

Corso di Psicosomatica

Scheda n. 8

Il modello di Joseph LeDoux

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Per gentile concessione del professor Silvio Merciai, docente al corso di Psicosomatica presso il corso di laurea in Psicologia dell'Università di Torino

*Il ruolo delle emozioni nei modelli di comportamento è stato illustrato in particolare (ma non solo, ovviamente!) da due Autori, che ne hanno insieme gettato le basi sperimentali e proposto sintesi di ampio respiro e di evidente suggestione: **Joseph LeDoux** ed **Antonio R. Damasio**.*

Questa scheda è dedicata ad illustrare per sommi capi il contributo del primo dei due ...

Si devono a **Joseph LeDoux** ed al suo laboratorio gli studi fondamentali sull'emozione della paura e sul ruolo centrale dell'amigdala (la sua opera principale è **The Emotional Brain. The Mysterious Underpinning of Emotional Life**. Simon & Schuster, New York, 1996 - tradotto in italiano con il titolo di **Il cervello emotivo. All'origine delle emozioni**. Baldini e Castoldi, Firenze, 1998).

Riporto di seguito alcuni passi della bella recensione-commento del libro scritta da *Furio Di Paola* per il numero speciale di *Psiche* dedicato ai *Dialoghi tra psicoanalisi, scienze e neuroscienze* (V:2, Luglio-Dicembre 1997):

Senza voler inventariare i non pochi motivi che si connettono in questo [suo] sforzo di sintesi, propongo di concentrare l'attenzione su tre temi che rendono subito evidente la portata dei problemi in gioco, nella peculiare rielaborazione che trovano in LeDoux.

*Il primo riguarda il concetto di 'sistema limbico' ... [Torneremo più avanti sull'argomento, riprendendo la posizione di Joseph LeDoux con le sue stesse parole ...]
La seconda questione concerne il rapporto tra emozione e cognizione che trova, nel modello anatomo-fisiologico proposto da LeDoux una chiara e motivata risposta, almeno nel caso dell'affetto di base studiato, che è la paura.
Il terzo aspetto riguarda l'apertissima questione della plasticità cerebrale in relazione alle psicoterapie - gli ipotizzabili effetti somatici (micro-somatici) di una cura non 'chimica' ma appunto 'verbale' - questione che LeDoux affronta in modo non convenzionale: proponendo infine, come possibile risposta, una ingegnosa congettura, coerente con le evidenze empiriche prima raccolte (nonché, vedremo, proficuamente discutibile). ...
... l'attenzione problematica che LeDoux riserva a Freud costituisce un'ulteriore, rilevante motivo di interesse di *The Emotional Brain*. ...*

Il nodo dell'elaborazione teorica e del modello complessivo di LeDoux è così riassunto da *Regina Pally*, nel suo contributo sull'emozione di cui abbiamo parlato nella scheda precedente:

*Fear evolved to enhance detection and response to danger. All animals react with fear to extremely aversive stimuli such as shock. In addition each species has specific stimuli that elicit fear in that species. Rats react with fear to open spaces, vervet monkeys to eagles flying overhead. However, no matter what stimulus elicits fear, neural systems have been conserved throughout evolution. The brain structures, hormones, autonomic nervous system responses and behaviours of fear are similar in all animals. Therefore fear studied in rats or monkeys can reasonably be used to understand aspects of fear in human beings (Davis, 1992).
The classic fear conditioning experiment done in rats involves pairing a previously neutral stimulus, such as a tone, with a mild foot shock. The animal immediately exhibits the fear response, including 'freezing', increased startle reflex, defecation, increased heart rate and cortisol. After only one exposure, the animal is 'conditioned' to interpret the tone as aversive, meaning it will respond with the exact same fear response on subsequent occasions to the tone alone. Human beings also show this kind of rapid conditioning to mild shock. Once established, fear conditioning is relatively permanent, as illustrated by the results of extinction. When the tone is presented frequently in the absence of shock, eventually the fear response 'extinguishes'. However, the response is only inhibited, not erased completely. If the rat is exposed to some other unrelated stress, such as water deprivation, conditioned fear to the tone returns to its original intensity.
The details of fear circuitry emerge from experiments in which individual brain regions are lesioned and their effect on fear conditioning observed (Le Doux, 1994, 1995, 1996).*

