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Amygdala and Emotional Learning in Vertebrates – A Comparative Perspective

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1. Introduction

The mammalian amygdala is a telencephalic structure of complex embryonic origin, composed of several nuclei that proceed from both the telencephalic pallial wall and the striated sheet, settling a border or transition zone between the telencephalic pallium and subpallium. In primates, the amygdala is located in the anterior temporal lobe, and in other mammals occupies a topologically equivalent position. Klüver & Bucy (1939) described a syndrome after bilateral ablation of the temporal lobes of rhesus monkeys which, among other symptoms, caused a dramatic decrease in the emotional reaction of fear, an effect attributed primarily to the ablation of the amygdaline system rather than other structures. This effect sparked interest in studying the possible involvement of the amygdala in emotional responses, such as learning and emotional memory that has been developed since the second half of the 20th century. In fact, bilateral lesions to the amygdala (or its equivalent anatomical structure) in other vertebrate groups (reptiles, birds, and non-primate mammals) cause serious interference with the normal development of emotional responses characteristic of the species such as aggressive, sexual, or parental behavior (Kling & Brothers, 1992). Studies about emotional and avoidance (active or passive) conditioning show that the complete or partial injury to amygdala causes serious disorders in these learned responses (LeDoux, 1995). Moreover, the amygdala is involved in memory and attention processes (Kapp et al., 1992), in the emotional evaluation of stimuli (Davis, 1992; Gallagher & Chiba, 1996; Gallagher & Holland, 1992; Halgren, 1992; LeDoux, 1992, 1995) and in emotional aspects of dreams (Calvo et al., 1987). In the past, its intricate structure and the paradoxical experimental results that sometimes were obtained in both humans and animals (Phelps & Anderson, 1997) have been especially confusing when trying to clearly identify its functions. Although, a common factor found in these data is its emotional aspect. It has now become clear that it is also involved in humans' social learning of fear and in anxiety disorders (Olsson & Phelps, 2007). In fact, it appears that stimuli that are not

consciously perceived activate the amygdala; this provides a fast and automatic processing of the stimuli for social cognition (Adolphs, 2009), especially with regard to facial expression (Adolphs et al., 1994).

As stated above, the amygdala is not a uniform structure, not only in terms of the structural differences but also by the intrinsic and extrinsic pattern of afferents and efferents of its different nuclei, including the divisions of the subnuclei (Amaral et al., 1992; Pitkänen et al., 1997), which also appears to be involved in different functions for different circuits. Lesion and stimulation studies may lead to varying results depending on the affected circuit, but always shows implicit emotional factors (Davis, 1992; Gallagher & Chiba, 1996; Gallagher & Holland, 1992; Killcross et al., 1997; LeDoux, 1992). In the human case, the amygdala could be considered an essential part of the brain involved in surveillance tasks of potentially ambiguous or unpredictable stimuli (Whalen, 1999).

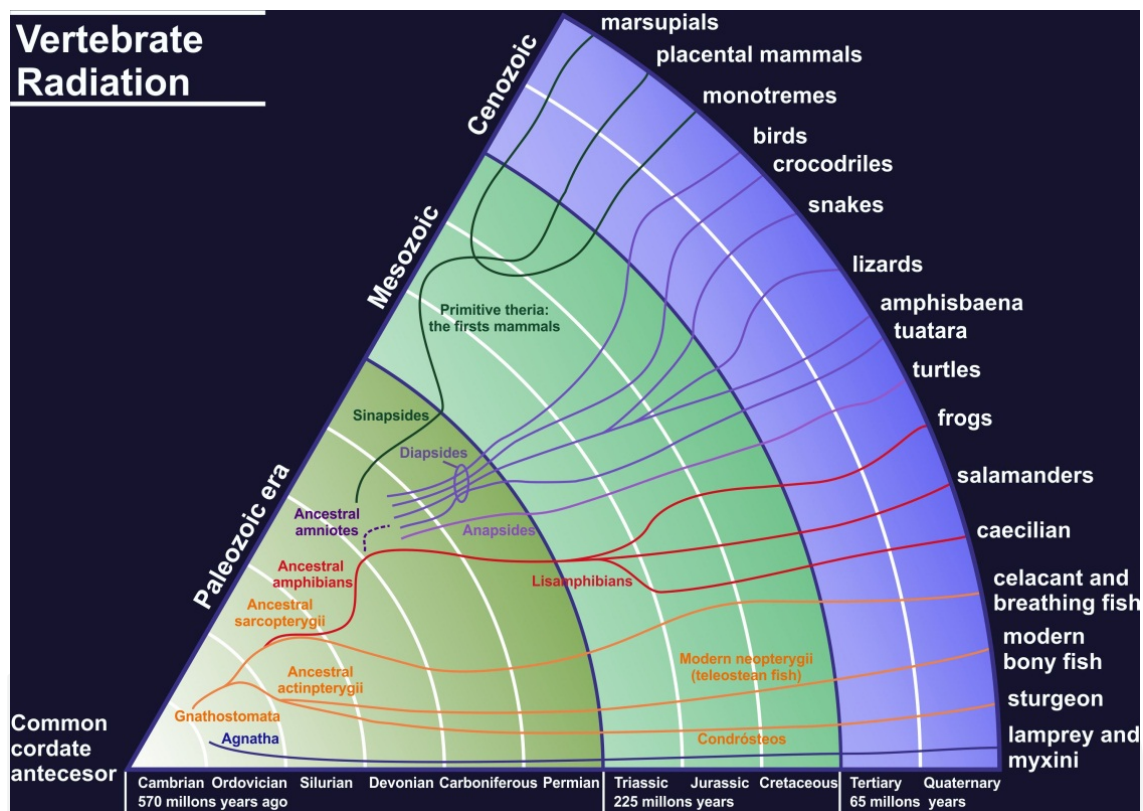


Figure 1. Phylogeny of vertebrates represented in a cladogram. Although the first fossil records are from the Ordovician period, the existence of vertebrates in the Cambrian is very likely. Fishlike vertebrates gave rise to the agnatha (jawless fish) and gnathostomes (jawed fish). The current vertebrates descended from one of these two groups. Gnathostomes were diversified into different classes, such as reptilians, birds, and mammals. Current bony fishes present brain structures with a cytoarchitecture and function very close to the amygdala in mammals. This data indicates that most likely a common ancestor to vertebrates had a brain with a structure functionally similar to the amygdala, and because of its adaptive value, it was conserved throughout the phylogeny.

