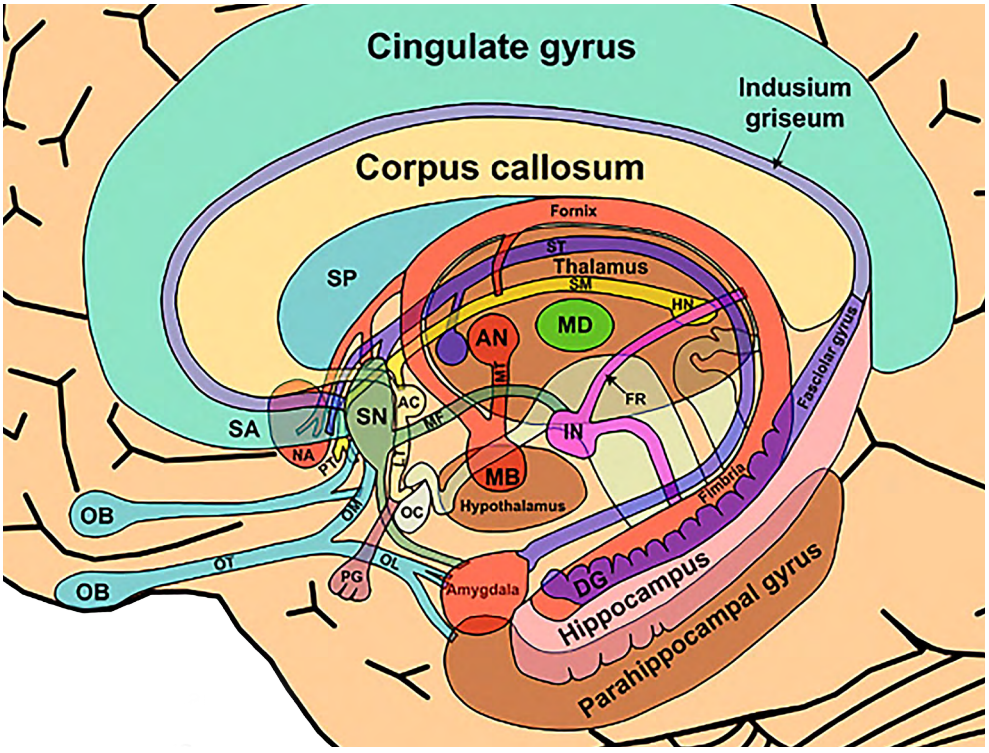
DEEP SYSTEMS

THE LIMBIC SYSTEM

The limbic system (also known as the paleomammalian brain) is a collection of brain structures located in the middle of the brain. It was first defined by Paul Broca in the 19th century as the structures between the cerebral hemisphere and the brainstem (i.e., the limbus, or border of the brain). The limbic system is not a discrete system itself but rather a collection of structures—anatomically related but varying greatly in function. The term has been in use for about 70 years, despite the belief of some neuroscientists that it should be abandoned for implying that the various structures represent a functionally unified system. Irrespective of terminology, collectively we can think of the limbic system as the centre for emotional responsiveness, motivation, memory formation and integration, olfaction, and the mechanisms designed to keep us safe. These are broad strokes, certainly—not to suggest that the neocortex is not involved in these functions, but these are the focal activities of the limbic system. For the purposes of this overview we will be considering the amygdala, hippocampus, and hypothalamus as the main limbic structures of clinical relevance to the practising psychotherapist. We will also touch on the thalamus, which feeds the limbic system with sensory input.

The basal ganglia, a set of subcortical structures located near the thalamus and hypothalamus, are also included in the limbic system and are involved in intentional movements. The limbic system is closely connected to the prefrontal cortex, and it is this prefrontal–limbic connection that is strengthened when practising mindfulness. The functional relevance of the limbic system to psychotherapy is obvious, as affect, memory, sensory processing, time perception, attention, consciousness, autonomic control, motor behaviour, and more are all mediated in and through this collection of structures.

AC Anterior commissure
AN Anterior nucleus of thalamus
DG Dentate gyrus
FR Fasciculus retroflexus
IN Interpeduncular nucleus
LT Lamina terminalis
MB Mammillary body
MD Mediodorsal thalamic nucleus
MF Medial forebrain bundle
MT Mammillothalmic tract
NA Nucleus accumbens
OB Olfactory bulbs
OC Optic chiasm
OL Olfactory striae lateral
OS Olfactory striae medial
OT Olfactory tract
PG Pituitary gland
PT Paraterminal gyrus
SA Subcallosal area
SM Stria medullaris
SN Septal nuclei
SP Septum pellucidum
ST Stria terminallis



