Role of amygdala in the experience of fear



The amygdalae (from the Greek for 'almond') are two groups of almondshaped nuclei located deep within the medial temporal lobes of the brain in complex vertebrates, including humans, (see Fig 1 below). Research has shown that the amygdalae perform a primary role in the processing and memory of emotional reactions, and are considered to be part of the limbic system. [pic] Fig 1: Location of Amygdala. (Image from: imemat. blogspot. com) The regions described as amygdalae are a combination of several nuclei with distinct functions. Among these nuclei are the basolateral complex, the cortical nucleus and the centromedial nucleus, (see Fig 2 below). The basolateral complex can be further subdivided into the lateral, the basal and the accessory basal nuclei. Anatomically, the amygdala and more particularly, its centromedial nucleus, may be considered as a part of the basal ganglia. The amygdala sends impulses to various parts of the brain, for example, to the hypothalamus to activate the sympathetic nervous system; to the thalamic reticular nucleus to increase reflex movement; and to the laterodorsal tegmental nucleus for the activation of various neurotransmitters such as dopamine, norepinephrine and epinephrine. The cortical nucleus is involved in the sense of smell and pheromone- processing. It receives input from the olfactory bulb and olfactory cortex. The lateral amygdalae, which send impulses to the rest of the basolateral complexes and to the centromedial nuclei, receive input from the sensory systems. The centromedial nuclei are the main outputs for the basolateral complexes, and are involved in emotional arousal in rats and cats. [pic] Fig 2: Nuclei of the rat amygdaloid complex. (ABmc = accessory basal magnocellular subdivision; ABpc = accessory basal parvicellular subdivision; Bpc = basal nucleus magnocellular subdivision; e. c. = external capsule; Ladl = lateral https://assignbuster.com/role-of-amygdala-in-the-experience-of-fear/

amygdala medial subdivision; Lam = lateral amygdala medial subdivision; Lavl = lateral amygdala ventrolateral subdivision; Mcd = medial amygdala dorsal subdivision; Mcv = medial amygdala ventral subdivision; <math>Mr = medialamygdala rostral subdivision; Pir = piriform cortex; s. t. = stria terminalis). (Image from: Physiol Rev 83: 805) The amygdala filters sensory information and acts as a sort of 'interpretation' channel. The basolateral amygdala receives sensory information from the thalamus and cortex and then forwards a signal to the appropriate target areas (see Figure 3 below). It is also known as the amygdala proper, and the several areas of the brain that it targets are part of a broader network that serves much more specialized functions. Because the basolateral amygdala is critical for emotion, a better understanding of the chemicals within these brain circuits should lead to improved pharmacological treatments for emotional dysfunction in psychiatric disorders. [pic] Fig 3: The basolateral amygdala. (Image from: Current Biology, Vol. 10, (4)) Within most of these disorders is a common symptom in that the patient often says 'I didn't think, I just reacted'. Straker, D. (2006) believes they may be exactly right. All sensory data, with the exception of the sense of smell, is sent by the body first to the thalamus which then forwards it to both the relevant part of the cortex and to the amygdala. The information is sent out over two parallel pathways: the thalamo- amygdala pathway (the 'short route') and the thalamo-corticoamygdala pathway (the 'long route'). The short route transmits a quick estimated representation of the situation, in which no cognition is involved. This pathway activates the amygdala which, through its central nucleus, generates emotional responses before the mind can form a complete representation of the stimulus. The amygdala does a quick threat

assessment by comparing the sensory data received with already stored fear responses. If any of these are triggered, then the amygdala floods the cortex with chemicals to stop it taking over. The result is action without conscious thought. (See Fig 4 below). Subsequently, the information that has travelled via the long route and been processed in the cortex reaches the amygdala and tells it whether or not the stimulus represents a real threat. Should a real threat be presented the amygdala will then activate the efferent structures responsible for physical manifestations of fear, such as increased heart rate and blood pressure, sweaty hands, dry mouth, and tense muscles. The parallel operation of our explicit (hippocampal) and implicit (amygdalic) memory systems explains why we do not remember traumas experienced very early in our lives. At that age, the hippocampus is still immature, while the amygdala is already able to record unconscious memories. Early childhood traumas can disturb the mental and behavioural functions of adults by mechanisms that they cannot access consciously. In complex vertebrates, including humans, the amygdalae perform primary roles in the formation and storage of memories associated with emotional events. Amunts et al (2005) indicate that, during fear conditioning, sensory stimuli reach the basolateral complexes of the amygdalae, particularly the lateral nuclei, where they form associations with memories of that particular stimuli. These associations between stimuli and the aversion may be mediated by long-term potentiation, a lingering potential for affected synapses to react more readily. Memories of emotional experiences that become imprinted in the reactions of synapses in the lateral nuclei produce fear behaviour through their connections with both the amygdalae's central nucleus and the bed nuclei of stria terminalis (BNST). These central nuclei are involved in the