The role of the amygdala and cortex

The amygdala, essential for conditioned fear, is the hub of inputs and outputs. Two main input pathways are

identified: (1) a rapid, shorter, subcortical route and (2) a slow, longer, cortical route ...). Each elicits an identical output, the 'fear response', but in reaction to different stimulus information. In route (1) sensory information goes from the thalamus directly to the amygdala and rapidly triggers fear in response to simple stimulus cues. In route (2) sensory information is routed from the thalamus to the cortex and hippocampus and is then projected to the amygdala, taking longer to trigger fear in response to more complex stimulus objects. What most surprised scientists is that the auditory cortex, which is required for conscious awareness of auditory stimuli is not required for conditioned fear! When the rat's auditory cortex is lesioned, fear conditioning still occurs, because subcortical route (1) remains in tact, even though the animal cannot consciously hear the tone. The implication is that emotion can be triggered by situations of which the person is unaware. Lesion studies reveal that although the cortex is not required to create a conditioned fear it plays a regulatory role in the process. The cortical route can inhibit a fear response triggered by the subcortical route. It also provides greater sensory discrimination. When two tones of different frequency are presented, but only one tone is paired to the shock, an animal with an intact auditory cortex reacts with fear only to the 'paired' tone. An animal with a lesioned auditory cortex reacts with fear to both tones. A cortical region called the medial prefrontal cortex is essential for extinction of fear. If this region is lesioned the rat's fear response to the tone will not be extinguished. These studies imply that although early fear responses to trauma may never completely disappear, conscious awareness may help diminish those fear responses. Studies confirm the role of the amygdala in human fear. Stimulating the amygdala causes feelings of fear (Halgren, 1992). Removal of the amygdala for treatment of epilepsy causes impaired fear conditioning (La Bar et al., 1995). People normally react with fear to a loud obnoxious noise, just as rats and human beings do to shock. With an intact amygdala, when a soft noise is paired with a loud obnoxious noise, subjects subsequently react with autonomic arousal upon hearing the soft noise alone. Patients without an amygdala do not develop a conditioned response to the soft noise. Damage to the amygdala impairs recognition of fear on the faces of others while the ability to detect other emotions is retained (Bechara et al., 1995). There are more connections from the amygdala to the cortex than from the cortex to the amygdala. This biases us towards fear, since automatic fear responses may be stronger than our ability to inhibit them volitionally.

The role of the hippocampus

The hippocampus provides information about contextual location (Okeefe & Nadel, 1978; Phillips & Le Doux 1992) (...). A rat in cage 'A' conditioned to a tone reacts with fear to the tone, but also reacts with fear when just placed in the cage! If later the rat is placed in cage 'B' and the fear response to the tone extinguished, the fear response will be elicited in full force by simply placing the animal back in cage 'A'. A man mugged once in front of his neighbour's house develops feelings of fear towards people who resemble the mugger, and also every time he walks past his neighbour's house. When the hippocampus is lesioned a rat still reacts with fear to the stimulus cue of the tone, but not to location cues of the cage. Contextual cues allow animals to learn to avoid danger even from cues only remotely related to danger, thus avoiding danger well before it happens. These findings imply that spatial cues activate fear in situations that are no longer dangerous, but in which the animal is merely in the same location as it was during a traumatic event. This may explain why rape victims assiduously avoid the specific situation (i.e. car park, flat) in which they were raped. Anything impairing the hippocampus can make fear generalise to other locations. Besides its role in memory, the hippocampus also regulates emotional arousal, since cortisol binding of receptors in the hippocampus sends a message to the hypothalamus to slow the release of cortisol (Joseph, 1996; Le Doux, 1996). However, states of high excitation that cause excessive neocortical arousal can lead to diminished hippocampal activity, presumably to protect the neocortex from becoming overwhelmed. Emotionally traumatic events lead to very high levels of cortisol that can actually damage hippocampal cells (Bremner et al., 1995; Sapolsky, 1996). Therefore, dampening of hippocampal activity and actual hippocampal damage may lead to a loss of cortisol regulation and impairments in the memory of traumatic situations. Conversely, amygdala activity, if anything, increases during emotional arousal (Corodimas et al., 1994). These findings imply that at the same time that stress 'represses' conscious explicit memory of a traumatic experience, it can enhance unconscious emotional memory of that experience!

