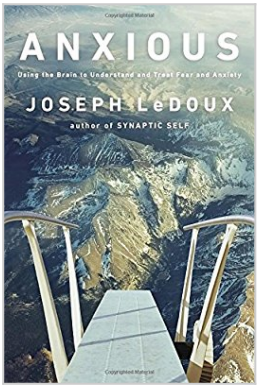

Anxious: Using the Brain to Understand and Treat Fear and Anxiety

By Joseph LeDoux

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"[*Anxious*] helps to explain and prevent the kinds of debilitating anxieties all of us face in this increasingly stressful world." —Daniel J. Levitin, author of *The Organized Mind* and *This Is Your Brain On Music*

A comprehensive and accessible exploration of anxiety, from a leading neuroscientist and the author of *Synaptic Self*

Collectively, anxiety disorders are our most prevalent psychiatric problem, affecting about forty million adults in the United States. In *Anxious*, Joseph LeDoux, whose NYU lab has been at the forefront of research efforts to understand and treat fear and anxiety, explains the range of these disorders, their origins, and discoveries that can restore sufferers to normalcy.

LeDoux's groundbreaking premise is that we've been thinking about fear and anxiety in the wrong way. These are not innate states waiting to be unleashed from the brain, but experiences that we assemble cognitively. Treatment of these problems must address both their conscious manifestations and underlying non-conscious processes. While knowledge about how the brain works will help us discover new drugs, LeDoux argues that the greatest breakthroughs may come from using brain research to help reshape psychotherapy.

A major work on our most pressing mental health issue, *Anxious* explains the science behind fear and anxiety disorders.

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Editorial Review

Review

“Every age believes itself to be the age of anxiety, as Auden’s famous poem first put it. But in his new book, *Anxious*, the neuroscientist and writer Joseph LeDoux suggests that that has never been a stronger claim to make than it is now . . . . If this is the age of anxiety, LeDoux is our Lewis and our Clark: It was LeDoux who laid down the first map of what is called the brain’s ‘fear circuit,’ the regions—centered on the amygdala and its adjacent structures—that together give rise to our ability to respond to threats and danger. But with his new book, he wants to redraw that map.”
—Casey Schwartz, *New York Magazine*

“Mr. LeDoux offers a careful tour through the current neuroscience of fear and anxiety. . . . [*Anxious*] will reward the informed reader.”
—Leonore Tiefer, *The Wall Street Journal*

“LeDoux presents a rigorous, in-depth guide to the history, philosophy and scientific exploration of this widespread emotional state. . . . Neuroscientists, psychologists, philosophers and psychiatrists will find this exquisitely referenced book particularly useful. It is also a must-read for young investigators, and anyone perusing the footnotes will be rewarded with an insider’s view of the state and evolution of anxiety research. LeDoux’s charming personal asides give an impression of having a conversation with a world expert. LeDoux ends on a high note, describing how cutting-edge research on the neural substrates of anxiety is being translated into new approaches for psychiatric treatment.”
—Susanne Ahmari, *Nature*

“LeDoux is not only a pioneer in the neurobiological analysis of fear in animals but also a scholarly and accessible writer. In *Anxious*, he systematically builds on his earlier works, covering with aplomb a vast literature on emotion, memory, attention, and consciousness. With that said, Anxious is a significant and important departure from the author’s earlier views on the neural underpinnings of fear. . . . In *Anxious*, LeDoux challenges the reader to think differently about the neural origins of fear and its disorders. In doing so, he offers a masterful synthesis of animal and human work and a novel roadmap for future work in both the laboratory and the clinic.”
—Stephen Maren, *Science*

“Anxious is an extraordinarily ambitious, provocative, challenging, and important book. Drawing on the latest research in neuro-science (including work in his own laboratory), LeDoux provides explanations of the origins, nature, and impact of fear and anxiety disorders.”
—Glenn Altschuler, *Psychology Today*

“Drawing on years of research, neuroscientist LeDoux delves into the subject of anxiety and fear, depicting both emotions as cognitive constructs. . . . [*Anxious*] will open up new worlds of thinking and feeling”
—Publishers Weekly

“Wonderfully erudite, informative, and splendidly well written. Helps to explain and prevent the kinds of debilitating anxieties all of us face in this increasingly stressful world. Any author who can weave Leonard Bernstein, W. H. Auden, The Rolling Stones, and Alfred E. Neuman into a single illustrative example is on

my short list for favorite writers ever."
—Daniel J. Levitin, author of *The Organized Mind* and *This Is Your Brain On Music*

"Joseph LeDoux [is] the William James of our era. . . . This marvelous book is science at its best. It traces the evolution of a key set of scientific insights based on progressively better empirical data, most of these derived from LeDoux's brilliant studies, and applies these new insights to a family of clinically important phenomenon. *Anxious* is an absolute must read for clinicians and basic scientists as well as for anyone else interested in anxiety and its disorders."
—Eric R. Kandel, Kavli Professor and University Professor, Columbia University; Senior Investigator, Howard Hughes Medical Institute; author of *In Search of Memory* and *The Age of Insight*; recipient of the 2000 Nobel Prize in Physiology or Medicine

"An exquisite and unique attempt to truly relate how neural cells lead to felt conscious states in the human mind—the toughest problem in all of science. LeDoux has thrown down the gauntlet and set the standard. I wish all of us working on the problem luck trying to beat this analysis."
—Michael Gazzaniga, Professor of Psychology, University of California, Santa Barbara, and author of *Tales from Both Sides of the Brain, Human*, and *The Social Brain*

"*Anxious* is a profound, exciting and immensely useful work about one of our most troubling—and puzzling—emotions. Joseph LeDoux takes us behind the scenes of our own minds to show us not only how anxiety is constructed in the brain but how it can be deconstructed. This is neuroscience at its very best: helpful and hopeful without a hint of hyperbole."
—Mark Epstein, M.D., author of *Thoughts without a Thinker* **and** *The Trauma of Everyday Life*

"In this tour de force, LeDoux artfully guides the reader from the unconscious defensive system, through attention and memory, to the conscious experience of fear and anxiety. His traverse from the unconscious to the conscious experience of emotion is rich in scientific detail and yet exquisitely readable. LeDoux completes his masterpiece with provocative discussions of therapies for anxiety. This book is a fascinating revelation of the evolution in LeDoux's own scientific thinking and in the field at large and is a must read for any student of learning, memory or emotion."
—Michelle G. Craske, Ph.D., Professor of Psychology and Director, Anxiety Disorders Research Center, UCLA

"LeDoux is a true leader in the field of cutting-edge neuroscience and psychology, yet he also has an uncanny ability to write beautifully and clearly. . . . A must-read for anyone interested in the intersection of the mind and brain, and how an understanding of psychology and neuroscience can change ourselves and the world around us!"
—Kerry J. Ressler, M.D., Ph.D., Professor of Psychiatry and Behavioral Sciences, Emory University; Scientific Council Chair, Anxiety and Depression Association of America; Investigator, Howard Hughes Medical Institute; Member, National Academy of Sciences

Praise for *Synaptic Self*
"*Synaptic Self* represents a brilliant manifesto at the cutting edge of psychology's evolution into a brain science. Joseph LeDoux is one of the field's pre-eminent, most important thinkers."
—Daniel Goleman, author of *Emotional Intelligence* and *Primal Leadership*

"A clear, up-to-date, and impressively fair-minded account of what neuroscience has established about human nature."
—Howard Gardner, John H. and Elisabeth A. Hobbs Professor of Cognition and Education at

Harvard University and author of *Frames of Mind* and *Intelligence Reframed*

"*Synaptic Self* is a wonderful tour of the brain circuitry behind some of the critical aspects of the mind. LeDoux is an expert tour guide and it is well worth listening. His perspective takes you deep into the cellular basis of what it is to be a thinking being."
—Antonio R. Damasio, University Professor, David Dornsife Chair in Neuroscience and Professor of Psychology and Neurology, Director of the USC College Brain and Creativity Institute and author of *The Feeling of What Happens* and *Descartes' Error*

"In this pathbreaking synthesis, Joseph LeDoux draws on dazzling insights from the cutting edge of neuroscience to generate a new conception of an enduring mystery: the nature of the self. Enlightening and engrossing, LeDoux's bold formulation will change the way you think about who you are"
—Daniel L. Schacter, William R. Kenan, Jr. Professor of Psychology at Harvard University, author of *The Seven Sins of Memory* and *Searching for Memory*

"Starting with a synopsis of the evolving nature of the "self" in philosophy, psychology, and physiology . . . [LeDoux] addresses that most unwieldy of subjects through the empirical divinations of neuroscience. The core of his argument rests on synapses, the empty gaps that neurons bridge to form circuits. LeDoux's remarkably accessible descriptions of the process crackle like the electrical storms that rain chemical ooze on the brain. . . . [*Synaptic Self*] goes a long way in ordaining the steps to humanity's timeless tango with tautology."
—Andy Battaglia, *A.V. Club*

"[A]n important contribution."
—*Publishers Weekly*

Praise for *The Emotional Brain*

"Highly accessible, a stimulating and thoughtful work [that] is essential reading for any serious student of human nature."
—Raymond J. Dolan, *Nature*

"[*The Emotional Brain*] is vivid and convincing in its description of a central mechanism of emotion, and is directly applicable to understanding anxiety, the most common ingredient of emotional disorders. It's a terrifically good book."
—Keith Oatley, *New Scientist*

"With clarity and convincing logic, *The Emotional Brain* presents a new view of emotion that is derived in large part from the author's own ground-breaking research. . . . LeDoux shows how the study of the brain leaves our understanding of emotion richer than it was before."
—Steven E. Hyman, M.D. Director, National Institute of Mental Health

"Engrossing and engaging."
—Richard Restak, *The New York Times Book Review*

About the Author
JOSEPH LEDOUX is the Henry and Lucy Moses Professor of Science at New York University, where he is a member of the Center for Neural Science and Department of Psychology. He directs the Emotional Brain Institute at New York University and at the Nathan Kline Institute, and is the author of *Synaptic Self* and *The*

Emotional Brain. A member of the National Academy of Sciences, LeDoux lives in Brooklyn, New York.

PREFACE

When I completed my previous book, *Synaptic Self*, which was published in 2002, I wasn't certain I wanted to write another book for a general audience. I had gotten the idea that the way to really have an impact on the field was to write a textbook in my particular area, behavioral and cognitive neuroscience. My agents, John Brockman and Katinka Matson, urged me not to, as did my editor at Viking, Rick Kot, each of them warning me that I would regret it as a publishing experience. After struggling with the project for almost a decade, I had to admit that they were correct. I discovered that the textbook format was far too restrictive—it had to be fresh and innovative . . . so long as it was just like every other competing book. After each chapter was reviewed by a number of teachers from a mix of universities, colleges, and junior colleges around the country, I began to feel little connection to the edited text that was resulting and concluded that my role was more to be a name on the cover than to actually drive the content.

A few years ago I ran into Rick at a reading by our friend Rosanne Cash, who wrote *Composed* under his editorship, and he asked with a wry smile, "How's that textbook going? I've been waiting for you to bail out of that and do another book with me." I was thrilled that he was still interested in working with me, and I negotiated, with some help from Eric Rayman, my way out of the textbook and prepared a new proposal for Katinka. *Anxious* was the result. Rick loved the idea, and so here we are.

Anxious is different from my other books. While *The Emotional Brain* and *Synaptic Self* can be thought of as a series of connected essays that hang together around a single theme, in *Anxious* each successive chapter builds on the previous ones to argue for a new view of emotion, especially the emotions fear and anxiety. Although the book is called *Anxious*, fear and anxiety are complexly entwined, and must be understood both separately and together.