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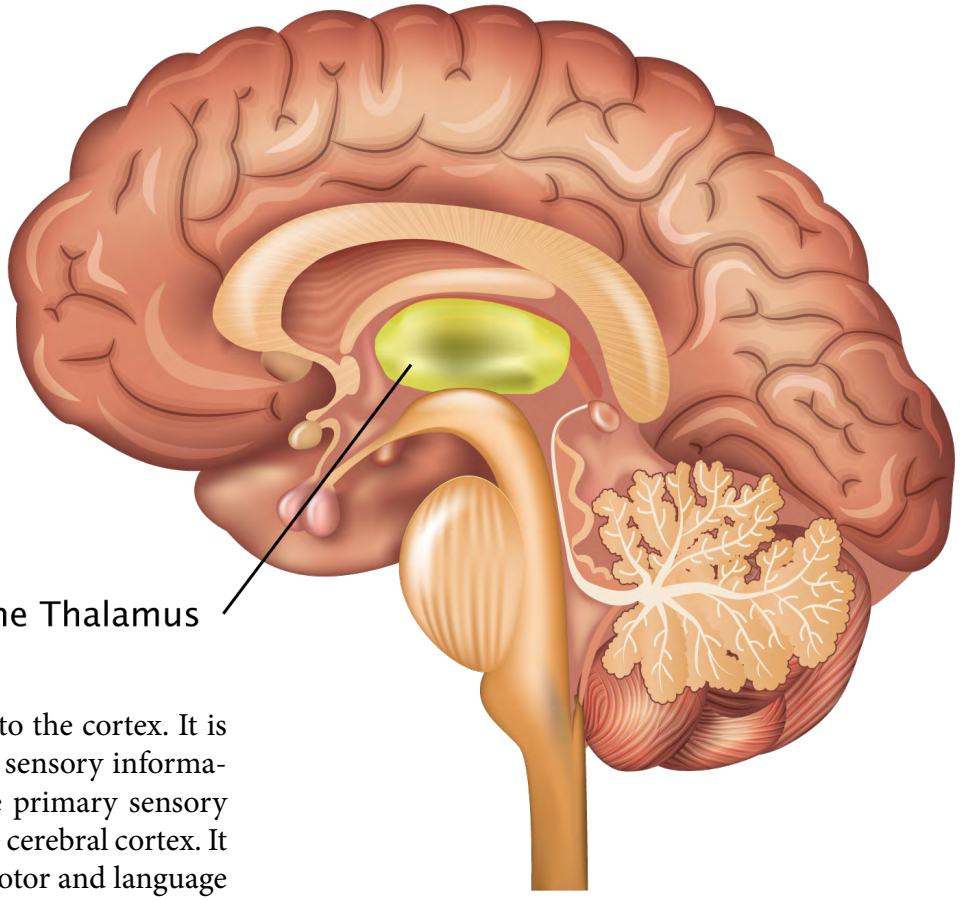
This diagram shows some of the complexity of the limbic system. Note how the olfactory bulbs feed directly into the amygdala, giving smell a particularly important role in emotional memory and evaluation of circumstances—we can smell danger faster than our "smart brain" will recognize a problem, and we can automatically recall the emotions of a past love at the smell of the perfume they used to wear. It is also striking that the size of these structures has little correlation to their power and importance in this amazing system.

THE THALAMUS

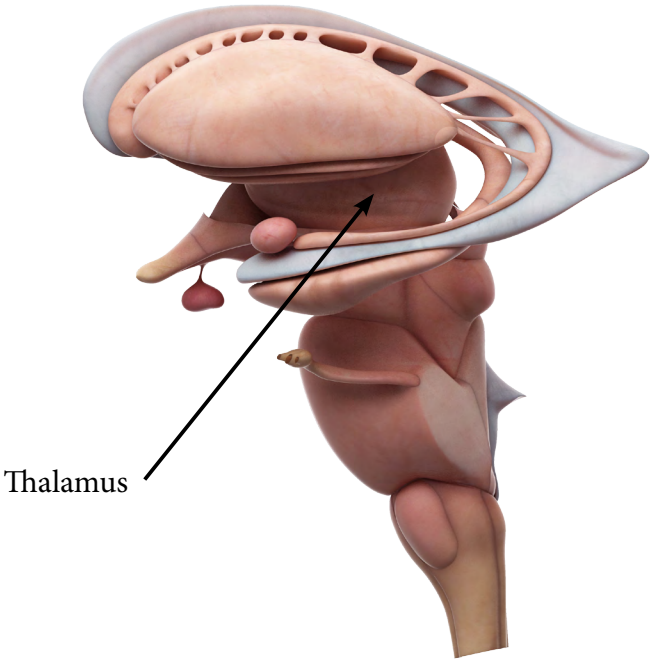


The thalamus (from the Greek word meaning "chamber") is centrally located between the cerebral cortex and the midbrain and is known for its role in relaying sensory and motor signals to the cerebral cortex, and in the regulation of sleep, consciousness, and alertness—rather like a hub of information flow from the senses to the cortex. It is believed that the thalamus processes sensory information in addition to relaying it to the primary sensory areas and receiving feedback from the cerebral cortex. It plays major roles in the support of motor and language systems and (in connection with the hippocampus) the spatial memory that is critical for episodic memory.

