Coming to terms with fear

Joseph E. LeDoux

Center for Neural Science and Department of Psychology, New York University, New York, NY 10003; Department of Psychiatry and Department of Child and Adolescent Psychiatry, NYU Langone Medical Center, New York, NY 10016; and The Nathan Kline Institute for Psychiatric Research, Orangeburg, NY 10962

This contribution is part of the special series of Inaugural Articles by members of the National Academy of Sciences elected in 2013.

Contributed by Joseph E. LeDoux, January 9, 2014 (sent for review November 29, 2013)

The brain mechanisms of fear have been studied extensively using Pavlovian fear conditioning, a procedure that allows exploration of how the brain learns about and later detects and responds to threats. However, mechanisms that detect and respond to threats are not the same as those that give rise to conscious fear. This is an important distinction because symptoms based on conscious and nonconscious processes may be vulnerable to different predisposing factors and may also be treatable with different approaches in people who suffer from uncontrolled fear or anxiety. A conception of so-called fear conditioning in terms of circuits that operate nonconsciously, but that indirectly contribute to conscious fear, is proposed as way forward.

Pavlovian conditioning | emotion | survival circuits | global organismic states | consciousness

Hunger, like, anger, fear, and so forth, is a phenomenon that can be known only by introspection. When applied to another...species, it is merely a guess about the possible nature of the animal's subjective state.

Nico Tinbergen (1)

Neuroscientists use “fear” to explain the empirical relation between two events: for example, rats freeze when they see a light previously associated with electric shock. Psychiatrists, psychologists, and most citizens, on the other hand, use...“fear” to name a conscious experience of those who dislike driving over high bridges or encountering large spiders. These two uses suggest...several fear states, each with its own genetics, incentives, physiological patterns, and behavioral profiles.

Jerome Kagan (2)

My research focuses on how the brain detects and responds to threats, and I have long argued that these mechanisms are distinct from those that make possible the conscious feeling of fear that can occur when one is in danger (3–6). However, I, and others, have called the brain system that detects and responds to threats the fear system. This was a mistake that has led to much confusion. Most people who are not in the field naturally assume that the job of a fear system is to make conscious feelings of fear, because the common meaning of fear is the feeling of being afraid. Although research on the brain mechanisms that detect and respond to threats in animals has important implications for understanding how the human brain feels fear, it is not because the threat detection and defense responses mechanisms are fear mechanisms. It is instead because these nonconscious mechanisms initiate responses in the brain and body that indirectly contribute to conscious fear.

In this article, I focus on Pavlovian fear conditioning, a procedure that has been used extensively to study the so-called fear system. I will propose and defend a different way of talking about this research, one that focuses on the actual subject matter and data (threat detection and defense responses) and that is less likely to compel the interpretation that conscious states of fear underlie defense responses elicited by conditioned threats. It will not be easy to give up the term fear conditioning, but I think we should.

Pavlovian Fear Conditioning: A Technique and a Process

Fear is the most extensively studied emotion, and the way it has most often been investigated is through Pavlovian fear conditioning. This procedure involves presenting a biologically neutral conditioned stimulus (CS), often a tone, with a noxious or harmful unconditioned stimulus (US), typically a mild electric shock. As a result, the CS comes to elicit species-typical (presumably innate) behavioral responses (e.g., freezing behavior) and supporting physiological adjustments controlled by the autonomic nervous system (e.g., changes in heart rate, blood pressure, respiration) or by endocrine systems (e.g., adrenocorticotropic hormone, cortisol, epinephrine) (7–12). Through fear conditioning, researchers thus have control of the antecedent conditions (the independent variables, namely the CS and US) and can measure the outcomes (dependent variables, such as freezing behavior or autonomic nervous system responses).

The fear-conditioning procedure works because it taps into a process called associative learning that is a feature of circuits in the nervous systems of many if not all animals (4, 13–16) and may also exist in single-cell organisms (17, 18). When associative learning occurs in the circuit engaged by the fear conditioning procedure, the learning process itself is also called fear conditioning. The fear-conditioning process allows the US to alter the effectiveness of the CS in activating circuits that control defense responses in anticipation of harm.

Fear conditioning has many attractive features as a laboratory tool. It is rapidly acquired (19), and is long-lasting, often persisting throughout life (20). Also, it can be used across a wide range of animals, including vertebrates and invertebrates (4, 13, 14, 16), allowing explorations of the extent to which similar mechanisms underlie the conditioning process in diverse organisms (21).