As an overview, here are the key points that *Anxious* addresses. First, the science of emotion, and especially the science of fear and anxiety, now finds itself at an impasse, dictated by the way we discuss emotions in relation to the brain. For example, researchers use words like "fear" to describe the brain mechanisms that cause rats to freeze when in danger, and also to describe the conscious feeling that humans experience if they think that they will be seriously harmed physically or psychologically. The general idea is that a fear circuit in the brain is responsible for the feeling of fear, and when it is activated, whether in a rat or a human, the feeling of fear occurs, along with responses characteristic of fear (such as freezing, facial expressions, changes in body physiology). The feeling of fear is often said to mediate between the threatening event and those responses. Because these circuits are conserved throughout mammals, including humans, we can study human fear by measuring freezing in rats. The key circuits crucially involve the amygdala, which is generally described as the seat of fear in the brain.

In fact, most of what I have just described is wrong. Because my work and writings are in part responsible for these misconceptions, I feel some responsibility to try to straighten out the story before it goes further off track. One of the main goals of this book is to provide a new view of fear and anxiety, one that more accurately distinguishes what we can learn from animals from what we can best learn from humans, and what fear itself really refers to in the context of the human brain.

Don't get me wrong: I am not arguing that we have to study brain mechanisms related to emotions exclusively in humans. There is much we have learned, and can continue to learn, and can, in fact, only learn, from animal research. But we do need a rigorous conceptual framework for understanding what the

animal work does and does not mean for understanding the human brain. I offer my view of such a framework, which I think provides a new perspective on fear and anxiety, and the disorders related to these states.

The suggestions I make in this book concern in part the words we use to describe certain phenomena, but my argument is not simply about semantics. Words have extended meanings that imply a great deal. For example, some researchers who study fear in rats by measuring freezing behavior say that they are not studying what most people think of as fear, but rather some nonsubjective physiological state that they *call* fear. While this scientific redefinition of fear makes it more tractable as a research problem, it has three disadvantages. First, using fear in a nonconventional way to describe a physiological state that connects threats with responses often leads researchers to write and talk about this state as if it was referring to the conscious feeling of fear. Second, even when the researchers adhere to that definition, everyone thinks that they are actually studying the feeling of fear. And third, we in fact do need to understand the feeling of fear, and ignoring it is not the solution.

As scientists we have an obligation to be precise in how we describe our research. This is especially important when the work is being used to conceptualize human problems—in this case, fear and anxiety disorders—and develop treatments for them. But because conscious feelings of fear and anxiety arise from circuits in the brain that differ from the circuits that control the expression of defensive behaviors like freezing, and are likely vulnerable to different factors, they need to be understood separately. Certainly the circuits that control defense responses and give rise to feelings of fear interact, but this does not mean that they are the same.

Failure to make such distinctions accounts for poor outcomes of studies that have attempted to develop new pharmacological treatments for fear and anxiety in animals, as the studies assess the effects of drugs on behavioral responses but then expect the drugs to actually make people feel less fearful or anxious. We have long known that there is discordance in how treatments affect the way people feel when threatened as opposed to the behavioral and physiological responses they express in such situations.

One of the key issues to point out is that people can be shown pictures of threats in such a way that they are not conscious of the stimulus, and have no conscious feeling of fear. But their amygdala is activated by the threat, and bodily responses, such as changes in perspiration, heart rate, or pupil size occur, showing that the detection and response to threat is independent of conscious awareness. If we don’t need conscious experience to control responses to threats in humans we should be cautious about concluding that conscious states cause rats to respond to threats. I am not saying that rats or other animals lack consciousness. All I am saying is that we should not simply assume that because they may respond the way we do when threatened they feel what we do. The problem is that scientific studies of animal consciousness are not easily performed.

Implied above is that fear and anxiety are conscious feelings. As such, we need to understand consciousness in order to understand fear and anxiety. Several chapters of *Anxious* are devoted to giving a progress report on the current state of our understanding of consciousness in neuroscience, psychology, and philosophy (at least from my perspective). Included is the controversial topic of animal consciousness, which, as I just mentioned, is extremely difficult to study scientifically. I suggest guidelines about how we might be more scientific in our approach to this subject.

My view of consciousness dates back to my graduate work on split-brain patients, which I conducted with my mentor, Michael Gazzaniga, at SUNY Stony Brook. We concluded that one important role of consciousness is to make sense of our complex brains. Much of what our brain does, it achieves nonconsciously. Our conscious minds then construct an explanation of what we experience. In this sense, consciousness is a self-narrative built from bits and pieces of information we have direct conscious access to

(perceptions and memories) and also from the observable or “monitorable” consequences of nonconscious processes. Emotions are, as some now say, cognitive or psychological constructions.

Finally, I discuss issues related to therapy. One key argument I make is that contrary to popular opinion, the behavioral procedure called extinction is not the main process at work in exposure therapy. Extinction plays a role, but exposure therapy actually involves many more mechanisms, and it is possible they actually interfere with the ability to extinguish. Another principle I challenge is that avoidance is always a bad thing for people with anxiety, for I believe that a form of proactive avoidance can be very useful. These and a number of other ideas for improving psychotherapy come directly from animal research. The key is to know what we can and can’t learn from animals, and to not conflate the two.

• • •

I have dedicated this book to the many graduate students, postdoctoral, and technical researchers in my lab over the years who have contributed to the work with which I have been credited, for they deserve as much credit as I, and in some cases more. In alphabetical order they are:

Prin Amorapanth, John Apergis-Schoute, Annemieke Apergis-Schoute, Jorge Armony, Elizabeth Bauer, Hugh Tad Blair, Fabio Bordi, Nesha Burghardt, David Bush, Christopher Cain, Vincent Campese, Fernando Canadas-Perez, Diana Cardona-Mena, William Chang, June-Seek Choi, Piera Cicchetti, M. Christine Clugnet, Keith Corodimas, Kiriana Cowansage, Catarina Cunha, Jacek Debiec, Lorenzo Diaz-Mataix, Neot Doron, Valerie Doyere, Sevil Durvaci, Jeffrey Erlich, Claudia Farb, Ann Fink, Rosemary Gonzaga, Yiran Gu, Nikita Gupta, Hiroki Hamanaka, Mian Hou, Koichi Isogawa, Jiro Iwata, Joshua Johansen, O. Luke Johnson, JoAnna Klein, Kevin LaBar, Raphael Lamprecht, Enrique Lanuza, Gabriel Lazaro-Munoz, Stephanie Lazzaro, XingFang Li, Tamas Madarasz, Raquel Martinez, Kate Melia, Marta Moita, Marie Monfils, Maria Morgan, Justin Moscarello, Jeff Muller, Karim Nader, Paco Olucha, Linnaea Ostroff, Elizabeth Phelps, Russell Philips, Joseph Pick, Gregory Quirk, Franchesa Ramirez, J. Christopher Repa, Sarina Rodrigues, Michael Rogan, Liz Romanski, Svetlana Rosis, Akira Sakaguchi, Glenn Schafe, Hillary Schiff, Daniela Schiller, Robert Sears, Torfi Sigurdsson, Francisco Sotres-Bayon, Peter Sparks, Ruth Stornetta, G. Elizabeth Stutzmann, Gregory Sullivan, Marc Weisskopf, Mattis Wigestrand, Ann Wilensky, Walter Woodson, Andrew Xagoraris. Also included are Elizabeth Phelps, my long-standing collaborator, and her team at NYU, as they have done human versions of our rodent studies and verified that our findings apply to people.

For assistance with the ancient roots of the modern word “anxiety,” I am grateful to my son, Milo LeDoux, who was trained in classics at the University of Oxford, and is now a student at the University of Virginia School of Law, and Peter Meineck, clinical associate professor of classics at NYU and founder of the Aquila Theatre. The cognitive therapist Stefan Hofmann of Boston University helped me tremendously by providing key papers for me to read to help me better understand cognitive therapy and its relation to extinction. Isaac Galatzer-Levy, a colleague from the NYU Langone Medical Center Department of Psychiatry, read several chapters and made helpful comments.

I am also grateful to my illustrator, Robert Lee, for his patience in working through my various incomplete and sometimes incoherent rough drafts of the art.

Special thanks to William Chang, my longtime assistant, who has suffered graciously through many writing projects, and without whom completion of this project would have been far more onerous a task.

I have been continuously funded by the National Institute of Mental Health since 1986, and much of the research discussed here was made possible by its support. Recently, I have also been supported by the National Institute of Drug Abuse. In the past I have also received funding from the National Science

Foundation. I am grateful to Robert Kanter and Jennifer Brour for their support.

In 1989 I joined the faculty of Arts and Sciences at NYU, where I have been a member of the Center for Neural Science and Department of Psychology. In recent years I have received appointments in psychiatry and in child and adolescent psychiatry at NYU Langone Medical School. NYU has been a loyal and generous friend to me and my research.

In 1997, through a collaboration between NYU and New York State, I was appointed as director of the Emotional Brain Institute. This is a multisite program with laboratories at NYU and at the Nathan Kline Institute for Psychiatric Research. Through support of this program by NYU and New York State we hope to make new gains in understanding fear and anxiety. Some of the studies described in this book have been conducted in this context.

John Brockman, Katinka Matson, and everyone at Brockman Inc. are incredible agents. I am grateful for all they have done for me over the years, starting with *The Emotional Brain.*

At Viking, I can't lavish enough praise on Rick Kot. He was the editor of *Synaptic Self* as well, and I hope of future books that may be lurking deep down in the synaptic recesses of my brain. Rick's assistant, Diego Núñez, has been terrific in helping navigate the end-of-book steps. And Colin Weber deserves special credit for designing such a compelling jacket image—so compelling, in fact, that it led to discussions about whether it might be too "scary" for anxious people.

I want to express my love and thanks to my brilliant and beautiful wife, Nancy Princenthal. Nancy and I were engaged in major book projects at the same time, both headed toward publication in the spring/summer of 2015. In spite of special challenges that she faced in completing her biography of the late artist Agnes Martin, she was a friend, companion, critic, and editor when each role was needed.

• • •

How did the title *Anxious* finally come about? In 2009 my band, The Amygdaloids, released an album titled *Theory of My Mind* on the Knock Out Noise label, on which Rosanne Cash sang two songs with me. One of the pieces that we recorded that didn't make it onto the album was called "Anxious." I always liked the song, and had been thinking of releasing it separately. That's when the idea that the book should be called *Anxious* hit me. And it didn't take long to make the next mental leap: Why not release both *Anxious* the book and *Anxious* the CD simultaneously, since my songs are related to the themes in the book? Colin Weber generously agreed to allow me to use the book jacket art for the CD cover. Below you will see a barcode that can be scanned for a onetime free download of the songs on *Anxious*.

Use a "scanning app" with your smartphone and scan the code provided here to obtain a free download of songs from *Anxious* (the CD) by The Amygdaloids. The app should take you directly to The Amygdaloids' website, where you will see instructions on how to download the songs. This offer is limited to 18 months from initial publication of the hardcover edition of the book in the U.S. and expires on January 14, 2017. If any problems arise, email a copy of the copyright page (backside of title page) of the book to amygdaloids.anxiousdownload@gmail.com with the subject "Anxious CD Download."

Enjoy the book, and the music.

CHAPTER 1

THE TANGLED WEB OF ANXIETY AND FEAR

“He who fears he shall suffer already suffers what he fears.”

