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TURMERIC (*Curcuma longa L*) EXTRACT EFFECT ON MEMORY FUNCTION

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ABSTRACT

Memory is the entire process of mental function centered in the brain for raises return knowledge specific about the experience through stages of catch or receive, give code, save, reproduce, and call or bring back. Someone's memory can decrease along increase age. Many factors can influence, among other things: stress, dysfunction pattern-affected acetylcholine life and the environment can cause memory ability to decrease. Stress is a reaction of the body to various situations as threatening as possible and causes a manner physique or physiology that will disturb homeostasis including memorizing. We can consume capable drugs to increase memory and work slows down the descent process of memory. However, using drugs synthetically continuously can raise side effects.[1] Using natural material as a trusted drug can reduce effect side effects compared to synthetic drug use.[2] A possible treatment alternative is consuming turmeric (*Curcuma longa L*). Curcumin, the main component biologically active *Curcuma longa L*, shows various pharmacological activities, including anti-inflammatory, antioxidant, activity immunomodulatory, and neuroprotective. Mechanism enhancement induced memory consequence Turmeric (*Curcuma longa L*) improves synaptic function by increasing the expression of PSD-95 protein and increasing communication locally between the pyramidal neurons.

Keywords: turmeric, *Curcuma longa L*, memory function

1. INTRODUCTION

Everyone needs memory in carrying out daily activities to support their activities. In psychology, memory is a process of encoding, storing, and recalling information (retrieval) by humans and other organisms. Kindly physiology, memory results from a change in synaptic propagation from one neuron to the next due to prior neural activity.[3] Memory is the entire process of mental function centered in the brain for raises return knowledge specific about the experience through stages of catch or receive, give code, save, reproduce, and call or bring back.[4]

Part of the brain that plays a role in the memory process is the *hippocampus*. The memory process is related to the interaction between opioids and their agonists in *the hippocampus* and cerebral cortex.[5] In addition, acetylcholine, which is part of neurotransmitter systems, also plays a role in the memory process.[6] There are three types of memory, that is short-term memory, long-term memory, and working memory. Someone's memory can decrease along increase age. It happens with the elderly and can happen with young age too. Many factors can influence, among other things: stress, dysfunction pattern-affected acetylcholine life and the environment can cause the ability of memory to drop.[5]

Stress is a reaction of the body to various situations as threatening as possible and causes a manner physique or physiology that will disturb homeostasis.[6,1] Brain is the main organ that interprets and responds at a time and is the target of hormone stress Because the brain determines which stimulus is threatening.

The brain also plays a role in arranging function, physiology, and behavior in demand as a response to stressors.[7]

We can consume capable drugs to increase remembering, and work slows down the descent of the memory process. However, using drugs synthetically continuously can raise side effects.[1] Using natural material as a trusted drug can reduce effect side effects compared to synthetic drug use.[2] A possible treatment alternative is consuming turmeric (*Curcuma longa L*).

Curcuma longa L., a plant native to India, has been used as a drug for several disturbances to health for at least 2,500 years.[8] Main component of this drug ancestor is aromatic-turmerone (21.4%), alpha- santalene (7.2%), and aromatic-curcumin (6.1%). However, some reports show that the most active component of *Curcuma* is curcuminoids and essential oils. [9] Curcuminoids contain curcumin (diferuloylmethane), desmethoxycurcumin, and bisdemethoxycurcumin but curcumin is the most components studied from *Curcuma* for characteristic pharmacological and yellow color.

Curcumin, the main biologically active component of *Curcuma longa L*, shows various pharmacological activities, including anti-inflammatory, antioxidant, activity immunomodulatory, and neuroprotective.[10] Recently, the potential effect of antidepressant curcumin in various animal models of depression has been increasingly recognized.[11] Curcumin seems to exert an action antidepressant with modulated system monoaminergic and also got hinder the release of glutamate in nerve terminals from the rat pre-frontal cortex. This effect is similar to the classic antidepressant fluoxetine.[12] The protective effect of curcumin on memory function to stress conditions not yet lots are known. The article will discuss the influence of curcumin on production factor growth, triggering neuroplasticity to increase memory.

2. METHOD

The method used in the literature search in this literature review was systematically carried out according to the keywords " Curcuma *Longa L" and "turmeric" and "Memory Enhancement*." Through machine PUBMED, searches with inclusion criteria include articles in the last five years from 2017-2022, full-text access, original articles, or article reviews in English or Indonesian.

Procedures performed in this research are: (1) Searching for articles using the keywords and criteria above, (2) Eliminating duplicate articles, (3) Screening the substance of the article by looking at the title and abstract, (4) Extracting data according to suitability title of the article with the aim of research to obtain relevant articles (5) Perform analysis and synthesis of the substance of the article. The total amount of the final literature used to review the effects of *Curcuma longa L* on enhancement memory is 12 articles.