Fear Conditioning During the Age of Behaviorism

Fear conditioning is often said to endow the CS with the ability to elicit fear. It is, after all, called fear conditioning. However,

Significance

Research on Pavlovian fear conditioning has been very successful in revealing what has come to be called the brain’s fear system. The field has now matured to the point where a sharper conceptualization of what is being studied could be very useful as we go forward. Terms like “fear conditioning” and “fear system” blur the distinction between processes that give rise to conscious feelings of fear and nonconscious processes that control defense responses elicited by threats. These processes interact but are not the same. Using terms that respect the distinction will help focus future animal research on brain circuits that detect and respond to threats, and should also help clarify the implications of this work for understanding how normal and pathological feelings of fear come about in the human brain.

Author contributions: J.E.L. wrote the paper.

The author declares no conflict of interest.

Freely available online through the PNAS open access option. See QnAs on page 2860.

1E-mail: ledoux@cns.nyu.edu.
what different researchers have meant by fear has been a moving target since the procedure was first used.

The story begins with John Watson, the father of behaviorism (22). As is well known, the behaviorists banished consciousness from psychology, focusing instead on observable events. However, they did not eliminate mental state terms—fear was still studied but was viewed as something other than a feeling.

Watson, following Ivan Pavlov (23), viewed fear as a conditionable reflex (22) and used Pavlov’s defensive conditioning procedure to condition the fear reflex in a young boy (24). B. F. Skinner, another behaviorist, adopted a different approach, instrumental (operant) conditioning, in which behavior is learned by its consequences (25). Fear became a behavioral disposition determined by a history of aversive reinforcement.

Watson and Skinner were opposed to assumptions about unobservable events inside the head. However, Edward Tolman found a way to call upon inner factors and still be a behaviorist (26). The inner factors were psychological but not conscious; they were “intervening variables” defined by the empirical relation between observable independent and dependent variables. Fear, for example, was an intervening variable that accounted for the expression of defensive behaviors in the presence of a threat. Importantly, intervening variables were not entities (states or processes) but instead descriptions of the relation between observable events. Tolman emphasized that this approach could lead to study introspecting and nonintrospecting organisms alike (27).

While behaviorism was flourishing, so was Sigmund Freud’s psychoanalytic theory, which emphasized drives as inner forces of motivation (28). Clark Hull (29) integrated Freud’s drive theory with Tolman’s intervening-variable approach, arguing that reinforcement of behavior during learning results from reduction in a physiological drive state. For example, food deprivation increases drive, and behaviors that lead to food are reinforced by the reduction in drive that follows eating of the food. Intervening variables were at their most part, abstract psychological constructs for Tolman but were physiological states for Hull.

Two of Hull’s protégés, O. Hobart Mowrer and Neal Miller, developed the view that fear is a learned drive state that comes to be elicited by the CS after Pavlovian conditioning with a shock US (30–33). They used an instrumental task called avoidance conditioning in which rats learn to perform responses that reduce shock exposure. Skinner said that avoidance conditioning was reinforced by escape from the shock, but Mowrer and Miller proposed that avoidance is reinforced by reduction in a CS-elicited fear drive. Early in training, stimuli in the chamber become CSs that are associated with the US. Exposure to the CSs then elicits a fear state that motivates performance of behaviors that eliminates CS exposure, thus reducing the state. (A related two-process theory was proposed by Konorski and Miller; see ref. 34.)

Over the subsequent decades, much research was done to evaluate the role of fear in avoidance (35–44). Drives came to be called central (i.e., brain) motive states (34, 45, 46). Because so little was known about the brain, Donald Hebb referred to central states as existing in a conceptual nervous system rather than the central nervous system (47). However, drive proved problematic as an all-purpose explanation of motivation because organisms are also impelled to act by external incentives (48, 49, 50). Still, central states survived because incentives were also said to activate motivational states that control behavior. Robert Bolles, for example, argued that avoidance does not reflect reduction in fear drive, but instead results, because the CS activates a fear system that generates a fear state, and this limits behavioral options to species-specific defense responses (9, 39).

The defense response selection rules turn out to be more complex than this (51, 52) but are still said to involve activation of a fear system by stimuli that predict harm (19, 53) and also, for some, a central state of fear that causes defense responses (38, 41, 54–57).

Originally, intervening variables were conceived of as abstract constructs (a means of connecting observable independent and dependent variables), with no “surplus meaning” that implied psychological or physiological entities (states or processes) that intervene between stimuli and responses (58). However, drive and fear theories led to much discussion about the relative merits of pure intervening variables vs. intervening variables that implied hypothetical entities to explain behavior (58–60). Tolman later acknowledged that at times he was actually referring to hypothetical constructs (59). Although hypothetical constructs are generally viewed as acceptable when empirically grounded in observable events, they cause problems when reified and given a status that is not empirically verifiable (e.g., when the construct is named with a common language term that implies a psychological state, and the construct then takes on assumed attributes of the state in theory and data explanation) (58–60). Fear as a nonsubjective physiological state that intervenes between stimuli and responses is a potentially verifiable construct (35). However, when fear takes on its received meaning as a conscious feeling, and researchers start looking for properties associated with human fearful feelings in animals, the more problematic kind of hypothetical construct exists.

