The Hippocampus: Anatomy, Function and Clinical Correlation

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Abstract

The hippocampus is a brain region situated deep within the temporal lobe. The hippocampus houses the archipallium cortex, which includes components such as the dentate gyrus, cornu ammonis, and subiculum. Each structure contains different layers and relates to structures around the hippocampus through afferent and efferent nerves.

The coupling of afferent and efferent fibers is crucial in supporting the hippocampal function related to memory formation, learning, spatial navigation, and emotion regulation. Previous studies have shown that several neurological diseases are associated with hippocampus abnormalities. The transformations in the hippocampus, spanning from molecular to morphological alterations, have been extensively researched. Progress in science and technology has facilitated a more detailed examination of the hippocampus structure.

This article will explore the typical anatomical configuration of the hippocampus, its functionality, and its connections with neighboring structures. Furthermore, it will also address hippocampus alterations in various disease states.

Keywords: Hippocampus, Alzheimer's Disease, Epilepsy, Depression

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Introduction

The hippocampus is involved in cognitive function, learning, and memory and regulates the stress response. The cells within the hippocampus exhibit distinct features, notably a pyramid form, particularly in the cornu ammonis (CA) region. The hippocampus's cornu ammonis region comprises CA1, CA2, and CA3. The CA3 and CA1 layers are responsible for learning and memory functions. However, the role of the CA2 layer remains uncertain. The pyramidal cells of the cornu ammonis are vulnerable to various pathological circumstances, including the impact of stress, seizures, and similar factors. Extended stress can result in neuronal degeneration in both layers of the hippocampus (1). Cortisol is a protein that plays a role in adaptation and stress response. Its levels rise in situations of chronic stress (2). The hippocampus contains many cortisol receptors that have a remarkably high sensitivity. In the hippocampus, memory formation occurs through short-term changes in the electrical properties of neurons and long-term modifications in the structure of synapses. Short-term alterations manifest as long-term potentiation (LTP) at the synapse, whereas enduring modifications take the form of neuropil development and synaptogenesis (3). BDNF is a significant neurotrophin that plays a crucial role in long-term potentiation (LTP). Previous studies have shown that long-term exposure to chronic stress reduces BDNF expression in the hippocampus (4). It is crucial to comprehend the anatomical structure and function of the hippocampus in order to analyse the alterations that occur in diseased circumstances. By comprehending the different alterations in structure and function, we aim to create a treatment approach to prevent or reduce hippocampus changes associated with specific clinical disorders.

Anatomy of Hippocampus

The hippocampal formation, which includes structures such as the indusium griseum, longitudinal striae, gyrus fasciolaris, hippocampus proper, and part of uncus, is a complex region. Nevertheless, according to some sources, the hippocampal formation comprises the hippocampus proper, dentate gyrus, subiculum, and entorhinal region. The hippocampus comprises cornu ammonis (CA), dentate gyrus, and subiculum. The hippocampus formation comprises the archipallium and forms along the inferomedial aspect of the temporal lobe towards the lateral ventricle by the C-shaped choroidal fissure (5). The hippocampus volume in adults is approximately 3-3.5cm^3, with a length of around 5 cm for each hemisphere. This volume is a mere fraction of the neocortex's volume, typically between the range of 320-420cm^3 (6).

The Indusium griseum and longitudinal striae are residual structures of the hippocampal development. The indusium griseum is a thin layer of grey neural tissue that covers the corpus callosum. This layer crosses over the medial and lateral longitudinal striae on both midline sides (7,8). The indusium griseum surrounds the genu and rostrum of the corpus callosum and the paraterminal gyrus at the front. The indusium griseum spans from the gyrus fasciolaris, wraps around the splenium of the corpus callosum, and then links to the

dentate gyrus. The indusium griseum is connected to the cingulate gyrus in the lateral region via the callosal sulcus (7–9).

The dentate gyrus is situated anatomically between the parahippocampal gyrus and the hippocampus. The structure in question is a thin strip of grey matter that runs from the gyrus fasciolaris along the top surface of the parahippocampal gyrus. The dentate gyrus consists of the architectural fascia dentata, which is organized into three layers in the following order: the outermost molecular layer, the intermediate granular layer, and the interior multiform layer. The multiform layer expands into the CA4 section, also known as the hillus, of the cornu ammonis. The hillus and dentate fascia collectively form the dentate gyrus. The dentate gyrus extends continually as the caudal portion of the dentate gyrus, referred to as the Giacomini band, crosses the uncus posteriorly and medially. The Giacomini band divides the uncus into two discrete and clearly defined areas: The uncinate gyrus is anterior to the band.