It is considered that the amygdala is not an exclusive structure of mammals, since genoarchitectonic, neurochemical, embryological, anatomical, and behavioral studies in a

comparative perspective (Aggleton, 1992; Bruce & Neary, 1995; Macphail, 1996; Medina et al., 2011; Moreno & González, 2006; 2007; Parent, 1986; Sriedter, 1997; Wulliman & Rink, 2002) have proposed several homologies for this structure in other vertebrate groups (fish, amphibians, reptiles, and birds). Although there has been no complete agreement on the homology of all parts of this structure (pallial and subpallial portion) among all vertebrate groups over time (Braford, 1995; Bruce & Neary, 1995; Northcutt, 1995; Sriedter, 1997), the existence of a basic scheme of the amygdala in fish, reptiles, birds, and mammals, irrespective of the diverse differentiation of this structure along the evolution of the different groups of tetrapods has been established by examining the comparative genoarchitecture (Medina et al., 2011; Moreno & González, 2006; 2007). In this sense, it is possible that the amygdala or analogous structure was present in an ancestor of current vertebrates. Its function was necessary for the adaptative processes, and it was conserved along the vertebrate radiation (figure 1).

2. The amygdala. Homologies among vertebrates

As noted previously, the amygdala of mammals and other tetrapods is divided into two main parts, one pallial or cortical, and another subpallial or striated. In addition, it is divided into different nuclei, and these divisions also seem to be found in groups other than mammals, such as reptiles and birds (Bruce & Neary, 1995; DeOlmos et al, 1985; Lohman & Smeets, 1990; McDonald, 1992; Medina et al., 2011; Moreno & González, 2007; Northcutt, 2008; Striedter, 1997; Wulliman & Rink, 2002; Zeier & Karten, 1971). In the case of chondrichthyan fish the so-called Nucleus A is recognized as a possible homologue of the tetrapod pallial amygdala, both for its position and its pattern of connectivity (Northcutt, 1995; 2008; Smeets, 1990). In actinopterygians, the telencephalon undergoes an eversion process that raises a serious difficulty in establishing homologies with the amygdala (Braford, 1995; Gage, 1983; Scalia & Ebbesson, 1971). However, the cytoarchitecture, the neurohistochemistry, studies of embryonic development, the pattern of connectivity and topological studies (Braford, 1995; Chanconie & Clairambault, 1975; Morgan, 1974a, 1974b; Murakami et al., 1983; Northcutt, 1981; 1995; Northcutt & Braford, 1980) suggest that the region Dmv of the teleost telencephalon is a good candidate to be considered the homologue of the pallial amygdala (Northcutt, 1995; Northcutt & Braford, 1980), although other authors suggest that Dmv could be homologous to the basal ganglia or the hippocampus of mammals (Murakami et al., 1983; Parent et al., 1978). Moreover, the pattern of connectivity, with strong sensory thalamic inputs and reciprocal connections with the hypothalamus, gives a distinctly limbic character to Dmv (Braford, 1995; Ehteler & Saidel, 1981; Ito et al., 1986; Murakami et al, 1983; Striedter, 1991). Another circumstance that would support the homology of Dmv with the pallial amygdala is its topological situation. The Dmv telencephalon is located between the ventral subpallial area (possible striated region) and the dorsoposterior area (main olfactory receptor area), a similar position to the pallial amygdala of other vertebrates, between the olfactory cortex and the subpallial amygdala (Braford, 1995; Bruce & Neary, 1995; Neary, 1990; Neary & Bruce, 1993; Northcutt & Kicliter, 1980). Other authors (Kyle & Peter, 1982; Kyle et al., 1982) have pointed to the region Vs-

pVv (ventral area supracomissural ventral-ventral area) as homologous to the corticomedial amygdala, based on the fact that it receives olfactory afferents (Oka, 1980), the concentration of steroid hormone receptors (Kim et al., 1978; see Medina et al., 2011 for a review of steroid hormone-regulated as a component of the amygdala), and the results of electrolytic lesions (Kyle et al., 1982). This data is also supported by functional data as damage to this structure affects the male reproductive behavior and other signs of social interest such as parental care or aggressive behavior, the same pattern of deficits that is found with lesions of the corticomedial amygdala in mammals (Isaacson, 1976; Kling & Brothers, 1992). In fact, it has been proposed that the most medial region of the ventral area of the telencephalon in teleosts fish correspond to the subpallial amygdala (Northcutt, 1995; 2008; Northcutt & Braford, 1980).

3. Functions of the amygdala in emotional learning and memory in mammals

3.1. Amygdala and fear conditioning

The conditioning of the fear response in short delay procedures is affected by the lesion of specific areas of the amygdala, mainly the central, basal, and lateral nucleus (Davis, 1992; LeDoux, 1992; 1995). This conditioned fear response is established very easily and this characteristic seems to be preserved through evolution in all vertebrate groups (LeDoux 1992; Portavella et al, 2004a), and consists of three components: behavioral (eg usually appears a "freezing" response), autonomic (eg bradycardia response) and hormonal (eg increased levels of ACTH) (Davis, 1992). In the human case, the involvement of the amygdala in fear conditioning has been shown by both consciously and unconsciously processed stimuli (Morris et al., 1989). Thus, the lesion of the amygdala produces a serious effect on the ability to recognize expressions of fear (Adolphs et al., 1994; 2005), and thus interferes seriously in the early detection of emotional stimuli. Recent studies using functional imaging techniques have analyzed the role of different parts of the human limbic system in the processing of emotional discrimination of faces and natural scenes (Fusar-Poli et al., 2009; Sabatini et al., 2011)

To better understand the role of the amygdala in fear conditioning, it is necessary to know how the main efferents and afferents of the amygdalar structures are organized and their intrinsic connections (Amaral et al., 1992; Pitkänen et al., 1995; Pitkänen et al., 1997; Savander et al., 1995). In fact, the study of the function and the connectivity go hand in hand, since not all lesions nor interventions have the same effects in different amygdaloid nuclei; although some pallial nuclei (lateral, basal and accessory basal nuclei) and subpallial (central nucleus) appear to be more decisive in this type of conditioning (see LeDoux, 2000 for a review). In mammals, the lateral nucleus receives direct thalamic afferents from different sensory modalities (eg intralaminar nuclei, supragenulate nucleus and medial division of the medial geniculate nucleus), so it receives somesthetic, visual, and auditory thalamic afferents (Campeau & Davis, 1995; Gallagher & Chiba, 1996; Rogan & LeDoux,