The expression “state of fear,” practically begs the reader to think of rats feeling afraid of the CS and to think that this feeling is the cause of defensive behavior. However, because the research discussed above was done by researchers who were working along the behaviorist tradition, it seems likely that they were thinking along the lines of empirically verifiable constructs and not in terms of unverifiable feelings in their animal subjects. In fact, a variety of empirically based interpretations of fear were proposed (9, 35–44). On the other hand, Mowrer, a leading figure in this field, explicitly endowed the central state of fear with subjective properties that were said to cause behavior. For example, Mowrer wrote that “consciously experienced fear... must invariably be present, in some degree, as the cause of the observed behavior” (30), and “we do not have to say that the rat runs in order to avoid the shock; we can say instead that the rat runs because (or by-cause) of fear” (31). However, even authors who seemingly adhered to empirically based approaches wrote about fear in a way that could easily be interpreted to mean a subjective feeling. For example, Bolles mentions the “frightened rat” (39), McAllister and McAllister say the CS is an elicitor of fear” (38), and Kamin and colleagues describe rats as being “very fearful of the CS.” Adding to the ambiguity is the fact that in a given paper the word fear would sometimes be in quotes and sometimes not, implying that two kinds of fear were being discussed, but without explaining the different uses (37).

To try to gain some clarity on the nature of what fear really meant in this literature, I contacted several of the behavioral researchers who played an active role in this work and asked whether they were thinking of fear as a conscious feeling, along the lines of Mowrer, or as an intervening variable that did not imply subjective states. Robert Rescorla, Bruce Overmier, Donald Levis, and Michael Fanselow responded, each noting that they did not view fear as conscious feeling but instead as an empirically defined term based on observable events. (These comments were obtained through e-mail correspondence. Respondents agreed to be quoted.) For example, Rescorla noted: “I do not think that reference to subjective experiences (by which I mean private experiences not subject to independent inter-observer verification) is especially useful.” Fanselow said: “I feel that part of our job is to redefine the concept of motivation in a scientific manner and that those new definitions should replace the layman’s informal view. I don’t see how subjective experience helps us do that.”

With the constraints of behaviorism loosened, animal consciousness is no longer a taboo topic (61–66). Lacking the conceptual and historical foundations needed to navigate the ambiguous use of fear and the subtle issues that were being ignored with the behaviorist focus on the more objective, intervening variable, readers today (including scientists, journalists, and lay people) are easily drawn toward the conventional meaning of fear as a conscious feeling and to the everyday belief that fearful feelings cause us to respond in a certain way to threats. Let us look at contemporary brain research to see why this view is neither necessary nor desirable.
Conditioned Fear as a Circuit Function

In the 1950s, avoidance conditioning became the main task used to explore brain mechanisms of fear and aversive learning (67–72). However, this work led to inconclusive results (4, 68, 69, 73). By the 1980s, researchers interested in learning in mammals and other vertebrates turned to Pavlovian conditioning (74, 75), inspired by the success of simple conditioning approaches in studies of invertebrates (14, 16, 76, 77). This strategy worked remarkably well, and Pavlovian fear conditioning became the “go-to” method in mammals for studying aversive learning (4, 54, 78), as well as for studies of the relation between emotion and memory (4, 79).

The neural circuits and cellular, synaptic, and molecular mechanisms underlying the acquisition and expression of conditioned fear responses have been characterized in detail (4, 5, 53, 80–82). (For a different perspective on the circuitry, see refs. 49 and 85.) The lateral nucleus of the amygdala (LA) receives sensory inputs about the CS and US. Before training, the CS only weakly activates LA neurons. After the CS is paired with the US, the ability of the CS to activate the LA increases. When the CS later occurs alone, CS activation of the LA leads to neural activity that propagates through amygdala circuits to the central nucleus (CeA). Output connections of CeA then result in the expression of defensive behavior and physiological responses, as well changes brain arousal. Plasticity also occurs in the central nucleus of the amygdala (84–86) and in CS sensory processing areas (87). At the cellular and molecular levels, fear conditioning occurs when LA neurons that process the CS are weakly activated at the same time that the US strongly depolarizes the LA neurons (5, 53, 80, 81, 88, 89). This results in an increase in the strength of the synapses that process the CS, allowing it to more effectively activate amygdala circuits. Molecular mechanisms engaged result in gene expression and protein synthesis, stabilizing temporary changes in synaptic strength and creating long-term memories. Many of the molecular findings were pursued following leads from invertebrate work (14, 77, 90), following.