How the prefrontal cortex chooses how to respond

The amygdala activates pre-packaged automatic responses. The prefrontal cortex, most developed in human beings, can shift from the automatic responses of the amygdala to decisions and choices about what response is indicated based on prior experience (Krasnegor et al., 1997). The prefrontal cortex, in relation to its working memory function (see Pally, 1998 for a discussion of memory) anticipates the outcome of various response options and considers what might go wrong if one's plan fails. Human anxiety may be the high cost of the ability to anticipate danger and think about how things can go wrong, independent of them actually being present. Lower animals suffer the consequences of wrong choices but don't worry about it beforehand.

Diamo di nuovo la parola a Furio Di Paola:

Ciò vuol dire che l'amigdala dà corso comunque e subito alle reazioni somatiche al potenziale pericolo, per discriminare poi più in dettaglio lo stimolo percettivo (che potrebbe anche rivelarsi un falso allarme). Cammino in un bosco - è uno degli esempi, questa volta con stimolo visivo, di LeDoux - e scorgo qualcosa di sottile e flessuoso simile a un serpente; questo stimolo poco definito raggiunge in modo quick and dirty - per la via bassa - l'amigdala, che attiva subito, via ipotalamo e sistema nervoso autonomo, le reazioni somatiche immediate di allarme. Intanto - per la via alta - lo stimolo è elaborato più in dettaglio, giunge poi [di nuovo] all'amigdala meglio definito, permettendo di valutare più accuratamente se si tratti proprio di un serpente (e non di un

innocuo ramoscello). Dal concorso di entrambi i processi esemplificati dipenderà un apprendimento emotivo, mentre la maggiore velocità del primo ha un immediato valore di sopravvivenza ("... è meglio aver preso un ramoscello per un serpente, che non aver risposto a un possibile serpente", pag. 166). ... "Mentre il controllo conscio sulle emozioni è debole, le emozioni possono sommergere la coscienza. Ciò dipende dal fatto che l'organizzazione del cervello a questo punto della nostra storia evolutiva è tale che le connessioni dei sistemi emotivi a quelli cognitivi sono più forti delle connessioni dei sistemi cognitivi a quelli emotivi" (pag. 19). ... LeDoux sviluppa l'ipotesi che l'interazione ippocampo-amigdala, nel contesto della rete di connessioni indipendenti di ciascuna struttura con il resto del cervello, sia la questione critica per comprendere il processo delle memorie emotive. Il sistema dell'amigdala presiederebbe alla memoria emotiva implicita (emotional memory); il sistema dell'ippocampo alla memoria esplicita intorno alle emozioni (memory of an emotion). ... "Per quanto ne so, lo stress non interferisce con l'efficienza dell'amigdala, e anzi ... lo stress può rinforzarne le funzioni. E' dunque del tutto possibile che si abbia scarsa memoria conscia di un'esperienza traumatica, e al tempo stesso che si formino memorie emotive implicite, inconscie, molto potenti - attraverso il condizionamento alla paura mediato dall'amigdala. ... queste forti paure inconscie possono diventare molto resistenti all'estinzione. Possono, in altre parole, divenire fonti inconscie di intensa angoscia che esercitano influenze opache e perverse per tutta la vita" (pag. 245). ... L'ipotesi di LeDoux [a proposito del ruolo della psicoterapia e della sua possibile efficacia] si può riassumere partendo dal suo assunto di base ...: le pratiche psicoterapeutiche, scrive nel penultimo capitolo, non sono che "un altro modo di riconnettere il cervello (just another way to rewire the brain, pag. 263). ... "La terapia non è che un altro modo di produrre potenziamento sinaptico, nei cammini cerebrali che governano l'amigdala. Le memorie emotive dell'amigdala, come si è visto, sono radicate indelebilmente nei suoi circuiti. Il meglio che si può sperare è regolare la loro espressione. E il modo per farlo è ottenere che la corteccia controlli l'amigdala" (pag. 265).

Su possibili non dissimili segnali anche la recensione di *Franco De Masi*, comparsa sul numero XLV:1 (gennaio-marzo 1999) della *Rivista di Psicoanalisi*, dove sono peraltro più ampiamente discusse le possibili interazioni tra i modelli neuroscientifici e l'impianto teorico della psicoanalisi, con particolare attenzione al pensiero di W. R. Bion.