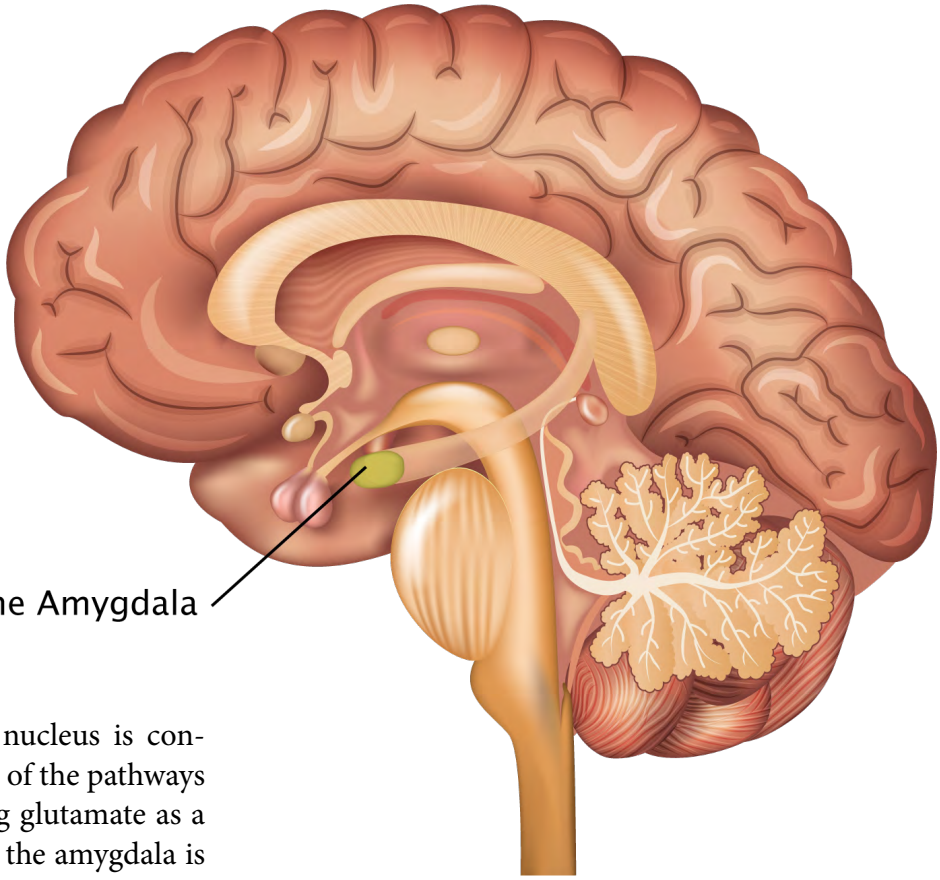
Interestingly, a common genetic variation in humans is that of the serotonin transporter 5-HTTLPR. People who inherit two short alleles (SERT-ss) also have more neurons and larger volume in parts of the thalamus. The SERT-ss inheritance is linked to a greater vulnerability to depression, PTSD, and suicide. The dorsal thalamus also plays a role in the inhibition of compulsive behaviour—we will examine this more closely later, when we consider the so-called OCD loop that involves the orbitofrontal cortex, striatum, and thalamus.

The thalamus is important for sleep regulation, in particular slow-wave sleep cycles, and coordinates parts of the cortex as sleep changes from state to state—the latter in orchestration with the activity of the hippocampus.

In sum, the thalamus appears to play a greater role than merely the relay of sensory information and is integral to brain function in a cortico–thalamo–cortical pathway of processing.

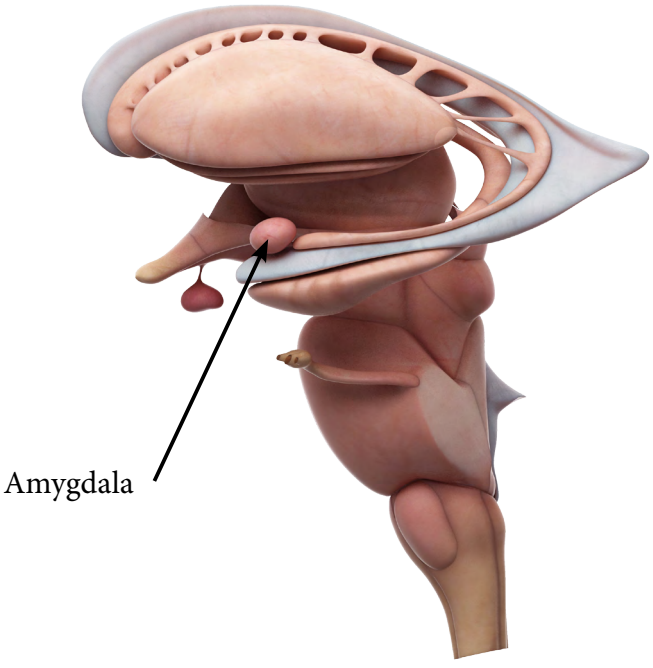


THE AMYGDALA



The amygdala is located in both hemispheres of the brain and is involved in a range of cognitive processes. The lateral amygdala receives input from visual, auditory, and somatosensory systems: the central nucleus is connected with the brainstem that controls innate behaviour and associated physiological responses, while the medial nucleus is connected with the olfactory system. Most of the pathways into the amygdala are excitatory, using glutamate as a transmitter. Information flow through the amygdala is modulated by a number of transmitters, including norepinephrine, serotonin, dopamine, and acetylcholine.