Fear conditioning thus became a process that is carried out by cells, synapses, and molecules in specific circuits of the nervous system. As such, fear conditioning is explainable solely in terms of associations created and stored via cellular, synaptic, and molecular plasticity mechanisms in amygdala circuits. When the CS later occurs, it activates the association and leads to the expression of species-typical defensive responses that prepare the organism to cope with the danger signaled by the CS. There is no need for conscious feelings of fear to intervene. The circuit function is the intervening variable. Yet, I and others mulled the waters by continuing to call the circuits involved in detecting and responding to threats the fear system (4, 19, 54).

Nonconscious Conditioned Fear in Humans

Embedded in the intervening variable approach was the assumption that the relevant factors (the observable facts) in the brain could in principle be accounted for in introspecting and nonintrospecting organisms. And the neuroscience perspective described above provided a biological account that made it unnecessary to call upon conscious fear to account for the data. However, findings from studies of fear conditioning in humans made it unnecessary to further tiptoe around consciousness, because the relation between conscious and nonconscious processing can be directly evaluated in our species.

Research on patients with brain damage revealed that fear conditioning creates implicit (nonconscious) memories that are distinct from explicit/declarative (conscious) memory (4, 5, 91, 92). Thus, damage to the hippocampus in humans disrupts explicit conscious memory of having been conditioned but has no effect on fear conditioning itself, whereas damage to the amygdala disrupts fear conditioning but not the conscious memory of having been conditioned (93, 94). Furthermore, behavioral studies in healthy humans have found that conditioned or unconditioned threats presented subliminally elicit physiological responses without the person being aware of the stimulus (95–99) and without reporting any particular feeling, even when instructed to try to introspect about feelings (98). The conditioning process can also be carried out nonconsciously (99–101) and without awareness of the CS–US contingency (102). Acquisition effects are sometimes weaker (101), but this is likely attributable to the degraded input required to prevent awareness than to the limits of nonconscious processing per se because complex cognitive and social processes that control human behavior are often carried out without conscious awareness of their occurrence in daily life (103–105). Also, functional imaging studies have shown that the amygdala is activated when conditioned or unconditioned threats are presented with or without CS awareness (97, 98, 101, 106–112). Under certain conditions of attentional load, subliminal activation of the amygdala is reduced but not eliminated (e.g., ref. 113). Amygdala, but not hippocampal, activation also occurs when subjects are unaware of the CS–US contingency (114). Finally, in people with blindness attributable to damage to visual cortex, visual threats elicit body responses and amygdala activation without awareness of the stimulus and without any obvious feeling of fear (115–118).

If conditioned fear responses do not require consciousness in humans, we should not call upon conscious mental states to explain how a CS elicits freezing and autonomic conditioned responses in animals. Behavioral and physiological responses elicited by a CS tell us about the CS and control the responses. Fear (in the sense of a conscious feeling) is not in that causal sequence. Conscious fear can occur when the conditions are favorable, but such conscious states come about through different processes that involve different circuits. The function of the neural circuit that underlies fear conditioning is to coordinate brain and body resources to increase the chance of surviving the encounter predicted by the CS with minimal adverse consequences (3, 9, 42, 51, 119), not to make conscious fear.

A striking example of the problems caused by the ambiguous use of the term fear comes from a recent study showing that a woman with bilateral amygdala damage could still experience “feelings of fear” (120). This was surprising to the authors and to journalists. Science, Nature, and other esteemed publications published stories with dramatic headlines about the study. However, the only reason this would be surprising is if one believed that the amygdala is the wellspring of fearful feelings and that amygdala-controlled responses are reliable markers of these feelings (121). That the amygdala is responsible for fear is, in fact, a widely held belief (120). However, as we have seen, neither amygdala activity nor amygdala-controlled responses are telltale signatures of fearful feelings. As long as we use the term fear to refer to the neural mechanisms underlying both conscious feelings and nonconscious threat processing, confusion will occur. Conscious fear can cause us to act in certain ways, but it is not the cause of the expression of defensive behaviors and physiological responses elicited by conditioned or unconditioned threats. We should not have called it a fear system.

Going Forward

Research under the banner of fear conditioning has been extremely productive. We are at the crossroads between a fledgling and mature field. I propose that this transition could be greatly facilitated by adopting terms that distinguish processes that give rise to conscious feelings of fear from processes that operate nonconsciously in detecting and responding to threats.

The story of fear research shows how hard it is to keep conscious fear out of the causal sequence of behavior. In research, scientists measure responses to threats. However, some then conclude that conscious fear underlies the responses and thus that the responses can signal the presence of conscious fear in people and animals.