—MICHEL DE MONTAIGNE1

“While I was fearing it, it came, but came with less of fear. . . .

’Tis harder knowing it is due, than knowing it is here.”

—EMILY DICKINSON2

Anxiety is a normal part of life—there’s always something to worry about, dread, fret over, or be stressed by. But we aren’t all anxious to the same degree. Some people are “nervous Nellies” and worry about everything; others are “cool as a cucumber” and seem to just take it all in stride.

My mother was a worrier. Not in the extreme, but she could be preoccupied and fidgety and sometimes complained about sleepless nights. She had a good reason to be this way. My father was more or less carefree, the kind of guy who could put the day behind him and fall asleep within minutes of his head hitting the pillow. If she didn’t worry, their business, a mom-and-pop store, could not have thrived. She kept everything together, both at work and at home. And while loving and kind, she also sometimes suffered under the pressures of keeping all the balls in the air on a daily basis. My own temperament lies somewhere between theirs, and when I feel the stress of daily life pulling me toward anxiety and worry, I try to channel a bit of my father’s disposition to balance things out. But it’s a temporary measure, as I tend to revert to who I am, to my own level of anxiety, quite quickly.

Not surprisingly, one’s general level of anxiety is a fairly stable personality trait,3 a significant component of temperament.4 We vary from our personal hot spot from time to time, but we always return to our resting place. It’s as if “conservation of anxiety” is a law of human nature.

What makes us each have his or her own individual anxiety level? In part, it is because we each experience and respond to the world differently. Anxiety is very subjective: What’s really stressful to one person may hardly matter to another. It’s not as simple a matter as just having the ability to let the small stuff slide. People who are dispositionally anxious see more things as stressful than less anxious people; for the more anxious, fewer experiences fall in the “small stuff” category.

But simply stating that we are each different only begs the question: What is it that makes us each psychologically distinct? The answer, of course, is that we each have a one-of-a-kind brain. As I explained in *Synaptic Self*,5 while all human brains are similar in overall structure and function, they are wired differently in subtle, *micro*scopic ways that make us individuals. These differences come about both because of the unique combination of genes we get from our two parents and because of the experiences we have as we go through life. Nature and nurture are partners in shaping who we are, and that partnership is played out in each of our brains.

ANXIETY: ANCIENT YET NEW6

The English word “anxiety” and its European equivalents (e.g., *angoisse* in French, *angoscia* in Italian, *angustia* in Spanish, *Angst* in German, and *angst* in Danish) come from the Latin *anxietas*, which, in turn, has roots in the ancient Greek *angh*.7 Although *angh* was sometimes used by the Greeks to mean burdened or troubled (i.e., *ang*uished), it was primarily employed in reference to physical sensations, such as tightness, constriction, or discomfort. For instance, the word “angina,” a medical condition in which chest pains occur

in relation to heart disease, comes from *angh*.8

Literary and religious writings and works of art over the ages reveal that people have always recognized the mental state we commonly refer to as anxiety today, even though they did not typically label it using *angh* or its linguistic descendants.9 For example, the famous Greek sculpture *Laocoön and His Sons*, shown in Figure 1.1, illustrates anxiety (anguish, worry, and/or dread) in faces of Laocoön and his offspring, who are entwined with and are being bitten by snakes as punishment by the gods for having attempted to expose the ruse of the Trojan horse.10 Ares, the Greek god of war, had two sons, Phobos (the god of fear) and Deimos (the god of dread), who accompanied him into battle to spread their namesake emotions.11 In the New Testament, the reader is told in Matthew 6:27, "You cannot add any time to your life by worrying about it." The philosopher and theologian Thomas Aquinas noted in the thirteenth century, "When a man dreads the punishment which confronts him for his sin and no longer loves the friendship of God which he has lost, his fear is born of pride, not of humility."12 Indeed, in the Christian world, anxiety was often connected with sin and redemption.13 In the 1800s, for example, Søren Kierkegaard, who at the time was a little-known Danish theologian and philosopher, conceived of anxiety as the key to human existence: a sense of dread over our freedom to choose. It began, Kierkegaard said, when Adam struggled between Eve's apple and God, and remains a factor in every choice that humans make.14

Figure 1.1: The Anguish of Laocoön and His Sons.

In spite of its long history, however, the word "anxiety" was not primarily thought of as a troubled, worried state of mind and a source of psychopathology until the early twentieth century. This transformation began when Sigmund Freud made anxiety the centerpiece of his psychoanalytic theory of mental disorders.15 Earlier psychopathologists such as Emil Kraepelin16 had formulated ideas about anxiety, but it was Freud who introduced the concept of pathological anxiety to the world at large.17

According to Freud, anxiety is the root of most if not all mental maladies18 and central to any understanding of the human mind: "There is no question that the problem of anxiety is . . . a riddle whose solution would be bound to throw a flood of light on our whole mental existence."19 He saw anxiety as a natural and useful state but also a common feature in mental problems that plague people in everyday life. Ever since, anxiety has been viewed as a state of mind characterized by worry, dread, anguish, and apprehension.

Anxiety was, for Freud, first and foremost "something felt," a special "character of unpleasure."20 Like the Greeks, he made a point of distinguishing *Angst* (anxiety) from *Furcht* (fear). Anxiety, Freud said, relates to the state itself, and disregards the object that elicits it, whereas fear draws attention precisely to the object.21 Specifically, Freud noted that anxiety describes a state of expecting danger or preparing for it, and of dreading it, even though the actual source of harm may be unknown; fear, however, requires a definite object of which to be afraid.22 He also distinguished between *primary anxiety*, which has an immediate object (essentially fear), and *signal anxiety*, which is objectless and involves a more diffuse or uncertain feeling that harm may come in the future (essentially anxiety).

In Freud's view anxiety is born out of a need to keep impulses based on stressful thoughts and memories, mostly about childhood, out of consciousness. Through the defense mechanism of repression, these impulses are hidden in the unconscious mind. When repression fails, the troubling impulses reach consciousness, and neurotic anxiety results. The impulses then need to be repressed again or "satiated" through neurotic "enactments" to relieve anxiety. The goal of Freud's psychoanalytic method was to bring the cause of the neurotic anxiety, or what came to be called *anxiety neurosis*, into consciousness and eliminate its clandestine, subversive power.

Existential philosophers such as Martin Heidegger23 and Jean-Paul Sartre24 offered a different view of mental life, and especially anxiety, one centered on consciousness.25 Sartre, for example, rejected Freud's emphasis on the pathological and unconscious aspects of mind. He famously said, "*l'existence précède l'essence*" (existence precedes essence), by which he meant that we create ourselves by the conscious choices we make.

The existentialists viewed anxiety as an integral part of human nature, rather than a disorder. In this they were greatly influenced by the writings of Kierkegaard. In *The Concept of Anxiety*, published in 1844, before Freud was born, Kierkegaard made a distinction between fear, which has a specific object (similar to Freud's *Furcht*, or primary anxiety), and anxiety, a kind of unfocused, objectless, future-oriented fear (comparable to Freud's *Angst* and signal anxiety but with much less emphasis on pathology and a greater focus on consciousness).26 Because of its lack of an objective focus, Kierkegaard argued that anxiety (dread) was caused by "nothingness": the despair that comes from the realization that we are not grounded in the world and are defined only by the practices in which we engage. It is through choice that we prevent the return to nothingness.27 Kierkegaard became well known only after the existentialists adopted him, and Freud apparently was not aware of his writings when developing psychoanalytic theory.28

Kierkegaard believed that experiencing anxiety was essential for a successful life, for without it one could not advance. As he noted, "Whoever is educated by anxiety is educated by possibility."29 The well-adjusted person faces anxiety and moves ahead.30 His emphasis on the importance of anxiety to success is borne out by research showing that there is an optimal relation between cognition and anxiety in performing life's tasks; with too little anxiety, one is not motivated, but with too much, impairments result.31 As pointed out by leading anxiety researcher David Barlow, without anxiety, "[t]he performance of athletes, entertainers, executives, artisans, and students would suffer; creativity would diminish; crops might not be planted. And we would all achieve that idyllic state long sought after in our fast-paced society of whiling away our lives under a shade tree. This would be as deadly for the species as nuclear war."32

Therapies arose from both the Freudian and existentialist camps but with different goals. Freud's psychoanalysis sought to rid the person of unconscious psychic conflict caused by past experiences; he viewed the analyst as an archeologist digging through layers to uncover the past. Existential therapy viewed anxiety and other sources of inner strife as a condition of human life that is best coped with by using our freedom to make choices about our actions as we go forward in life. Mainstream psychiatry today is biologically oriented and in this sense more aligned with Freud's view that anxiety can become a pathological condition for which treatment is required to heal the troubled brain. Yet, while contemporary biological psychiatry recognizes the importance of Freud's seminal contributions,33 it is divorced from his psychoanalytic theory.34

Figure 1.2: Anxiety in Popular Culture of the Mid-Twentieth Century.

(Clockwise, from upper left) W. H. Auden's 1947 poem, The Age of Anxiety; Leonard Bernstein's 1947–1949 Symphony, The Age of Anxiety; the cover of Mad magazine from 1956, introducing Alfred E. Neuman's trademark expression, "What? Me worry?"; and advertisements for the 1967 film Valley of the Dolls, the 1977 film High Anxiety, and the 1958 film Vertigo. (Center) The Rolling Stones' 1966 45 rpm hit, "Mother's Little Helper."

The popularity of both Freud and Sartre helped make anxiety a cultural byword in the United States following World War II35 (Figure 1.2). In 1947 the poet W. H. Auden published a book-length poem called *The Age of Anxiety*.36 Although the piece itself was complex and difficult and said to have actually seldom

been read,37 its title had a tremendous impact. The composer Leonard Bernstein almost immediately produced a symphony with the same name.38 The phrase “age of anxiety” has since been used to characterize everything perilous about the modern world39 and has appeared in the titles of many books, paired with subjects as diverse as science, motherhood, “the transforming vision of Saint Francis,” and “mindblowing sex.” In 1956 *Mad* magazine celebrated anxiety by putting a cartoon character, Alfred E. Neuman, and his motto, “What? Me worry?” on the cover. In film, Freud’s view of anxiety was a popular theme for Alfred Hitchcock, figuring prominently in *Spellbound* (1945), *Stage Fright* (1950), and *Vertigo* (1958). In the 1960s Woody Allen made anxiety his signature foible, the centrifugal force of his cinematic humor. Mel Brooks capitalized on the cultural fascination with anxiety, spoofing Hitchcock’s *Vertigo* and its Freudian themes in *High Anxiety* (1977). The Rolling Stones’ 1966 hit “Mother’s Little Helper” was about British housewives getting through the day on Valium (a highly prescribed antianxiety drug at the time). The use of drugs to control anxiety also played a key role in *Valley of the Dolls*, Jacqueline Susann’s popular novel that was made into an equally popular film. (“Dolls” was Susann’s nickname for the pills abused by the characters.) In Alan J. Pakula’s film *Starting Over* (1979), when the main character has a panic attack in Bloomingdale’s, his brother pleads with the other customers for a Valium; everyone in the area pulls out a pill bottle.40 The psychoanalyst Rollo May, who earlier helped fuse Freud and Kierkegaard in psychiatry,41 proclaimed in 1977 that “[a]nxiety has certainly come out of the dimness of the professional office into the bright light of the market place.”42 Simply entering the word “anxiety” into a Google search returns more than 42 million hits.

