Progressions

My Fifty Years Thinking about Emotional Consciousness in the Brain

Joseph E. LeDoux
New York University, Brooklyn, New York 11249

This piece provides an overview of my fifty-year career as a neuroscientist. I spent much of this time trying to understand what emotions are and how they relate to human behavior and consciousness. I am pleased that my work on the amygdala helped bring emotion into the field. But late in the game I changed my views about the amygdala and emotions, causing some unrest in the field. Note that this is a personal, not an encyclopedic, overview of the neuroscience of emotion. In other words, this is my story about emotions and the brain, especially in relation to emotional consciousness.

Introduction

Emotions define the highs and lows of our lives and are at the core of human experience. Unfortunately, the scientific study of emotions has long been plagued by arguments about what emotions are and how they function within the brain. Until we pin down what emotions are, we won't be able to relieve the emotional problems that plague the lives of so many of people. At the end of the day, our understanding of emotion in the brain is only as good as our conceptualization of what an emotion is. If we don't know what we are looking for, we will surely fail to find it, and we will mislead ourselves and others in the process.

The modern science of emotion has roots in the latter part of the nineteenth century, especially through the writings of Darwin (1872)and James (1884). Their ideas led to two leading psychological theories of emotion in the 1960s. Darwin fostered Basic Emotions Theory (Tomkins, 1962), and James paved the way for the Cognitive Theory (Festinger, 1957; Arnold, 1960; Schachter and Singer, 1962). In the late 1960s, Neuroscience was emerging as a new discipline in parallel with the thriving cognitive movement in psychology. But unlike psychology, emotion was not a topic of much interest in neuroscience, at least compared to research on sensory and motor functions, language, thinking and reasoning, and learning and memory.

Changing Lanes

I received an undergraduate degree in business administration in 1971 and in 1974 a master's degree in marketing, both from Louisiana State University. Midstream during my undergraduate years, a political revolution was taking place, and business was not a fashionable topic in the left-leaning political climate. Psychology was much more acceptable, and I was able to stay in step with my generation by taking as many psychology classes as I could to understand and protect consumers from corporate America. In the Fall of 1973, I registered for a course titled "Learning and Motivation." I thought it might be relevant to my work on consumer behavior. But I was wrong. The class, taught by Professor Robert Thompson, was about his research on the brain mechanisms of memory in rats. My initial inclination was to drop the class. But as each session went by, I became more and more intrigued and spent as much time as I could in his lab. I didn't do much in the way of hands-on research, but I learned a lot and decided that I wanted to study the brain. Things were pretty loose back then and by the end of the spring semester I had been accepted into the Psychology PhD program at SUNY Stony Brook.

My mentor was Michael Gazzaniga, who had become famous for his work on consciousness in split-brain patients a decade earlier. In the spring of 1978, 4 years after receiving my master's in marketing, I had earned a PhD studying consciousness in split-brain patients. A key conclusion from my thesis was that the human brain had evolved the capacity to generate stories: narrations that prevent unconsciously controlled behaviors from challenging our sense of mental unity. Right around the same time, *The Integrated Mind*, a book that Mike and I wrote about our work on consciousness in these patients, was published (Gazzaniga and LeDoux, 1978).

A minor part of my PhD research in the late 1970s involved emotions. Mike observed that not much work was being done on the brain and emotion at the time. Some scientists were flirting with emotion in the brain, and there were also researchers working on topics related to emotion but with no explicit connection to emotion (addiction, stress feeding, sexual behavior). In short, there was not sufficient activity to move emotion forward in neuroscience.

I saw this lack of focus and interest in emotion as an opportunity waiting to be embraced. Because the methods for studying

Received Nov. 18, 2024; revised May 4, 2026; accepted May 28, 2026.

This article provides a summary of my scientific career, especially the early years. It is based on my personal memories, as well as information I published in research articles, reviews, and books over the years. Of note, some text in the present piece about my science career overlaps with text in the science part of my in-press book, *Starting Over: A Neuro-Emotional, Cajun, Rock'n'Roll Memoir* (MIT Press, April 2027). This is because in the process of writing the article, I was also editing the final draft of *Starting Over*. The result was I bit of synergy between some parts of the article and the science parts of the book. Also of note, the nine figures I used in the present article are owned by me and are a subset of a larger set of figures that I designed and had illustrated for *Starting Over*.

This work was supported by the Vulerable Brain Project.

The author declares no competing financial interests.

Correspondence should be addressed to Joseph LeDoux at ledoux@cns.nyu.edu

https://doi.org/10.1523/JNEUROSCI.0172-26.2026

the human brain were limited back then, I decided I would use my limited experience with Thompson at LSU to study rats. I had no illusion that I could study conscious emotion in rats, but I thought that they would at least be useful for understanding the brain mechanisms underlying human emotional behavior, since the circuits involved were long thought to be shared between humans and other mammals (Darwin, 1872; Cannon, 1929; Maclean, 1949). But there was a problem. I needed a lab.

Mike moved to Cornell Medical School in Manhattan in the fall of 1978, and I continued to work with him studying patients with neurological disorders. Knowing my interest in studying emotion, he put me in touch with Don Reis, who directed the Neurobiology Lab at Cornell Med. His research goal was to understand and treat hypertension, i.e., high blood pressure. When we met, he explained that he had a top-notch neuroscience group consisting of anatomists, physiologists, and molecular biologists. All he needed was a behavioral researcher to round out the lab's skill set.

He was understandably hesitant to hire me since I had relatively little experience in animal behavior. But three things were in my favor. First of all, talking about emotion made him nostalgic. A decade earlier as a postdoc, he published a paper on sham rage, an emotional behavior controlled by the hypothalamus (Cannon, 1929; Reis and Fuxe, 1968). The second thing was that I had some understanding, or at least enough understanding, of how to do behavioral studies of rats from my few months with Thompson at LSU. And the third was something I did not find out until I actually joined the lab. My predecessor had died suddenly, and Reis needed a replacement. I wasn't the perfect candidate. I was just the right guy at the right time.

Researching Emotional Behavior

When I started in the Reis Lab, I was assigned the task of taking on the work of my deceased predecessor. And to do that I had to be trained on how to record blood pressure and heart rate in freely behaving rats (Fig. 1). No, you don't put a tiny cuff on their arm. Instead, you have to insert a catheter into an artery and connect the other end to a device that records the level of blood pressure. Once I could do that, I started studying how measurements of blood pressure and heart rate varied while rats went about their daily activities of eating, drinking, grooming, exploring, and sleeping. That was okay, but I was eager to get into the brain.

Research on the brain mechanism of behavior in mammals had typically asked the question "What does a brain area like the hippocampus or cerebellum do?" But studies of split-brain patients taught me to think about how brain functions emerge from information flow between brain areas and how that ceases when the connections between those areas are damaged. I adopted a behavioral task called defense conditioning, invented by Ivan Pavlov (Pavlov, 1927) in the early twentieth century, and renamed as Fear Conditioning by John Watson, the father of behaviorism (1925). With it, a harmless sound, sight, or touch paired with a mild shock becomes a warning signal of danger and later elicits defensive behaviors like freezing or withdrawal. In the 1970s it was used by Eric Kandel (1976) to follow the flow of information, step by step, from the sensory system that processes the conditioned stimulus to the motor system that controls the response. Pavlovian fear conditioning seemed to be the perfect task for me to follow the information flow underlying emotional, especially fear, behavior from sensory to motor systems in mammals, particularly in rats (Fig. 2).