We have conscious feelings of fear when we act afraid, and it is natural to assume that these feelings are causal in our behavior and in the behavior of others (122), including animals (61–66). However, much research in psychology and neuroscience shows that people exercise less conscious control of their behavior than
they believe (103–105, 123–125). Careful analyses show that im- plcit processes often underlie (126), and in some cases account for (127), presumed mental states in animals. Conscious mental states should not, in the absence of direct evidence, be the first choice of explanation of behavior, even in humans. Also, when the processes in question are represented similarly in the brains of humans and animals, and do not require consciousness in humans, we should be especially cautious in giving conscious states a causal role in these processes and the responses they control in animals.

The story of fear research also illustrates the perils of using an everyday term about human subjective experience, like fear, as a nonscientific scientific term. When those not “in the know” about the nonscientific meaning of fear (whether they are other scientists, lay people, or journalists) encounter the term “fear,” they naturally conclude that the research is about conscious fear- ing. Loose talk by those who believe otherwise promotes mis-understanding. Researchers today can commonly be heard to say: “we used freezing as a measure of fear” (I have done this myself.) The burden is on scientists who think of fear in non-subjective terms to be clear about what they mean because the default, everyday meaning of fear needs no such help. It is not sufficient to simply say, “science is complicated, so those outside the field cannot be expected to know what is really going on.” We depend on public funding for science, and the public has a right to expect that we try to explain what we are doing as clearly as possible. This is especially important for topics that have clinical relevance, as is the case for fear conditioning.

It is routine for novelists and poets to assume that their readers will turn to shared assumptions captured by everyday language to understand ambiguous statements. Ambiguity is not only tolerated but can be a virtue in literature and poetry but should be avoided in science (128). Francis Bacon cautioned centuries ago against the conceptual dangers of imprecise sci- entific terminology and the potential for reification (128). We should heed his warning. Otherwise, those not in the loop will fail naturally to see that fear does not always mean conscious fear.

I am not suggesting that we banish the “F” word from our scientific vocabulary and research. On the contrary, I think that we need to come to terms with fear because the conscious feeling of fear is a key part of human experience and an important factor in psycho-pathology. Neither am I suggesting that animal research is irrelevant to understanding human conscious feelings of fear. Animal research is essential. However, we need a conception that allows us to un- derstand how nonconscious processes in other species contribute to conscious fear in humans. (This includes animals that are relatively close relatives of humans, like other primates as well as species far from our evolutionary roots, such as worms, flies, and others.)

The required conception is unlikely to be achieved by looking for human mental states in animals. Why should we expect that our introspections will lead to an accurate portrayal of the or- ganization and operation of ancient processes in our own brains or in the brains of other organisms (129). Experiences we label and talk about as fear are not directly tied to the circuits that detect and respond to threats (see Nonconscious Conditioned Fear in Humans), and are not reliably correlated with body responses elicited by threats (130). Claims by some that animals must have conscious feelings because of the continuity of behavior across species (61–66) assume that behavior and con- scious feelings are coupled in the brain. However, if this not the case, and it does not appear to be, we cannot use information about defense responses to tell us whether animals are experi- encing fear. Lloyd Morgan long ago warned against “humanizing the brute,” arguing that just because scientists necessarily start their exploration of animal behavior from their own subjective experiences does not justify the attribution of similar experiences to other animals (131). This kind of attribution is desirable, he says, when we interact socially with other humans but question- able when trying to understand animal behavior. Assumptions about unconscious subjective states are more complex than the assumptions about unconscious entities of physics or astronomy (128, 132).

I am not proposing that animals lack conscious feelings. I just do not think that this is an issue that can be resolved scientifically, as Tinbergen also implied in the opening quote. We are on safe ground when we compare observable variables across species (defense responses elicited by threats). We can also be confident when we assume unconscious variables (feelings of fear) in other people, because all people have brains with the same functions and because we can compare notes with each other verbally. However, whether other animals feel fear when threatened is another matter. Different species have different brains, and even when the same brain areas and circuits are present, these do not necessarily per- form the same exact functions. Also, as we have seen, responses elicited by threats are not telltale signs of fear, even in humans.

Nevertheless, as I discuss below, we can learn quite a bit that is relevant to human feelings from studies of animals without making any assumptions about consciousness. Some will surely counter that this is too limiting. However, if we do not limit the discussion, confusion inevitably results. Those who observe our field lose track of what we are studying and what it means and are left to draw their own conclusions, which are understandably based on their everyday understanding of fear. Going forward, we need clear terms and concepts to advance the field.

Coming to Terms with Fear

There is a really simple solution to these problems. We should reserve the term fear for its everyday or default meaning (the meaning that the term fear compels in all of us—the feeling of being afraid), and we should rename the procedure and brain process we now call fear conditioning.

