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Possible Role of mGluR5 Receptor in Synaptic Plasticity and Synaptic Transmission of Memory and Learning

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Abstract*—Synaptic plasticity is the capacity of neurons to alter their network connections in brain while neuronal damage occurs within, due to inflammatory and neurodegenerative diseases. Synaptic transmission occurs at synapses and transmits the neuronal information through exchange of chemical, electrical signals. mGluR5 is the subtype receptor of group I metabotropic glutamate receptor and plays a vital role in modulation of synaptic plasticity. The mGluR5 subtype receptors are associated with consolidation of neural networks during learning. Present review is focused how the of mGluR5 are responsible for the source of learning and memory. Here in the review, we discussed about unique role mGluR5 of memory and learning and Synaptic plasticity*.

***Keywords*—** *mGluR5, Memory, Synaptic plasticity, Learning*.

I. Introduction

Synaptic plasticity is a structural mechanism by which the brain reworks and realizes and learns new things and performing a fundamental role in shaping cognitive functions like, learning and memory. The ability to strength memories and transmits through synapses is called synaptic transmission. Neural synapses and circuits of nervous system are mainly formed by 2 types of synaptic plasticity, long term potentiation (LTP) which has long-lasting enhancing of synaptic transmission following high-frequency stimulation of a synapse that, plays in vital role in cellular mechanism of learning and memory. Long-term depression (LTD) is lasting decrease in synaptic strength following low-frequency stimulation [1]. Usually LTP implies with N-methyl-d-aspartate receptor (NMDA) which is glutamate type of receptor that plays essential role in synaptic plasticity and these receptors are efficient at neural signal transmissions [2]. The establishment of memory is concerns with stabilization of synaptic changes provoked by LTP and LTD and strengthen by synaptic connections and the enrolment of extra new neural circuits that, makes memory additional stable and resilient to interference [3]. Synaptic plasticity regulations deprivations can lead to neurological disorders like Alzheimer's disease where impairments the memory and schizophrenia and autism spectrum disorders. Knowing about certain pathophysiological mechanism involved in synaptic plasticity can establish the therapeutic interventions [4].

II. Materials & Methods

A review of the literature was conducted, focusing on studies that discuss on the role of mGluR5 receptor in synaptic plasticity and synaptic transmission of memory and learning and their deficits related to mGluR5. Google Scholar, ScienceDirect, Web of Science, PubMed/Medline and Research Gate were used to collect data as evidence from already available published literature.

III. Discussion

Synaptic plasticity defines as, the capability of neurons to modulate its intensity and strength by their connection network with other neurons by neurophysiological process involved in expansion and restructuring after damage [5]. mGluR5 is the subtype receptor of group I metabotropic glutamate receptor and plays a vital role in modulation of synaptic plasticity. The function of mGluRs in are responsible for the source of learning and memory, characterized in the hippocampus [6]. MGluR5 is a subtype of type I mGluRs, is a G protein coupled receptor (GPCR) which is responsible for a variety of nervous system diseases like dementia and Alzheimer's disease. mGluR5 regulates the induction and maintenance of long-term potentiation (LTP) in the CA1 region of hippocampus. In Parkinson's disease, Inhibition of mGluR5 regulates the glutamate associated overactivity and postsynaptic excitatory synaptic transmission [7,8]. Glutamate is the primary excitatory neurotransmitter in the brain and activates both ionotropic glutamate receptors and G protein coupled metabotropic glutamate receptors mGluRs [9]. Inhibition of mGluR5 expression can impair the spatial learning in experimental rats [10]. Ionotopic glutamate receptors play a vital role in excitotoxic cell death and related with neurodegeneration [11].

mGluRs is important factor that showed processing in synaptic plasticity that maintain neuronal development for learning and memory [12]. mGluR5 mRNA protein expression is gradually reduced in aging [13]. mGluR5 signalling changes were noticed in various neurodegenerative diseases like Parkinson 's disease, Huntington 's disease and Alzheimer 's disease (AD) [14]. mGluR5 is the subtype receptor of group I metabotropic glutamate receptor and plays a vital role in

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modulation of synaptic plasticity. The function of mGluRs in synaptic plasticity and synaptic transmission are responsible for the source of learning and memory, characterized in the hippocampus [15]. The role of mGluRs in learning-facilitated plasticity has not yet established, but mGluR1 and mGluR5 synchronize synaptic excitability and essential for synaptic plasticity [16]. Formation of the memory mostly occurs due toto rely on synaptic plasticity [17]. learning and memory processes is associate with long term potentiation (LTP synaptic plasticity [18]. LTP depends on the activation of n-methyl-d-aspartate receptors (NMDARs) and they arise from the functional characteristics of this receptor are the one of the synaptic plasticity properties [19]. Modified NMDA-mediated synaptic plasticity decreases temporal correlation between converging inputs to coupled neurons, that leads to additional activity-dependent disconnection of networks in prefrontal cortex [20]. Lack of mGluR5 in the mice affected hippocampal long-term potentiation and hence attributed for spatial learning impairments and fear conditioning [21]. Alterations in synaptic plasticity leads to memory deficits diseases, particularly neurodegenerations in Alzheimer's disease [22].

IV. Conclusion

Synaptic plasticity and transmission are a major mechanism by where memory formation, consolidation and transmission take place through synapses. The group I metabotropic glutamate receptor and its subtype mGluR5 is showed that to cooperate a fundamental role in the alteration of synaptic plasticity. Present study attempted to review the possible role of synaptic plasticity and its transmission in the presence of mGluR5 receptors in memory and learning.

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