production of many typical fear responses, including freezing (immobility), tachycardia (rapid heartbeat), increased respiration, and stress-hormone release. Damage to the amygdalae impairs both the attainment and the expression of Pavlovian fear conditioning, which is a form of classical conditioning of emotional responses.

[pic]Fig 4: The Amygdala Bypass System. (Image from: www. changingminds, org) Advances in neuroimaging technology such as fMRI, have allowed neuroscientists to show just how much of a role the amygdala plays in many psychological disorders. Donegan et al. (2003) studied patients with Borderline personality disorder who showed significantly greater left amygdala activity than the normal control subjects. Some of these borderline patients even had difficulties classifying neutral faces or classed them as being threatening. In support of these findings, in 2006, researchers at Monash University, Australia, observed increased levels of activity in the amygdala when patients with social phobia were shown images of threatening faces or when they were confronted with frightening situations. These activity levels in the amygdala were in direct correlation with the severity levels of the social phobia. Similarly, depressed patients showed more activity in the left amygdala when interpreting emotions for all faces, and especially for fearful faces, although this hyperactivity was normalized when patients were prescribed antidepressants. Cultural studies such as Williams et al (2006) showed that normal subjects exposed to images of frightened faces or faces of people from another race will show increased activity of the amygdala, even if that exposure is subliminal. However, according to Tsuchiya et al (2009), the amygdala is not necessary

for the processing of fear-related stimuli, since people with bilateral damage show rapid reactions to fearful faces. Early research on primates has also provided explanations for the functions of the amygdala in relation to emotional disorders. An early study by Brown & Shafer (1888) observed rhesus monkeys with a lesioned temporal cortex (including the amygdala) and found that they suffered from significant social and emotional deficits. Kluver & Bucy (1939) later expanded upon this observation by showing that large lesions to the anterior temporal lobe produced not only fearlessness, but also severe emotional disturbances including increased sexual behaviour and a propensity to place objects in their mouths. Some monkeys also displayed an inability to recognize familiar objects and would approach both animate and inanimate objects indiscriminately, while also exhibiting fearlessness towards the researchers. This behavioural disorder was later named Klüver-Bucy syndrome. However, their study can be criticised in that these lesions were so large and crude when compared to today's techniques, that researchers weren't exactly sure of the structures responsible for these significant changes in behaviour. Improved techniques, such as using the neurotoxin ibotenic acid to make more precise lesions are partly responsible for the more detailed understanding of the amygdale today. | | | [pic] | | Fig 5: Sensory data routes, the fear response and the amygdala. (Image from: http://thebrain. mcgill. ca/flash/index a. html) Previous studies have examined activation of the amygdala in response to emotional facial stimuli, but these have been carried out in either the U.S. or Western Europe, although none of these explored cross-cultural differences. Although culture shapes several aspects of human emotional and social experience, including how fear is perceived and expressed to others, very little is known about how

culture influences neural responses to fear stimuli. In response to this gap in the research, a study by Chiao et al (2008) found that the bilateral amygdala's response to fear faces is, in fact, modulated by culture. Using fMRI, they measured the amygdala's response to fear and non-fear faces in two distinct cultures, Native Japanese in Japan and Caucasians in the United States. Both culture groups showed greater activation in the amygdala to fear expressed by members of their own culture, (their in-group), than in any of the other emotional measures such as anger, happiness or neutrality. (See Fig 6 below). [pic] Fig 6: The amygdala's response to fearful facial expressions is culture- specific. (Image from: Chiao et al 2008). As mentioned earlier, sensory data, apart from the sense of smell, is sent by the body to the thalamus and then forwarded to both the cortex and the amygdala. In relation to this sense of smell, when faced with a threatening situation, many organisms, including insects, fish and mammals, release volatile pheromones, signalling the danger to other members of the same species. Nearly 70 years ago, Karl von Frisch (1941), described the alarm response in a species of small freshwater fish called the European minnow (Phoxinus phoxinus). Frisch, who was one of the founders of the scientific study of animal behaviour, demonstrated that when a minnow was eaten by a predator, a chemical released from its damaged skin would be reacted to by other minnows that were close by. They would at first dart about randomly, form a tight school and then retreat from the source of the chemical. Frisch called this substance schreckstoff, meaning 'scary stuff', and we now know that similar chemicals are used throughout the animal and plant kingdoms. A team of researchers from the University of Lausanne in Switzerland (Brechbuhl et al, 2008) have shown that mice detect alarm