Sul lavoro di LeDoux e ad integrazione di quanto sopra ricordato, si vedano [A Talk with Joseph LeDoux](#), una breve e chiara nota esplicativa - [Joseph LeDoux on the Amygdala](#) - ed una descrizione in prima persona delle sue ricerche - [Joseph E. LeDoux – Memory and Emotion](#). Riporto una citazione dalla sua intervista, perché rilevante ai fini dell'integrazione tra neuroscienze e psicoanalisi:

By cognitive memory I'm going to assume you mean explicit conscious memory, the kind of memory we usually have in mind when we use the word memory in everyday speech. Emotional memory and explicit memory happen at the same time, but separately. For example, the amygdala mediates emotional memory and the temporal lobe memory system mediates explicit memory. Here's an example. Imagine driving down the road and having an accident. You hit your head on the steering wheel and the horn gets stuck on. You're bleeding and in pain. It's awful. Sometime later, you hear the sound of a horn. The sound goes to your amygdala and activates your autonomic nervous system (raising your blood pressure and heart rate, making you sweat), tenses your body muscles, releases stress hormones into your blood, and so on. The sound also goes to the temporal lobe system and reminds you of the accident, of who you were with and where you were going. It also reminds you that it was awful. But these are all just facts about the situation. They are memories of the emotional experience rather than emotional memories. In general, one difference between emotional and cognitive processing is that emotional processing often leads to bodily responses, whereas cognitive processing leads to more cognitive processing. Cognitions are seldom characterized by specific kinds of responses, but emotions usually are. It's important that we understand as much as we can about the biology of these systems. Many people have problems with their emotional memories; psychologists' offices are filled with people who are basically trying to take care of and alter emotional memories, get rid of them, hold them in check. If anything, emotional memory is more basic than explicit conscious memory. For example, it takes place at an earlier age. It's conceivable, and in fact seems very likely, that a child could be abused very early in life and develop unconscious emotional memories through the amygdala prior to the point where the temporal lobe memory system has kicked in. If that's true then emotional memories are being formed for things that will never be consciously understood, because the system that mediates conscious memory isn't available to encode the experience and can therefore never retrieve it. We need to understand how unconscious emotional memories are formed - not only because they occur in early childhood, but because emotional memories are created throughout our lives. And it appears that these memories are indelible. They can be extinguished in the laboratory or treated in the psychiatrist's office, but they can usually be brought back. And recently we've been able to find a mechanism in the amygdala that might be responsible for this. It's sort of complicated, but the finding goes like this. We record neural activity in the amygdala before and after conditioning. Cells fire more to the tone afterwards. With extinction the firing rate goes back to baseline. However, in addition to measuring these stimulus-evoked responses, we measure the correlation in the time when different cells fire spontaneously (no stimulus present). After conditioning, some cells that were not correlated become correlated. And for some of the cells the correlations remain past extinction. In other words, the feared stimulus no longer elicits activity in the amygdala, but the amygdala cells continue to be functionally coupled. It's as if extinction (and therapy) doesn't erase the memory, it just weakens the ability of the stimulus to activate the memory. So in order for the stimulus to again be effective all you have to do is change the synaptic strength of the connection between the stimulus and the memory rather than

recreate the memory.

...emotional reactions can be elicited independent of our conscious thought processes. For example, we've found pathways that take information into the amygdala without first going through the neocortex, which is where you need to process it in order to figure out exactly what it is and be conscious of it. So, emotions can be and, in fact, probably are mostly processed at an unconscious level. We become conscious and aware of all this after the fact. Conscious feelings of fear are thus not a necessary step in the link between a dangerous stimulus and emotional responses. We're probably not as in control of our emotions as we sometimes think we are, or wish to be...The connectivity of the amygdala with the neo-cortex is not symmetrical. The amygdala projects back to the neo-cortex in a much stronger sense than the neo-cortex projects to the amygdala...The implication is that the ability of the amygdala to control the cortex is greater than the ability of the cortex to control the amygdala. And this may explain why it's so hard for us to will away anxiety; emotions, once they're set into play, are very difficult to turn off. Hormones and other long-acting substances are released in the body during emotions. These return to the brain and tend to lock you into the state you're in at the time. Once you're in that state it's very difficult for the cortex to find a way of working its way down to the amygdala and shutting it off. This is why therapy is probably such a long and difficult process, because the neocortex is using imperfect channels of communication to try and grab hold of the amygdala and control it... The amygdala can control the neocortex very easily, because all it has to do is arouse lots of areas in a very non specific way. But for the cortex to then turn all of that off is a very difficult job. The evolution of the brain is at a point where we don't have the connectivity that would be necessary for cognitive systems to more efficiently control our emotions.