So what should fear conditioning be called? There are two viable options. Pavlov’s original term, “defense conditioning,” is one. This expression reflected Pavlov’s focus on stimulus sub- stitution—transfer of control of the defensive reflex from the US to the CS. Whereas the CS does come to control defensive freezing, this is not the same response elicited by the US—the US elicits jumping, flight, and other responses (9, 51, 57). Pavlovian aversive conditioning instead is more appropriately conceived of as involving a process in which the meaning of the CS has been changed (133). For this reason, I prefer the second option, “threat conditioning.” This phase implies that a stimulus that was not threatening becomes so. One could argue that threat is in the eyes of the beholder and thus defense conditioning is more neutral. However, a threat can be defined objectively as a stimulus that elicits defense responses. Thus, although either is better than fear, threat has advantages over defense as a description of the process.

We can thus say that the association of the CS with the US changes the meaning of the CS, making it a threat and giving it the ability to flow although amygdala circuits and elicit defense responses. The particular response that occurs depends on fac- tors such as perceived proximity to the threat in space and time (51, 52). Autonomic and endocrine responses that also occur are part of the physiological preparation for responding to the threat and are part of the “defense response complex.”

Much has been written about the language of psychology, where the use of everyday terms based on human introspection invites each reader to interpret the words in their own way (59, 128, 134, 135). Some argue for a new scientific language to re- place everyday folk concepts (128, 135). I have not proposed anything so radical. I have stuck with everyday terms (threat, defense) that describe observable events (stimuli and responses). I also keep the everyday term fear because it reflects states that we know are part of conscious experience, at least in humans.

The problem is not the terms but the way we use them. Spec- ifically, problems arise when we conflate terms that refer to conscious experiences with those that refer to the processing of stimuli and control of responses and assume that the brain mechanisms that underlie the two kinds of processes are the same. By making mild changes that capture these distinctions, we have an easy fix that has the potential for eliminating much of the terminological confusion in the field. We may someday find
an abstract scientific language for describing all this. In the meantime, we should use the language we have more carefully.

One could argue that we should not go down this road unless we are willing to do it for other psychological processes labeled with mental state terms. I would argue that this is indeed correct.

There are practical implications of getting the terminology correct. Pavlovian conditioning research is used to understand, and, in some cases, guide treatment of, psychiatric disorders (130, 136–140). Understanding how conditioning in animals relates to conscious symptoms, as opposed to underlying processes that indirectly contribute to conscious symptoms, is important, because explicit and implicit symptoms may be susceptible to different treatments. Approaches that alter the potency of threats by manipulation of the storage or retrieval of implicit memories are offering variations of, and alternatives to, exposure therapy (138, 140–144). Whereas these operate on nonconscous systems by directly changing how these detect and respond to threats, therapies based on insight or cognitive-change work, in part, through systems that give rise to conscious awareness and that that have limited access to processes underlying implicit memory. Both approaches have a place. Recognizing what each does in the brain may better focus efforts to treat specific needs of the individual, and recognizing which aspects of human brain function animal research is most relevant to gives a more realistic view of what to expect from the CS–US approach, which is a clinical approach

Survival Circuits and Global Organismic States

Having argued for a different way of talking about Pavlovian aversive conditioning, I will put the ideas described above into different terms. One of the assumptions I am only introduced: survival circuits and global organismic states (3).

A neural circuit that underlies the expression defense responses elicited by conditioned and unconditioned (presumably innate) threats can be called a “defensive survival circuit” (3), which is similar to what has been called a defense system (19, 34, 119, 130). There are a number of defensive circuits in the brain (146). These together constitute one of several classes of survival circuits, including circuits for acquiring nutrients and energy sources, balancing fluids, thermoregulation, and reproduction (3, 19, 119, 147, 148). These circuits contribute to the organismic state in different degrees under different conditions, as well as the body, and are necessarily slower to unfold. Invertebrates have different circuit schemes than vertebrates but nevertheless have circuits that perform similar survival functions and that appear to be precursors of survival functions in vertebrates (15, 21, 150–152). Related survival functions also exist in single cell organisms, and thus predates neurons and circuits (3) and likely depend on mechanisms that are primitive precursors of neuronal elements in animals (153).

A notable consequence of activating a survival circuit is that a global (body-wide) state emerges in the organism, components of which maximize well-being in situations where challenges or opportunities exist (3, 148). “Global organismic states” in mammals and other vertebrates, like the survival circuits that initiate them, are elaborations of similar states in invertebrates (151, 154, 155).

The state that results when an organism is in danger, as we have seen, has been called a central state of fear. This construct played a role in Pavlovian conditioning and aversive instrumental conditioning. Now that neuroscience has made progress in replacing Hebb’s conceptual nervous system with circuits and mechanisms that underlie Pavlovian aversive conditioning, and is beginning to do the same for instrumental aversive tasks (49, 156–158), we can ask more specifically about what such a state does. I think renaming the state would help facilitate this research and its interpretation. The expression “defensive organismic state” captures the spirit and emphasis of most central fear state hypotheses (9, 38, 41, 54–57), without pulling the reader or listener’s mind toward the conclusion that the state in question involves a subjective feeling of fear.