In contrast, the intra-limbic gyrus is positioned posterior to it. The hippocampal sulcus separates the dentate gyrus from the parahippocampal gyrus. The fornix fimbria, which envelops the dentate gyrus, is restricted from the dentate gyrus by the fimbriodentate sulcus (10–13). The fornix is a crucial arc-shaped cluster of nerve fibers that connects the hippocampus with the mamillary body of the hypothalamus and septum.

The hippocampus is a prominent curved structure located on the inner surface of the temporal lobe, forming an elevated shape. The hippocampus comprises the folded dentate gyrus, cornu ammonis, and subiculum (Figure 1) (5). The hippocampus consists of three main parts: the head, body, and tail. The skull is the widest component. The hippocampal sulcus separates the cornu ammonis and dentate gyrus, with the subiculum located below the sulcus. The subiculum smoothly continues into the six-layered neocortex of the parahippocampal gyrus (11–13). During development, the cornu ammonis and dentate gyrus move towards the inferior horn of the lateral ventricle near the hippocampal sulcus. This condition causes the molecular layers of the dentate gyrus and subiculum to come close to each other (5,14).

The hippocampus resembles a seahorse, with the ventricular surface covered by the alveus, a slender layer of grey matter. The alveus is formed by the axons of hippocampal pyramidal cells, which come together in the middle of the hippocampus to create the fimbriae of the hippocampi (12,13,15). The fimbriae of the hippocampi extend posteriorly to surround the dentate gyrus and establish a connection with the splenium of the corpus callosum, which subsequently connects with the fornix. The fimbria-dentate sulcus separates the fimbria from the dentate gyrus. The fimbria surrounds the thalamus and is connected to it by the choroidal fissure, which contains the choroidal plexus. This fissure acts as the barrier between the fornix and the thalamus (11–13).