pheromones by means of a recently identified sensory system in the nose by examining a structure called the Grueneberg ganglion (GG), which in mammals is located on both sides in the tip of the nose, close to the openings of the nostrils. When the GG was first discovered by Hans Grueneberg in 1973, its anatomy was not known in such detail and so it was thought to be a non-sensory structure. It is only very recently that the olfactory system has come to be viewed as containing 3 distinct 'channels', each with a unique structure and function. The main channel is involved in detecting aromatic molecules; the second channel is called the vomeronasal system, and is an 'accessory' olfactory system which is now known to be involved in the detection of pheromones; the GG constitutes a third component of the olfactory pathway, one that was thought to be involved in mother-pup recognition and suckling behaviour, because it is present at the time of birth. The researchers sought to investigate the role of the GG in behaviour. Because of its location, the GG is easily accessible, so they were able to cut the axons of GG neurons in live mice (axotomy), thus preventing any signals from reaching the brain. But after numerous tests for nipple finding and other possible functions, the team actually found that the ganglion played a role in danger communication. [pic] Fig 7: Scanning electron microscope images of the mouse Grueneberg ganglion. Left: a cluster of neurons (GC) in a meshwork of fibroblasts (Fb) Right: and a higher magnification of a single GG neuron (green), with its axon (red) and thin ciliary process (blue). Scale bars: 20 microns (L) and 5 microns (R). (Image from: Brechbuhl et al, (2008)). 30 days after the axotomy, the researchers then compared how mice with and without their Grueneberg ganglia responded to alarm pheromones. According to Broillet, the contrast was very

striking. Normal mice with the ganglia showed fear immediately by ' freezing' while mice without the ganglia seemed to be unaffected and they carried on as before, apparently unaware of the danger signals that affected the normal mice. Although their sense of smell did not seem to be affected as they were able to sniff out cookies hidden in their cages as well as the normal mice. This study clearly shows that in mice the GG is involved in detecting alarm pheromones, rather than in mother-pup interactions, as was previously thought. It is able to perform this primitive function thanks to a specialized yet very basic structure as the GG consists simply of a small group of cells separated from the external environment by a waterpermeable sheet of epithelial cells. Its location, far away from the main olfactory system, enables rapid detection of alarm pheromones. Such a mechanism is crucial an organism's survival rate, and the GG is found in every mammalian species examined so far, including humans. However, whether or not alarm pheromones affect, or even exist in humans, has been a subject for debate in the scientific community. Since pheromones are not detectable by the human sense of smell, scientists believe that pheromones are sensed by the vomeronasal organ (VNO), part of the olfactory system and located inside the mouth or nose. For many years, the existence of the VNO produced much speculation because it had only been found occasionally in adult humans, and when it was found, it was believed to be vestigial. However, Johnston et al, (1985) conducted a study in which the noses of 100 human adults were examined post-mortem and the VNO was found in the septums of 70% of those examined. Since then, much evidence has been gathered to support these findings of a presence of the VNO in most adult humans, but many scientists still believe it to be a functionless

organ that was inherited from some ancestor of humans. However, recent genetic research has shown the possibility of a receptor in the nose that could sense pheromones. When searching the human genome for genes that had similar sequences to those of rodent pheromone receptors, a team of researchers from The Rockefeller University in New York and the Yale University School of Medicine identified for the first time a candidate pheromone receptor gene in humans. The findings, reported in Nature Genetics, may shed new light on the molecular basis of social communication between humans, including the fear response. In conclusion, despite the saying, ' have no fear', to live without the ability to experience and recognise fear is to be deprived of a vital neural mechanism that enables appropriate social behaviour, and possibly even survival.