FROM FEAR TO ANXIETY AND BACK AGAIN

Scientists and mental health professionals today are greatly influenced in their views of fear and anxiety by both Freud and Kierkegaard, who each regarded fear and anxiety as perfectly normal, yet unpleasant, feelings. In fear, as we have seen, the focus is on a specific external threat, one that is present or imminent, whereas in anxiety the threat is typically less identifiable and its occurrence less predictable—it is more internal, and in the mind more of an expectation than a fact, and can also be an imagined possibility with a low likelihood of ever occurring.43 Tables 1.1 and 1.2 summarize common similarities and differences between fear and anxiety.

A simple analysis of the English language suggests that the words “fear” and “anxiety” can describe a range of emotions (Figure 1.3). We’ve seen some of these above: fear, panic, terror, anxiety, anguish, dread, worry. There are actually more than three dozen English words that are either synonyms, variants, or aspects of “fear” and “anxiety.”44 Some of these are shown in Figure 1.4.

Words usually exist because they account for something important in the lives of the people who use them—Inuit, as is well known, have many words for snow. Fear and anxiety, it would seem, are significant to us. Indeed, each generation since Auden’s time has claimed a special relation to anxiety, insisting that it is more anxious than the last.45

Based on Table 1.1 in Rachman (2004)

Based on Table 1.2 in Rachman (2004) and Table 1.2 in Zeidner and Matthews (2011)

How are we to deal with the semantic complexity of these terms and the implications of linguistic imprecision for our understanding of the underlying mechanisms of fear and anxiety? Some emotion researchers treat all (or at least many) of the terms as measures of intensity of varying degrees of fear: On the

low end are *concerned, nervous, jittery, apprehensive,* and *worried,* with *threatened, scared,* and *frightened* in the middle, and *panicked and terrified* at the other end.46 Another approach retains the centrality of fear and anxiety as categories of aversive experience and identifies specific members of these two families. *Frightened, panicked, scared, and terrified* are viewed as states that have an objective cause and an imminent consequence and thus are considered forms of fear, whereas *anguish, worry, dread, nervousness, concern, trepidation,* and *troubled* are viewed as variants of anxiety because the source, or cause, is more amorphous and the consequences less certain.

Figure 1.3: The Lexicon of Fear and Anxiety.

Figure 1.4: Some Variations on the Themes of Fear and Anxiety.

(FROM MAKARI [2012].)

But even this simple solution hints at a source of confusion in the study of fear and anxiety. While these terms are sometimes used to define *categories* (families) of experience, they are also often used more specifically to refer to distinct *types* of experience: In this context "fear" is considered as only one particular form of fearful experience among many possible others, whereas "anxiety" is likewise one particular form in the range of anxious experiences. It is also unclear the extent to which the examples in each category are truly distinct states of fear or anxiety or just slight variations, or even synonyms, of the identical states. But in spite of such complications, we at least have guidelines to help us separate the two broad categories: Fear states occur when a threat is present or imminent; states of anxiety result when a threat is possible but its occurrence is uncertain.

DEFINING FEAR AND ANXIETY

While we can often separate fear from anxiety conceptually on the basis of the nature of the threat, in our daily lives fearful and anxious states are not completely independent. It is probably impossible to feel fear without also being anxious—as soon as you are afraid of something, you begin to worry about what the consequence of the danger at hand will be. For example, the sight of an agitated person near you waving a gun compels the feeling of fear, but worry (or anxiety) quickly takes over as you fret over what the person will do. As Montaigne notes in one of the epigraphs to this chapter: "He who fears he shall suffer already suffers what he fears."

Likewise, when you are anxious, the perceived threat potential of stimuli related to your anxiety can rise such that things you typically encounter that might not usually trigger fear now do so. For example, if you encounter a snake in the course of a hike, even if no harm comes anxiety is likely aroused, putting you on alert. If farther along the trail you notice a dark, slender, curved twig on the ground, an object you would normally ignore, you might now momentarily be prone to view it as a snake, triggering a feeling of fear. Similarly, if you live in a place where terror alerts are common, benign stimuli can become potential threats. In New York City, when the alert level rises, a parcel or paper bag left under an empty subway seat can trigger much concern.

Ultimately, the question we have to ask is: Can we really make a distinction between fear and anxiety given that both are anticipatory responses to danger and thus closely entwined? I think we can, and must. As I will describe in later chapters, somewhat different brain mechanisms are engaged when the state is triggered by

an objective and present threat as opposed to an uncertain event that may or may not occur in the future. An immediately present stimulus that is itself dangerous, or that is a reliable indicator that danger is likely to soon follow, results in fear. Anxiety may well also be present, but if the initial state is triggered by a specific stimulus, it is a state of fear. However, when the state in question involves worry about something that is not present and may never occur, then the state is anxiety. Fear can, like anxiety, involve anticipation, but the nature of the anticipation in each is different: In fear the anticipation concerns if and when a present threat will cause harm, whereas in anxiety the anticipation involves uncertainty about the consequences of a threat that is not present and may not occur.

Fear and anxiety, as I will argue later, both involve the self. To experience fear is to know that YOU are in a dangerous situation, and to experience anxiety is to worry about whether future threats may harm YOU. This involvement of the self in fear and anxiety is a defining feature of these and other human emotions.

DISORDERED ANXIETY AND FEAR

While fear and anxiety are perfectly normal experiences, sometimes they become maladaptive—excessive in intensity, frequency or duration—causing the sufferer distress to the extent that his or her daily life is disrupted.47 When this happens, an *anxiety disorder* exists.48 For historical reasons, as I will soon explain, problems involving maladaptive fear and anxiety are typically grouped together under the label "anxiety disorders." Table 1.3 compares normal and pathological expressions of fear and anxiety.

What constitutes an anxiety disorder in the United States is dictated by the *Diagnostic and Statistical Manual* (DSM) of the American Psychiatric Association.49 Although the World Health Organization has its own system, the two are for the most part compatible.50 The DSM has recently gone into its fifth edition (DSM-5), but to understand its classification of anxiety disorders, it will be useful to examine prior versions first.51

The DSM classification system, which was introduced in the mid-twentieth century, was initially dominated by psychoanalytic ideas, which resulted in mental disorders being categorized into states of either psychosis or neurosis. Psychotic conditions were considered to involve thought disturbances, including delusions and/or hallucinations, a break with reality, and, in general, an inability to function in normal social situations. Neuroses involved several conditions in which one suffered from distress (sometimes debilitating distress) but without significant distortions of thought, or loss of touch with reality. The neurotic conditions most related to fear and anxiety included anxiety neurosis (excessive worry, dread), phobic neurosis (irrational fears), obsessive neurosis (repetitive thoughts), and war neurosis (mental problems in soldiers that stemmed from stress, exhaustion, and specific battlefield experiences).

Based on http://www.adaa.org/understanding-anxiety

With the arrival of DSM-III in 1980, anxiety neurosis was divided into two separate states, a partition based on research findings by the psychiatrist Donald Klein.52 Klein had been studying a new experimental drug, imipramine, to treat hospitalized schizophrenic patients in the hope of reducing their high levels of anxiety. The patients claimed that their anxiety levels were unchanged, but the staff noted a dramatic decrease in the frequency with which these patients would show up at the nurses' station complaining of physiological symptoms (inability to breathe, racing heart, dizziness) and psychological distress (feeling terrified that they were about to die). These brief bouts of intense fear (or, as they came to be called, *panic attacks*) were lessened after several weeks of treatment. Benzodiazepines, drugs like Valium, by contrast, reduced chronic anxiety but did not help with panic attacks. Findings such as these led Klein to distinguish between two broad kinds of anxiety disorders: *generalized anxiety disorder* (GAD) and *panic disorder*. While Freud anticipated this distinction, because he discussed anxiety as a general condition that sometimes had

physiological symptoms similar to those in a panic attack, he did not distinguish these as different subcategories of anxiety neurosis.

Let's look at these two conditions in a bit more detail. Generalized anxiety (worry, nervousness, apprehension, dread) is what most laypeople have in mind when they use the term "anxiety." People with GAD have prolonged, uncontrollable, and excessive worry and tension about life situations (including family, work, finance, health, romance, and other circumstances) to the point of interfering with normal routines.53 Panic disorder, by contrast, is typified by brief, intense attacks during which a person is overcome with the feeling that he or she is suffocating or experiencing a heart attack—remember that *angh*, the Greek root of the English word "anxiety," referred to physical sensations more than the mental states of worry and dread that occur in GAD.54

DSM-IV, which was published in 1994, integrated conditions related to some of the other forms of neurosis (specifically phobic, obsessive, and war neuroses) with GAD and panic disorder. Two broad categories of phobic conditions were included: *specific phobias* (in which one experiences anxiety about encountering certain objects, such as snakes or spiders, or physical situations, such as high elevations or tight, closed spaces) and *social phobias* (anxiety about attending social events such as parties or situations in which one has to speak publicly). Also added was *obsessive-compulsive disorder* (OCD), which involves recurrent thoughts (e.g., concerns about germs) that intrude into consciousness and are accompanied by repetitive actions (e.g., excessive hand washing) that are aimed at reducing distressing feelings. Finally, *posttraumatic stress disorder* (PTSD) was also categorized as an anxiety disorder. In this condition, thoughts and memories about a past, often life-threatening event led to feelings of detachment, sleep problems, and hypersensitivity to trigger cues. Although soldiers throughout history have suffered psychologically from experiences in battle, PTSD came to be recognized as a specific condition after the Vietnam War. This term replaced earlier designations, such as *war neurosis, nostalgia,*55 *shell shock, battle fatigue,* and *combat stress reaction.* But the PTSD diagnosis was not limited to battlefield conditions and instead also included a response to any kind of severe traumatic experience, such as an automobile or other accident, or rape, torture, or other forms of physical abuse.

With the arrival of DSM-5 in 2014, some retractions and reorganization occurred. In addition to using an Arabic rather than a Roman numeral to identify the edition, two of the DSM-IV disorders in the anxiety disorder domain were removed from DSM-5 and placed into separate categories: PTSD became part of *trauma and stressor-related disorders* and OCD part of *obsessive-compulsive and related disorders*.

The term "anxiety disorders" thus originally arose to subsume two states of anxiety (GAD and panic) and was retained when additional conditions were added. But the label "anxiety disorders" gives short shrift to the fact that most of the conditions included also involve fear (e.g., fear of specific objects or situations in specific and social phobic disorders; fear elicited by somatic sensations, such as heart palpitations or shortness of breath, in panic disorder). Therefore, I prefer to describe these states as *fear and anxiety disorders*, conditions in which maladaptive fear and/or anxiety plays a central role. With this in mind, I break with the DSM-5 categorization and include PTSD in my discussion of fear and anxiety because it involves maladaptive fear (fear of trauma-related cues).56 Some of the accepted characteristics typically associated with these fear and anxiety disorders are shown in Figure 1.5.

Together, fear and anxiety disorders are the most prevalent of all psychiatric problems in the United States, affecting about 20 percent of the population, more than twice the number who suffer from mood disorders such as depression and bipolar disorder, and twenty times the number with schizophrenia.57 The economic cost of fear and anxiety disorders is estimated to exceed $40 billion annually.58 These conditions have a significant impact on the workforce. For example, a study from Australia found that anxiety and affective disorders resulted in 20 million work impairment days annually, mostly involving absences.59

But the problem is actually more pervasive than the 20 percent anxiety prevalence statistic implies. Problems with threat processing and maladaptive fear and anxiety are factors in many other psychiatric conditions. GAD and depression often occur together, and fear and anxiety can play a role in schizophrenia, borderline personality disorder, autism, and eating and addictive disorders. Moreover, many individuals are impaired by uncontrollable fear or anxiety without having received an official psychiatric diagnosis of their condition. These issues can also trouble those whose health is compromised by illnesses such as cancer, heart disease, and other chronic physical ailments. Even many people who are considered otherwise healthy in mind and body can from time to time suffer from bouts of excessive fear and worry. A better understanding of the nature of these conditions, and the brain mechanisms involved, would be extremely helpful to just about everyone.