Costantino Iadecola was my best friend in the Reis lab. He was studying the brain mechanisms underlying hypertension in rats

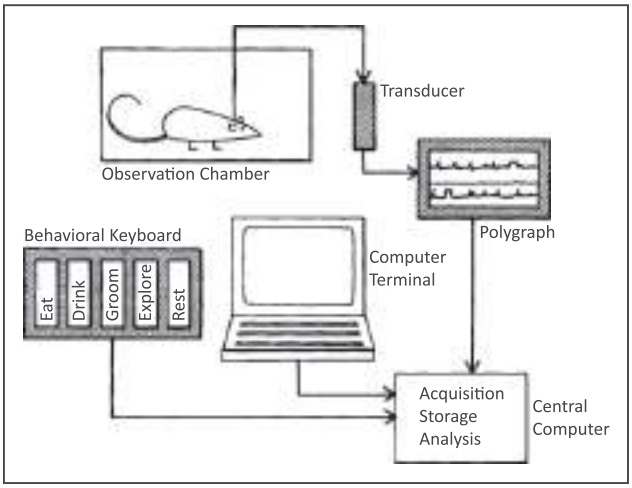

Figure 1. Recording behavior and blood pressure in freely behaving rats. Blood pressure is recorded via a catheter inserted into the thoracic artery in the neck. Pressure measurements are fed through a transducer and presented on a monitor. At the same time, behaviors are recorded on a keyboard. The two sets of data are sent to a central computer for storage and analysis.

by measuring blood flow with a very new brain mapping techniques, and I convinced him that it would be interesting to use his procedure to identify neural pathways activated by emotional stimuli in rats. The method had not been used to study brain and behavior, and Reis gave us the green light, as he always liked being first.

With this procedure you inject a radioactively labeled drug called iodoantipyrine (IAP) into an artery and it travels in the bloodstream to the brain. The amount of blood flowing into a brain area is measured by the metabolic activity of the neurons in the area. The more active the neurons are, the more metabolic fuel they use, and the greater will be the amount of radioactivity. If you present a stimulus for ~30 s after the injection, and then kill the animal immediately, the amount of radioactivity in a given brain area is an indicator of the amount of blood flow, which approximates the level of neural activity the stimulus elicited in the area right before death.

At the time, a similar radiographic approach called the 2-deoxy-D-glucose method was very popular. But it required much longer exposure to stimuli. As a result, this procedure is only suitable for examining the neuronal correlates of mental or behavioral activities that can be effectively sustained in a prolonged steady state—30–45 min. The 30 s stimulus exposure of the IAP method made it far more useful for relating neural activity to ongoing behavior.

Using the IAP method, we found high levels of neural activity throughout the auditory areas of the brain but also found high levels in brain areas previously implicated in emotional behavior, including the hypothalamus (Cannon, 1929; Hess and Brugger, 1943; Bard and Mountcastle, 1948) and the amygdala (Weiskrantz, 1956; Blanchard and Blanchard, 1972; Rolls, 1992). The results were published in the *Science Magazine* (LeDoux et al., 1983).

During the 2 years we were doing the imaging study, I also pursued a more traditional approach to brain and behavior. I used lesions to disrupt auditory processing triggered by a pavlovian fear conditioned tone. But there were two choices about which area to lesion first. You could start at the origin of the auditory system and lesion the nerves connecting the ear to the brain, or you could start with lesions of the auditory cortex,

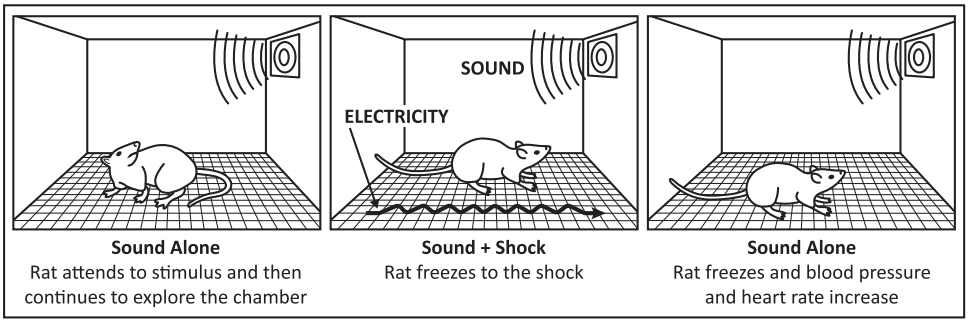

Figure 2. Pavlovian fear conditioning in rats. When a salient but harmless stimulus (a sound) is paired with a noxious stimulus (electric shock), the harmless stimulus begins to evoke the same response as the noxious stimulus, in this case, freezing.

the highest level of auditory processing. Conventional wisdom said that in order for an animal, whether rat or human, to meaningfully respond to an external stimulus, that stimulus had to be processed by the neocortical component of that sensory system. So, it seemed that starting with the auditory cortex would be the best way to approach the problem since we'd ultimately have to go there anyway

But there was a hitch. While the auditory cortex had been well demarcated in humans, monkeys, cats, and some other mammals, its boundaries in rats were not well understood. The IAP method provided a rough approximation of the location of the auditory cortex, but I wanted more precision. Fortunately, a methodological revolution was underway in neuroanatomy as a result of new chemical methods for tracing connections between neurons in different brain areas. Reis, of course, had these procedures in his lab. When you inject one of these "tract-tracers" into a brain area it is absorbed by the neurons in that area and transported down the axons to their target connections. In our case we injected the tracer into the auditory thalamus, which was known to send auditory information to the auditory cortex in all mammals. The location of the tracer defined the auditory cortex.

We used this information to make lesions that damaged the entire auditory cortex. Given that the auditory cortex was assumed to be required for meaningful information processing of sounds, what we discovered was quite surprising. Complete damage to the auditory cortex in both hemispheres had no effect on pavlovian fear conditioning (Fig. 3).

Next, we lesioned the auditory thalamus, which eliminated the ability of rats to express freezing and cardiovascular responses to the tone paired with shock. Now we were really on to something. All we had to do was figure out which areas besides the auditory cortex might be targets of the auditory thalamus. To do this we did additional tracer studies and found that besides connections with the auditory cortex, there were thalamic connections with several subcortical areas, including the hypothalamus and amygdala and striatum, which confirmed the IAP results. It's nice when different approaches tell you the same thing! We then lesioned each and found that lesions of the hypothalamus had no effect, but lesion of the amygdala and striatum did. This tract-tracing/lesion study was my first publication in the *Journal of Neuroscience* (LeDoux et al., 1984).