The term “defensive motivational circuits” might be useful as a description circuits that, in the presence of threat-predicting cues, control defensive instrumental behaviors (goal-directed actions such as avoidance and other coping responses). The defensive reaction and action circuits likely interact (5, 49, 73). And both contribute to defensive organismic states.

A defensive organismic state is triggered by activity in survival circuits that detects threats and generates automatic defense reactions (3). The detection circuits are either prewired to respond to species-typical threats or are wired via experience (associative learning) to detect novel stimuli that predict sources of harm (3, 19, 146). When activated by a threat, a variety of responses results: species-typical behaviors (e.g., freezing) (7–10, 19), peripheral physiological responses of the autonomic nervous and endocrine systems (10–12, 75, 78) that produce signals that feedback to the brain (32, 37, 159), and changes in brain activity, including synaptic transmission within and between circuits, and increases in general arousal due to widespread release of aminergic neuromodulators (78, 160). Collectively, these responses constitute the defensive organismic state. Such states are multidimensional, not unitary (3, 9, 41, 119); different components may be activated to different degrees under different conditions, and interactions between them are necessary for the state to unfold.

The global defensive organismic state can be thought of as a “metaconditioned response” that depends on the more specific constituent conditioned responses. In this view, the global state is not a cause of the specific conditioned responses, as is sometimes assumed (9, 38, 54–57), but rather a consequence. This needs some clarification because “state” is often used ambiguously. There is, of course, some neural state that occurs locally in the survival circuit when it is activated by a threat and that accounts for the initial defense response. Also, during conditioning, the CS and US both elicit neural states that interact as part of associative learning. Some, if not most, who have used the nonsubjective central state of fear construct likely have had this local state notion in mind. Models that argue that a US-induced affective state is associated with the CS during learning may also have a local neural state in mind (34, 48, 49, 161). Calling these local responses central states of fear confuses them with the global states being discussed here that affect widespread brain areas, as well as the body, and are necessarily slower to unfold. Although the defensive organismic state, as I view it, does not account for rapidly triggered default defensive responses, like freezing, it does contribute to the organismic state that emerges as the threatening situation evolves over time (9, 19, 51, 52).

However, an unresolved issue is the extent to which global organismic states, as opposed to their component processes, have a causal role in behavior. Is, in other words, the global state greater than the sum of its component parts? Such states are, in principle, measurable and are thus not simply reified constructs. However, whether the state itself has a causal role beyond the neural activity occurring in the specific circuits that process threats, retrieve memories, generate arousal, select responses, and motivate and reinforce behavior is unclear. Because of this uncertainty, I prefer the expression defensive organismic state over related expressions [i.e., central motive state (45, 46), central fear state (9, 38, 54–57), or defensive motivational state (34, 41, 130)] that imply that the state itself organizes and controls behavior.

Regardless, however, to the extent that defensive organismic states, or their components, contribute response selection, motivation, reinforcement, or other processes, it is not because they constitute a state of consciously experienced fear. The latter is an almost unavoidable, yet mostly unintended, implication of much of the current literature. A conscious state of fear may occur, but that is not the factor that selects, motivates and/or reinforces behavior. A similar argument has been made for appetitive instrumental behaviors (eating, drinking, and sexual behavior, and behaviors related to use of addictive drugs)—circuits and cellular and molecular mechanisms involved in reinforcing
and motivating these are not the same mechanisms that give rise to conscious states of pleasure (83, 162, 163).

I thus assume, until proven otherwise, that a defensive organic state and its constituent components are implicit (non-conscious). If so, to be felt as fear, the state or its components have to become a presence in conscious awareness (3-6). This can only happen in organisms that have the capacity to be aware of brain representations of internal and external events, and may also require the ability to know in a personal, autobiographical sense that the event is happening to them (164). In short, someone has to be home in the brain to feel fear. Infants can react in “emotionally” long before they can feel emotion (165). Similarly, it is possible, in fact likely, that animals can react “emotionally” without feeling emotional (even if they in some situations do feel emotional). In the end, as I have noted, the question of whether animals react but do not feel, or whether they both react and feel, is, in my opinion, not something we can determine scientifically.

By using different terms for conscious feelings and the non-conscious events that can, in some organisms, contribute to feelings in the presence of threats, much of the ambiguity and confusion about the neural mechanisms that detect and control responses to threats, and neural states that may result, is avoided. Furthermore, research on these mechanisms can be conducted without having to struggle with questions about whether the animal does or does not experience fear. These mechanisms can be studied the same in humans and other animals, including invertebrates. Fear itself, although, is best studied in humans.