1995). The lesion of this nucleus produces a severe deficit in the expression of conditioned fear response, and also an inability to learn it (see Rosen, 2004). Also specific neurotoxic lesions in the basolateral complex (lateral and basal nuclei) prevent the animal from acquiring a conditioned fear response (Campeau & Davis, 1995; Rabinak & Maren, 2008). Inputs from cortical regions to the lateral nucleus send processed information of the same stimuli (sensory, association, and polymodal cortex), through a thalamo-cortico-amygdala via (for a review see Sah et al., 2003). The importance of the thalamus-amygdala via is proven by the fact that ablation or disconnection of sensory cortical region does not prevent fear conditioning to a single sound stimulus (DiChara et al., 1970; LeDoux et al., 1986). However, when the set of stimuli is more complex (such as in a program of differential conditioning where there are two different sounds as stimuli or a reinforced conditioned stimulus (CS +) and a non-reinforced one (CS-), and the animal during the training and reversal has to learn which one is paired with the EI (electric footshock), the lesion to this circuit disrupts the performance on this type of differential learning tasks (Jarrell et al., 1987). It has also been shown that the context can become a fundamental cue for learning. This contextual conditioning has been established when for example, the fear response occurs without the presence of the CS or the US (unconditioned stimulus), by merely placing an animal in the box in which it has been receiving these two paired stimuli. In this case, the presence of certain structures such as the hippocampus and related structures is essential, because the lesion of this structure is sufficient to block the fear response to the context (Nadel, 2008; Phillips & LeDoux, 1992; Selden et al., 1991; Wiltgen et al., 2006). Lesions of retrosplenial cortex (Keene & Bucci, 2008) and of the entorhinal cortex (Ji & Maren, 2008) also disrupt contextual fear conditioning. The entorhinal cortex is implicated in processing background contextual information given that NMDA lesions impair the processing of contextual background fear conditioning but not the fear conditioned to an auditory stimulus (Majchrzak et al., 2006). The hippocampus maintains connections to the lateral and basal nucleus via subiculum and with the basal nuclei (Ottersen, 1982). In this sense, the mere disconnection of this afferent or lesion to the hippocampus is enough to prevent and suppress emotional conditioning context (Maren & Fanselow, 1995; Phillips & LeDoux, 1992). All available data point to the lateral nucleus as a nucleus of sensory input to the amygdala (Rogan & LeDoux, 1995), which according to LeDoux (1995) could act as an interface and a major site for integration of information from the different inputs of sensory information during the conditioning of the fear response. The afferents of the lateral nucleus are directed to other amygdalar structures including the pallial basal and basal accessory nuclei too (Pitkänen & Amaral, 1991).

The basal nucleus (basal and basal accessory) receives input from the hippocampus and cortical association areas such as inferotemporal cortex and other structures (Amaral et al., 1992), and sends efferents to the sensory association cortex and the central nucleus (Aggleton, 1985; Amaral et al., 1992; Price & Amaral, 1981). Electrolytic or NMDA-induced lesion of the basolateral complex of the amygdala blocked fear conditioning to both visual and auditory CSs (Campeau & Davis, 1995). The use of substances that produce temporal inactivation such as muscimol (a GABA type A agonist) has introduced some controversy,

since some studies show that injecting this substance in the basal and lateral nuclei produced different results depending on the time of injection. If the drug is injected before conditioning it results in acquisition deficits, although the same manipulation made immediately after conditioning has no effect (Helmstetter & Bellgowan, 1994). Nevertheless, with a similar design Muller et al. (1997) showed that injecting muscimol to functionally inactivate the basal or the lateral nucleus produced little conditioned fear to contextual or auditory conditioned stimuli. Herry et al. (2008) and Amano et al. (2010) have also shown that inactivation of the basal nuclei with muscimol does not block the expression of the conditioned fear. Fanselow & Kim (1994) did the same with the local infusion of d-APV (a NMDA receptor antagonist), obtaining similar results. Cousins & Otto (1998) analyzed the relation between the fear response to olfactory and contextual stimuli. The results showed that excitotoxic lesions (infusion of high concentration of NMDA) disrupted the conditioned response to odor and context. All the data supports the involvement of the amygdala in the acquisition of a conditioned fear response, such as in its expression, once conditioning is established.

The central nucleus, in contrast, has essentially extra-amygdala efferents, and maintains important connectivity with brainstem nuclei and areas, such as the periaqueductal gray matter, and lateral hypothalamus (Davis, 1992), which in turn control the response patterns that involve the emotional response of fear. In fact, the neurotoxic lesion of this structure impairs the learning of a conditioned fear response (Campeau & Davis, 1995).

3.2. Plasticity in the amygdala and fear conditioning

In rats, the plastic processes that take place in amygdala structures during learning of fear conditioning have been studied on the presence of postsynaptic long-term potentiation (Clugnet et al., 1990; Sah et al., 2008). Some of these studies found that these processes were not mediated by NMDA receptors (Chapman & Bellavance, 1992), as occurs in the hippocampus (Bliss & Collingridge, 1993; Hicks, 1995; Lynch et al., 1991; Madison et al., 1991; Malenka & Nicholl, 1993; McEntee & Crook, 1993; Morris et al., 1989; Witkin, 1995). Nevertheless, later studies found the existence of long-term potentiation processes mediated by NMDA receptors located in the lateral amygdala (Gean et al., 1993). Furthermore, these receptors located in the lateral amygdala are implicated in the processing of the conditioned stimulus in fear conditioning (Farb & LeDoux, 1997; Li et al., 1996). The use of antagonists of these receptors causes serious interference in the conditioning of the fear response to simple stimuli as the context, but not retention or expression of a learned response (Fanselow & Kim, 1994; Gerwitz & Davis, 1997; Maren et al., 1996). “In vivo” and “in vitro” studies show how fear conditioning induces a process of long-term potentiation (LTP) mediated by NMDA receptors (Rogan et al., 1997; McKernan & Shinnick-Gallagher, 1997). It has also been shown that NMDA receptor blockade interferes with the process of extinction of a conditioned fear response (Falls et al., 1992). These results and their relationship to learning processes are parallel to those found in the hippocampus, and indicate that a long-term depression (LTD) also mediated by NMDA receptors, which has been linked to the extinction of responses acquired in other structures like the hippocampus, takes place in the