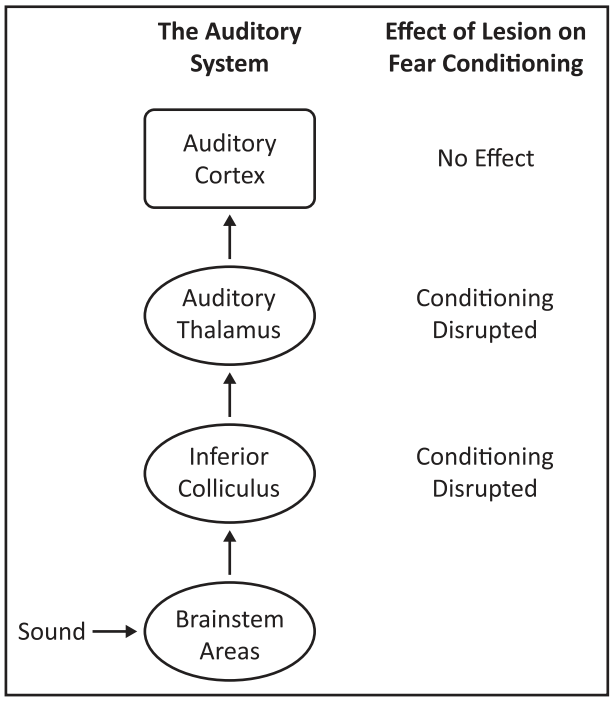

Figure 3. The auditory pathways underlying fear conditioning. The auditory nerve transmits information about sound to the auditory brainstem, which transmits information to the inferior colliculus, which transmits information to the thalamus, which in turn sends the information to the auditory cortex. Lesions of the inferior colliculus or auditory thalamus disrupt auditory conditioning, but lesion of the auditory cortex does not. Such results give clues about the pathways underlying conditioning.

"Emotion Is Not a Neuroscientific Topic"

With my 1983 *Science* and 1984 *Journal of Neuroscience* papers published, I was sure that I had what I needed to get my first NIH grant proposal funded. Alas, the grant was rejected flat out for two reasons. One was because "emotion is not a neuroscientific topic"—clearly a behaviorist assumption of emotion as conscious feelings. The other reason was that I was using pavlovian fear conditioning without including the "crucial nonassociative control group" necessary to show that the conditioned response was learned associatively. But both concerns seemed irrelevant since I was not studying either conscious emotions or learning.

I quickly figured out what was going on. The young field of neuroscience, in an effort to earn its stripes as a serious scientific area, had left "emotion" off the menu—it was too subjective, too "Freudian." Most researchers studying brain and behavior were trained in the behaviorist tradition and called themselves behavioral neuroscientists. They populated the study of learning and memory, and it is very likely that the reviewer who rejected my grant submission was in that camp. I decided that if minimizing my use of the word "emotion" and adding a nonassociative

control group was all I needed for funding, that's what I would do. And indeed, the grant was funded the next round. But I knew emotion was not going to be accepted any time soon. So, to protect my good fortune, I essentially disguised myself as a pavlovian fear conditioning researcher who was focused on learning and memory. I only mentioned emotional consciousness in review articles. As an aside, I think the reason there was not much emotion research in neuroscience was because it was not easy to get funding, and many who did get funding, like me, succeeded by turning to studies of learning and memory.

Learning and Memory as My Cover

The hottest topic in behavioral neuroscience in the mid-1980s was the role of the hippocampus in learning and memory. All one had to do to realize this was to attend the annual meeting of the Society for Neuroscience, where rows and rows of posters focused on the hippocampus, touching on various subthemes, especially place cells, spatial learning, and long-term potentiation. I walked the aisles with both awe and curiosity. I had a poster on fear conditioning and the amygdala, a topic that was hardly represented at all at the meeting. It was there that I met Bruce Kapp, who was also working on fear conditioning and the amygdala and had a poster next to mine. Michael Davis also had a poster on using fear conditioning to measure the extent to which a pavlovian conditioned stimulus, a light in this case, potentiated startle responses. His poster was about the role of the visual cortex in fear-potentiated startle, but he soon ended up studying the amygdala as well. The three of us became close friends and colleagues and played key roles in making research on the amygdala and fear conditioning a hot topic in the field of learning and memory.

I followed up on my 1984 paper in the *Journal of Neuroscience* to examine in more detail which subcortical areas were potentially involved in controlling emotional behaviors. As was the case with the auditory cortex, the auditory thalamus was not well defined in rats. We therefore started by identifying its boundaries and subareas based on work in other mammals (Morest, 1964; Winer and Morest, 1983a,b).

Our 1984 study had used anterograde tracing of connections from the auditory thalamus to subcortical areas. With "retrograde tracing" we found that injections into the amygdala and striatum labeled cells within the auditory thalamus but injections into the hypothalamus only showed connections to cells surrounding the auditory thalamus. Perhaps most important, the retrograde tracing results also showed that the areas of the auditory thalamus that send connections subcortically to the amygdala and striatum were different from areas that connect with the auditory cortex. The study was published in *The Journal of Comparative Neurology* (LeDoux et al., 1985). An important follow-up study showed that lesions of the amygdala disrupted fear conditioning, but lesions of the striatum did not (Iwata et al., 1986). Bingo! We therefore had good evidence that the amygdala was a key structure that received auditory inputs and controlled emotional behavior. But to be clear, the fact that we had found a subcortical thalamic auditory route to the amygdala did not mean that the auditory cortex did not also send inputs to the amygdala in fear conditioning. When we completely lesioned auditory cortex, the thalamic pathway worked fine and when we lesioned the thalamic pathway, the cortical pathway worked fine (Romanski and LeDoux, 1992).

To summarize, our studies identified two sensory routes to the amygdala. The thalamic pathway sends crude but fast auditory information to the amygdala, while the cortical pathway sends more complex auditory information more slowly. The two routes came to be called the low and high roads to the amygdala (Fig. 4). Over the years fMRI, electrical recordings, and tracing studies in humans supported our conclusion about a thalamic shortcut, a low road to the amygdala (Morris et al., 1999; Luo et al., 2010; Tamietto and de Gelder, 2010; Rafal et al., 2015; Kragel et al., 2021).

Some years earlier, Bruce Kapp had found that central amygdala output connections to the brainstem controlled heart rate responses elicited by the conditioned stimulus (Kapp et al., 1978, 1979; Schwaber et al., 1980). But it was not known if freezing and blood pressure were also controlled by outputs of the central amygdala. Tracing studies by others had shown that the central amygdala was connected with the lateral hypothalamus and the periaqueductal gray area, both of which had been implicated in emotional behaviors and blood pressure control. We therefore explored the effects of lesions of these areas on fear conditioning. The results, published in the *Journal of Neuroscience* (LeDoux et al., 1988), showed that the central amygdala did indeed control all three of the responses we were studying, but it did so by way of different output connections: freezing depended on connections of the central amygdala to the rostral periaqueductal gray, and from there to brainstem areas that control skeletal motor neurons; blood pressure depended on connections to the lateral hypothalamus, and from there to the rostral ventral lateral medulla, which controls the sympathetic nervous system; and heart rate, as Kapp showed, depended on connections to brainstem areas that control the parasympathetic nervous system (Fig. 5).