The amygdala forms part of the limbic system. It is most commonly recognized as the emotional processing centre that receives incoming sensory information and processes it for an emotional response. The response may be a defence to a perceived threat, a critical function of this "early-warning system". The amygdala learns how to respond to various stimuli based on its reference to implicit memory and makes decisions on how to initiate an emotional reaction to such stimuli. The emotional memory learned and utilized by the amygdala is episodic–autobiographical memory that can be notably implicit or unconscious, in contrast with explicit or declarative memory processed by the hippocampus.



The left and right amygdalae have separate memory systems, but they work together to evaluate incoming information and process an emotional response, encoding, storing and retrieving memories that are associated with certain cues in the environment. The right amygdala is more strongly associated with nega-

tive emotions such as fear and sadness, whereas the left amygdala has been associated with both positive and negative emotional responses.

The amygdala has an attentional role, focusing our attention on the most important stimuli in the environment. It helps us define a stimulus and primes our immediate response, for example in recognizing a dangerous stimulus and initiating a stress response. Processing social cues (e.g., evaluating faces) is also part of what the amygdala does prior to these cues being processed in higher cortical areas like the medial prefrontal cortex. Evaluating faces (e.g., the trustworthiness of a face) is an important social processing skill that is carried out very quickly in the limbic system. The amygdala also plays a part in processing reward learning and the resulting motivation, and modulating emotional states such as aggression, maternal instincts, and sexual and digestion behaviours as well as attention, perception, and explicit memory—generally thought to be part of processing the emotional significance of what we are encountering. Such processing causes the amygdala to respond with the release of hormones, or neuromodulators, that can alter the cognitive processing in cortical areas as well as activate the body for an appropriate response via the hypothalamus.

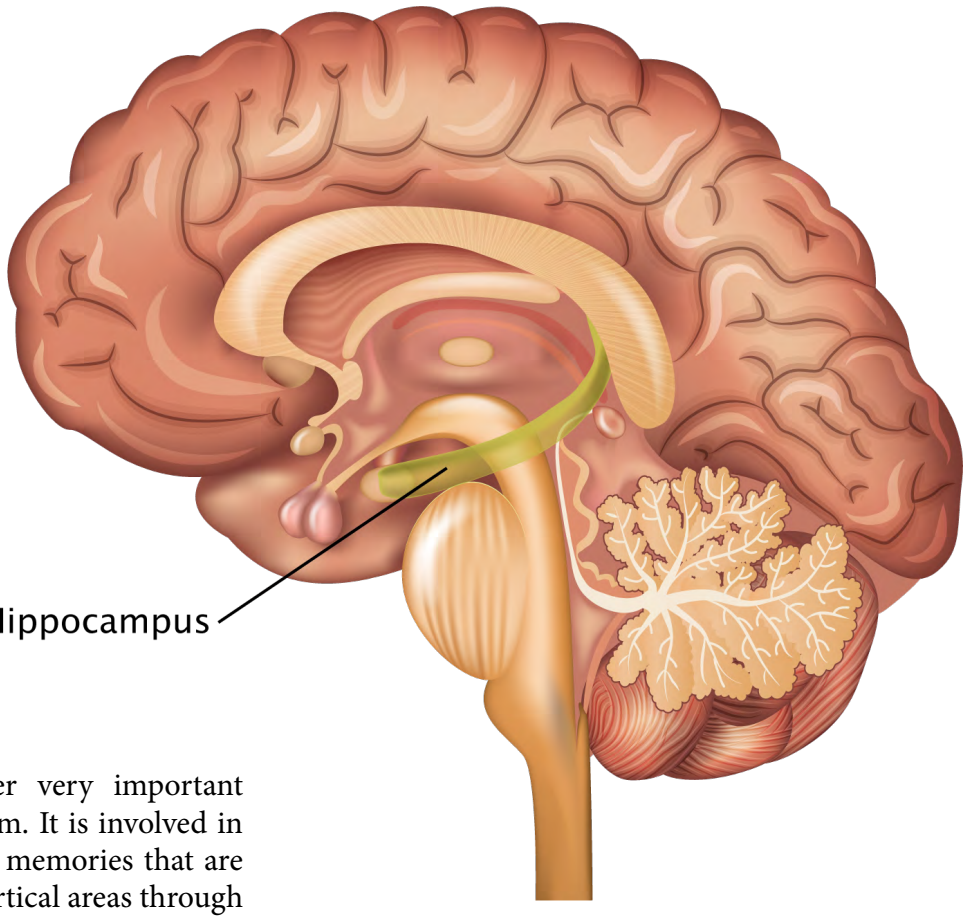
In terms of information flow, sensory input goes to the thalamus and then directly to the amygdala (except olfaction, which goes directly to the amygdala), while it is sent also via a slower path to the neocortex. The amygdala is especially activated by surprising, ambiguous, and uncertain situations or stimuli. If the amygdala perceives a correspondence between the record of experiences in the hippocampus and incoming information, and judges that the stimulus warrants a fight, flight, or freeze response, then it will trigger the hypothalamic–pituitary–adrenal (HPA) axis and "hijack" the prefrontal cortex (PFC), partly in the form of blood flow being redirected from the PFC to the limbic system. This amygdala activity processes information milliseconds earlier than the neocortex, so in the case where implicit memory matches an incoming stimulus, the amygdala acts before any possible direction from the neocortex can be received. On the other hand, if the amygdala does not perceive a match to the incoming stimulus, it acts according to the directions received from the neocortex. This means that when the amygdala perceives a threat, it initiates a response to keep us safe from that threat, although this may not be the most adaptive response. We know a lot more about the fear response of the amygdala than its other functions, but there is likely much more going on in this structure than we yet realize.