amygdala too (Dudek & Bear, 1992). In fact, it has been proposed (LeDoux, 1995), that the convergence of sensory information of the conditioned and the unconditioned stimuli in the amygdala would follow Hebbian patterns (Hebb, 1949) and it would affect other areas involved in the process of emotional conditioning. In the last 20 years, a great amount of evidence (Johansen et al., 2011) showed that two kinds of molecular mechanisms are responsible for fear conditioning in the amygdala, specifically in the lateral amygdala: hebbian plasticity and neuromodulatory processes. In this sense, studies of fear conditioning to auditory stimuli suggest the existence of response potentiation in the lateral nucleus after learning. In addition, these plastic changes have also been observed in the auditory cortex. This change would be mediated by the circuit thalamus - lateral nucleus - basal nucleus - central nucleus - nucleus basalis of Meynert, since this latest nucleus sends a strong cholinergic innervation to the cerebral cortex (Price & Amaral, 1981; Russchen et al., 1985). It is very probable that this circuit has a key role in producing fast brain activity rhythms of low voltage (Bukasi et al., 1988). These results has been verified in studies that analyzed the involvement of the central nucleus of the amygdala in brain activation (Kapp et al, 1984; 1990; 1992) showing that activation of the amygdala and the prosencephalic basal nucleus produced cerebral asynchrony mediated by this cholinergic innervation. Therefore, the neuromodulatory processes in the glutamate amygdalar circuits (Ferry & McGaugh, 2008; Johansen et al., 2011) are mediated by monoamine transmitters (adrenaline, norepinephrine, and dopamine). The consolidation of this learning is dependent on plastic changes of amygdala synapses. The synthesis of a broad amount of proteins underlies consolidation of fear conditioning, such as receptor glutamate subunits, calmodulin, protein-kinases, and signaling pathway (see Helmstetter et al., 2008).

Experimental results show that the specific lesion of a thalamic region (that sends its efferents to the amygdala as the medial division of medial geniculate nucleus) can prevent the animal to be conditioned to a stimulus of that sensory modality (auditory), but not to another of different modality, such as visual (Campeau & Davis, 1995). On the other hand, different results show that the thalamo-amygdala and the thalamo-cortico-amygdala pathways are equipotential with the facilitation of classical conditioning to a simple stimulus (Johansen et al., 2011). These data indicate that there may be multiple and multi-modal representation of the CS-US associations in the amygdalar structure and the intrinsic circuits for fear conditioning (Gallagher & Chiba, 1996; LeDoux, 1995).

The neural system proposed by several authors for fear conditioning (Davis, 1992; LeDoux, 1995; Pitkänen et al, 1997) gives a central role to the amygdala as a receiving center of different information about stimuli, such as features of the stimuli (thalamus-lateral nucleus), perceptual phenomena (sensory association areas-lateral nucleus), spatial location and/or contextual and explicit memory (hippocampus-parvocellular division of the basal and lateral nucleus), polymodal representations (polymodal association cortex-lateral nucleus), and proprioceptive information (hypothalamus-medial nucleus). In this way, the response elicited by the circuit through the central nucleus would be induced by multiple representations of the existing stimulus in various structures, including the amygdala (Johansen et al., 2011).

3.3. Amygdala and avoidance conditioning

The studies on the involvement of the amygdala in avoidance learning performed between the decades of the 50's and the 70's were somewhat confusing (Brady et al., 1954; Campenot, 1969; Caruthers, 1969; Fonberg, 1973; Grossman, 1972; Grossman et al., 1974; Isaacson, 1976; Kaada, 1972; Kling et al., 1960; McGaugh & Gold, 1976; Pellegrino, 1968; Ursin, 1965). After a critical reading of these works many of the lesions performed in the different studies affect more than one area, the procedures were not exactly comparable to one another, and it was unclear what the animals were really learning and in what context. However, while some results appear contradictory, it seems that it can be establish the differential involvement of some of the amygdaloid nuclei in the different types of avoidance behavior: passive, discriminative active -lever press- or one-way and two-way active discrimination (Ambrogli et al., 1991; Fonberg, 1973; Grossman et al., 1974; Handwerker et al., 1974; Horvath, 1963; Killcross et al., 1997; Liang & McGaugh, 1983; McIntery & Stein, 1973; Roozendaal et al., 1997; Sanchez-Riolobos 1986; van-der-Zee et al., 1997; Werka et al., 1978; Wiersma et al., 1998). The contradictory results mentioned above could be due to the fact that the amygdala has been considered a functional structure instead of a set of functional subsystems. The most important issues to consider when interpreting the different results on different types of avoidance behaviors are the result of overtraining, and hence the degree of consolidation of learning, individual differences (high-level avoidance animals and low-level avoidance animals within the same strain of animals), and of course, the verification of experimental procedures.

In addition, the similarity of lesions between different species, and defining which structures are affected are important data to analyze, since it has been shown that all these factors can dramatically influence the effects on acquisition or retention of this learned avoidance responses on apparently similar procedures as is exposed below.

3.4. Amygdala and olfactory conditioning

Fear conditioning studies have been directed primarily to analyzing the relationship between visual/auditory cues and the US, with little attention to olfactory cues. Studies with these types of cues are relatively recent although olfactory cues are of great evolutionary importance for laboratory animals such as rats. As with visual and auditory cues, studies that have used odors as CS associated with a foot-shock have recorded a strong emotional response, showing even greater resistance to extinction processes (Richardson et al. 2002). Electrophysiological studies also showed that the synaptic modifications in the BLA are dependent on the contingency between CS and US (Rosenkranz & Grace, 2002). Furthermore, this plasticity is also dependent of NMDA receptors during the acquisition process and is dependent of AMPA during recovery (Walker et al. 2005).