A key issue at that point was whether the central amygdala might be the major link from the auditory system to the downstream outputs. But our tracing studies did not find robust auditory connections to the central amygdala. Instead, we found that

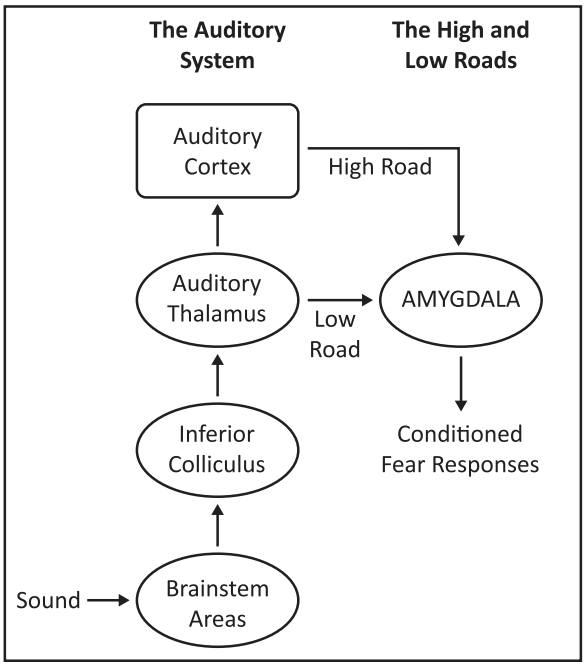

Figure 4. The low and high roads to the amygdala. Tract tracing and other studies have shown that auditory information reaches the amygdala via two pathways: relatively simple auditory information travels directly from the thalamus to the amygdala to enable quick responses, but more complex information arrives more slowly, via the auditory cortex.

auditory inputs mainly entered the amygdala via its lateral nucleus (LeDoux et al., 1985). When our lateral amygdala results were combined with the central amygdala findings, we had a rough picture of how a conditioned tone makes its way through the auditory system to the lateral amygdala, then to the central amygdala, and from there to the control of various emotional responses. But at that time we did not have strong behavioral data to support the idea.

An Opening for Emotion

My lab's work focused exclusively on objectively measurable conditioned fear responses—freezing, blood pressure, and heart rate. But, as I've mentioned, I also wrote about subjective emotional consciousness in reviews, typically toward the end of the chapter. This made me feel like I was still somewhat in the emotion game. Yet, the truth was, these academic publications had low distributions and were not well read. But in 1987 that changed. I wrote a chapter titled "Emotion" in a book called *Higher Functions*, a volume of the prestigious *Handbook of Physiology* (LeDoux, 1987). The editor of the volume was Fred Plum, a leading neurologist of his day, and the Handbook Editor was Vernon Mountcastle, one of most prominent neurophysiologists of his day. In my chapter I summarized the history of emotion research from the late nineteenth century to the present and used fear conditioning research to illustrate how modern neurobiological techniques are helping us understand emotional behavior circuits in the brain. And, of course, at the end of the chapter I pontificated about emotional consciousness in the brain.

Given the lowly status of emotion in neuroscience, why did Mountcastle and Plum, both powerful neuroscientists, publish such a chapter? They both became brain researchers in the mid-twentieth century, Mountcastle by studying physiology in animals and Plum by his work on neurological patients. In medical settings, they were isolated from behaviorist doctrine. Mountcastle, in fact, had done research on emotion early in his career (Bard and Mountcastle, 1948), and Plum, through his work on consciousness in coma patients, was interested in emotional suffering (Plum and Posner, 1966). But I still had to tread carefully, as I never knew when anti-emotion and/or an anti-consciousness behavioral neuroscientist might be reviewing my grants and research articles. But the support by Mountcastle and Plum gave me the nerve to be more aggressive about "emotion." In fact, I started putting "emotion" in the title of review articles I wrote. Mike Davis, my behaviorist-trained amygdala colleague, once asked why I talked about emotion so much. But a few years later, even he was also using the "E" word.

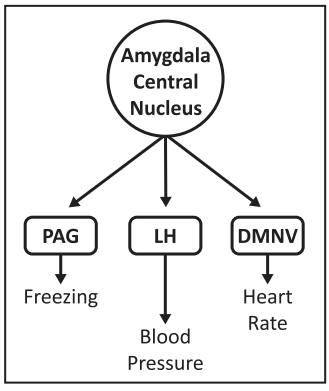

Figure 5. Different outputs of the central nucleus of the amygdala control different kinds of Responses. Heart rate is transmitted in one output, blood pressure another, and freezing still another. See main text for details.

A Lab of My Own

By 1989 I had been in the Reis lab for more than a decade and felt it was time to move on. But I wanted to stay in New York City, so I applied for a position in the new Center for Neural Science at New York University. Much to my surprise, I got the job. I was both very excited and anxious about how things would go in this rigorous, physiology-based, math-loving environment. The fact that I was using state-of-the-art scientific methods and was well funded had gotten me hired. As luck would have it, I got an immunization against my anxiety 2 weeks before my appointment started. On August 15, an article about my research by science writer Daniel Goleman came out in the Science Times section of the *New York Times* (Goleman, 1989). The title was, "Brain's Design Emerges as a Key to Emotions." Goleman was particularly attracted to the subcortical thalamic shortcut to the amygdala that allows us to react before we know what we are reacting to. My work was mainly known by a handful of neuroscientists who were also studying emotion in the brain, such as Jeffrey Gray, Edmund Rolls, John Aggeleton, James McGaugh, Trevor Robbins, Barry Everitt, Antonio Damasio, Richard Davidson, and Jaak Panksep. Thanks to Goleman, that changed instantly, as I suddenly had new scientific and lay followers.

During much of my first year at NYU, I spent time wrapping up four studies that started in Reis's lab. They were all about the lateral nucleus of the amygdala. I had had, since the early 1980s, a sense that the lateral amygdala was an important sensory input zone of the amygdala for fear conditioning (LeDoux et al., 1983). But a set of four studies showed just how important the lateral amygdala was. One study used anterograde and retrograde tract tracing to unambiguously demonstrate that the LA was the main amygdala recipient of auditory information from the thalamus (LeDoux et al., 1990b). We confirmed these connections physiologically by recording neural activity in the lateral amygdala in response to stimulation of the auditory thalamus (Clugnet et al., 1990). Next, we showed that lesions limited to the lateral amygdala disrupted auditory fear conditioning of freezing, blood pressure, and heart rate (LeDoux et al., 1990a). And the fourth study demonstrated that high-frequency stimulation of axons from the auditory thalamus to the lateral amygdala induced lasting synaptic plasticity in the form of long-term potentiation (LTP) in the lateral amygdala (Clugnet et al., 1990). It was quite exciting that all four papers were published in the same volume of the *Journal of Neuroscience*, with three of them back-to-back in the same issue. These four studies helped the amygdala become the second most popular brain area in research on learning and memory. But most important to us was that we now had evidence that the conditioned tone makes its way through the auditory system to the lateral amygdala, then from there to the central amygdala to control the different responses (Fig. 6).