Figure 1.5: Major Symptoms of Fear / Anxiety Disorders.

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What determines who will be likely to suffer from a fear or anxiety disorder? For example, why does only a relatively small proportion of people exposed to a trauma develop PTSD?60 David Barlow has proposed that three factors make people vulnerable to these disorders61 (Figure 1.6). One is genetics or other *biological factors* in the brain. The heritability of anxiety is estimated to be between 30 percent and 40 percent, which is considerably less than that of some other conditions.62 But the rates rise when one examines particular anxious traits, such as the tendency to be inhibited and withdrawn in situations involving uncertainty. Genetic influences on anxiety and other mental disorders are complex and involve interactions between multiple genes. Individual differences in brain organization that arise from environmental influences, and interactions between genetic and environmental factors, are also important. Another source of vulnerability involves *general psychological processes,* such as an individual's tendency to perceive situations as unpredictable and uncontrollable. The third factor listed by Barlow is *specific learning experiences*. If a child is given excessive attention when ill, he may continue to use "sick behaviors" as a way to attract attention and sympathy. Similarly, if a child observes a parent or other adults using such strategies, he might adopt them as well. Early life situations in which one experiences negative consequences that cannot be controlled following situations involving uncertainty may well predispose one to feel less in control in later life. It should be noted that psychological processes and learning experiences are, in the end, also biological in nature, because they are products of the brain and as such are also subject to genetic influences and the influence of gene-environment interactions, or what is called epigenetics.

Figure 1.6: Vulnerability to Pathological Anxiety.

(Based on Barlow [2003].)

The social scientists Allan Horwitz and Jerome Wakefield have called for prudence in the use of the term "disorder" when talking about mental problems. In their books, *All We Have to Fear*63 and *The Loss of Sadness,*64 they point out that "disorder" implies that something physical is not working as it should. In people who are having trouble with anxiety, they argue, the brain is often doing what it is supposed to do—it's just doing it in the wrong context. Fear and anxiety triggered by strangers, snakes, heights, and the like served our ancestors well by helping them avoid dangers but can cause distress in the modern world. Horwitz and Wakefield are particularly concerned about the rising rates of diagnosis of fear and anxiety disorders and the alarming increase in the use of medications to treat brains that are working fundamentally correctly, from an evolutionary point of view. They do accept that disordered fear and anxiety can exist and describe criteria to distinguish disordered from normal states. Regardless of what one thinks of their gauges

for defining disorders, their book is important because it raises important social issues about psychiatric diagnosis and treatment.

THE CENTRALITY OF THREATS

This book is called *Anxious*, and it discusses how feelings of anxiety and related states (including worry, concern, dread, disquiet, apprehension, nervousness) come about. But the close connection between fear and anxiety demands that these two emotions be understood together. A key factor that links them is that they both depend on mechanisms in the brain that detect and respond to threats to well-being.

Threats, whether present or anticipated, real or imagined, demand action. As many others have noted, threat detection provides preparation for fight or flight.65 We are all familiar with the *fight-flight response*, the defensive emergency reaction that is triggered when we encounter present or anticipated threats, and that moves into overdrive when we are under stress (see Chapter 3). This whole-body reaction is mobilized to help us survive an encounter with danger. When it is in play, our conscious mind is consumed with fear or anxiety, and often with both. Threat processing is at the heart of fear and anxiety.

Particularly important is the fact that threat processing is altered in each of the fear and anxiety disorders (Figure 1.7).66 In this book I will be less concerned with the disorders themselves than with the question of how threat processing contributes to maladaptive feelings of fear and anxiety in these disorders.67 People who suffer from them are hypersensitive to threats, which seize and hold their attention, a condition sometimes called hypervigilance. They are also impaired in distinguishing things that are dangerous from those that are safe and overestimate the significance of perceived threats. Even when threats are not present, they worry excessively that threats will occur and constantly scan the environment to try to understand why they feel distressed. They go to extremes to escape from or avoid threats, so much so that these avoidance strategies interfere with daily life.

Figure 1.7: Alterations of Threat Processing Occur in Many Psychiatric Disorders.

SHOWING MY HAND

Any understanding of fear and anxiety presumes an understanding of emotion. So before we go further, I want to make clear what my own view of the subject is, using the emotion fear for illustrative purposes. In many ways my core view of emotion has not changed since the 1980s.68 But recently I have begun to discuss it somewhat differently in an effort to sharpen the conceptualization of this complex psychological function and its relation to brain mechanisms.69

Traditionally, emotion theories have focused on conscious feelings.70 For example, in the late nineteenth century, William James, the father of American psychology, proposed that fear is a conscious feeling that occurs when we find ourselves responding to danger; the feeling of fear, for him, was the perception of body signals that are unique to defending against danger.71 Not all theorists have agreed with James about how conscious feelings come about, but many have concurred that the feeling *is* the emotion. Freud, as mentioned above, said that anxiety is “something felt” and also noted, “It is surely the essence of an emotion that we should feel it.”72 More recently, the Dutch psychologist Nico Frijda claimed that emotions are primarily “hedonic experiences.” Lisa Barrett, James Russell, Andrew Ortony, and Gerald Clore, and others, emphasize that emotions are psychologically constructed conscious experiences.73 Clore notes that “emotions are never unconscious.”74

Other theorists, though, have found conscious experience to be unnecessary, or even a detriment, to understanding emotion. For example, in the early twentieth century, behaviorists argued strongly that consciousness, being unobservable, had no place in psychology; they insisted that behavior alone should be the focus of inquiry.75 This led to the idea that fear was a relation between stimuli and responses rather than a specific feeling.76 When behavioral psychologists later turned to physiology in an effort to understand how stimuli and responses are connected in the brain, fear became a central motivational state—a physiological state of the brain that organized responses to dangerous stimuli. But like the behaviorists, these physiological theorists, for the most part, also shunned conscious experiences—central states were physiological intermediaries between stimuli and responses, not subjectively felt states.77 While this approach provided a way of studying the emotions like fear similarly in animals and humans, it achieved this goal by ignoring the feeling of fear, which is what most people think fear is.

But even those who argue that emotions *are* conscious experiences sometimes claim that such experiences are just one aspect or component of emotion. For example, the Swiss psychologist Klaus Scherer views emotion as a process consisting of cognitive appraisals, expressive responses, physiological changes, and conscious feelings.78 In this view, fear is what happens when we cognitively appraise a situation as dangerous, express certain behaviors in response to it, are physiologically aroused, and feel fear. This approach seems logically cumbersome to me because it regards fear as both the overall process and the specific feeling of being afraid; fear (the experience) is thus a component of fear (the process).

Yet another theory is that emotions are hardwired in the brain and unleashed in the presence of trigger stimuli.79 In this view, championed by those who adhere to basic emotions theory, innate behavioral reactions, physiological responses, and conscious feelings all flow from a fear center or network. As I will argue later, although threats do indeed release innate behavioral and physiological patterns, the feeling of fear is not itself innate, a view consistent with the ideas of psychological construction theories of emotion.

Fear, anxiety, and other emotions are, in my view, just what people have always thought they were—conscious feelings. We often feel afraid while we freeze or flee in the presence of danger. But these are different consequences of threat detection—one is a conscious experience and the other involves more fundamental processes that operate nonconsciously. The failure to distinguish the conscious experience of fear and anxiety from more basic unconscious processes, I argue, has led to much confusion. The more basic processes contribute to emotional feelings, but they evolved, not to make conscious feelings, but instead to help organisms survive and thrive. For the sake of avoiding confusion, the more basic nonconscious processes should *not* be labeled as “emotional.”

Feelings of fear, in my view, result when we become consciously aware that our brain has nonconsciously detected danger.80 How does this happen? It all starts when an external stimulus, processed by sensory systems in the brain, is nonconsciously determined to be a threat. Outputs of threat detection circuits then trigger a general increase in brain arousal and the expression of behavioral responses and supporting physiological changes in the body. Signals from the behavioral and physiological responses of the body are sent back to the brain, where they become part of the nonconscious response to danger (sensory components of these responses can be “sensed” just like sights or sounds). Brain activity then comes to be monopolized by the threat and by efforts to cope with the harm it portends. Threat vigilance increases—the environment is scanned to figure out why we are aroused in this particular way. Brain activity related to all other goals (eating, drinking, sex, money, self-fulfillment, etc.) are suppressed. If, via memory, environmental monitoring reveals that “known” threats are present, attention becomes focused on these stimuli, which are consciously “blamed” for the aroused state. Memory also allows us to know that “fear” is the name we give to experiences of this type (starting in childhood, we build up templates of what it is like to be in states we label with emotion words). When the various factors or ingredients are integrated in consciousness, an emotion, specifically the conscious feeling of fear, is thus compelled. But this can only happen if the brain in

question has the cognitive wherewithal to create conscious experiences and interpret the contents of these experiences in terms of implications for one's well-being. Otherwise, the brain and body responses are a motivational force that guide behavior in the quest to stay alive, but the feeling of fear is not a part of the process. This does not mean that the feeling of fear is a mere by-product. For once it exists, it opens up the resources of the conscious brain to the quest to survive and thrive.

I am not alone in arguing for this view of emotions as cognitively assembled conscious feelings.81 The recent notion that emotions are "psychological constructions"82 is perhaps the cognitive-based theory of emotions that is closest to my own view.

One important implication of the ideas to be developed in this book is that they reveal a disconnect between the troubling feelings of fear and anxiety that motivate people to seek help and the way research on those feelings—including research on how to find new treatments to allay them—is conducted and interpreted. Consciousness is no longer a taboo subject in science, and much progress has been made in this area in recent years. Yet research on fear and anxiety disorders in animals, and also in humans, that I and others have conducted is often focused on how the brain detects and responds to threats, processes that operate nonconsciously. Although this work is very relevant to understanding conscious fear and anxiety, it has to be understood in the proper context. Responses to threats, in spite of common practice, are not foolproof markers of conscious feelings, even in humans, and likewise should not be assumed to be so in animals.

A major aim in this book is to provide a framework that will allow a better understanding of the connection between research, therapy, and conscious feelings. But to do that we have to be careful about when we should call upon consciousness and when we should not. We can't understand fear and anxiety if we ignore consciousness, but neither should we overemphasize its role.

LOOKING AHEAD

Having established the tangled web of fear and anxiety from the point of view of threat processing by the brain in the present chapter, I will summarize in the following one how my current views evolved in the course of my three-decade struggle to scientifically understand the emotional brain. Subsequent chapters cover defense in the animal kingdom and the brain mechanisms that enable animals, including humans, to detect and respond defensively to threats. I then address the question of what we have inherited from animals. Contrary to the popular opinion of laypeople and many scientists, I argue that we have not inherited feelings like fear or anxiety from animals; we have instead inherited mechanisms that detect and respond to threats. When these threat-processing mechanisms are present in a brain that can be conscious of its own activities, conscious feelings of fear or anxiety are possible; otherwise threat processing mechanisms motivate behavior but do not necessarily result in or involve feelings of fear or anxiety. Organisms that have the capacity to be conscious can feel fear; otherwise they cannot have such experiences. Thus, if we want to understand feelings of fear and anxiety, we have to understand consciousness, and several chapters on this topic follow. One examines the physical basis of consciousness, another examines the role of memory in consciousness, and a third explains how conscious feelings of fear and anxiety emerge when nonconscious consequences of threat processing are consciously experienced. The final three chapters turn to brain mechanisms related to fear and anxiety and their disorders, and offer a reconception of these disorders. The final chapters also suggest how research on brain mechanisms can offer new ways to help people better cope with such troubling feelings.