In addition to the BLA, other areas like the mPFC, perirhinal cortex and hippocampus specifically influence the expression of activity of the amygdala during fear conditioning odor. While the perirhinal cortex is essential for the initial processing of odor stimulus (Herzog & Otto, 1998), CA1 and CA3 of the hippocampus have been linked to the encoding

and retrieval of context information (Hunsaker & Kesner, 2008). The subiculum is the principal structure linking the hippocampus with the entorhinal cortex and many other cortical and subcortical areas. Specifically, the v-Sub may act as an interface between the hippocampus as a contextual information processor and cortical and subcortical processing systems related to motivation such as the ventral striatum and amygdala (Floresco et al., 2001; Quintero et al., 2011; Traverso et al., 2010). Parsons & Otto (2008) showed that the inactivation of dorsal hippocampus using an olfactory contextual conditioning, caused acquisition and retrieval deficits. In addition, neurons from mPFC are essential to acquire the association between CS and UC. These neurons show strong activity to foot-shock odor association. This activity is dopamine dependent, given that selective blockade of dopamine receptors reduces emotional response to the CS (Laviolette et al. 2005).

3.5. Effects of lesions to the amygdala

Kaada (1972) postulated that there were two different regions in the amygdala for the control of active avoidance behavior in rats, the defense amygdalar region, and the basolateral complex. Partial or total lesion to the defense amygdalar region (in rats and cats including the ventral part of the internal capsule and the ventral amigdalofugal pathway and pallial and subpallial structures), suppressed defense responses including the escape response (Blanchard & Blanchard, 1972; Kemble et al., 1984; 1990; Ursin et al., 1981). So, in their view it would have to block active avoidance responses in a classical active avoidance response paradigm. In contrast, lesion to the basolateral complex, which had inhibitory influences, impairs passive avoidance behavior but it does not affect the performance in an active avoidance paradigm behavior. Several experimental results support this view: in cats, both the electrolytically lesion of the anterior portion of the lateral nucleus and the interruption of the amigdalofugal ventral pathway produced a severe deficit in active avoidance conditioning, but not in passive avoidance (Ursin, 1965). Also, electrolytic lesion to the central nucleus and to the adjacent and dorsal regions to the basolateral complex in cats affected the retention of a two-way active avoidance response, but it did not affect one way active avoidance nor passive avoidance (Horvath, 1963). Similar data were obtained in rats (Pellegrino 1968). In this study, a lesion to the basolateral complex produced a severe disability for the maintenance of a passive avoidance response. Also, in mice, a large electrolytic lesion produces a severe deficit in both active and passive avoidance responses (Takashina et al., 1995). According to Werka et al. (1978) a lesion to the lateral cortex and central and lateral nuclei described deficits in the retention of one-way active avoidance. Killcross et al. (1997) demonstrated a process that allowed them to separate two responses related to fear conditioning (a conditioned suppression response to an aversive stimulus and an active avoidance response to the harmful stimulus) that lesions to the central nucleus affect the maintenance of the conditioned suppression response, but not the acquisition of the active avoidance response. In contrast, lesions to the basolateral nucleus produced the opposite effect. This latest result would establish a parallel processing of two types of fear dependent response, indicating that in the amygdala there are different mechanisms and different areas for processing the stimuli and the fear response. This idea would lead to a

more critical discussion about the nature of avoidance responses, but what concerns us is that the amygdala is establishing an important relationship with this type of learning. Other studies present conflicting results, for instance Brady et al. (1954), showed that a large electrolytic lesion to the amygdala impairs cats to acquire an active avoidance response, but if the lesion was done after the learning, it did not affect the maintenance of the response. Similar results were obtained with passive avoidance procedure after bilateral electrolytic lesions (Liang et al., 1982). Also, in rats the lesion to the central nucleus produced a deficit in the acquisition but not in the retention of the response (Davis, 2000; Grossman et al., 1974; McIntery & Stein, 1973). In addition, different molecular processes seem underlying to acquisition and retention in central amygdala. While the inhibitory consolidation processes are under control of NMDA and cannabinoid CB1 receptors (Ghiasvand et al., 2011), serotonergic system is involved in the modulation of retention in the passive-avoidance task (Schneider et al., 2003). Another set of results have raised deeper questions about the involvement of various amygdaloid nuclei, where the amygdaloid cortex lesion severely affected the acquisition of one and two-way active avoidance response. In contrast, lesions of the cortical, medial, central, intercalated, lateral, and basolateral nucleus did not affect active avoidance, although lesions to the central, intercalated, and basolateral nucleus impair passive avoidance learning (Grossman et al., 1974). A recent electrophysiological study shows that the expression of conditioned freezing depends on increased activity levels of the medial region of the central amygdala but it does not depend on the activity of the lateral region (Duvarci et al., 2011). These differences in results could be due to various reasons (e.g. lesion differences, collaterally affected areas, use of different strains, etc). Another cause is the effect of overtraining, since according to Fonberg et al. (1962) it prevents the effects of the lesion on the amygdala. At the same line, Thatcher & Kimble, (1966) showed no effect on the retention of a learned avoidance response after overtraining. In contrast, a recovery deficit is observed in absence of overtraining (Goldstein, 1974; Thatcher & Kimble, 1966). It is possible to observe a very similar case in inhibitory avoidance. Reversibly inactivation or lesion to the amygdala affects retention if it is done soon after the training, but has no effect if it is performed much later (Liang et al., 1982; McGaugh et al., 2000; Parent & McGaugh, 1994; Wilensky et al., 2000; 2006).

However, although the amygdala is believed to be essential for the acquisition of Pavlovian fear conditioning, studies using excitotoxic lesions have recently called this view into question. Thus, different nuclei of the amygdala could contribute to the modulation of memory consolidation of an avoidance response (active or passive) in a different way:

- a. Pallial amygdala. Lesions to the basolateral nucleus (lateral, basal, and accessory basal) produce severe deficits in the acquisition of both active and passive avoidance, although the effects on retention are lower in the passive than in the active (Ambrogli et al., 1991). The same lesions have no effect on the conditioned response nor in a passive avoidance response with contextual cues (Selden et al., 1991). A previous study comparing the effects of electrolytic lesions to those produced by ibotenic acid showed that only the electrolytic lesions by radiofrequency impairs for an active avoidance response (Jellestad & Cabrera, 1986). Nevertheless, this study did not define what

amygdalar structures were indeed damaged. Moreover, it postulates the idea of the involvement of a pathway through the nucleus instead of the cell groups present in those structures.

- b. Subpallial amygdala. Lesions to the central nucleus produce deficits in both an active avoidance response (Riolobos & Garcia, 1987) and a two-way active avoidance (Sánchez-Riolobos, 1986).