LTP had mostly been used to study memory in the hippocampus. But it seemed that pavlovian fear conditioning in the lateral amygdala might be better suited than hippocampal spatial learning tasks for connecting LTP, synaptic plasticity, and memory together. The reason was that the connection between weak and strong stimuli was more obvious in the lateral amygdala than the hippocampus. This led some researchers who were using knock-out and transgenic mice to study LTP in the hippocampus

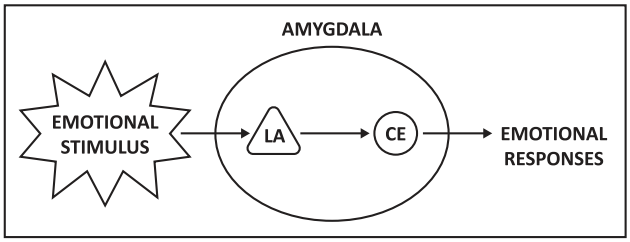

Figure 6. The lateral nucleus (LA) is the sensory gateway to the amygdala and the central nucleus (CE) is the behavioral and physiological output (see details in main text). Lesions and tract tracing studies suggested that auditory information primarily reaches the amygdala via the lateral nucleus, which distributes the information to other amygdala areas.

to turn instead to studies of the lateral amygdala to identify the genes and proteins involved in LTP and fear conditioning.

Finally, Emotion Made It Onto the Neuroscience Menu

During my early tenure at NYU (1989–1995), my laboratory maintained a prolific publication schedule, writing about 50 scientific articles and reviews. In the latter, I emphasized an important distinction, namely, between emotional memory and memory about emotion.

Unconscious/implicit memories (emotional memories) were acquired and stored by the amygdala, while conscious/explicit memories (memories about emotion) were acquired and stored by the hippocampus and related areas of the medial temporal lobe.

In 1994 my work on the "thalamic short-cut" was popularly illustrated by a hiker freezing before a rattlesnake. It hit the mainstream via a *Scientific American* article, "Emotion, Memory and the Brain" (LeDoux, 1994). That, along with the coverage by Dan Goleman in the *New York Times*, thrust me into the public eye. I was thinking about writing a book for the lay public on the implications of the work my lab had done. At the time, writing for a lay audience was a controversial move for a serious scientist, especially a young one; many colleagues viewed it as "undignified." However, I saw peers like Steven Pinker, Mike Gazzaniga, and Antonio Damasio (Damasaio, 1994) finding great success. With Mike's encouragement I decided to take the leap.

The Emotional Brain was published in 1996 (LeDoux, 1996) by Simon and Schuster. Looking back, I believe that my book and Damasio's collectively catalyzed a new era of interest in emotion research among scientists and the public alike. This shift also sparked a 25 year collaborative journey with Elizabeth Phelps, allowing my research on emotional behavior to expand beyond rats to humans (Labar et al., 1995; 1998; LeDoux and Phelps, 2000, 2008; Phelps and LeDoux, 2005; Schiller et al., 2010; Wen et al., 2024).

In the mid-1990s my lab was becoming very involved in the cellular and molecular mechanisms in the amygdala that underlie LTP and fear conditioning (Farb et al., 1995; Li et al., 1995; Weisskopf and LeDoux, 1999; Weisskopf et al., 1999; Bauer et al., 2001; Schafe and LeDoux, 2000; Schafe et al., 2000; Blair et al., 2001; Rodrigues et al., 2001; Rogan et al., 2001). By 2000, our focus shifted from memory consolidation to reconsolidation (Nader et al., 2000; Schafe and LeDoux, 2000; Doyere et al., 2007; Monfils et al., 2009; Schiller et al., 2010). And by 2010, we were using optogenetics to dive deeper into mechanisms that definitively related LTP to fear conditioning (Johansen et al., 2011).

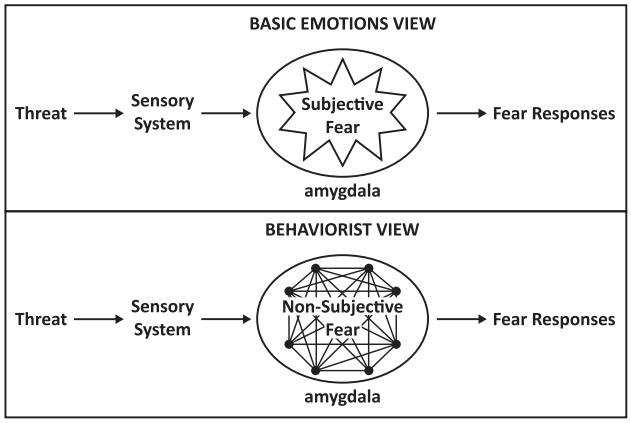

Figure 7. Two traditional views of fear in the amygdala. Researchers in the legacy of Darwin viewed subcortical areas like the amygdala and periaqueductal gray area as responsible for both the conscious experience of fear and its behavioral and physiological correlates. However, researchers trained in the tradition of behaviorism had little interest in conscious feeling and simply used the same term, "Fear" for behavioral and physiological responses.

Rethinking Emotion

Emotion was a thriving research topic in animal and human neuroscience throughout the 2000s and into the 2010s. A good deal of research in my lab continued on the use of pavlovian fear conditioning to study reconsolidation (Diaz et al., 2013; Schiller et al., 2013), and we also did considerable work on the relation of fear conditioning to avoidance behavior (Cain and LeDoux, 2008; Choi et al., 2010; Campese et al., 2013; Martinez et al., 2013). But I was concerned. There was nothing wrong with the research we and others were doing on the amygdala. The problem was with the way the results were being conceived by the whole filed at large. The underlying assumption was that the amygdala was a "fear center." And not just in science. Putting "amygdala and fear" into a Google search bar back then yielded millions of hits. The word "amygdala" showed up in novels, blogs, movies, TV shows, podcasts, plays, songs, legal defenses, and on and on. The amygdala fear center had become a bona fide cultural meme among scientists and in the lay public.

But there were two versions of the fear center in the 2000s (Fig. 7). For some scientists, especially those with a Darwinian, basic emotions perspective, the amygdala and related areas were viewed as being responsible for both conscious feelings of fear and behavioral and physiological correlates (Panksepp, 2005). But for those from a behaviorist perspective who had no interest in consciousness, the amygdala simply controlled behavioral and physiological fear responses nonsubjectively. In short, both perspectives put fear in the amygdala, with it being a subjective experience that underlies emotional behaviors in one case and it being primarily a controller of objective emotional behaviors in the other.

In 2012, I published "Rethinking the Emotional Brain" in *Neuron* (LeDoux, 2012). In it, I rejected both of the amygdala fear center ideas, relabeling the amygdala a defensive survival circuit (Fig. 8).

I used the amygdala subjective fear center idea as one example of the confusing state of affairs in emotion research. Specifically, I proposed that the feeling of fear and the behavioral and physiological responses that co-occur with it depend on different circuits. Because the two circuits are activated simultaneously by the same external stimulus, we intuit that the feeling of fear must be the cause of our responses. But, I argued, this is a

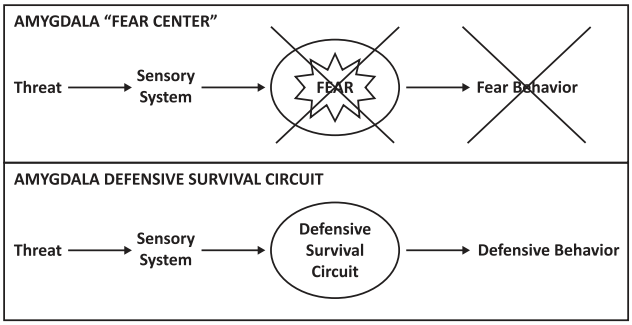

Figure 8. A new view. The amygdala is home to an ancient defensive survival circuit that controls behavioral and physiological responses when one is in danger. It is neither a subjective fear center nor a nonsubjective fear center.