Vorrei concludere questa scheda affidando infine direttamente la parola a Joseph LeDoux, che ha recentemente pubblicato una sintesi del suo lavoro con il titolo di **Emotion Circuits in the Brain**, comparsa sugli *Annual Reviews - Neuroscience* (2000, 23: 155-184):

After decades of neglect, neuroscience has again embraced emotion as a research topic. This new wave of interest raises the question of why emotion was overlooked for so long. It is instructive to consider this question before examining what has been learned about emotional circuits, as some of the factors that led brain researchers to turn away from this topic may again hamper progress unless they can be grappled with.

Why Did Interest in Emotion Wane?

During the first half of the twentieth century, brain researchers were immensely interested in the brain mechanisms of emotional behavior. Some of the early pioneers in neuroscience worked in this area, including Sherrington, Cannon, Papez, and Hebb. Responses that occur when we defend against danger, interact with sexual partners, fight with an enemy, or have a tasty bite to eat promote the survival of individuals and their species. Emotional responses are thus inherently interesting and important. So what happened? Why did research on the brain mechanisms of emotion come to a halt after midcentury?

For one thing, emotion research was a victim of the cognitive revolution. The emergence of cognitive science shifted the interest of those concerned with the relation between psychological functions and neural mechanisms toward processes (perception and memory, for example) that were readily thought of in terms of computer-like operations. From the start, cognitive scientists claimed that their field was not about emotion and other such topics (...). The cognitive approach came to be the dominant approach in psychology and brain science, and research interest in emotion dwindled.

Another factor that hindered work on emotions in neuroscience was that the problem of how the brain makes emotions seemed to have been solved in the early 1950s by the limbic system concept (MacLean 1949, 1952). This appealing and convincing theory was the culmination of research on the brain mechanisms of emotion by many researchers, extending back to the late nineteenth century (...). Studies of how the brain mediates cognitive processes seemingly had a long way to go to catch up with the deep understanding that had been achieved about emotions, and researchers flocked to the new and exciting topic of cognition and the brain to begin filling the gap.

Cognitive questions also seemed more tractable than emotional ones, due in part to the dark cloud of subjectivity that hung over the topic of emotion. Although subjective experience and its relation to neural mechanisms is a potential difficulty for any area of psychology, cognitive scientists figured out how to study mental processes without having to solve the mind-body problem. They showed, for example, that it is possible to study how the brain processes (computes and represents) external stimuli without first resolving how the conscious perceptual experiences come about. In fact, it is widely recognized that most cognitive processes occur unconsciously, with only the end products reaching awareness, and then only sometimes (see Kihlstrom 1987). Emotion researchers, though, did not make this conceptual leap. They remained focused on subjective emotional experience. In spite of the fact that most research on emotions and the brain was, and still is, conducted with experimental animals, creatures in which subjective states are difficult if not impossible to prove, theoretical discussions of emotions and the brain typically reverted back to the age-old question of feelings. This approach puts the mind-body problem right smack in the middle of the path of progress.

The main lesson to be learned from this brief excursion into history is that emotion researchers need to figure out how to escape from the shackles of subjectivity if emotion research is to thrive. It is ironic that cognitive science, which led to the neglect of emotion research, may also be able to help in its resurrection by providing a strategy that allows the study of emotion independent of subjective emotional experiences. It is possible, for example, to ask how the brain processes emotional information (i.e. detects and responds to danger) without necessarily first solving the question of where conscious feelings come from. Contrary to popular belief, conscious feelings are not required to produce emotional responses, which, like cognitive processes, involve unconscious processing mechanisms (...). If we want to understand feelings, it is likely going to be necessary to

figure out how the more basic systems work. Failure to come to terms theoretically with the importance of processing systems that operate essentially unconsciously has been a major impediment to progress in understanding the neural basis of emotion. To overcome this, brain researchers need to be more savvy about the nature of emotions, rather than simply relying on common sense beliefs about emotions as subjective feeling states.