All these data together support the differential involvement of pallial and subpallial nuclei in active avoidance learning (McGaugh et al., 2000; Wilensky et al., 2000; 2006).

3.6. Effects of electrical stimulation

Early studies showed that electrical stimulation of the amygdala induced amnesia for different learning processes (Grossman, 1972; Isaacson 1976; Kaada, 1972) included avoidance learning. The specific stimulation of the central nucleus, immediately after training in an avoidance task, produced a large deficit in the retention of that task and a blockade of fear conditioning (Gold et al., 1975; McDonough & Kesner, 1971). The same results were observed in the retention of passive avoidance tasks (Gold et al, 1973a; Kesner, 1982), and in a one-way active avoidance after unilateral stimulation in the basomedial nucleus (McGaugh & Gold 1974). Gold et al. (1973) showed that 1 hour after training, stimulation produced a deficit in the avoidance response, but 6 hours later produced no effect in the same avoidance test. This data suggests that there is a plastic phenomenon related to learning, which would be disrupted by the stimulation shortly after training, but not once its effects are consolidated. Other studies also described amnesic effects in both one-way avoidance and discriminated avoidance (press a lever) after post-training stimulation (Hanwerker et al., 1974; McGaugh & Gold, 1976). Nevertheless, other studies have found memory facilitating effects after stimulation in passive avoidance tests with a weak shock (Gold & Van Buskirk, 1975). In other studies, a low intensity shock showed similar results in tasks such as conditioned emotional response. Lidsky et al. (1970), Gold & McGaugh (1975), and McGaugh & Gold (1976) proposed that the different motivational state caused by a severe or a mild shock condition would produce the differential effect of electrical stimulation. Subsequent studies have exposed as the amnesic effects of electrical stimulation can be modulated by hormone levels, which would support this hypothesis. The removal of the adrenal gland (decline on systemic levels of catecholamines) showed that electrical stimulation does not induce amnesia but showed an improvement in the retention of an inhibitory avoidance response in both active and passive condition (Bennett et al., 1985). Also, in a similar study, Liang et al. (1985) injected norepinephrine immediately after training and just before intraamygdaline electrical stimulation. Animals without adrenal medulla showed the same deficits as the control animals, which could determine a modulation of systemic norepinephrine on memory through or in connection with the amygdala.

In fact, we know the involvement between the amygdala (mainly central nucleus) and the CHR (corticotrophin releasing hormone) - ACTH (adrenocorticotrophic hormone) - corticosteroids cascade and norepinephrine release, and its correlation with the response of

fear, anxiety and stress (Davis 1992). Several studies have linked the conditioning of stressful situations, amygdalar lesions and intraamygdaline inoculation of systemic ACTH or CHR. Bush et al. (1973) showed that an electrolytic lesion of the amygdala produced extensive damage to a passive avoidance response, and this effect was reversed by systemic injection of ACTH. Krivanek (1971) showed that injection of norepinephrine, pentylentetrazole (GABAergic antagonist) and ACTH produced a facilitation of avoidance behavior. Moreover, in an experiment with adrenalectomized rats, intra amygdalin electrical stimulation produced an amnesic effect in active and passive avoidance in control animals, while animals without adrenals improved their performance and retention in these tasks (Bennett et al., 1985). Another series of studies have combined individual differences in avoidance tasks and hormone action in the amygdala. Wiersma et al. (1998) determined that the animals of the RHA strain (high rate of avoidance) and the RLA (low avoidance rate) had different behavioural, physiological, and neurobiological responses under stress-free conditions after CRH microinfusion into the central nucleus of the amygdala. RHA increased heart-rate activity and decreased resting only in a stressful situation (inescapable shock) while a slight behavioral activation was observed in RLA. These results show a correlation between the ability to learn avoidance tasks, intra-individual differences, and hormonal regulation in the amygdala in an emotional context.

3.7. Social and neuroimage studies of primates and humans

In the studies of mammals (see above) it appears that the amygdala is involved in normal (LeDoux, 2012; Parkes & Westbrook, 2011; Phelps & LeDoux, 2005) and pathological (Magdaleno-Madrigal et al., 2010, Roozendaal et al., 2009) emotional processing. In primates including humans it integrates more complex functions of social and cognitive nature: such as anxiety disorders (Holzchneider & Mulert, 2011; Kim et al., 2011), decision making (Seymour & Dolan, 2008) or fear, and social learning (Olsson & Phelps, 2007). In species with a highly complex social environment, like in the human case, the amygdala has a crucial role. Neuroimaging studies has been shown that the amygdala functions as a relevance detector that allows humans to perceive incongruence in the emotional expressions, or fear (Phelps & LeDoux, 2005), although others argue that the amygdala activation is more indirect in these cases (van der Gaag et al., 2007). In humans, the amygdala shares common functions with other species, and also acquires more subtle properties in the management of emotional content information that can be shared at least in part with other social primates (Pessoa & Adolphs, 2010), in which circuit model "low road" and "high road" of emotional processing becomes more complicated in terms of increased complexity of sensory processing and perception in humans and primates, taking in this case a greater involvement of the cortex. However, there is a general conservation of function, with an implementation of new features that accompany human neural development itself (Adolphs, 2009; LeDoux, 2012; Olsson & Phelps, 2007; Pessoa & Adolphs, 2010). Likewise, in the pathological aspect of emotion, fMR studies have shown correlated changes in volume of the amygdala and secretion of cortisol in psychiatric disorders such as unipolar depression (Schuhmacher et al. 2012) and other behavioral disorders as described in other chapters of the present book.

4. Function of the amygdala homologous in non mammals vertebrates

Lesions or stimulation of the amygdala in reptiles and birds (or structures considered homologous to the amygdala) produce changes in social behavior or learning processes (Martínez-García et al, 2007). These changes are similar and show a comparable function to the amygdalar system of mammals. In the case of crocodilian reptiles (*Caiman sclerops*), lesions of the amygdala results in decreased frequencies of aggressive patterns (Keating et al., 1970). Likewise, lesions to the areas considered homologous to the amygdala in a iguanide in a social environment (*Sceloporus occidentalis*) caused a syndrome similar to that of mammals as it produced deficits in attention and response (loss of initiative), in patterns of dominance and submission, and a decrease in the response to fear stimulus (Tarr, 1977). Reproductive and aggressive behaviors, as well as associated seasonal variations, have been the main object of study in snakes (*Thamnophis sirtalis parietalis*). These studies show that the sphericus, considered homologous to the amygdala in these reptiles (Bruce & Neary, 1995; Sriedter, 1997), presents sexual dimorphism which is more evident of the males (Crews et al., 1993). Moreover, when the nucleus sphericus is lesioned before the start of the stage of hibernation, there is a facilitation of courtship patterns in the male after the hibernation that correlates with increased blood levels of androgens, indicating a potential facilitating effect of the lesion (Krohner & Crews, 1987).