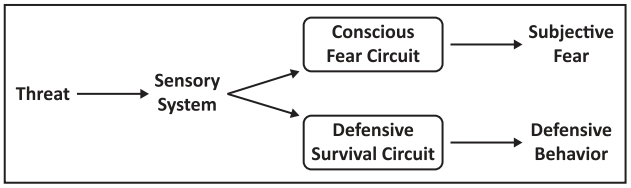

Figure 9. Subjective feelings of fear and objective behavioral and physiological responses are triggered by the same threatening stimulus. Subjective fear objective responses seem so entwined because they are both products of the same situation—the presence of danger (see main text for the consequences of that conflation).

confusion resulting from the mere correlation of the two events in time. I also questioned the objective fear concept, proposing that mental state terms like "fear" only be used to describe conscious emotional states or feelings and that behavioral and physiological correlates be referred to as defensive survival responses (Fig. 9). Both camps pushed back. Basic emotions theorists argued that subjective fear is a subcortical process (Panksepp et al., 2017) and behaviorist leaning researchers argued that subjective fear is a quaint fiction (Fanselow and Pennington, 2017).

I believed that the failure of psychiatry to deliver effective treatments for disorders involving fear and anxiety was due to the conflation of the amygdala's control of ancient defensive survival circuits with cognitive circuits that construct conscious feeling of fear and other emotions. The two occur in parallel and are not one and the same. Drugs that change animal behavior and physiological responses can help tame behavioral and physiological symptoms but will never effectively relieve the subjective feelings of fear or anxiety that prompt people to seek help in the first place.

I continued this effort, especially focusing on the clinical importance of distinguishing subjective conscious feelings from responses based on survival circuits, publishing my ideas in articles in various journals, including *PNAS* (LeDoux, 2014, 2020; LeDoux and Brown, 2017), *Trends in Cognitive Sciences* (LeDoux, 2017), *Current Biology* (LeDoux, 2020, 2021), *Nature Reviews Neuroscience* (LeDoux and Daw, 2018), *Nature Reviews Psychology* (Lau et al., 2022), *Current Trends in Behavioral Sciences* (Mobbs and LeDoux, 2018), *American Journal of Psychiatry* (LeDoux and Pine, 2016), *Philosophical Transactions of the Royal Society of London B* (LeDoux, 2020), *Molecular Psychiatry* (Taschereau-Dumouchel et al., 2022), and *Trends in Neurosciences* (Rust and LeDoux, 2023), as well as in books targeted to lay and scientific audiences, including *Anxious* (LeDoux, 2015), *The Deep History of Ourselves* (LeDoux, 2019), and *The Four Realms of Existence* (LeDoux, 2023). The latter book closed the circle of my 50 year interest in consciousness with a mechanistic hypothesis about how narrations prevent unconsciously controlled emotional behaviors from challenging our conscious sense of mental unity.

Coda

In retrospect, after all these decades I have a better understanding of why some neuroscientists in the 1980s were suspicious about whether emotion could be studied the same way that processes like sensation, movement, perception, and memory were. To the extent that I succeeded in helping emotion prosper in neuroscience, it was because I framed much of my early work around the topic of pavlovian conditioning, studying emotional behavior as a sensory-motor and memory problem, and hence separate from the emotional consciousness problem. But the zeitgeist went in a different direction, either conflating the brain mechanisms underlying emotional behavior with those that construct emotional experiences or ignoring emotional experiences altogether. I am now retired, but I continue my efforts to make emotion a more rigorous topic in neuroscience.

I believe that a new approach is needed, one that fuses the science of emotion with the science of consciousness. Such a science of emotional consciousness does not presently exist for two reasons. One is that researchers who study emotion in the brain have had relatively little interest in and knowledge about the science of consciousness. And the other is that consciousness researchers are far more interested in the conscious perception of visual stimuli than in emotional consciousness. Both fields are diminished by mutual ignorance of the other, and much could be gained if emotion researchers accepted that emotions are fundamentally conscious experiences, and if consciousness researchers accepted that emotions are our most important conscious experiences.

I was especially pleased to be asked to by the Society for Neuroscience to write this piece and to deliver the 2025 History Neuroscience Lecture on "The Troubling Problem of the Emotional Brain." This lecture offered a historical context for challenging issues in modern-day work on Emotion and Consciousness that has roots in similar problems in the late nineteenth century. One problem was the assumption that if a behavior seems conscious in humans, the person must be conscious, and another was if animal behaves in a way similar to a human in a similar situation, it too must be conscious. Empirical grounding for both claims were lacking and led to the ban of consciousness in the first half of the twentieth century (LeDoux et al., 2020; LeDoux, 2021; 2023; 2025). The ban is long gone, but the same problems are returning. Consciousness is again being assumed based on intuition rather than rigor (LeDoux, 2023, 2025; Taschereau-Dumouchel et al., 2026). I am not saying I have the answer. But I do believe that we need to ratchet up on rigor.

I conclude with a point I made at the beginning about the misuse of language:

> "If we don't know what we are looking for, we will surely fail to find it, and we will mislead ourselves and others in the process."

References

Arnold MB (1960) *Emotion and personality*. New York: Columbia University Press.