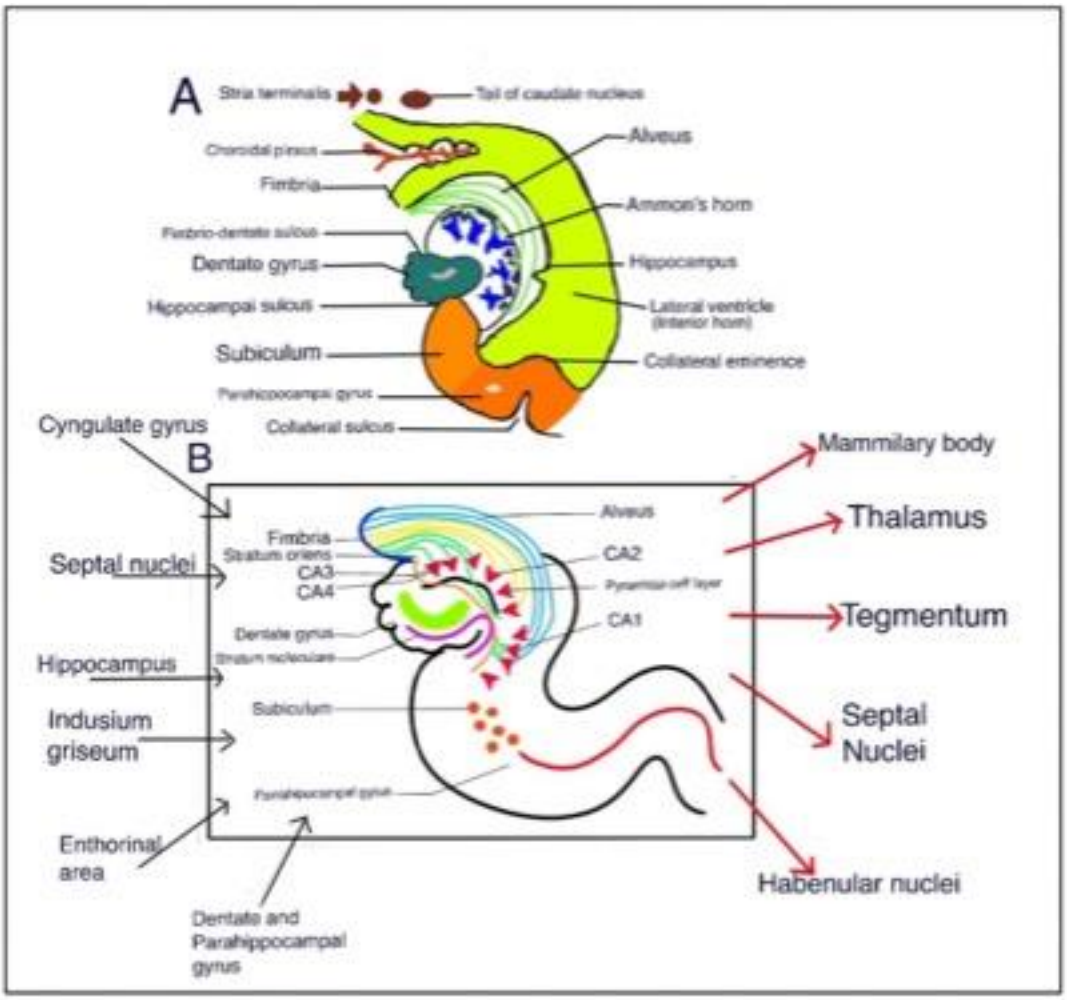

Figure 1. (A) Anatomy of the hippocampus; (B) Connections of the hippocampus with surrounding structures

The hippocampus and cerebral cortex possess distinct roles, physical features, and cytoarchitecture. The hippocampus belongs to the archicortex, whereas the cerebral cortex is derived from the neocortex. The hippocampus is situated on the bottom of the lateral ventricle, whereas the cortex cerebri is the outermost region of the brain. The cortex cerebri surface consists of gyri and sulci, but the hippocampus is convex and lacks gyri and sulci. The hippocampus spreads in the anterior region to create a paw-like shape called "pes hippocampi" (6).

Arterial supply to the hippocampus

The hippocampus plays a vital role in the process of memory formation. Malfunction in this domain can lead to conditions such as Alzheimer's disease and epilepsy (5,10,16). The hippocampus mainly receives blood supply via the posterior cerebral artery and anterior choroid artery. The posterior cerebral artery is the final branch of the basilar artery, which unites with the posterior communicating artery to complete the circle of Willis. The posterior cerebral artery is anatomically

segmented into four distinct sections and gives rise to three main arteries: cortical branches, postero-lateral striate branches, and posterior choroid artery. The second segment of the posterior cerebral artery gives rise to two branches called the anterior inferior temporal artery and anterior hippocampal-parahippocampal artery. These branches send blood to the entorhinal area (5,16,17). The parahippocampal gyrus and hippocampus are supplied with blood via the parietooccipital arteria trunk, which is a branch that originates from the fourth posterior cerebral artery (5).

The anterior choroid artery arises from the distal segment of the internal carotid artery and runs towards the subarachnoid space. The segment that stretches from the anterior choroid artery to the inferior horn of the lateral ventricle is known as the cisternal segment. The anterior choroidal artery gives rise to the choroid plexus in the inferior horn of the lateral ventricle. In addition, the anterior choroidal artery supplies blood to the optic tract, uncus, globus pallidus, lateral geniculate body, and internal capsule. The amygdala and hippocampus receive blood flow from the perforating branches of the anterior choroidal artery (5).

The anterior hippocampal artery enters the uncal sulcus in order to deliver blood to the head of the hippocampus. Conversely, the posterior hippocampal artery provides nourishment to the body and tail of the hippocampus (16,17). The anterior hippocampus artery arises from the surface of the piriformis lobe and supplies blood to the nearby entorhinal area. The posterior hippocampal artery runs through the hippocampal sulcus, giving out important branches that enter the hippocampus and smaller branches that supply blood to the margo denticulatus and fimbriodentate sulcus. The longitudinal branches of the posterior hippocampal artery form connections along the hippocampal sulcus. The intrahippocampal artery is divided into four segments and supplies blood to certain areas. The first step involves the significant ventral feeding of the stratum pyramidalis, stratum lacunosum, molecular layer of the dentate gyrus, and CA1 and CA2 regions. The second component, known as the dorsal portion, provides the CA3 and CA4 sections as well as the granular layer of the dentate gyrus. The ventral and dorsal regions contribute to the proximal portion of the dentate gyrus, fimbriodentate sulcus, and the adjacent area (5,16,17).

