alzheimer's disease through diverse molecular mechanisms. Amino acids with neuroprotective properties such as glutamine, D-serine, selenomethionine, SAM, D-ribose-L-cysteine, S-allyl-cysteine, NAC, Se-methyl-selenocysteine, L-theanine and taurine—have shown potential in preventing

or alleviating Alzheimer's disease-related pathology. In contrast, amino acids such as homocysteine and β-N-methylamino-L-alanine (BMAA) have been identified as contributors to promoting Alzheimer's disease pathology. The underlying mechanisms by which these amino acids influence Alzheimer's disease development include modulation of mitochondrial function, regulation of neuroinflammation and microglial activation, control of oxidative stress levels, interference with Aβ plaque formation, tau protein hyperphosphorylation and the protection or disruption of neuronal and synaptic integrity. These findings highlight the critical role of specific amino acids in either mitigating or exacerbating key pathological processes in Alzheimer's disease and may provide insight for future therapeutic strategies.

Ethics

This study was a secondary analysis based on the currently existing data and did not directly involve with human participants or experimental animals. Therefore, the ethics approval was not required in this paper.

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Conflicts of interest

The authors declare that there is no conflict of interest.

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Data access

The data that support the findings of this study are available from the corresponding author upon reasonable individual request.

Author ORCID numbers

Sapto Yuliani (SY):
0000-0002-8454-2370
Rahmah Annura (RA):
0009-0009-6453-3520
Lolita Lolita (LL):
0000-0001-9876-1905
Dwi Utami (DU):
0000-0003-1833-6250
Danang Prasetyaning Amukti (DPA):
0000-0002-4256-6534

Author contributions

Conceptualisation: SY
Methodology: SY
Formal analysis: RA, LL, DW, DPA
Investigation: RA, LL
Data curation: RA, LL
Writing - original draft: SY
Writing - review and editing: SY, DPA
Visualisation: DW
Supervision: SY

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