Bard P, Mountcastle VP (1948) Some forebrain mechanisms involved in expression of rage with special reference to suppression of angry behavior. Res Publ Assoc Res Nerv Ment Dis 27:362–404.
Bauer EP, LeDoux JE, Nader K (2001) Fear conditioning and LTP in the lateral amygdala are sensitive to the same stimulus contingencies. Nat Neurosci 4:687–688.
Blair HT, Schafe GE, Bauer EP, Rodrigues SM, LeDoux JE (2001) Synaptic plasticity in the lateral amygdala: a cellular hypothesis of fear conditioning. Learn Mem 8:229–242.
Blanchard DC, Blanchard RJ (1972) Innate and conditioned reactions to threat in rats with amygdaloid lesions. J Comp Physiol Psych 81:281–290.
Cain CK, LeDoux JE (2008) Brain mechanisms of Pavlovian and instrumental aversive conditioning. In: *Handbook of anxiety and fear* (Blanchard RJ, Blanchard DC, Griebel G, Nutt D, eds), pp 103–124. Jordan Hill: Academic Press.
Campese V, McCue M, Lazaro-Munoz G, LeDoux JE, Cain CK (2013) Development of an aversive Pavlovian-to-instrumental transfer task in rat. Front Behav Neurosci 7:176.
Cannon WB (1929) *Bodily changes in pain, hunger, fear, and rage*. New York: Appleton.
Choi JS, Cain CK, LeDoux JE (2010) The role of amygdala nuclei in the expression of auditory signaled two-way active avoidance in rats. Learn Mem 17:139–147.
Clugnet MC, LeDoux JE, Morrison SF (1990) Unit responses evoked in the amygdala and striatum by electrical stimulation of the medial geniculate body. J Neurosci 10:1055–1061.
Damasaio A (1994) *Descartes's error*. New York: Grosett, Putnam.
Darwin C (1872) *The expression of the emotions in man and animals*. London: Fontana Press.
Diaz-Mataix L, Ruiz Martinez RC, Schafe GE, LeDoux JE, Doyere V (2013) Detection of a temporal error triggers reconsolidation of amygdala-dependent memories. Curr Biol 23:467–472.
Doyere V, Debiec J, Monfils MH, Schafe GE, LeDoux JE (2007) Synapse-specific reconsolidation of distinct fear memories in the lateral amygdala. Nat Neurosci 10:414–416.
Fanselow MS, Pennington ZT (2017) The danger of LeDoux and pine's two-system framework for fear. Am J Psychiatry 174:1120–1121.
Farb CR, Aoki C, LeDoux JE (1995) Differential localization of NMDA and AMPA receptor subunits in the lateral and basal nuclei of the amygdala: a light and electron microscopic study. J Comp Neurol 362:86–108.
Festinger L (1957) *A theory of cognitive dissonance*. Evanston, IL: Row, Peterson.
Gazzaniga MS, LeDoux JE (1978) *The integrated mind*. New York: Plenum.
Goleman D (1989) *Brain's design emerges as a key to emotions*. New York: The New York Times, The New York Times.
Hess WR, Brugger M (1943) Das subkortikale Zentrum der affektiven Abwehrreaktion. Helv Physiol Pharmacol Acta 1:33–52.
Iwata J, LeDoux JE, Meeley MP, Arneric S, Reis DJ (1986) Intrinsic neurons in the amygdaloid field projected to by the medial geniculate body mediate emotional responses conditioned to acoustic stimuli. Brain Res 383:195–214.
James W (1884) What is an emotion. Mind, 188–205.
Johansen JP, Cain CK, Ostroff LE, LeDoux JE (2011) Molecular mechanisms of fear learning and memory. Cell 147:509–524.
Kandel ER (1976) *Cellular basis of behavior: an introduction to behavioral neurobiology*. San Francsisco: W.H. Freeman and Company.
Kapp BS, Frysinger RC, Gallagher M, Schwaber JS (1978) The central nucleus of the amygdala: bulbar projection and involvement in heart rate conditioning. Sot Neurosci Abstr 24:1–23.
Kapp BS, Frysinger RC, Gallagher M, Haselton JR (1979) Amygdala central nucleus lesions: effect on heart rate conditioning in the rabbit. Physiol Behav 23:1109–1117.
Kragel PA, Ceko M, Theriault J, Chen D, Satpute AB, Wald LW, Lindquist MA, Feldman Barrett L, Wager TD (2021) A human colliculus-pulvinar-amygdala pathway encodes negative emotion. Neuron 109:2404–2412 e2405.
LaBar KS, LeDoux JE, Spencer DD, Phelps EA (1995) Impaired fear conditioning following unilateral temporal lobectomy in humans. J Neurosci 15:6846–6855.
LaBar KS, Gatenby JC, Gore JC, LeDoux JE, Phelps EA (1998) Human amygdala activation during conditioned fear acquisition and extinction: a mixed-trial fMRI study. Neuron 20:937–945.
Lau H, Michel M, LeDoux JE, Fleming SM (2022) The mnemonic basis of subjective experience. Nat Rev Psychol 1:479–488.
LeDoux JE, Thompson ME, Iadecola C, Tucker LW, Reis DJ (1983) Local cerebral blood flow increases during auditory and emotional processing in the conscious rat. Science 221:576–578.
LeDoux JE, Sakaguchi A, Reis DJ (1984) Subcortical efferent projections of the medial geniculate nucleus mediate emotional responses conditioned to acoustic stimuli. J Neurosci 4:683–698.
LeDoux JE, Ruggiero DA, Reis DJ (1985) Projections to the subcortical forebrain from anatomically defined regions of the medial geniculate body in the rat. J Comp Neurol 242:182–213.
LeDoux JE (1987) Emotion. In: *Handbook of physiology 1: the nervous system Vol V, higher functions of the brain, vol.* (Plum F, ed), pp 419–459. Bethesda: American Physiological Society.
LeDoux JE, Iwata J, Cicchetti P, Reis DJ (1988) Different projections of the central amygdaloid nucleus mediate autonomic and behavioral correlates of conditioned fear. J Neurosci 8:2517–2529.
LeDoux JE, Cicchetti P, Xagoraris A, Romanski LM (1990a) The lateral amygdaloid nucleus: sensory interface of the amygdala in fear conditioning. J Neurosci 10:1062–1069.
LeDoux JE, Farb C, Ruggiero DA (1990b) Topographic organization of neurons in the acoustic thalamus that project to the amygdala. J Neurosci 10: 1043–1054.
LeDoux JE (1994) Emotion, memory and the brain. Sci Am 270:50–57.
LeDoux JE (1996) *The emotional brain*. New York: Simon and Schuster.
LeDoux JE (2014) Coming to terms with fear. Proc Natl Acad Sci U S A 111: 2871–2878.
LeDoux JE (2015) *Anxious: using the brain to understand and treat fear and anxiety*. New York: Viking.
LeDoux JE (2017) Semantics, surplus meaning, and the science of fear. Trends Cogn Sci 21:303–306.
LeDoux JE (2020) Thoughtful feelings. Curr Biol 30:R619–R623.
LeDoux JE, Michel M, Lau H (2020) A little history goes a long way toward understanding why we study consciousness the way we do today. Proc Natl Acad Sci U S A 117:6976–6984.
LeDoux JE (2021) What emotions might be like in other animals. Curr Biol 31:R824–R829.
LeDoux JE (2023) *The four realms of existence: a new theory of being human*. Cambridge, MA: The Belknap Press of Harvard University Press.
LeDoux JE (2025) What the functions of consciousness are depends on what one thinks consciousness is. Philos Trans R Soc Lond, B, Biol Sci 380: 20240311.
LeDoux JE, Brown R (2017) A higher-order theory of emotional consciousness. Proc Natl Acad Sci U S A 114:E2016–E2025.
LeDoux JE, Lau H (2020) Seeing consciousness through the lens of memory. Curr Biol 30:R1018–R1022.
LeDoux JE, Phelps EA (2000) Emotional networks in the brain. In: *Handbook of emotions*, Ed 2 (Lewis M, Haviland-Jones JM, eds). New York: Guilford Press.
LeDoux JE, Phelps EA (2008) Emotional networks in the brain. In: *Handbook of emotions*, Ed 3 (Lewis M, Haviland-Jones JM, Barrett LF, eds), pp 159–179. New York: Guilford Press.
LeDoux JE, Pine DS (2016) Using neuroscience to help understand fear and anxiety: a two-system framework. Am J Psychiatry 173:1083–1093.
LeDoux J (2012) Rethinking the emotional brain. Neuron 73:653–676.
LeDoux J (2019) *The deep history of ourselves: the four-billion-year story of how we got conscious brains*. New York: Viking.
LeDoux J, Daw ND (2018) Surviving threats: neural circuit and computational implications of a new taxonomy of defensive behaviour. Nat Rev Neurosci 19:269–282.
Li XF, Phillips R, LeDoux JE (1995) NMDA and non-NMDA receptors contribute to synaptic transmission between the medial geniculate body and the lateral nucleus of the amygdala. Exp Brain Res 105:87–100.
Luo Q, Holroyd T, Majestic C, Cheng X, Schechter J, Blair RJ (2010) Emotional automaticity is a matter of timing. J Neurosci 30:5825–5829.
MacLean PD (1949) Psychosomatic disease and the "visceral brain": recent developments bearing on the Papez theory of emotion. Psychosom Med 11:338–353.
Martinez RC, Gupta N, Lazaro-Munoz G, Sears RM, Kim S, Moscarello JM, LeDoux JE, Cain CK (2013) Active vs. reactive threat responding is associated with differential c-Fos expression in specific regions of amygdala and prefrontal cortex. Learn Mem 20:446–452.
Mobbs D, LeDoux JE (2018) Survival behaviors and circuits. Curr Opin Behav Sci 24:168–171.