Venous drainage of the Hippocampus

The deep hippocampal vein, sometimes called the intrahippocampal vein, consists of two categories. The initial category is the intrahippocampal sulcal vein, which arises from the CA1 and CA2 areas, stretches towards the superficial hippocampal sulcus, and gathers drainage from the molecular layer. The second kind is the subependymal intrahippocampal vein, situated on the surface of the hippocampus ventricle. The deep hippocampal veins, which arise from CA2 and the subiculum, are responsible for emptying into the subependymal intrahippocampal vein. The superficial veins of the hippocampus provide two longitudinal venous pathways that extend across the fimbriodentate sulcus and the

superficial hippocampal sulcus. The venous channel originating from the fimbriodentate sulcus is connected to the subependymal intrahippocampal vein. In contrast, the venous pathway arising from the superficial hippocampal sulcus is coupled with the deep intrahippocampal vein. Usually, the veins that provide blood to the hippocampus are constricted, which raises the chances of blood clot development and subsequent damage to the pyramidal cells in the hippocampus (5).

Histology of the Hippocampus

The dentate gyrus serves as the input channel within the hippocampal formation system. The dentate gyrus's microscopic composition comprises three layers: the molecular layer, granular layer, and polymorphic layer, arranged from outermost to innermost. The dentate gyrus has a semilunar shape, being convex towards the molecular layer and concave towards the CA region (5).

The dendrites of granular neurons receive impulses from the parahippocampal gyrus, more precisely from the entorhinal cortex through the perforant pathway. Axons originating from granular neurons form synapses through mossy fibers onto the apical dendrites of pyramid cells in the CA region. The Cornu Ammonis consists of multiple layers, one of which is the alveus. The alveus is formed by efferent fibers from CA pyramid cell axons when specific collateral axons re-enter the hippocampus. The stratum oriens houses a limited number of inhibitory interneurons known as basket cells. Stratum pyramidalis is the primary cellular component of the CA, consisting of 10-30 layers of pyramidal cells. Stratum radiatum is composed of apical dendrites of pyramid cells and stellate cells. Stratum lacunose-molecular contains axons and interneurons, with inhibitory interneurons projecting to the retrosplenial cortex from this layer. The stratum oriens is crossed by axons and dendrites of pyramid cells, recurrent axon collaterals, and commissural fibers (5,11,13).

The main function of the pyramidal layer is to generate excitatory signals. Pyramidal cells have an apex connected to the alveus, whereas the base is directly connected to the outer molecular layer. Alveus and fimbriae are formed by axons originating from the central region at the base of the pyramidal cell. The dendrites originating from the base extend into the stratum oriens, whereas the basal dendrites receive commissural fibers from the comparable area in the contralateral hippocampus. The dendrites that stretch from the apex branch out extensively, and the apical dendrite receives commissural fibers from a specific area of the opposite hippocampus. The apical dendrite receives afferent fibers from the entorhinal area and mossy fibers from the dentate gyrus. Neighboring pyramidal cells establish recurrent connections that form synapses with the higher dendrite (5,11,13).

Regulation and Connections of the Hippocampus

The cornu ammonis of the hippocampus is divided into four separate regions referred to as CA1, CA2, CA3, and CA4. The CA1 region is the largest area, including several structures,

such as the presubiculum on the outside and CA2 on the inner side. Most neurons in CA1 are comprised of pyramidal cells, making up 90% of the total. These pyramidal cells are glutaminergic projection neurons. The remaining neurons in CA1 are interneurons. CA2 is adjacent to CA1 in a lateral direction and to CA3 in a medial direction. The structure receives flux from the supra mammillary region of the hypothalamus, but it only receives modest mossy fiber input from the dentate gyrus. CA3 forms a direct connection with the hilus of the dentate gyrus, which is located adjacent to CA2 on the inner side. The CA3 neurons get input from mossy fibers that originate in the granule cells of the dentate gyrus. In contrast, the axons of CA3 pyramidal cells extend to the alveus, fimbria, and fornix. Specific axons in the CA3 region receive extra fibers called Schaffer's collaterals, which form connections with the dendrites of CA1 pyramidal cells. CA1 pyramidal neurons have axons that establish connections with subiculum neurons. The axons of subiculum neurons give rise to the fibers of the fimbria and fornix via the alveolar pathway (5).

The hippocampus receives input from several structures, including the cingulate gyrus via the cingulum, the indusium griseum and septal nuclei via the fornix, the opposite hippocampus via the hippocampal commissure, the outer part of the entorhinal area via the perforant pathway, and the inner entorhinal area and subiculum to the alveus via the alveolar path (Figure 2) (5). The entorhinal cortex conveys impulses to the dentate gyrus, responsible for recognizing patterns and remembering memories (18). The axons of the perforant pathway arise mostly from layers II and III of the entorhinal cortex, with limited contribution from the deeper layers IV and V. Axons originating from layers II and IV extend to granule cells and CA3 pyramidal cells. On the other hand, axons originating from layers III and IV can reach CA1 pyramidal cells and the subiculum.