In the case of depression, the amygdala is often found to be enlarged from continual hyperactivation, with studies showing that there is an increased metabolic activity in the amygdala of depressed subjects. There is a positive correlation between the degree of amygdala activation and the severity of the depression. The depressive symptoms do not seem to dampen the anxiety-readiness for negative events or the ability to recall negative memories to ruminate upon.

Hypersensitivity and overactivity of the amygdala are at the core of anxiety-based disorders such as generalized anxiety disorder, phobias, PTSD, and other limbic-driven states that inhibit positive, rational (cortical) responses to stressors. Down-regulating amygdala reactivity and the resulting HPA axis stress-response cascade is of primary importance when treating clients suffering from fear-driven conditions. Cognitive therapeutic techniques are of little value to someone who cannot function cognitively (or "be cognitive"), so a bottom-up approach should be employed to bring the PFC back online, as it were. Creating an environment of safety and calm becomes the first step in helping the client regulate their amygdala reactivity. Allison and Rossouw (2013) describe the importance of a safe and controlled environment to effect change:

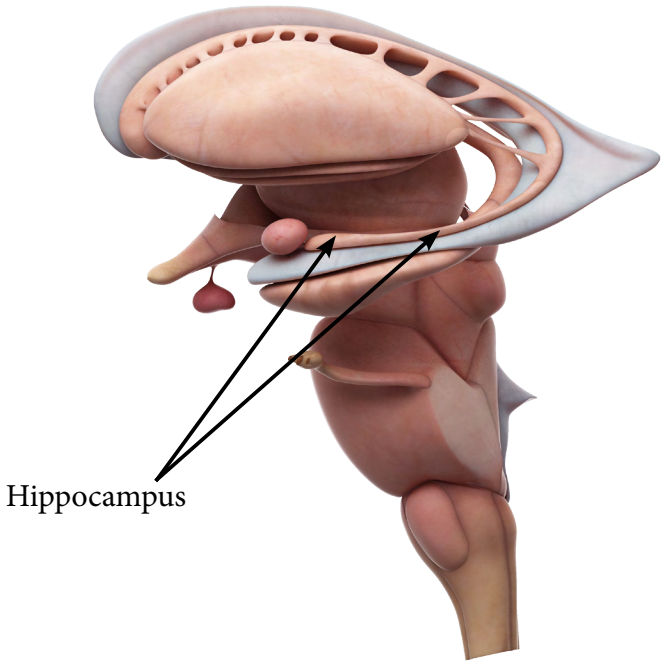
> A therapeutic environment facilitates an enriched safe environment where psychotherapy has the potential to facilitate neural change and proliferation. Safety is essential for people in distress because it down-regulates the hypothalamus–pituitary adrenal system. When the fear response, which is triggered from the pons, amygdala, basal ganglia, hypothalamus, pituitary and adrenal glands, is activated, the distress activates the release of the corticotrophin releasing factor, adrenocorticotrophic hormone, adrenalin and cortisol. If these patterns are activated frequently, the patterns of firing will become well established, resulting in a default neural activation when a trigger is received. Through psychotherapy it is possible to facilitate down-regulation of the stress response system and encourage the development of new patterns of neural activation. Hence it is vital to enable change through the provision of a safe environment in which the individual can experience controlled incongruence, or stress, to prevent activation of the default distress response. A controlled environment is essential; however, if change is facilitated too quickly, the stress signal may be activated and the habitual pathological patterns facilitated. (p. 24)

THE HIPPOCAMPUS



The hippocampus is another very important structure in the limbic system. It is involved in the formation of declarative memories that are processed and transferred to neocortical areas through the process of memory consolidation and, notably, in the contextual anchoring of experience in time and space. The process of how the hippocampus transfers information to the neocortex to consolidate memory may be explained by what is called the hippocampal–neocortical dialogue. Structured interactions between the hippocampus and neocortex happen during slow-wave sleep, when sharp wave patterns dominate the hippocampus and there are bursts of neuron activity in synchrony with state changes in the cortex. This may be part of the process of consolidating memory into long-term state while you sleep.