Zeier & Karten (1971) have argued that the posteromedial portion of the archistriatum, now called arcopallium (The Avian Brain Nomenclature Consortium, 2005), is the homologous to pallial amygdala (basolateral amygdala) in birds. The experimental results support the homology proposal (Dafters, 1975; Goodson & Bass, 2001; Lowndes & Davies, 1994).

In the case of the turtle dove (*Streptopelia risoria*), it is known that electrical stimulation of archipallium produces emotional responses of fear, offense, and defense (The Avian Brain Nomenclature Consortium, 2005; Vowles & Beasley, 1974). In addition, lesions to this structure abolish the conditioned emotional response (as the cardiac acceleration that occurs paired with a shock) in *Columba livia* (Cohen, 1975, Dafter, 1976). When an active avoidance is used, the arquipallial lesion produces the same deficit for the acquisition of a avoidance response that the lesion of the mammalian amygdala (Dafter, 1975). In the case of active avoidance, the arquipallial lesion produces the same deficit in the acquisition of the avoidance response that lesion of the mammalian amygdala (Dafter, 1975). Phillips & Youngren (1968) showed that unilateral excitotoxic lesions of this structure in chicks that are 5 days old showed a decrease in distress vocalizations (the chirp). Anatides have a similar effect, since in the mallard (*Anas platyrhynchos*) lesions to archipallium produce a generalized effect as defined by author of "domestication" or put in another way, lack of fear of emotional reactivity (Phillips, 1964; The Avian Brain Nomenclature Consortium, 2005). The same effect occurs in *Columba livia* after lesions of the posterior portion of archipallium. In contrast, lesions to the anterior portion facilitate the emotional reaction of fear (Zeier, 1971). In a study in chickens (*Gallus gallus*), Maser et al. (1973) showed that lesions to the anterolateral portion produced an increase in reactive immobility typical of fear responses.

In psittacidae (*Agapornis roseicollis*), lesions of the medial part induces a decrease of fear, and it facilitates the approach toward dangerous stimuli of the animal (Phillips, 1968). These results seem to clearly differentiate two portions of the birds' medial archipallium, with opposite functions from the point of view of control of emotional responses. This is corroborated by the results of a study of a fringillidae, the zebra finch (*Taenopygia guttata*). In this study the activity of brain areas was measured by the uptake of 2-deoxyglucose-C14, after subjecting male animals to various stressful circumstances (Bischof & Herrmann, 1986). One group was housed alone in a cage. Animals with experience in courtship were assigned to a second group. A third group of subjects did not have experience in courtship and a fourth group was subjected to a situation in which the experimenter pretended to try to catch them in their cages. The analysis showed a high rate of uptake in the caudal portion of archistriapallium in the group without experience in courtship and the one of capture inside the cage: the two most stressful situations. Also lesions to the taenia nucleus of the amygdala (homologous to part of the medial amygdala of mammals) seriously affect the socio-sexual behavior of male Zebra finches (Ikebuchi et al., 2009).

In the case of teleosts fish, the results from the experiments of telencephalic lesions and stimulation that produce alterations in reproductive and aggressive behaviors (de Bruin, 1980; Kyle & Peter, 1982; Kyle et al., 1982) present a picture of behavioral changes that can be easily compared to changes mediated by the amygdala function. For instance, the agonistic behavior of male Betta fish has been widely studied. The sight of another male typically stimulates a series of agonistic behavioral displays toward the intruder often followed by physical aggression. The context used in these studies is known as agonistic context. The partial ablation of the dorsomedial telencephalon (Dm) in *Betta splendens* produces a loss of reactivity (facilitation of habituation) in an agonistic context (Marino-Neto & Sabbatini, 1983). However, lesion experiments often extend the damage to the basal portion of Dmv. If we admit the existence of telencephalic structures with limbic function equivalent to those of tetrapods, we would also have to admit that lesions in different limbic areas produce similar effects in fish and tetrapods.

The results of ablation studies in teleost fish show that the Dmv region, proposed as homologous to the pallial amygdala of land vertebrates, has a key role in Pavlovian conditioning. These studies showed a clear lesion effect in both the acquisition and maintenance of a two-way active avoidance behavior (Portavella et al., 2004a, 2004b; Portavella & Vargas, 2005). Thus, lesions to the Dmv region produce a deficit equivalent to those described in the case of complete ablation of the telencephalon in earlier studies in Pavlovian conditioning (Aronson, 1948; de Bruin, 1977; 1980; Fiedler, 1967; 1968; Hale, 1956; Kamrin & Aronson, 1954; Karamyan et al., 1967; Kassel & Davis, 1977; Kassel et al., 1976; Noble, 1939; Noble & Bourne, 1941; Overmier & Gross, 1974;; Segaar, 1961; 1965; Segaar & Nieuwenhuys, 1963; Ribbink, 1972). Also, the deficit showed in both the acquisition and retention in avoidance behavior cannot be attributed to the lack of activity or initiative of the animal after lesion, because the level of escape is very high. In addition, the absence of effects on spatial memory or motor response (Portavella & Vargas, 2005) rules out a possible