Various routes extend from the entorhinal cortex (layer III of the perforant pathway) to CA1 neurons. CA1 pyramidal cells project axons to the subiculum and within the entorhinal cortex layer. The Shaffer collateral is the conduit through which axons from region CA1 connect to area CA3. This pathway is crucial in memory formation and emotional regulation within the Papez circuit. Conduction in this circuit is excitatory, namely glutaminergic, and plays a role in synaptic plasticity (5,6). One more stimulating pathway is the recurrent collateral that transmits signals to CA3. This circuit plays a crucial role in retaining memory, such as when a teacher delivers a lesson, gets interrupted by a phone call, and continues with the lesson.

The efferent fibers from the hippocampus will connect with other structures via the fornix. The efferent fibers from the hippocampus travel through the dorsal fornix to reach the gyrus fasciolaris, indusium griseum, cingulate gyrus, and septal nuclei. Moreover, hippocampal efferent fibers project to the para terminal gyrus, pre-optic and anterior hypothalamic nuclei through the pre-commissural fornix; to the hypothalamic nuclei, anterior nuclei of the thalamus, and mammillary body via the post-commissural fibers; and ultimately to the habenular nucleus via the stria medullaris thalami (5).

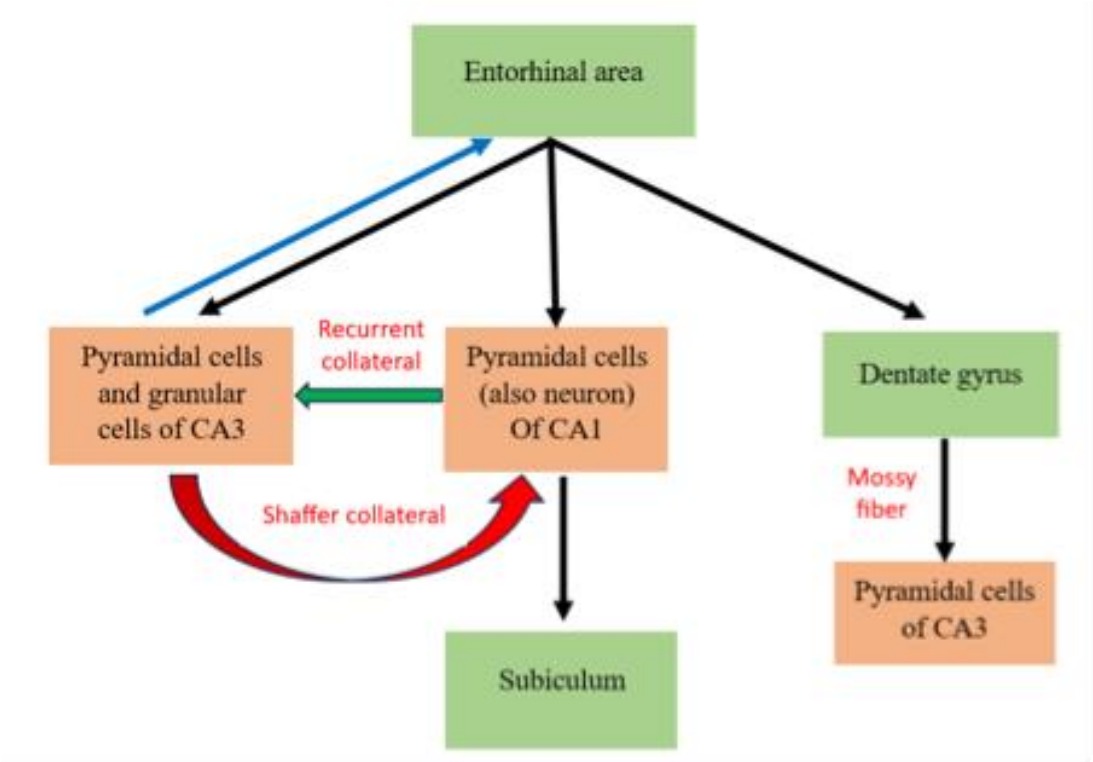

Figure 2. Perforant pathway

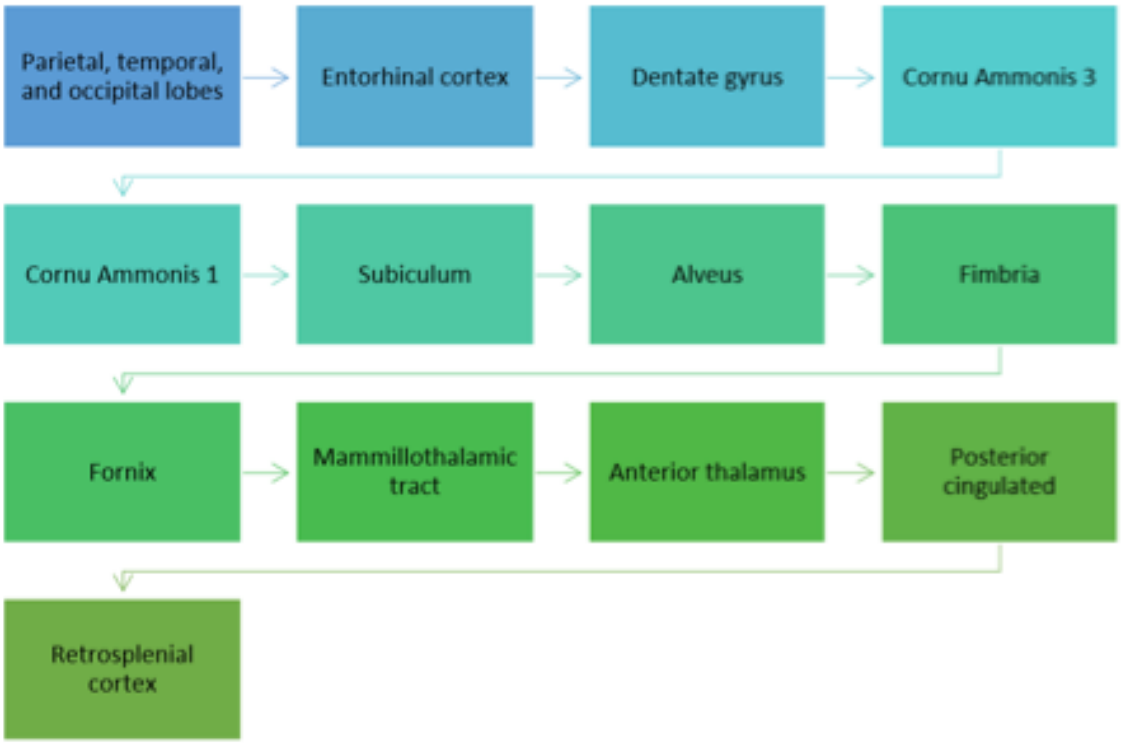

Figure 3. Polysynaptic Pathway

Correlation of Clinical Implications with Hippocampal Dysfunction

Alzheimer's Disease