Fear Itself

Restricting the term fear to the conscious experience that occurs when an organism is threatened eliminates the awkward distinction that is required when theorists assume that some emotional feelings are innately wired in brain circuits and others are psychologically or socially determined (61, 159, 166, 167). I do not think of emotions in this dualistic way, where fear is a bottom-up state that is unleashed in a prepackaged pure form of experience stored in a hardwired subcortical circuit, and other feelings are cognitively constructed.

My conception is more aligned with theories that propose that feelings result from the cognitive processing of situations in which we find ourselves (168–175). In my view, the feeling of fear occurs in the same way as the feeling of compassion or pride—through cognitive processing of neural raw materials. Some feelings involve raw materials provided by activation of survival circuits and their consequences, but others do not. Fear often does, but pride or compassion typically does not. What distinguishes kinds of emotional experiences is the combination of raw materials that are in play. What distinguishes emotional experiences from nonemotional experiences is the fact that emotional experiences have raw materials that nonemotional experiences lack. What distinguishes the various kinds of fears (fear of a snake, of social situations, of being late for an appointment, of having a panic attack, of an examination, of falling in love, of failure on a task, of not leading a meaningful life, of the eventualty of death) is also the combination of raw materials involved (4, 5, 170, 172, 176). Some fears depend on survival circuits but others do not. The “survival-circuit-dependent” kind of fear is the romanticized version but is not the only kind of fear we have. Fear is what happens when the sentient brain is aware that its personal well-being (physical, mental, social, cultural, existential) is challenged or may be at some point. What ties together all instances of fear is an awareness, based on the raw materials available, that danger is near or possible. A theory of fear has to account for fears that do and do not involve survival circuit activity.

Fear can be thought of as emerging in consciousness, much the way the character of a soup emerges from its raw materials, its ingredients. Start with salt, pepper, garlic, onions, carrots, and chicken. Add roux and chicken soup becomes gumbo, or add curry paste, and it shifts it in a different direction. None of these are soup ingredients. They are just things that exist in nature and that can be combined to make soup or many other things. Similarly, emotions emerge from nonemotional ingredients, events that exist in the brain and body as part of being a living organism of a particular type (e.g., survival circuit activity, brain arousal, body responses and feedback, memories, thoughts, predictions). No one ingredient is essential to fear. Variation in the kind and amount of ingredients determine whether you feel fear, as opposed to some other emotion, and also determine the variant of fear you feel. Barrett has expressed a related view (134).

We do not know whether other organisms have feelings of fear or other states of consciousness. However, even if they experience conscious states of awareness, these states are likely to be very different from ours (177). Our experiences depend, in part, on our capacity for natural language, as well as other cognitive capacities (134). The idea that language and culture shape experience (178), including emotional experience, is currently thriving in psychology (176, 179–183). We have English words to distinguish more than three dozen variants of fear-related experiences (184). Animals, lacking our language and culture, cannot experience the world the way we do. Their feelings, if they have them, cannot be like those made possible by our brain’s capacities to conceptualize, categorize, label, and interpret, and to introspect about and consciously experience, our outer and inner worlds.

As Kagan says in the opening quote, the mechanisms that allow organisms to respond to threats are different from the mechanisms that give rise to conscious fear. Using terms that acknowledge this difference will help avoid confusion about what we study and what it means as our field moves forward.


104. Gazzaniga MS (2012)


140. Parsons RG, Ressler KJ (2013) Implications of memory modulation for post-traumatic


137. Milad MR, Quirk GJ (2012) Fear extinction as a model for translational neuroscience:

128. Mandler G, Kessen W (1964)


120. Feinstein JS, et al. (2013) Fear and panic in humans with bilateral amygdala damage.


5825

124(Pt 6):1241


125. Wilson TD, Dunn EW (2004) Self-knowledge: Its limits, value, and potential for im-


132. Fletcher GJO (1995) Two uses of folk psychology: Implications for psychological sci-

131. Morgan CL (1890


155. Lebestky T, et al. (2009) Two different forms of arousal in Drosophila are oppositely


136. Lewis M (2013)


156. Amorapanth P, LeDouex JE, Nader K (2000) Different lateral amygdala outputs me-


150. Garrison JL, et al. (2013) Oxytocin/vasopressin-related peptides have an ancient role in

143. Schiller D, et al. (2010) Preventing the return of fear in humans using reconsolidation


165. Lewis M (2013)


104. Gazzaniga MS (2012)


140. Parsons RG, Ressler KJ (2013) Implications of memory modulation for post-traumatic


137. Milad MR, Quirk GJ (2012) Fear extinction as a model for translational neuroscience:

128. Mandler G, Kessen W (1964)