Anxiety and its partner, fear, are, as Freud said, riddles, and seeking their solution will take us through many aspects of how the brain and its mind work. Many topics in psychology and neuroscience, ranging from basic mechanisms of defensive behavior in animals to decision making by humans, from automatic nonconscious processing to conscious experience, from perception and memory to feelings, will be covered. Some of these

involve complex brain mechanisms, but rest assured that the principles, which will be my focus, for the most part are easily grasped.

CHAPTER 2

RETHINKING THE EMOTIONAL BRAIN

"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 KAGAN1

After working on what I referred to as the "emotional brain" for more than thirty years, I concluded that this terminology needed rethinking.2 Jerome Kagan's quote in the epigraph above hints at my reasoning but doesn't go far enough. Kagan implies that there are two different kinds of fear states, with different underlying brain systems, that are elicited by fear-arousing stimuli, one involving conscious feelings and the other involving behavioral and physiological responses. In contrast, I believe we should restrict the use of emotion words like "fear" to *conscious* feelings, such as the feeling of being afraid. Brain systems that detect threatening stimuli and control behavioral and physiological responses elicited by these stimuli should not be described in terms of fear. The latter systems operate nonconsciously in humans, and although they contribute to feelings of fear, they are not fear mechanisms per se. This chapter explains why I think the distinction between mechanisms that detect and respond to threats outside the realm of consciousness, as opposed to mechanisms that create conscious feelings of fear, is so important for our conception of fear and its partner, anxiety.

IN THE BEGINNING

When I started my research on the emotional brain in the early 1980s, ideas like the limbic system theory were popular.3 It proposed that our reptilian ancestors were dominated by reflexes and instincts. Then, with the emergence of mammals, a new brain system (the limbic system) evolved to make feelings, enhancing the adaptive potential of the newest vertebrates. Later in mammalian evolution the neocortex appeared and provided a thinking brain that made reasoning and emotional control possible. Although the limbic system concept inspired much research, the goal was often as much about validating the concept as about understanding the emotional brain. If some region outside the limbic system happened to be implicated, rather than calling into question the limbic system theory, the criteria for inclusion of brain areas were simply changed. As a consequence, the limbic system theory lost its connection to the theory of brain evolution upon which it was based.4 (Unfortunately, the limbic system theory continues to be prominent in both lay and scientific discussions of the emotional brain in spite of its evolutionary basis having been discredited.)

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I thought that a different approach was needed, one that made minimal starting assumptions about how emotions are organized in the brain. The approach I took was to follow the flow of information in the brain from the sensory system that processes a stimulus to the muscles that control the responses to it. Somewhere along that pathway would be mechanisms that detect the significance of the stimulus and trigger the

appropriate responses. Limbic areas might well be implicated, but the point was to have an objective approach to the circuitry rather than one that presumed to know the answer before the research was done.

I was led to this “information flow” conception by prior experiences I had had at SUNY Stony Brook while doing doctoral research with Michael Gazzaniga.5 We studied patients in whom the two sides of the brain were separated in an effort to control epilepsy. In most people, information in one hemisphere is automatically and instantly transferred to the other hemisphere, a process that enables the two sides of the brain to work together seamlessly in daily life.6 In our *split-brain patients*, however, a stimulus presented to one hemisphere remained in that hemisphere (Figure 2.1). For example, if you show a split-brain patient’s right hemisphere a picture—say, of an apple—he will be unable to name the stimulus because the ability to speak is located in the left hemisphere. He can reach into a bag containing several items with his left hand (which is connected to the right hemisphere) and identify the apple by touch and retrieve it with ease. The right hand (which is connected to the left hemisphere) can’t do this with any degree of accuracy because the left hemisphere did not see the stimulus. When studying split-brain patients, you can’t help but picture signals flowing from point to point in the brain to construct what we are seeing, remembering, thinking, and feeling, and in controlling behavior.

Figure 2.1: Information Flow in the Split-Brain.

In the human brain, visual stimuli that appear to the left side of center are transmitted to the right hemisphere, and stimuli to the right side of space are transmitted to the left hemisphere. Connections between the two hemispheres (not shown) allow stimuli seen by one to be seen by the other. Through these connections, each hemisphere can construct a complete view of perceptual space, and visual stimuli presented in the left field of view and thus directed to the right hemisphere can be talked about by the left hemisphere, where speech is typically controlled. In the split-brain patient, because the connections between the two hemispheres are surgically sectioned as a treatment for epilepsy, if an apple is presented to the left side of space and thus directed to the right hemisphere, it remains in the right hemisphere and the patient cannot verbally describe the stimulus, as the left hemisphere does not have access to the sensory information.

Conceiving of brain functions in terms of information flow had become inevitable with changes in the psychological zeitgeist. For decades behaviorists had dominated psychology, shunning all talk of mind, consciousness, and other unobservable inner factors (whether in the mind or brain) in explaining behavior.7 A science of psychology, they said, must be based on observable events—stimuli and responses. But by the 1970s behaviorism had been supplanted by the cognitive approach, which treated the mind not as a place where conscious experiences occur so much as an information-processing system that connects stimuli with responses, and that does not necessarily involve consciousness.8 Split-brain studies fit perfectly within this intellectual framework.

After I received my PhD in 1978, Gazzaniga and I both moved to Manhattan to work at Cornell Medical School. Initially, I was exploring the consequences of brain damage on language and attention,9 but my real interest, which had begun with a study we had conducted in a split-brain patient, was in the brain mechanisms of emotion.10 When we showed the patient’s right hemisphere an emotional stimulus, the left hemisphere could not name it but could rate its emotional valence. This suggested that the cognitive processes involved in perceiving what a stimulus is are separable in the brain from the processes that evaluate its emotional significance. I wanted to figure out how emotional significance was added to a stimulus as it flowed, in the form of information, through the brain. Because there was no way to pursue detailed brain mechanisms in humans, I turned to studies of rats.

The field of neuroscience was officially born as a discipline when the Society for Neuroscience was formed

in 196911 and by the end of its first decade was coming of age. In 1979, with Gazzaniga's help, I got a position in the Neurobiology Laboratory at Cornell, run by Don Reis, which had all the latest and greatest tools for studying the brain. At the time, everyone in our young field was aware of Eric Kandel's pioneering research on learning and memory.12 Kandel had begun his work in the 1960s with studies of how memory is made in the brains of rats, though he soon concluded that brain research was not at a sufficiently advanced state to be able to tackle a complex issue like memory in complex animals. He accordingly recast his research in a way that would enable him to make progress with the tools available at the time. Specifically, he picked a simple organism (a sea slug, the invertebrate *Aplysia californica*) and focused on several simple forms of learning in which this organism engages. He then determined the complete neural circuit by which sensory information flows to motor output during the behaviors, and he isolated the cells and synapses that change during learning and the molecular mechanisms in the cells and synapses that made the cellular changes possible. With this "what, where, and how" strategy, Kandel revolutionized research on learning and memory, earning him a Nobel Prize in 2000.

A word or two is in order here about the behavioral backbone of the Kandel strategy. There are two basic approaches to studying learning in the laboratory: classical and instrumental conditioning. As is well known, in the early twentieth century Ivan Pavlov discovered that after pairing a sound with food, the sound alone would elicit the salivary reflex in dogs.13 Thus, classical (or Pavlovian) conditioning brings an inborn reflex under the control of a novel stimulus. By contrast, in instrumental conditioning, pioneered by Edward Thorndike in the late nineteenth century, a new response is learned because of the success of that response in obtaining a positive, or avoiding a negative, outcome.14 A typical example is a rat that learns to press a lever to receive food. This was called instrumental conditioning because the response is instrumental in achieving the outcome (it is also called operant conditioning).15 When Kandel started his research, most work on learning and memory in the brain was being done using instrumental conditioning because it was thought to be more relevant to complex human behavior than simple Pavlovian conditioned reflexes.16 But Kandel recognized that research on the neural basis of instrumental conditioning in mammals was going nowhere fast and that more progress might be made by using Pavlovian conditioning and other simple learning procedures in an organism with a less complex nervous system.17 That insight made possible his pioneering work.

Based on Gluck et al, 2007.

By the time I began my research on emotion in Reis's laboratory, well over a decade had passed since Kandel had started his project, and the field of neuroscience had advanced considerably. It was now possible to map connections between neurons in different brain areas, record cellular responses from neurons, disrupt neural activity, and measure molecules related to learning, not just in invertebrates but also in the brains of mammals and other vertebrates. Inspired by Kandel's success, researchers were thus beginning to explore memory in the brains of complex animals using Pavlovian conditioning.18

Some researchers had also begun to use Pavlovian conditioning to study emotional behavior, especially defensive or fear behavior, in mammals.19 I was particularly taken with the behavioral studies of Pavlovian fear conditioning in rats being done by Robert and Caroline Blanchard and by Robert Bolles and his students Mark Bouton and Michael Fanselow.20 These researchers showed that when an innocuous stimulus, such as a tone, was paired with mild electric shocks, it elicited freezing behavior, whether tested after a few minutes, or days or weeks later (Figure 2.2). Freezing is an innate defensive response that is just as important to animals as its more familiar partners, fight and flight.21 (Indeed, as discussed in the next chapter, the fight-flight reaction is now often described as freeze-fight-flight.) The tone also produces increases in blood pressure, heart rate, and respiration and releases hormones like adrenalin and cortisol,22 providing

physiological support for energy-demanding defensive behaviors.

The meaning and even the value of the term “innate” in relation to behavior has been debated over the years.23 It is now widely recognized that individual experience affects the way genetic programming is expressed, leading to the lack of a clear boundary between what is innate and what is learned. Although some shun the use of the term “innate,” others think that it is useful, as some behaviors are decidedly more dependent than others on characteristics that are expressed so consistently within a species that the opportunity to learn the behavior seems limited. Freezing is one such example.

Fear conditioning is an example of associative learning, a process by which the brain forms memories about the relation between events. In the language of psychological learning theory, the tone in the example above is a conditioned stimulus (CS), the shock an unconditioned stimulus (US), and the responses elicited by the CS after conditioning are conditioned responses (CRs). During fear conditioning, the brain thus learns the relation between the CS and the US. After conditioning, the CS tone becomes a warning signal that danger is imminent. When the CS appears, it thus elicits the conditioned fear responses because it activates the CS-US association, which controls freezing and other fear CRs. Although freezing is said to be a conditioned response, the response is not learned. What gets conditioned is the ability of the CS to elicit the response.

Figure 2.2: Fear Conditioning: The Procedure.

So-called fear conditioning is a variant on Pavlovian conditioning in which an innocuous conditioned stimulus (CS), often a tone, is paired with an aversive unconditioned stimulus (US), typically footshock. My laboratory has used this procedure extensively in rats, but it can be used similarly in a wide variety of animals, including humans. On the first day of typical study the rat is exposed to the CS alone (habituation). The next day one or more CS-US pairings occur (conditioning). One or more days later, the conditioned response is tested by presenting the CS alone (CS test). As described later, in order to better separate processes that give rise to feelings of fear from those that underlie the detection and response to danger, we have begun to use the expression “threat conditioning” in place of “fear conditioning.”