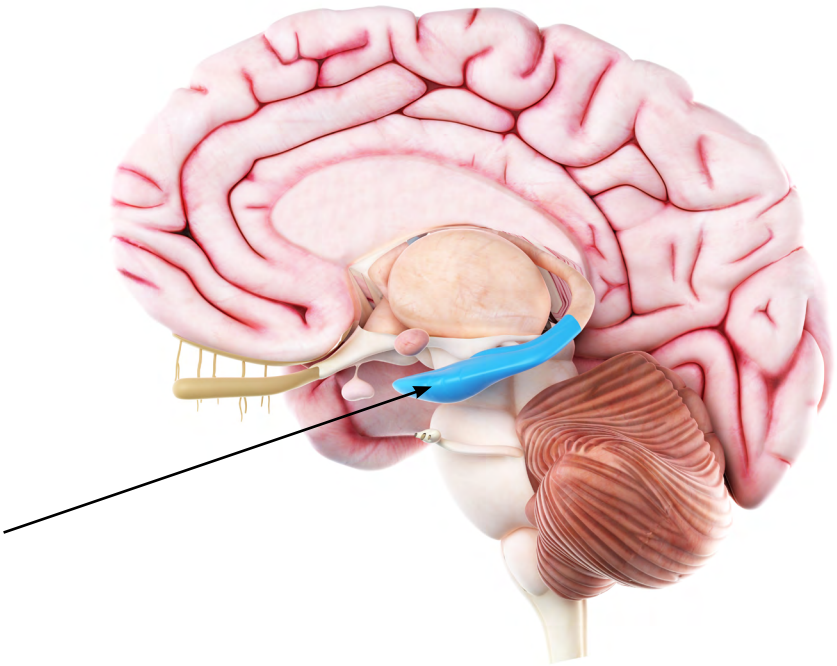
Some researchers conceptualize the hippocampus not as a storage location for memory, but as a control centre that connects areas of the neocortex to activate the effective recall of memory. From this perspective we might think of the hippocampus as the brain's Google search engine, allowing fast and efficient searching of established memories in the neocortex to help assess and plan. A well-known function of the hippocampus is the capacity to learn and retrieve spatial memory: the what, when, and where qualities of an experience.



Researchers have found that neurogenesis—the creation of new neurons—happens in the hippocampus with new learning. There is an upsurge of new neurons and associated circuits when a new task is learned, particularly in the case of physical tasks, and in enriched, stimulating environments. If the hippocampus is damaged, however, this can have significant effects on overall cognitive functioning, especially on spatial memory, and on explicit memory in general. Damage to the hippocampus can occur through prolonged exposure to stress hormones such as glucocorticoids (to which the hippocampus is particularly sensitive), highlighting the seriousness of chronic stress and its physiological impact. Basic feedback mechanisms in the hippocampus that help modulate the release of glucocorticoids during stress may be compromised, compounding the effects of chronic stress by leading to chronically elevated cortisol levels. It has been observed that some people suffering post-traumatic stress disorder (PTSD) have smaller hippocampal volume than the general population, and one study has found that this volume did not necessarily increase after therapy and symptom reduction (Lindauer et al., 2005). A compromised hippocampus may not anchor emotional reactions to a traumatic event to a specific time and space, and such reactions can reoccur inappropriately, as is typical in PTSD.

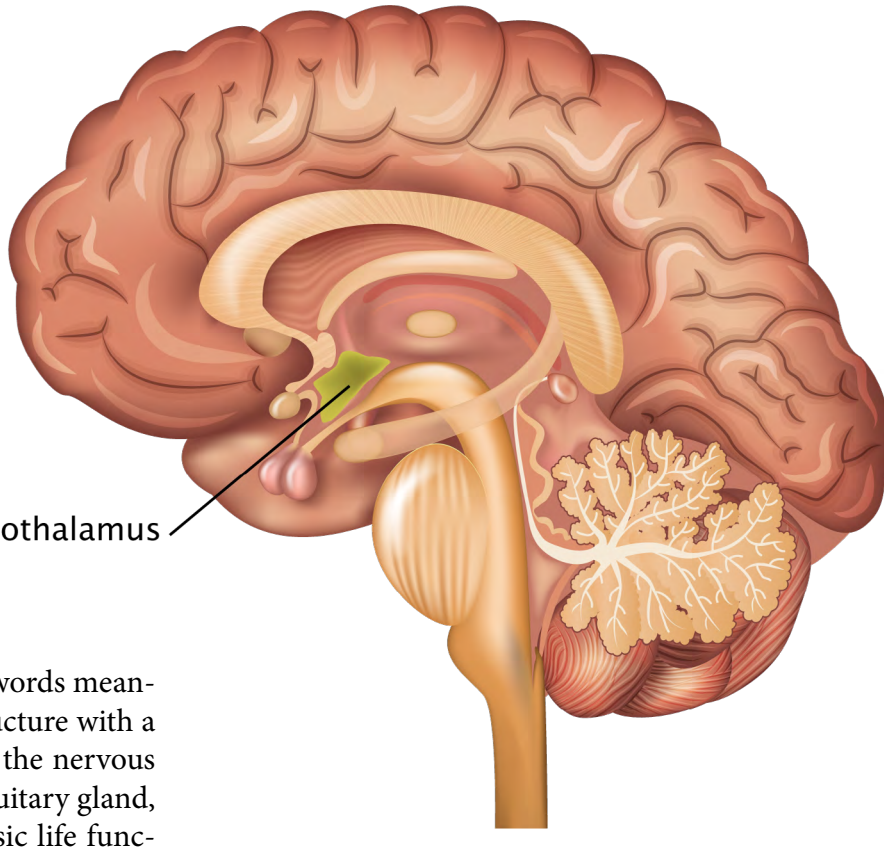
The hippocampus is also implicated in major depression, with findings of hippocampal volume being reduced as much as 8–19% in those with major depression, the reduction in volume being positively correlated with the duration of the depressive state. Similar correlations can be found with bipolar disorder and borderline personality disorder. It is possible that reduced hippocampal volume may be a risk factor rather than a consequence of chronic or excessive stress, so that individuals with a smaller hippocampus will be more prone to fear responses when exposed to traumatic events and less able to regulate that fear response (i.e., to keep the HPA axis stress-response cascade in check) and effectively integrate explicit and implicit memory of the trauma into long-term memory.