Research on emotion can also help cognitive science. A pure cognitive approach, one that omits consideration of emotions, motivations, and the like, paints an artificial, highly unrealistic view of real minds. Minds are not either cognitive or emotional, they are both, and more. Inclusion of work on emotion within the cognitive framework can help rescue this field from its sterile approach to the mind as an information-processing device that lacks goals, strivings, desires, fears, and hopes.

Once a processing approach to emotion is taken, emotion and cognition can be studied similarly: as unconscious processes that can, but do not necessarily, lead to conscious experiences. This would open the door for the integration of emotion and cognition, and such integration should be a major goal for the immediate future.

Should We Integrate the Cognitive Brain with the Limbic System?

The rise of cognitive science led to important advances in understanding the brain mechanisms of perception, attention, memory, and other cognitive processes. One might be tempted to say that the way to foster the synthesis of cognition and emotion into a new science of mind would be to put all this new information about the cognitive brain together with the definitive view of the emotional brain provided long ago by the limbic system concept. However, this would be a mistake. In spite of the fact that the limbic system concept remains the predominant view about how the brain makes emotions, it is a flawed and inadequate theory of the emotional brain.

The limbic system concept was put forth in the context of an evolutionary explanation of mind and behavior (...). It built upon the view, promoted by comparative anatomists earlier in the century, that the neocortex is a mammalian specialization—other vertebrates have primordial cortex but only mammals were believed to have neocortex. And because thinking, reasoning, memory, and problem solving are especially well developed in mammals, particularly in humans and other primates that have relatively more neocortical tissue, these cognitive processes must be mediated by the neocortex and not by the old cortex or other brain areas. In contrast, the old cortex and related subcortical ganglia form the limbic system, which was said to mediate the evolutionarily older aspects of mental life and behavior, our emotions. In this way, cognition came to be thought of as the business of the neocortex and emotions of the limbic system.

The limbic system theory began to run into trouble almost immediately when it was discovered, in the mid-1950s, that damage to the hippocampus, the centerpiece of the limbic system, led to severe deficits in a distinctly cognitive function, long-term memory (Scoville & Milner 1957). This was incompatible with the original idea that the primitive architecture of the limbic system, and especially of the hippocampus, was poorly suited to participate in cognitive functions (...). Subsequently, in the late 1960s, it was discovered that the equivalent of mammalian neocortex is present, though rudimentary, in nonmammalian vertebrates (...). As a result, the old/new cortex distinction broke down, challenging the evolutionary basis of the assignment of emotion to the limbic system and cognition to the neocortex (...).

The limbic system itself has been a moving target. Within a few years after inception, it expanded from the original notion of "old cortex" and related subcortical forebrain nuclei to include some areas of the midbrain (...), and even some regions of neocortex (...). Several attempts have been made to salvage the limbic system by defining it more precisely (...). Nevertheless, after half a century of debate and discussion, there are still no agreed upon criteria that can be used to decide which areas of the brain belong to the limbic system. Some have suggested that the concept be abandoned ...

In spite of these difficulties, the limbic system continues to survive, both as an anatomical concept and as an explanation of emotions, in textbooks, research articles, and scientific lectures. This is in part attributable to the fact that both the anatomical concept and the emotional function it was supposed to mediate were defined so vaguely as to be irrefutable. For example, in most discussions of how the limbic system mediates emotion, the meaning of the term emotion is presumed to be something akin to the common English language use of the term (because no other definition is given). However, the common English use of the term emotion is at best a poor theoretical notion, for emotion is a rich and complex theoretical concept with many subtle aspects, some of which are nonintuitive and thus inconsistent with the common use of the term (...). On the neural side, the criteria for inclusion of brain areas in the limbic system remain undefined, and evidence that any limbic area, however defined, contributes to any aspect of any emotion has tended to validate the whole concept. Mountains of data on the role of limbic areas in emotion exist, but there is still little understanding of how our emotions might be the product of the limbic system.

Particularly troubling is the fact that one cannot predict, on the basis of the original limbic theory of emotion or any of its descendants, how specific aspects of emotion work in the brain. The explanations are all post hoc. Nowhere is this more apparent than in recent work using functional imaging to study emotions in the human brain. Whenever a so-called emotional task is used, and a limbic area is activated, the activation is explained by reference to the fact that limbic areas mediate emotion. And when a limbic area is activated in a cognitive task, it is often assumed that there must have been some emotional undertone to the task. We are, in other words, at a point where the limbic theory has become an off-the-shelf explanation of how the brain works. However, this explanation is grounded in tradition rather than data. Deference to the concept is inhibiting creative thought about how mental life is mediated by the brain.