Monfils MH, Cowansage KK, Klann E, LeDoux JE (2009) Extinction-reconsolidation boundaries: key to persistent attenuation of fear memories. Science 324:951–955.
Morest DK (1964) The neuronal architecture of the medial geniculate body of the cat. J Anat 98:611–630.
Morris JS, Ohman A, Dolan RJ (1999) A subcortical pathway to the right amygdala mediating "unseen" fear. Proc Natl Acad Sci U S A 96:1680–1685.
Nader K, Schafe GE, LeDoux JE (2000) The labile nature of consolidation theory. Nat Rev Neurosci 1:216–219.
Panksepp J (2005) Affective consciousness: core emotional feelings in animals and humans. Conscious Cogn 14:30–80.
Panksepp J, Lane RD, Solms M, Smith R (2017) Reconciling cognitive and affective neuroscience perspectives on the brain basis of emotional experience. Neurosci Biobehav Rev 76:187–215.
Pavlov IP (1927) *Conditioned reflexes*. London: Oxford University Press.
Phelps EA, LeDoux JE (2005) Contributions of the amygdala to emotion processing: from animal models to human behavior. Neuron 48:175–187.
Plum F, Posner JB (1966) *Diagnosis of stupor and coma*. Philadelphia: FA Davis.
Rafal RD, Koller K, Bultitude JH, Mullins P, Ward R, Mitchell AS, Bell AH (2015) Connectivity between the superior colliculus and the amygdala in humans and macaque monkeys: virtual dissection with probabilistic DTI tractography. J Neurophysiol 114:1947–1962.
Reis DJ, Fuxe K (1968) Evidence that brain noradrenalin release in essential for sham rage behavior produced by acute brainstem transection in cat. Trans Am Neurol Assoc 93:268–270.
Rodrigues SM, Schafe GE, LeDoux JE (2001) Intra-amygdala blockade of the NR2B subunit of the NMDA receptor disrupts the acquisition but not the expression of fear conditioning. J Neurosci 21:6889–6896.
Rogan MT, Weisskopf MG, Huang Y-Y, Kandel ER, LeDoux JE (2001) Long-term potentiation in the amygdala: implications for memory. In: *Neuronal mechanisms of memory formation,* (Holscher C, ed.), pp 58–76. Cambridge: Cambridge University Press.
Rolls ET (1992) Neurophysiology and functions of the primate amygdala. In: *The amygdala: neurobiological aspects of emotion, memory, and mental dysfunction* (Aggleton JP, ed), pp 143–165. New York: Wiley-Liss.
Romanski LM, LeDoux JE (1992) Equipotentiality of thalamo-amygdala and thalamo-cortico-amygdala circuits in auditory fear conditioning. J Neurosci 12:4501–4509.
Rust NC, LeDoux JE (2023) The tricky business of defining brain functions. Trends Neurosci 46:3–4.
Schachter S, Singer J (1962) Cognitive, social and physiological determinants of emotional states. Psych Rev 69:379–399.
Schafe GE, Atkins CM, Swank MW, Bauer EP, Sweatt JD, LeDoux JE (2000) Activation of ERK/MAP kinase in the amygdala is required for memory consolidation of Pavlovian fear conditioning. J Neurosci 20:8177–8187.
Schafe GE, LeDoux JE (2000) Memory consolidation of auditory Pavlovian fear conditioning requires protein synthesis and protein kinase a in the amygdala. J Neurosci 20:RC96.
Schiller D, Monfils MH, Raio CM, Johnson DC, LeDoux JE, Phelps EA (2010) Preventing the return of fear in humans using reconsolidation update mechanisms. Nature 463:49–53.
Schiller D, Kanen JW, LeDoux JE, Monfils MH, Phelps EA (2013) Extinction during reconsolidation of threat memory diminishes prefrontal cortex involvement. Proc Natl Acad Sci U S A 110:20040–20045.
Schwaber JS, Kapp BS, Higgins G (1980) The origin and extent of direct amygdala projections to the region of the dorsal motor nucleus of the vagus and the nucleus of the solitary tract. Neurosci Lett 20:15–20.
Tamietto M, de Gelder B (2010) Neural bases of the non-conscious perception of emotional signals. Nat Rev Neurosci 11:697–709.
Taschereau-Dumouchel V, Michel M, Lau H, Hofmann SG, LeDoux JE (2022) Putting the "mental" back in "mental disorders": a perspective from research on fear and anxiety. Mol Psychiatry 27:1322–1330.
Taschereau-Dumouchel V, Hwang JS, Lau J, LeDoux JE (2026) The ethical impasse of current consciousness science. Neuron 114:2084–2088.
Tomkins S (1962) *Affect, imagery, and consciousness*. New York: Springer.
Watson JB (1925) *Behaviorism*. New York: WW Norton.
Weiskrantz L (1956) Behavioral changes associated with ablation of the amygdaloid complex in monkeys. J Comp Physiol Psychol 49:381–391.
Weisskopf MG, Bauer EP, LeDoux JE (1999) L-type voltage-gated calcium channels mediate NMDA-independent associative long-term potentiation at thalamic input synapses to the amygdala. J Neurosci 19:10512–10519.
Weisskopf MG, LeDoux JE (1999) Distinct populations of NMDA receptors at subcortical and cortical inputs to principal cells of the lateral amygdala. J Neurophysiol 81:930–934.
Wen Z, Pace-Schott EF, Lazar SW, Rosen J, Ahs F, Phelps EA, LeDoux JE, Milad MR (2024) Distributed neural representations of conditioned threat in the human brain. Nat Commun 15:2231.
Winer JA, Morest DK (1983a) The medial division of the medial geniculate body of the cat: implications for thalamic organization. J Neurosci 3:2629–2651.
Winer JA, Morest DK (1983b) The neuronal architecture of the dorsal division of the medial geniculate body of the cat. A study with the rapid Golgi method. J Comp Neurol 221:1–30.