Much has been learned about the functions of the hippocampus through the study of a certain subject, Henry Gustav Molaison. Molaison, who in 1953 at the age of 27 had almost all of his hippocampus removed in an attempt to stop epileptic seizures, lost his ability to hold on to new semantic and episodic memories, being unable to consolidate them into long-term memory. He was able to remember what had happened a long time previously, and the older the memory the better he seemed to remember—just the opposite of what is normally observed. Molaison's general knowledge of the world (his semantic memory) was intact, but everything that happened after his surgery seemed new to him: he could only remember new experiences for a few seconds. Even the ability to recognize himself as he aged was lost, although he could recognize himself in old pictures. Interestingly, Henry was able to learn complex motor skills, and retain these skills (what we know as procedural or implicit memory), but was unable to remember learning them (declarative or explicit memory).



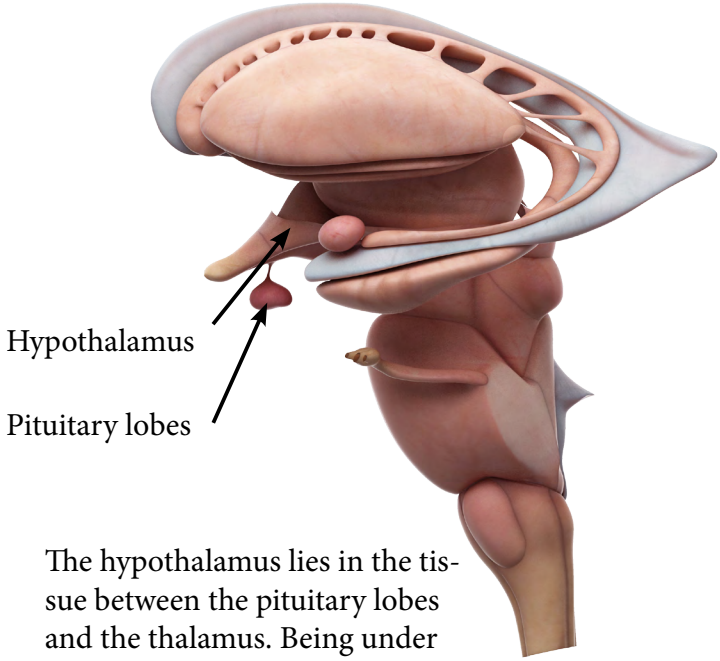
The left side hippocampus is represented here in blue. A compromised hippocampus may not anchor emotional reactions to a traumatic event to a specific time and space, and such reactions can reoccur inappropriately, as is typical in PTSD

THE HYPOTHALAMUS



The hypothalamus (from the Greek words meaning "chamber underneath") is a structure with a variety of vital functions that links the nervous system to the endocrine system via the pituitary gland, for the regulation and coordination of basic life functions. The hypothalamus, as the name would suggest, is located below the thalamus and above the brainstem. It receives sensory inputs that detect changes in both internal and external environments. It receives direct inputs from smell, taste, visual, and somatosensory systems and also senses blood temperature, blood sugar, mineral levels, and a variety of hormones. The hypothalamus is closely connected to other limbic structures such as the hippocampus, amygdala, and cingulate cortex, and thus forms part of the continuum of emotional responsiveness. The medial zone of the hypothalamus is involved in motivated behaviours such as defensive behaviours.

The hypothalamus acts as a control centre for certain metabolic processes and activations of the autonomic nervous system involved in fluid and electrolyte balance, energy metabolism, circadian rhythms, sleep, fatigue, thirst, body temperature, hunger, attachment behaviours (including sexual and reproductive behaviour), to name a few. It synthesizes and secretes certain neurohormones that stimulate or inhibit the secretion of pituitary hormones.