Although the limbic system theory is inadequate as an explanation of the specific brain circuits of emotion, MacLean's (1949, 1952, 1970) original ideas are very interesting in the context of a general evolutionary explanation of emotion and the brain. In particular, the notion that emotions involve relatively primitive circuits that are conserved throughout mammalian evolution seems right on target. Furthermore, the idea that cognitive processes might involve other circuits, and might function relatively independent of emotional circuits, at least in

some circumstances, also seems correct. These functional ideas are worth holding on to, even if we abandon the limbic system as a structural theory of the emotional brain.

Rivedendo i classici studi (non solo della sua scuola) a proposito dei circuiti della paura e della loro connessione con le teorie del condizionamento e dopo aver brevemente ricordato le attuali conoscenze circa la terminologia anatomica dell'amigdala

The amygdala consists of approximately 12 different regions, each of which can be further divided into several subregions (...). Although a number of different schemes have been used to label amygdala areas (...), the scheme adopted by Amaral et al (1992) for the primate brain and applied to the rat brain by Pitkänen et al (1997) is followed here. The areas of most relevance to fear conditioning are the lateral (LA), basal (B), accessory basal (AB), and central (CE) nuclei and the connections between these (...). In other classification schemes, B is known as the basolateral nucleus and AB as the basomedial nucleus. The term basolateral complex is sometimes used to refer to LA and B (and sometimes AB) together. ...

vengono poi descritte in dettaglio le varie connessioni, interne ed esterne, per passare poi a dare breve conto degli studi più recenti sulla plasticità dell'amigdala

With the basic elements of the circuitry understood from lesion studies, researchers have turned to questions about the nature of the plasticity within the amygdala that might underlie fear learning. Fear plasticity in the amygdala has been studied in three closely intertwined ways. First, single-unit recordings have been made in areas of the amygdala implicated in fear conditioning by lesion studies. Second, long-term potentiation (LTP), an experimentally advantageous but artificial form of plasticity, has been studied in these same areas. Third, drugs that block LTP have been infused into amygdala areas where LTP is believed to occur, and effects on the acquisition of conditioned fear behavior assessed. ...

e sui segnali intracellulari che costituiscono le basi biologiche della memoria a lungo termine, per concludere quindi con un cenno all'estendibilità delle scoperte sperimentali al caso dell'uomo e, conseguentemente, alle possibili implicazioni cliniche:

Although it is clear that studies of acute fear responses elicited by conditioned fear stimuli cannot account for all aspects of fear and fear disorders, there is growing enthusiasm for the notion that fear learning processes similar to those occurring in fear conditioning experiments might indeed be an important factor in certain anxiety disorders. For example, fear conditioning models of posttraumatic stress disorder and panic disorder (Pitman & Orr 1999, Goddard et al 1998) have been proposed recently by researchers in these fields. Earlier in this century, the notion that conditioned fear contributes to phobias and related fear disorders was fairly popular. However, this idea fell out of favor because laboratory fear conditioning seemed to produce easily extinguishable fear, whereas clinical fear is difficult to treat. The notion arose that fear disorders involve a special kind of learning, called prepared learning, where the CS is biologically significant rather than neutral (Seligman 1971, Marks 1987, Öhman 1992). Although preparedness may indeed contribute, there is another factor to consider. In studies of rats, Morgan et al (1993; but see Gewirtz & Davis 1997) found that easily extinguished fear could be converted into difficult-to-extinguish fear in rats with damage to the medial prefrontal cortex. This suggested that alterations in the organization of the medial prefrontal regions might predispose certain people in some circumstances (such as stressful situations) to learn fear in a way that is difficult to extinguish (treat) under normal circumstances. These changes could come about because of genetic or experiential factors, or some combination. ...

One of the key issues for the coming years is to integrate research on emotion and cognition. As already noted, this will not be achieved by simply linking research on the limbic system with research on the cortex. An approach that offers more anatomical precision on the emotion side is needed. Studies of fear conditioning provide a framework for beginning such an endeavor. Although this bottom up approach focused on fear may seem needlessly tedious, it is possible that once other emotions are understood in sufficient anatomical detail, some general principles that apply to other emotions will emerge. For the time being, it is best to restrict the discussion to fear circuits and their interactions with cognitive systems. Thus, in this section we consider how fear processing by the amygdala is influenced by and can influence perceptual, attentional, and memory functions of the cortex.