In spite of much elegant work on Pavlovian fear conditioning at the behavioral level, this procedure was not being used in any systematic way to study how fear mechanisms operate in the brain.24 Most research in this area was still employing complex instrumental conditioning tasks (especially tasks in which animals learned somewhat arbitrary responses to avoid shocks).25 I thought that with all the tools available in the Reis laboratory, I could use the Kandel strategy, in conjunction with Pavlovian conditioning procedures, to trace the flow of information that enabled a meaningless stimulus to elicit fear responses in mammals (rats) following fear conditioning. This should be possible, I reasoned, because the responses, which are expressed the same in all rats, are driven by a specific stimulus that was completely under my control as an experimenter. As a result, I might be able to trace the flow of the stimulus processing from the CS sensory system to the CR motor system. So that’s the approach I took initially at Cornell and then at NYU after I moved there to set up my own laboratory in 1989.26 And work by my laboratory and the laboratories of colleagues using Pavlovian fear conditioning was very successful in achieving, in a few short years, what instrumental avoidance conditioning had failed to do—identification of the brain areas and connections between them that constituted what came to be known as the brain’s fear system.

THE FEAR SYSTEM

I started my work on the neural basis of fear conditioning by determining which areas of the auditory system were required for the auditory CS to elicit freezing and blood pressure responses. Then, using anatomical

connection-tracing techniques, I pinpointed possible output targets of the key auditory processing areas. One of the targets suggested by the tracing studies was the amygdala. When we lesioned this area, or disconnected it from the auditory system, the fear conditioned responses were eliminated. Within the amygdala, we also found an area that receives the auditory CS input (the lateral amygdala, LA) and connects with an area (the central amygdala, CeA) that sends outputs to downstream targets that separately control freezing and blood pressure conditioned responses. Further, we were able to locate cells in the LA input region that received both the auditory CS and the shock US. This was an especially important discovery because the integration of the CS and US at the cellular level was thought to be required for fear conditioning to occur. After the circuit and cellular changes involved in the process were identified, we turned to the molecular mechanisms in the LA that underlie the learning and expression of conditioned fear, many of which were the same as those discovered by Kandel and others in invertebrates.27 In doing this work I have been fortunate to have had a fantastic group of people with me over the years, and I have dedicated this book to them.28 They have contributed not just in terms of their lab skills and work ethic, but also intellectually.

The laboratories of several close colleagues also made many important contributions to this area of research. Initially, Bruce Kapp, Michael Davis, and I were the main players in this game.29 But soon Michael Fanselow, who had started out studying fear conditioning at the level of behavior,30 turned to questions about brain mechanisms as well.31 Each had students who eventually also started laboratories,32 and others joined in this exciting new area of work.33 Fear conditioning became one of the most popular areas of research in neuroscience, and one known for having made great strides in relating brain to behavior.

Figure 2.3: Fear Conditioning: The Circuit.

a. The Simple Version. The basic circuitry underlying the acquisition and expression of fear (threat) conditioning involves conditioned stimulus (CS) and unconditioned stimulus (US) sensory transmission to the lateral nucleus of the amygdala (LA), where a CS-US association is learned and stored. LA communicates with the central nucleus of the amygdala (CeA), which then connects with areas that control conditioned fear responses. **b. A Slightly More Complex Version.** The LA connects with the CeA directly and by way of other amygdala areas, such as the basal nucleus (BA) and intercalated cells (ITC). CeA then connects with downstream targets that separately control freezing, autonomic nervous system (ANS), and hormonal conditioned responses. Additional details are described in Chapters 4 and 11.

A simplified and more complex version of the amygdala-centered fear conditioning circuit uncovered by this collective body of work is illustrated in Figure 2.3.34 A still more elaborate version of the circuitry will appear in later chapters. Through this research, the amygdala came to be viewed as a key component of the brain’s fear system.35

FEAR AS A STATE THAT INTERVENES BETWEEN THREAT STIMULI AND FEAR RESPONSES

That amygdala-based circuits described above were part of a fear system was widely accepted. But the question of what exactly a fear system does has turned out to be a tricky issue.

The most obvious answer to this question is that a fear system makes fear: A threat activates a fear system in the brain, and the result is a feeling of fear. This feeling then drives the expression of behavioral defense responses and physiological accompaniments (Figure 2.4). Consequently, the defensive response is often used as a sign that a person or animal is feeling fear.

Figure 2.4: The Darwinian (Commonsense) View of Fear.

Darwin adopted the everyday or commonsense view, treating fear and other emotions in people as “states of mind” that control emotional responses, and that have been inherited from our animal ancestors.

William James called this the commonsense view of fear: We run from a bear because we are afraid of it.36 Although he rejected it, the commonsense view has thrived. Charles Darwin was a proponent of this idea, calling fear a “state of mind” that accounts for the expression of fear behavior.37 This is also the view most laypeople have of fear, and when journalists write about the brain’s fear system, they typically adopt this perspective as well. Some scientists also argue that innate circuits involving the amygdala are responsible for the feeling of fear.38 But this is not the only perspective.

Behavioral research on fear conditioning took off in the 1940s and 1950s due to the rise of O. Herbert Mowrer’s influential theory that fear conditioning plays a key role in maladaptive fear and anxiety in people.39 Fear conditioning researchers treated fear as a state that intervenes between threats and defense responses,40 but not in the way conceived by Darwin’s and other commonsense theories. Coming from behaviorism, most researchers avoided reference to conscious state and feelings.41 Instead, fear was a central state, specifically a *defensive motivational state*,42 a physiological response in a hypothetical brain circuit43 (Figure 2.5). Most of these early central state researchers were not physiologists, and the physiological states were conceptual placeholders, what are sometimes called *intervening variables* or *hypothetical constructs*,44 rather than actual brain mechanisms. However, when researchers from this tradition started studying the brain, they applied the central state term to the circuits uncovered. Thus, amygdala activity came to be viewed as the neural embodiment of a central state called fear.45

Figure 2.5: The Central State View of Fear.

Physiological psychologists trained in the behaviorist tradition treat fear as a physiological state (rather than a subjective feeling—“state of mind”) that controls fear responses. However, calling the physiological state “fear” often leads to confusion about what fear really is, as even those who treat it in physiological terms often talk and write in a way that implies that the physiological state is the neural embodiment of the feeling of fear.

Central states, as a link between threats and fear responses, served a function similar to the Darwinian notion of feelings. But unlike Darwin, proponents of central states did not necessarily call upon a conscious feeling as the link between stimuli and responses. The entire question of conscious emotion was simply not considered relevant. For example, Michael Fanselow, a leading figure in the study of the central state of fear in animals, stated: “Our job is to redefine the concept of motivation in a scientific manner, and those new definitions should replace the layman’s informal view. I don’t see how subjective experience helps us do that.”46 According to Fanselow’s mentor, Robert Bolles, “Human experience . . . cannot be invoked to lend ancillary validity to a construct that is otherwise anchored to behavioral phenomena. . . . Its surplus meaning must always remain surplus.”47 And Robert Rescorla, another prominent behavioral fear conditioning researcher, 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.”48 Because many central state proponents removed subjective experience (consciousness) from the causal chain, central states were *de facto* nonconscious states for them. But not all researchers had this view. Mowrer, for example, was more like Darwin, treating fear central states as fearful subjective experiences that drive defensive behaviors. And even those who did eschew subjective states often wrote and spoke as if fear, the state, meant fear the

feeling, describing threatened rats as “afraid,” “frightened,” “frozen in fear,” “anxious,” and the like. How would a naïve reader or listener know that their fear did not mean “fear.”

FEAR AS A COGNITIVE CONSEQUENCE OF THREAT PROCESSING

I took a different approach from the ones described above. I thought that the Darwinian commonsense idea was flawed because it attributed too much to conscious fear, and the central state view was flawed because it ignored conscious fear. I believed conscious and nonconscious states both played roles, but the roles needed to be kept separate.

I was compelled toward my view by the split-brain studies I had done earlier. Gazzaniga and I noticed that the left hemisphere of a split-brain patient often commented on behaviors that were produced by the right hemisphere. One might expect that in people with this condition the left hemisphere would be surprised when it saw its body doing something for a reason that it (the left hemisphere) was not aware of. Instead, the left hemisphere took these unexpected behaviors in stride and wove them into its stream of thought. This was a fascinating phenomenon, so we designed some studies to explore it.49 Basically, we coaxed the right hemisphere to respond behaviorally and then simply asked the left hemisphere, “Why did you do that?” Without hesitation, time and time again, the left hemisphere came up with an explanation. For example, when the patient was urged to stand up via stimuli presented to his right hemisphere, in response to our query as to why he did that, his left hemisphere said he needed to stretch; when he waved, it was because he thought he saw a friend out the window; he scratched his hand because it itched. These were fabrications on the part of the conscious brain, explanations for why the body responses were being generated. Gazzaniga and I proposed that the human brain does this all the time.50 While we are not always privy to the motivations underlying the responses controlled by our brains, consciousness ties the loose ends together by coming up with an interpretation that unifies the mind and behavior by filling in the blanks of an otherwise incomplete mental pattern. Gazzaniga called this the *interpreter theory* of consciousness.51 And I used this idea to explain how nonconscious processes that underlie emotional responses contribute to the conscious feelings that we experience.

In the mid-1980s, around the time I was starting work on fear conditioning in rats, I developed a model, based on our split-brain conclusions, of unconscious emotional processing in the brain. Specifically, in a 1984 book chapter, I proposed that emotional stimuli, transmitted to the brain via sensory systems, are processed nonconsciously to initiate emotional responses.52 In 1996 I published the *Emotional Brain*, in which I framed the model in terms of brain mechanisms of fear, proposing that threat stimuli activate the amygdala in the process of eliciting fear responses; the amygdala processing, I argued, is automatic and requires neither conscious awareness of the stimulus nor conscious control of the responses.53 This conclusion was supported by a handful of findings at the time but has since been bolstered by many studies showing that the amygdala can process threats and trigger conditioned responses without a person’s being aware of the actual stimulus54 and without having any feeling of fear.55 This conclusion is consistent with our common experience of inadvertently responding to something and only afterward realizing that danger was present—as when one jumps back from a speeding bus and then, on the basis of this reaction, consciously realizes that he or she was in danger. The neural processes that enable us to consciously know that danger is present are slow compared with those that unconsciously control certain built-in protective (defense) responses. It is now recognized that the contrast between fast and automatic and slower, more deliberate processes is a fundamental organizational principle of the human mind and brain.56

In the 1984 chapter, I had also hypothesized how conscious feelings come about. I suggested that sensory processing in the brain splits into two channels, one that detects the emotional significance of stimuli and controls emotional responses and another that engages cognitive processing and leads to conscious feelings (see Figure 2.6). In the *Emotional Brain*, continuing the fear theme, I argued that the conscious feeling of

fear is due to the representation in consciousness, via attention and other neocortical cognitive processes, of unconscious ingredients that are consequences of activation of the amygdala threat-processing circuit. I proposed that we could study the nonconscious aspects of fear in animals and humans alike, but that the conscious feelings of fear are best studied in humans. (More about that later.)

The amygdala circuit, I proposed, thus contributes to fear in two ways. It has a direct role in detecting threats nonconsciously and controlling subsequent behavioral and physiological fear responses, but it also has an indirect role, via cognitive systems, in the emergence of a conscious feeling of fear. Specifically, I proposed that the nonconsciously controlled brain and body consequences are raw materials that, when cognitively interpreted, contribute to conscious feelings of fear. When I used the term "fear system," then, I was referring to this entire process, including the amygdala's role in both controlling fear responses and in providing ingredients that indirectly contribute to conscious feelings of fear.