Neuronal loss and gliosis in the hippocampus are commonly observed in Alzheimer's Disease (AD) as a neuropathological disorder. The layers that vanish on both sides consist of stratum radiatum, stratum lacunosum, stratum moleculare, subiculum, and stratum pyramidale, leading to the decreased hippocampal volume known as hippocampal atrophy. The pathophysiology of AD encompasses various pathways, including neuroinflammation, oxidative stress, and the buildup of Aβ peptides and tau proteins. The initial region impacted in cases of Alzheimer's disease is the entorhinal area, characterized by the buildup of tau proteins, subsequently progressing to the hippocampus (23,24). Neuronal degeneration initiates in the second layer of the entorhinal cortex, then spreads to the hippocampus and further impacts the temporal, frontal, and parietal cortices and subcortical nuclei. Over time, a disconnection arises between the dentate gyrus and other regions of the hippocampus, leading to a decline in cognitive abilities (24,25). Neurofibrillary tangles are also seen in Alzheimer's disease, starting in area CA1 and spreading to the subiculum, CA2, CA3, and dentate gyrus (24).

Excessive activity in the memory network, which includes the hippocampus, medial temporal lobe, and other cortex regions, is observed in individuals with Alzheimer's disease. Elevated neuronal activity augments amyloid precursor protein, leading to heightened Aβ peptide creation. Prior research on individuals with mild cognitive impairment and memory-impaired rodents demonstrated elevated generation and accumulation of peptide Aβ, forming plaques (26–28). Aβ plaques manifest near heightened neuronal activity in the Alzheimer's disease mice model (28). The aggregation of Aβ peptides can inhibit impulses that enter the hippocampus (24). Alterations in several neurotransmitters, such as noradrenergic, glutaminergic, and serotoninergic, occur in Alzheimer's disease as a reaction to neuronal depletion in the hippocampus (6).