3. RESULTS

3.1 Memory

Memory is the ability to save and remember return sensations, impressions, and ideas.[13] Memory is also acquired knowledge. For remember return then. The nerve change involved in retaining or storing knowledge is called footsteps memory.[14] Memory is divide*d* into primary and secondary memory. Primary memory is stored in awareness for a short time during sensory input. It still exists (short-period memory). Memory is secondary for an individual to remember incidents or circumstances that previously disappeared from awareness for a temporary time (long-period memory).[15]

Short-term memory goes on for several seconds to hours. New information obtained at first deposited in memory period short the capacity storage limited. Information in the short-time memory will experience one of two possibilities. It will

quickly be forgotten or consolidated in a longer-lasting memory period through active exercise or repetition.[14]

Studies have shown that exercising the same information repeatedly in the mind accelerates and potentiates the memory transfer rate period short to the memory period long, accelerating and improving consolidation. During certain periods, features necessary from sensory experience become more fixed in storage memory. Phenomenon This explains why somebody can remember A little learned information in a manner deep better than several extensive learned information only superficially.[13]

Total function memory and conversion from memory period short to long memory period depending on the hippocampus, an area of the brain with density receptor glucocorticosteroids supreme and representative level response highest against stress. Some theories explain Why memory period short can be formed. The first theory estimates that the memory period short causes existing activity nerves constantly by impulse nerves that run on the trial memory while in one reverberation neuron circuit. However, the theory not yet can be proven.[13]

Mechanism formation memory can be explained through experiments on *Aplysia* has proven that two forms of memory period short that is habituation and sensitization, caused by various modification canals in the presynaptic terminals of afferent neurons that play a role in the mediating pathway moderate behavior experience modification. Habituation is declined responsiveness to presentation repeated usual stimulus that is no stimulation produce award or punishment. Consequence habituation, canals Ca^{2+} No quick open when potential action comes, reduce entry Ca^{2+} to in the presynaptic terminal, which causes decline release neurotransmitter. As a result, potential postsynapse reduces compared to normal, so decline or loss response efferent controlled behavior postsynapse happens. Without exercise, further decreased responsiveness endures several hours. Habituation, the most common and trusted study, is the first learning process for a human baby.[16]

Sensitization is enhancement responsiveness to stimulation light after intense stimulation or damage. Income Ca^{2+} to in the presynaptic terminal increase through facilitation presynapse. Sensitization can happen as a response to presynaptic neuron transients, but if reinforced by the initial stimulus or dangerous ones, it can become part of a long memory period. Extension short on sensitization caused by $Ca2^{+}$ mediated by change resulting adenylyl *cyclase* increase production cAMP. Also, potentiation period length (PJP) plays a role in the protein synthesis of postsynaptic and presynaptic neurons.[17]

Neurogenesis is a process of neuronal development of histological and functional origin from a precursor. Neurogenesis happens during stage embryonic and perinatal and stops at the end of postnatal development.[18] Currently, much-proven research has limitations on these neurons. No entirely right; neurogenesis has proven it still occurs in adults, even in old age. In adults, neurogenesis is an important factor in tissue homeostasis brain. A disturbance in normal neurogenesis is suspected to be related to many diseases neuropsychiatric like depression and epilepsy.[19]

Neurogenesis in adults is formed mainly by progenitor cells (NSPCs). It occurs in a manner especially in two regions network brain that is region subgranular (SGZ) in the hippocampal dentate gyrus Where cell granule new dentate gyrus formed and region subventricular (SVZ) in the lateral ventricles where the new neurons form and then migrate through a *rostral migratory stream* (RMS) to the olfactory bulb become interneurons.[18] Although thus the

process of forming new neurons the Still rated own limitation because of 2 things that is lack of neurogenesis ability in most big region tissue in the brain caused exists factor originating inhibition of neuroglia, especially oligodendrocytes, and not exists stimulation factor growth after a postnatal period. In addition, the mechanism of new neurons being integrated in a manner that is functional with other parts of the network brain still needs to be explained. [49] Therefore, postnatal neurogenesis is believed to be mediated by a reinforcement mechanism synapse, namely the growth process and addition network synaptic.[18]

The process of neurogenesis and neuroplasticity in the brain of man mature has raised something new: disturbance neurology can be repaired functionally. Knowledge about factors that influence neurogenesis is significant for determining interventions or possible stimuli that mediate the formation of new neurons. The characteristic central of neurogenesis in adults is the sensitivity to suitable stimuli, physiological or pathological, in almost all stages, starting from the proliferation of precursor neurons, the process of maturation, and integration until survival neurons live. For one decade final, a lot study has proven to influence lots of factor in the process of neurogenesis.[20]

Organizational process synapse in the processes of neurogenesis and neuroplasticity relates tightly with the role of PSD-95 as *synapse-associated protein* 90 (SAP 90), which is an important protein component from the PSD fraction of front brain mice and also found localized to the dendrites of hippocampal neurons (Hunt *et al.,* 1996). PSD-95 is a member of *membrane-associated guanylate kinase (MAGUK*) binding receptor glutamate and is a functioning regulator of synapses. The variety of the type of protein that PSD-95 can bind exposes this protein's role in organizing PSD molecularly. More other functions and important capabilities of the PSD-95 to facilitate suite reaction chemistry between NMDA receptors with molecule other signals like *nitric oxide synthase, Kalirin-7, and Rac guanine nucleotide exchange factor* (GEF), which are further will regulate the morphology of dendrite spines.[21]