functional homology with the hippocampus and basal ganglia of tetrapods (Braford, 1995; Braford et al., 1992; 1993; Ehteler & Saidel, 1981; Murakami et al., 1983; Nieuwenhuys & Verrijdt, 1983; Northcutt, 1995; Northcutt & Braford, 1980; Parent, 1986; Parent et al., 1978). Thus, we concluded that the lesion produced a deficit in the associative process between the discriminative stimulus and the shock (Portavella et al, 2002; 2004a, 2004b; Portavella & Vargas, 2005; Vargas et al., 2009). Other results in fear context conditioning have showed that lesions of the dorsomedial telencephalic portion in *Betta splendens* produced a facilitation of habituation to the context (Marino-Neto & Sabbatini, 1983) and variations in levels of aggression (de Bruin, 1980). Taken as a whole, these data indicate that the Dm telencephalon area shares a great similarity with pallial amygdaloid nuclei and homologous structures in reptiles (Crews et al., 1993; Keating et al., 1970; Krohner & Crews, 1987; Tarr, 1977), birds (Cohen, 1975; Dafter, 1975; 1976; Ikebuchi et al., 2009; Martínez-García, et al., 2002; Phillips 1968; Phillips & Youngren, 1968; Vowles & Beasley, 1974; Zeier, 1971), and mammals (Ambrogli et al, 1991; Fonberg, 1973; Grossman et al., 1974; Hanwerker et al., 1974; Horvath, 1963; Killcross et al., 1997; Liang & McGaugh, 1983; McIntery & Stein, 1973; Pellegrino, 1968; Roozendaal et al., 1993; Sanchez-Riolobos, 1986; Takashina et al., 1995; van-der-Zee et al., 1997; Werka et al., 1978; Wiersma et al., 1998). These amygdala lesions affect not only the avoidance conditioning, but also the fear conditioning in mammals and birds (Cohen, 1975; Dafter, 1976).

One of two main approaches on the theories of avoidance in fish (Flood et al., 1976; Zhuikov et al., 1994) is a model adapted from the theory of the two processes of Mowrer (1947). This model recognizes the existence of a two processes. One of them is the conditioning process of a motivational state (fear). That is crucial to the Pavlovian conditioning. The other one is the association between the CS and the shuttling response (Zhuikov et al, 1994). Given these results, it could be postulated that the lesion in the dorsomedial region deprives the fish of such capacity, and it would be one of the causes of the main deficit for the maintenance of avoidance behavior (Portavella et al, 2002; 2004a, 2004b; Portavella & Vargas, 2005; Vargas et al., 2009).

5. Conclusion

Learning theories for the function of the amygdalar system are not an organized body of various alternatives, but show a set of nuclei that receive any type of sensory or perceptual relevant information inducing changes in the neuroendocrine system, and the emergence of emotional patterns mediated by this structure (autonomic, humoral, and behavioral). This set of nuclei would in turn form a central part of the emotional learning processes (Aggleton, 1992; Davis, 1992; Gallagher & Chiba, 1996; Gallagher & Holland, 1992; Halgren, 1992; Killcross et al., 1997; LeDoux, 1992; 1995; Olsson & Phelps, 2007; Phelps & Anderson, 1997). Likewise, though being intrinsically a very complex structure (Amaral et al., 1992; Pitkänen et al., 1997), it is clear that lesions of a large part of the nucleus of the pallial amygdala (basolateral amygdala) and also the subpallial part seriously affect all type of emotional learning. In fish, the ablation of Dmv telencephalon induced a clear deficit in a conditioned avoidance behavior, similar to that produced after a complete ablation of the

telencephalon (Portavella et al., 2004a, 2004b; Portavella & Vargas, 2005; Vargas et al., 2009). These data coupled with the effects of Dm lesions on aggressive behavior, breeding, habituation in situations of aggression in a resident/intruder paradigm (de Bruin, 1980; Marino-Neto & Sabbatini, 1983), as well as data from electrical stimulation of Dm in *Carassius* that show facilitation and inhibition of aggressive and reproductive patterns or startle response and escape (Savage, 1971), support the homology of the fish telencephalic Dmv with the pallial amygdala of mammals. This idea have been proposed on the basis of neuroanatomical and neurohistochemical evidence of similarities between the two structures developing an evolution-based model of brain organization (Braford, 1995; Ehteler & Saidel, 1981; Hornby et al., 1987; Ito et al., 1986; Medina & Reiner, 1995; Murakami et al., 1983; Northcutt, 1995; 2008; Piñuela & Nortcutt, 1994; Reiner & Northcutt, 1992; Striedter, 1991; Wulliman & Rink, 2002) and that of reptiles (Crews et al., 1993; Keating et al., 1970; Krohner & Crews, 1987; Tarr, 1977), birds (Cohen, 1975; Dafter, 1975; 1976; Ikebuchi et al., 2009; Phillips 1968; Phillips & Youngren, 1968; Vowles & Beasley, 1974; Zeier, 1971), and mammals (Ambrogli et al., 1991; Fonberg, 1973; Grossman et al., 1974; Hanwerker et al., 1974; Horvath, 1963; Killcross et al., 1997; Liang & McGaugh, 1983; McIntery & Stein, 1973; Pellegrino, 1968; Roozendaal et al., 1993; Sanchez-Riolobos, 1986; Takashina et al., 1995; van-der-Zee et al., 1997; Werka et al., 1978; Wiersma et al., 1998).

This exhibition along with the data of the previous sections would support the fact that structures homologous to the amygdala of the different groups of vertebrates, including humans, share similar functions, at least in terms of information processing of emotional content, which is essential for the survival of the species (LeDoux, 2012). This idea is based on results from genoarchitectonical studies showing the consistancy of genetical markers along vertebrates' brains evolution. In the case of amygdala of tetrapods (Medina et al., 2011) is composed of four different regions: ventral pallial amygdala (i.e. basal amygdala of mammals, dorsal ventricular ridge in reptiles and birds), striatal amygdala (i.e. the central nucleus in different groups), pallidal amygdala (medial amygdala of amniotes) and hypothalamical part present in different vertebrates groups (i.e. bed nucleus and extended amygdala). A part of specific gene expression of these four divisions is present in mammals, birds, reptiles, and amphibians. In the case of teleost fish, behavioral approaching (Portavella et al., 2004a, 2004b; Portavella & Vargas, 2005) showed similar learning deficits after brain lesions on homologues areas to mammalian amygdala, and genetical studies showed constancy in the expression of pallial and subpallial genes, and the presence of, at least, a medial amygdala (Gonzalez & Northcutt, 2009) and the hypothalamical components (Eaton et al., 2008). The behavioral studies commented in previous sections of this chapter show how essential functions for species survival such as fear conditioning, escape and avoidance behavior, aggressive, and sexual behaviors are dramatically impaired after mammalian amygdala lesions, or homologues structures in the case of birds, reptiles, amphibians, or teleost fish (see sections II and III of this chapter). These behavioral data and genetical studies, together with evidence of homologies seem to support the existence of a basic bauplan in the vertebrates' line, including man, which has been broadly conserved during evolution of the vertebrate nervous system, including structures such as the amygdala.

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