Epilepsy

Epilepsy is a brain disorder caused by a rapid and simultaneous imbalance between excitation and inhibitory signals. Temporal lobe epilepsy is the predominant kind of epilepsy, often linked with developing hippocampal sclerosis (HS), leading to potential cognitive and memory issues and a heightened chance of resistance to therapy (29,30). The International League Against Epilepsy (ILAE) categorizes HS into typical and atypical, based on alterations in histological structure caused by neuronal cell death and gliosis. The features of hippocampal sclerosis in epilepsy include reorganization of granule cells, changes in interneuronal populations, modifications in neuropeptide fiber networks, and the growth of mossy fibers (31). CA1 and the hilus region of the dentate gyrus and CA3 are the areas most frequently impacted. CA2 and granular cells of the dentate gyrus typically exhibit reduced

neuronal cell death. Axon elongation and expansion of neuronal cells in the granular cell layer of the dentate gyrus of the hippocampus are also observed in this situation (32).

In cases of temporal lobe epilepsy with hippocampal sclerosis, there are processes of structural reorganization, specific neuronal cell death, and the formation of new neurons that contribute to the development of epilepsy. This epilepsy involves specific neuronal damage in the dentate gyrus's hilus and the hippocampus's pyramidal cell layer. Despite this, certain granular cells in the dentate gyrus and a small section of the pyramidal cells in the CA region of the hippocampus are preserved. The existence of gliosis and neuronal loss results in the contraction and solidification of the tissue. Neuronal depletion in the entorhinal cortex and amygdala area results in mesial temporal sclerosis (33,34).

The hippocampal sclerosis contributes to the development of epilepsy. The theory of modified sprouting of mossy fiber axons from granule cells of the dentate gyrus corroborates this. Typically, the hippocampus receives excitatory input directly at granular cells of the dentate gyrus hippocampus from the entorhinal cortex. In contrast, inhibitory input originates locally from interneurons in the deep molecular layer. The granular cells of the dentate gyrus induce sprouting on mossy fiber axons, which are then projected to pyramidal neurons as a component of the efferent pathway of the hippocampus. Dentate gyrus granular cells often withstand hypersynchronous activation and prevent the onset of epileptic seizures originating from the entorhinal cortex. In sclerosis of the hippocampus, these cells cause the mossy fiber axons to directly return to the deep molecular layer, possibly because the neurons they extend have been lost. Evidence indicates that abnormal mossy fibers establish recurrent excitation circuits by creating a synapse on the dendrites of neighbouring dentate gyrus granular cells. Excitatory interneurons in the dentate gyrus, responsible for stimulating inhibitory interneurons, seem specifically depleted. Loss of excitatory cells can disturb inhibitory feedback and have a lasting impact on the granular cells of the dentate gyrus, leading to hyperexcitability. Moreover, evidence indicates that granular cells of the dentate gyrus can project directly onto inhibitory interneurons, which implies the presence of a potential compensating mechanism (33,34).

Injury to the hippocampus resulting from status epilepticus can lead to long-lasting hyperexcitability that continues even after the status epilepticus has ended. This persistent hyperexcitability results from the collective impact of many structural alterations (such as gliosis, neuronal cell death, mesial temporal sclerosis, and the development of aberrant axonal connections or "sprouting") that can give rise to epileptic foci in the brain. Germination leads to abnormal excitatory synapses. The granular neurons of the dentate gyrus are responsible for receiving all the activity that enters the hippocampus. Typically, these cells initiate individual action potentials and connect with the pyramidal neurons of the hippocampus, which in turn also generate individual action potentials when stimulated by input from the dentate gyrus. Status epilepticus results in the demise of

pyramidal cells due to heightened NMDA receptor activity and an excessive influx of calcium ions, but it preserves dentate gyrus neurons (35,36).

Consequently, the axon of the dentate gyrus neuron departs without a post-synaptic destination, consequently, re-innervating its dendrites and the dendrites of adjacent dentate gyrus neurons, establishing "auto-excitation" as an excitatory circuit. This leads to the dentate gyrus neurons receiving an overabundance of excitatory input, causing them to generate numerous action potentials, similarly affecting the surviving pyramidal neurons. This proliferation extends beyond the hippocampus and encompasses a variety of mechanisms that enhance the hyperactivity of brain circuits (35,37).