The amygdala receives inputs from cortical sensory processing regions of each sensory modality and projects back to these as well (Amaral et al 1992, Turner et al 1980, McDonald 1998). As shown above, these projections allow the amygdala to determine whether danger is present in the sensory world. But in addition to processing the significance of external stimuli, the amygdala can also influence sensory processing occurring in cortical areas. The amygdala only receives inputs from the late stages of cortical sensory processing, but it projects back to the earliest stages (Turner et al 1980, Amaral et al 1992). Thus, once the amygdala is activated by a sensory event from the thalamus or cortex, it can begin to regulate the cortical areas that project to it, controlling the kinds of inputs it receives from the cortex. The amygdala also influences cortical sensory processes indirectly, by way of projections to various "arousal" networks, including the basal forebrain cholinergic system, the brainstem cholinergic system, and the locus ceruleus noradrenergic system, each of which innervates widespread areas of the cortex (e.g. Aston-Jones et al 1996, Gallagher & Holland 1994, Holland & Gallagher 1999, Kapp et al 1992,

Weinberger 1995). Thus, once the amygdala detects danger, it can activate these arousal systems, which can then influence sensory processing. The bodily responses initiated by the amygdala can also influence cortical areas, by way of feedback either from proprioceptive or visceral signals or hormones (e.g. McGaugh et al 1995, Damasio 1994). Amygdala regulation of the cortex by either direct or indirect routes could facilitate the processing of stimuli that signal danger even if such stimuli occur outside the attention field (Armony et al 1996, 1998; Armony & LeDoux 1999).

In humans, damage to the amygdala interferes with implicit emotional memories but not explicit memories about emotions, whereas damage to the medial temporal lobe memory system interferes with explicit memories about emotions but not with implicit emotional memories (Bechara et al 1995, LaBar et al 1995). Although explicit memories with and without emotional content are formed by way of the medial temporal lobe system, those with emotional content differ from those without such content. The former tend to be longer lasting and more vivid (see Christianson 1989, Cahill & McGaugh 1998). Lesions of the amygdala or systemic administration of a beta-adrenergic antagonist prevent this amplifying effect of emotion on declarative memory (Cahill & McGaugh 1998), which suggests that the amygdala can modulate the storage of explicit memories in cortical areas. At the same time, the medial temporal lobe memory system projects to the amygdala (Amaral et al 1992). Retrieval of long-term memories of traumatic events may trigger fear reactions by way of these projections to the amygdala.

Although there has been relatively little work on the role of the amygdala in cognitive-emotional interactions, the importance of the amygdala as a bridge between emotion and attention was pointed out over thirty years ago (e.g. Pribram & Melges 1969). Given the extensive connections between the amygdala and cortical areas, this topic is begging for research. ...

Research on the emotional brain has progressed significantly in recent years, largely as a result of a highly focused approach centered on the study of fear mechanisms, and especially the mechanisms underlying fear conditioning. This work has mapped out pathways involved in fear learning in both experimental animals and humans, and it has begun to shed light on interactions between emotional and cognitive processes in the brain. Although the focus on fear conditioning has its limits, it has proven valuable as a research strategy and provides a foundation upon which to build a broader understanding of mind and brain.

At the same time, there is a disturbing rush to embrace the amygdala as the new center of the emotional brain. It seems unlikely that the amygdala is the answer to how all emotions work, and it may not even explain how all aspects of fear work. There is some evidence that the amygdala participates in positive emotional behaviors, but that role is still poorly understood. If an amygdala theory of emotion is on the horizon, let it get there by data rather than by faith.

Neuroscience meetings these days have numerous papers on the role of the brain in emotion, affect, hedonic tone, and the like. Unless these vague concepts can be operationalized, as was done in the work on fear, they are likely to impede, if not recede, the progress. The future of emotion research can be bright if we keep in mind the way that emotion became respectable again: by focusing on a psychologically well-defined aspect of emotion, by using an experimental approach that simplified the problem in such a way as to make it tractable, by circumventing vague and poorly defined aspects of emotion, and by removing subjective experience as a roadblock to experimentation. This is not to suggest that the hard problems should not be worked on but instead that they should be worked on in a way that advances the field.