PSD-95 plays a role in organizing the placement process between synapse with method bond with end chain carboxyl from molecules adhesion at the postsynapse like *neuroligins*, *netrin-G-ligands* (NGLs), *synaptic adhesion-like molecules* (SALMs), and *leucine-rich repeat transmembrane neuronal* (LRRTMs). There is a simultaneous bond between molecule adhesion, receptor glutamate, and conducting protein molecules, signaling the possible role of PSD-95 in the maturation process synapse functionally. Besides functionally influencing thorn dendrites, PSD-95 also morphologically affects thorn dendrites.[21] PSD-95 overexpression will cause a change in morphology, increased volume of spines dendrites, and formation of perforation synapse. The average area of the PSD-95 will be increased up to 8 times, followed by an increase in spine volume dendrites up to 3 times. Finally, the connection between synapses in the transmission signal will be strengthened. Function An interaction mediates this suspect between PSD-95 and *Nitric Oxide Synthase* (NOS), releases *Nitric Oxide* (NO), and activates cGMP signaling at the postsynaptic membrane.[21]

The important role of PSD-95 has been described in on possible influence on growth beginning until dendrite spines maturation. PSD-95 is a component of structural importance possessed by thorns' mature dendrites.

2.3 The effect of *Curcuma longa L* to Neuroplasticity

Curcuma longa L., a plant native to India, has been used as a drug for health for at least 2,500 years.[14] Component main from drug ancestor This is aromatic-turmerone (21.4%), alpha-santalene (7.2%), and

aromatic curcumin (6.1%). [15,12] However, some reports show that the most active component of *Curcuma* is curcuminoids and essential oils. [13,16] Curcuminoids contain curcumin (diferuloylmethane), desmethoxycurcumin, and bisdemethoxycurcumin [13,16] but curcumin is the most components studied from *Curcuma* for characteristic pharmacological and yellow color. [14,16]

Curcumin, the main biologically active component of *Curcuma longa L*, shows various pharmacological activities, including anti-inflammatory, antioxidant, activity immunomodulatory, and neuroprotective. Recently, the potential effect of antidepressant curcumin in various animal models of depression has been increasingly recognized. Curcumin seems to exert action antidepressant with modulated system monoaminergic and also hinders the release of glutamate in nerve terminals from the rat pre-frontal cortex. This effect is similar to the classic antidepressant fluoxetine. A study has shown that treatment of curcumin in a manner significantly reduces time immobility in test swim forced through activation expression factor Inherited neurotrophic from MAPK /ERK-dependent brain in mice.

Although curcumin has proven to become a compound naturally multi-target, which can modulate many lanes characterization detail from mechanism underlying nerves effect antidepressant part big Still Not yet known. Lots of proof shows that depression is associated with various change forms and functions of synapses in several brain regions. For example, on stress, neuropsychiatric processes associated with induce change morphology of dendrites and spines in the hippocampus and pyramidal neurons pre-frontal cortex (PFC) which shows change general in structure nerves and plasticity in conditions like stress and depression. However, inside the amygdala, a region of the brain, important others involved in depression, change opposite in structure nerve observed as a response to protocol stress. [22]

A recent report shows that curcumin is very inducing expression and activity of HO-1 in various brain regions with heterodimer activation of the Nrf2/ antioxidant pathway responsive element (ARE). In addition, research shows that curcumin also improves thioredoxin (Trx) protein expression and activity enzyme Trx in cortical neurons in mice. Both proteins, Nrf2 and Trx, have antioxidant action.[22] A study report shows that old mice subtracted long dendritic and reduced density bone behind the dendritic area limbic such as PFC, hippocampus, and BLA with a deficit in process memory.

However, the consumption period long from turmeric increase arborization dendritic and the number of thorns dendritic PFC, hippocampus, and BLA in animals old. This data show that turmeric increases communication localized among pyramidal neurons, which were analyzed by measuring long dendrites in the region.[23] Besides that, communication distance far is also evaluated through the analysis density of bone behind the distal dendritic, and its results show that turmeric also improves communication between limbic areas, mainly between the PFC and the hippocampus. Because it is possible for impressive that effect of chronic consumption of turmeric produces arrangement reset on level communication reflected dendritic with more memory processes well, mostly long-period memory.[23]

In another study, Western Blot analysis showed increased PSD-95 levels in the curcumin group (LDC, MDC, and HDC). Immunohistochemistry showed a significant increase in PSD-95 stained cells in the HDC (High Dose Curcumin) group. These results suggest that curcumin can improve synapse function by increasing the expression of synapse-associated protein PSD-95.[24]

4. CONCLUSION

Curcumin, components biological active main *Curcuma longa L*, shows various pharmacological activities, including anti-inflammatory, antioxidant, activity immunomodulatory, and neuroprotective. Mechanism enhancement induced memory consequence Turmeric (*Curcuma longa L*) improves synaptic function by increasing the expression of PSD-95 protein and increasing communication locally between the pyramidal neur

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