Research conducted on individuals with temporal lobe epilepsy and in laboratory animals with temporal lobe epilepsy induced by kainic acid demonstrates that both cytotoxicity resulting from overstimulation of NMDA receptors and apoptotic mechanisms involving the activation of caspase family proteins, which lead to hippocampal atrophy, occur promptly following the onset of chronic epileptic seizures. This conclusion is supported by studies indicating that NMDA receptor antagonists can decrease hippocampal damage during status epilepticus. Research on identifying caspase substrates that play a role in neuronal cell death triggered by epileptic waking remains rather obscure (30,32).

Depression

Multiple prior research has established a connection between the occurrence of major depressive disorders (MDD) and a decrease in hippocampus volume in comparison to individuals without the condition. Prolonged duration of depression, frequency of recurring episodes, and age of depression beginning were correlated with a reduction in hippocampus volume. One hypothesis that connects MDD with hippocampal volume is that neurotoxicity plays a role, as extended exposure to glucocorticoids heightens the vulnerability to neuronal harm, thereby accelerating brain damage. Regarding this idea, decreased hippocampus volume is a result of long-term depression, post-traumatic stress disorders, and chronic stress. Furthermore, disruptions in the HPA axis and irregularities in other neurobiological factors also have a role in diminishing hippocampus volume. These neurobiological illnesses encompass a decrease in neurotrophic factors like BDNF due to stress and a decrease in neurogenesis caused by stress. Stress can lead to decreased BDNF signaling in the hippocampus, where BDNF plays a crucial role in neural development (38–40).

During depression, alterations in hippocampal synaptic plasticity are also present in many hippocampal subregions, notably CA3 and the dentate gyrus. Chronic unpredictable stress (CUS) and the use of corticosteroids can decrease the transmission of excitatory signals from the temporoammonic-CA1 region in rats (41). CA1 experiences long-term plasticity as a result of the activation of GABAergic interneurons, specifically those that produce

parvalbumin and nitric oxide synthase (42). Intense stress triggers μ-opioid receptors on GABAergic neurons to enhance long-term depression generated by low-frequency stimulation at Schaffer collateral CA1 glutaminergic synapses in mice. Within an experimental animal model featuring depression generated by persistent unpredictable mild stress, a reduction in LTP, basal synaptic transmission, and dendritic spine density was observed at the CA3-CA1 synapse (43). Apoptosis was also observed in the CA1, CA4, and dentate gyrus regions in individuals diagnosed with MDD (40,44). Adult neurogenesis changes the excitation level in the dentate gyrus and enhances the ability to respond to chronic stress by suppressing adult granule cells in the ventral dentate gyrus. Stress and corticosterone may inhibit progenitor cell proliferation through the potential elevation of nitric oxide levels (40). Neurogenesis also encompasses the involvement of astrocytes in the dentate gyrus of the hippocampus. Depression caused by chronic mild stress can be linked to a decrease in astrocyte quantity and a reduction in microglia function. Microglia rebuild synapses by engaging in pre-synaptic phagocytosis and inducing spine head filopodia (45). In a research study using an animal model to generate depression by CUS, there was a reduction in the number of oligodendrocytes observed in the hippocampus's CA3 and dentate gyrus regions (46). Oligodendrocytes are involved in various crucial processes, including the facilitation of short-term and long-term plasticity in the hippocampus substantia alba and the regulation of axonal excitability and neuronal conduction.(40) Studies conducted on mice revealed that the interference with oligodendrocytes led to decreased memory consolidation (47).

Conclusion

The structure of the hippocampus is a crucial component of the brain's limbic. The hippocampus is essential and cannot be substituted by other brain components. The hippocampus is mostly composed of the subiculum, dentate gyrus, and CA, according to anatomical anatomy. The dentate gyrus is a constituent of the substantia grisea, comprising three layers: the polymorphic layer, the granular layer, and the molecular layer. The main function of the hippocampus is to facilitate memory formation. In order to carry out this function, the hippocampus is connected to various regions of the brain by a network of incoming and outgoing nerve fibers. Granular neurons get input from the parahippocampal gyrus through the perforant route, which starts in the entorhinal cortex. Granular neurons transmit mossy fibres to the apical dendrites of pyramidal cells in the cornu ammonis. The axons of pyramidal cells in the hippocampus give rise to the alveus, which thereafter expands as the fimbria and fornix. The fornix reaches to the septal area. The hippocampus is crucial in the formation of memories. To carry out this function, the hippocampus is connected to various regions of the brain through incoming and outgoing nerve fibers. Hippocampal abnormalities are linked to specific clinical disorders such as Alzheimer's disease, epilepsy, and depression.

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