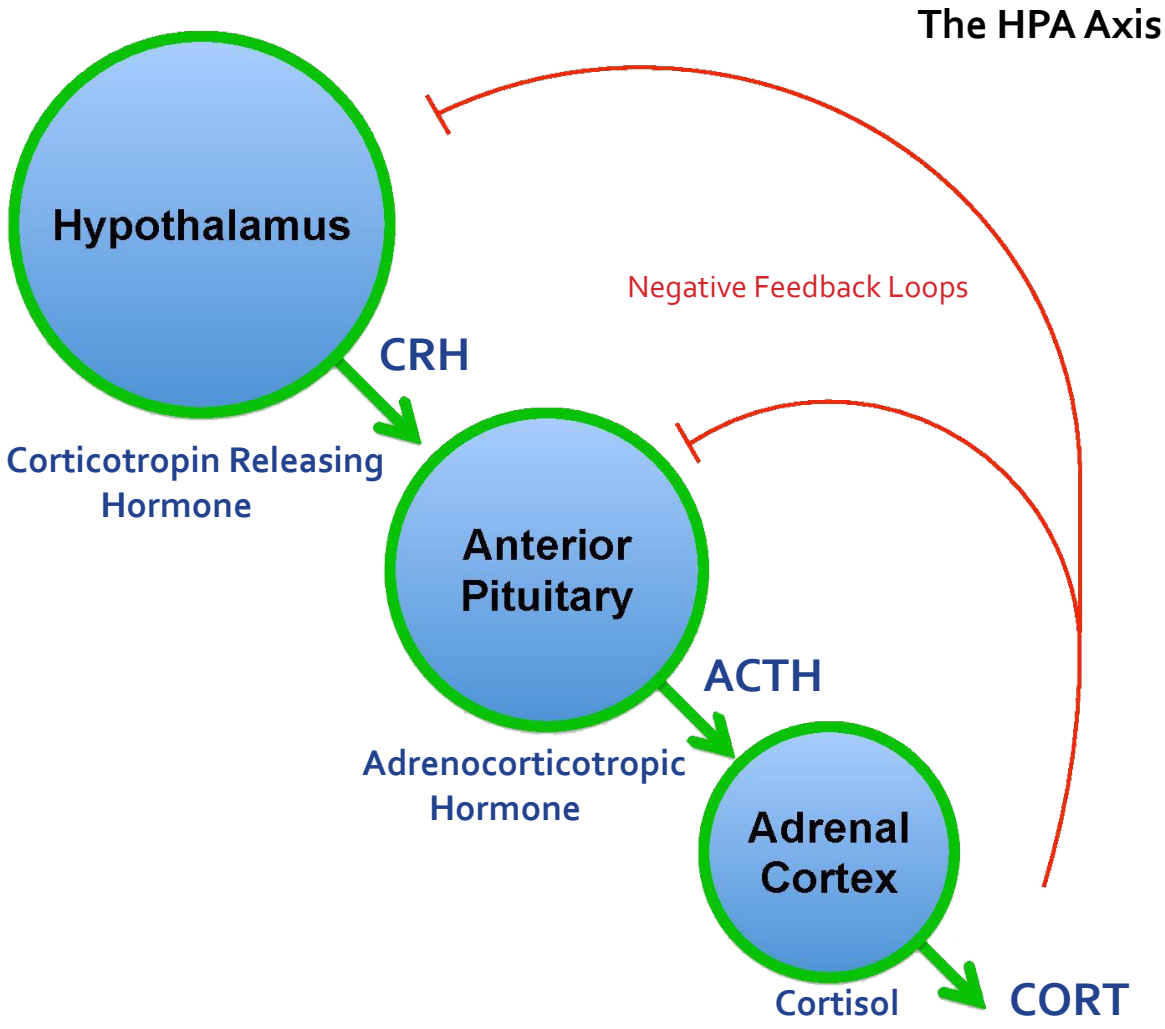


The hypothalamus lies in the tissue between the pituitary lobes and the thalamus. Being under the thalamus it has been named the *hypo*thalamus.

The HPA Axis Response

The hypothalamic–pituitary–adrenal (HPA) axis response is the physiological mechanism that activates the body to respond to a threat. When a threat is detected by the amygdala, a physiological response is mediated through the hypothalamus–anterior pituitary gland. A neurohormone—corticotropin-releasing hormone (CRH) that has been manufactured in the paraventricular nucleus of the hypothalamus—triggers the anterior pituitary gland to release adrenocorticotropin hormone (ACTH) into the blood stream. The ACTH then triggers the release of glucocorticoids such as cortisol from the adrenal cortex above the kidneys. Feedback mechanisms in the hippocampus, hypothalamus and pituitary gland monitor the rising levels of cortisol in the blood and modulate the HPA axis stress-response cascade accordingly.

In the case where someone feels they are dealing with an extreme, uncontrollably stressful situation (such as the severe physical or sexual abuse of a child, or constant life-threatening situations as in war), the feedback mechanisms that dampen the HPA axis stress-response cascade can be damaged, and glucocorticoid levels continue to escalate. These excessive amounts of glucocorticoids can damage activated glutamate synapses and pyramidal cells in the hippocampus (where there are many glucocorticoid receptors), destabilizing previously formed neural connections and thus neural/mental function in the hippocampus. This may be positively correlated with the reduction in volume of the hippocampi of individuals who have suffered extreme stress and been diagnosed with PTSD.



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HPA Axis Response Pathway