Figure 2.6: The Fear System: My Original View.

In 1984, I described emotional stimuli as being processed through two channels that diverge from the sensory pathway in the brain (LeDoux, 1984). One channel directs the stimulus to circuits that nonconsciously detect and respond to the stimulus, and the other channel directs the stimulus to cognitive systems that give rise to conscious emotional feelings. This idea is shown here in terms of threat stimuli and how they separately trigger so-called fear responses and feelings of fear. Over the next decade, the amygdala emerged as being responsible for the unconscious processing of threats, as described in the Emotional Brain in 1996. While I still adhere to the same basic idea about the two channels of threat processing, I no longer refer to this group of circuits as a "fear system." Saying that the amygdala is part of a fear system led to the view that the feeling of fear is a product of the amygdala, in spite of the fact that I argued that the amygdala is a nonconscious processor and that fear is a product of cognitive systems in the neocortex. I therefore now use more descriptive terminology to help make the distinction clear (see Figure 2.7 and Table 2.2).

In retrospect, I now believe that it was a mistake to use the expression "fear system" to describe the role of the amygdala in detecting and responding to threats, and also erroneous to talk about fear stimuli and fear responses in this context. To refer to the circuit that detects and responds nonconsciously to threats as part of a fear system, as I did, and as central state proponents have done, unnecessarily complicated things. Because the most commonly accepted meaning of fear is the conscious feeling of being afraid, those outside the field who came across research on the fear system naturally thought that it was a system that generated feelings of fear. Thus, even though research on the so-called fear system was really mostly about systems that operate nonconsciously in detecting and responding to threats, and thus at odds with the commonsense view, our findings were interpreted as supporting the commonsense contention that feelings of fear are unleashed from the amygdala by threats. It didn't help that fear system researchers often wrote in a way that strongly implied that the neural activity in the amygdala is the underpinning of the feeling of fear; for example, describing rats as "scared" or "frozen in fear" or "anxious." When this is done, the difference between the central state and the commonsense view of fear vanishes.

Don't get me wrong. I'm not innocent in all this. Although I called upon separate brain circuits to account for nonconscious threat detection versus conscious feelings, I also talked about the amygdala as being involved in fear. I was basically thinking in terms of conscious versus unconscious fear. But I slowly began to realize that this was confusing, and finally, in 2012, I wrote a long piece called "Rethinking the Emotional Brain" in the journal *Neuron*. In it, I introduced the ideas of survival circuits and global organismic states. These, I suggested, provide nonconscious ingredients that are cognitively interpreted in the assemblage of conscious feelings, so-called emotions. In 2013 I was elected to the National Academy of Sciences and asked to write

an inaugural article in the *Proceedings of the National Academy of Sciences*. I chose to write "Coming to Terms with Fear," which delved into issues about the language of fear and how fear arises as a cognitive interpretation of nonconscious ingredients generated by survival circuits that the global organismic states engender. These ideas are discussed in the following sections.

COMING TO TERMS WITH FEAR AND ANXIETY

Although research on fear conditioning has been very successful, we are now at a crossroads. We could easily continue along this same path and produce more findings, perhaps even important findings, but I believe we should take another approach, one based on a sharper conceptualization of what is actually being studied.

The language of fear, anxiety, and other emotions comes by necessity from our *folk psychology:* commonsense intuitions derived from introspections about how the mind works that have been handed down through the ages.57 Scientists often start from such words and the intuitions behind them. But as Francis Bacon argued hundreds of years ago, scientists should be vigilant when using such common language terms and especially should guard against tacitly granting reality to things simply because we have words for them.58 Everyone knows what the words "leprechaun," "unicorn," and "vampire" mean, but few believe they refer to actual beings.

The use of the vernacular language of fear to describe a system that detects and responds to threats is an example of what Bacon had in mind. It reifies fear and makes it *a natural kind,* something that is wired into the brain by evolution.59 This belief justifies a search in the brain for a specific, innate locus of the phenomenon called fear. Findings about how the brain detects and responds to threats are used to conclude where fear lives in the brain because it is assumed that the same system that controls such responses gives rise to the feelings associated with them. (This is the essence of the Darwinian commonsense approach.60) We are told that this primitive emotion, fear, is inherited from animals and that every human in the world, no matter where he or she lives, and even if isolated from the rest of humanity, has the same or a similar fundamental (primal) experience when in danger and expresses a fear response to it in the same way.61 The home of this universal fear is frequently assumed to be the amygdala, which until fairly recently was an obscure region of the brain but has now widely come to be known as the brain's "fear center."

Problems created by the ambiguous use of the word "fear" in scientific research can be illustrated by considering a 2012 finding that led magazines such as *Nature, Science, Wired, Scientific American,* and *Discover* to run dramatic headlines such as these: "Humans Can Feel Terror Even if They Lack the Brain's Fear Center," "Scaring the Fearless," "Evoking Fear in the Fearless," "Researchers Scare Fearless Patient," and "What Scared the Fearless Woman?" The fuss concerned the surprising finding that a woman with bilateral amygdala damage could still experience "feelings of fear."62 But the only reason this would be considered surprising was if one believed that the amygdala is the primary wellspring of fearful feelings and that amygdala-controlled responses are reliable markers of these feelings. As I've said, and will explain in detail later, amygdala-controlled responses are *not* unequivocal signatures of fearful feelings. When we scientists use the term "fear" to refer to the neural mechanisms underlying both conscious feelings and nonconsciously elicited responses, we are inviting confusion.

The problem is not limited to fear. Jeffrey Gray's *behavioral inhibition theory* is a prominent animal model of human anxiety.63 According to Gray and Neil McNaughton, the behavioral inhibition system of the brain is activated when goals are in conflict—for example, the need for food versus the risk of being exposed to predators. This conflict causes one's brain to attribute more risk, more harm potential, to stimuli and situations than we otherwise would, thus leading to a central state of behavioral inhibition that promotes risk avoidance rather than food seeking. Gray and McNaughton equated this brain state with anxiety because rats

took more risk in conflict situations when treated with drugs, such as benzodiazepines, that relieve anxiety in people. But were they referring to a conscious feeling of anxiety that involves dread, foreboding, and worry? Or were they scientifically defining anxiety to mean a nonconscious brain state of behavioral inhibition that leads to motivational conflict and behavioral arrest? Gray and McNaughton sometimes claimed the latter (the central state version) but also often wrote in ways that could be interpreted the other way (in terms of conscious feelings). Certainly many followers of this approach, and there are legions, believe that the anxious feelings are direct products of the behavioral inhibition system.

A recent study showed that benzodiazepines relieved a so-called behavioral inhibition response in crayfish64 (as a Cajun, I am always momentarily surprised when this is not spelled as "crawfish"). After receiving electric shock in a certain location, the crayfish remained immobile for an extended period (a behavior that was viewed as risk assessment) and then avoided the shock area, whereas the drugged crayfish were less inhibited (more exploratory). The authors claim that their results may lead to a new view of the emotional status of invertebrates. The study was published in *Science*, whose website led with the headline "Anxious Crayfish Can Be Treated Like Humans." The *New York Times* announced, "Even Crayfish Get Anxious," while the BBC, slightly more tempered, noted, "Crayfish May Experience a Form of Anxiety."

The theory of behavioral inhibition could, in fact, easily account for motivational conflict, behavioral arrest, and risk assessment in animals (including crayfish, rats, and people) without requiring the conscious experience of anxiety. Unfortunately, just as the meaning of defensive motivation gets tied up with subjective feelings when the motivational state and its brain system are labeled with the term "fear," the meaning of behavioral inhibition becomes entangled with subjective states when it or its brain system is labeled with the term "anxiety." Defensive motivation and behavioral inhibition are not the same as the conscious experience of fear and anxiety. That is not to say that the defensive motivation and behavioral inhibition states are unrelated to fear and anxiety, as they do make important contributions, but more is required to feel afraid or anxious.

In June 2014, a psychology website's headline read: "Fear Center in Brain Larger Among Anxious Kids."65 The story that followed described a study that measured the level of anxiety in a large group of children based on a questionnaire answered by their parents.66 The brains of these children were then imaged and the findings related to the parents' assessments. The results showed that the larger the amygdala of the child, the higher the level of anxiety rated by the parents. Let's consider what this actually means. In this study the parents did what animal researchers often do: They based a conclusion about anxiety, an inner feeling, on observations of behavior—their child seemed nervous, edgy, or had trouble concentrating or sleeping. Thus, although the size of the amygdala might well correlate with certain behaviors, whether it was related to feelings of anxiety was not tested. The website's headline was inaccurate in three respects: (1) What was being measured was behavioral activity, not the feeling of anxiety; (2) the kids were not anxious in the clinical sense, in spite of some being described as "anxious" in the story; and (3) the amygdala is not the fear center (and certainly not the anxiety center) if by fear or anxiety we mean a conscious feeling.

Fear and anxiety are hardly the only emotions that are viewed in these inaccurate and confusing ways. As we saw above, a number of emotions, including anger, sadness, joy, and disgust, are often considered to be wired into brain circuits.67 The same problem arises in these cases—the conflation of innate systems that detect and respond in predictable ways to significant stimuli with systems that give rise to conscious feelings.

The science of the conscious mind is different from other kinds of science.68 Physicists, astronomers, and chemists don't need to take seriously commonsense ideas about nature because people's beliefs and attitudes about the stars, matter and energy, and chemical elements don't affect the subject under investigation.69 The fact that we commonly say (and some may actually believe) that "the sun rises in the east" does not have any scientific bearing on the fact that sunrise is an illusion. But psychologists do have to pay attention to folk

psychology because people's common beliefs about the mind influence their thoughts and actions in daily life and are thus an important part of what psychology is all about.70 Folk psychology is a window into the things that interest people and affect their lives.71

Typically, when a science matures, whatever vernacular terms it may have used are replaced with scientific ones.72 Some argue that neuroscience will eventually do this for descriptions of mental states.73 Terms such as "fear," "joy," and "sadness" will, in this view, be replaced with proper scientific language that does not have familiar, everyday connotations.

But the psychologist Garth Fletcher made a helpful distinction between using folk psychological ideas as explanations about how the mind works as opposed to using folk psychology as a way to identify things about the mind we want to understand and pursue scientifically.74 He agrees that commonsense explanations about how the mind works will be replaced as psychological science progresses, but he thinks that the other side of folk psychology will continue to have a legitimate role because people's subjective experiences, their beliefs, fears, desires, and so on, affect how they approach their lives.

I side with Fletcher on this. If we want to understand conscious feelings, there's no getting around the use of words like "fear," "anxiety," "joy," "jealousy," "pride," and so forth. We run into problems when we label nonconscious processes with the words about conscious feelings. The conscious state takes on characteristics of the nonconscious processes: We assign the *feeling* of fear the responsibility for the defense responses elicited by threats. At the same time, nonconscious processes take on properties of the conscious feeling: the process of detecting and responding to threats comes to be the function of fear. The result is that it becomes very difficult to disentangle the concepts. We need a solution to get us out of this terminological quagmire.

A PROPOSAL

When scientifically discussing fear and anxiety, we should let the words "fear" and "anxiety" have their everyday meaning—namely, as descriptions of conscious experiences that people have when threatened by present or anticipated events. The scientific meaning will obviously go deeper and be more complex than the lay meaning, but both will refer to the same fundamental concept. In addition, we should avoid using these words that refer to conscious feeling when discussing systems that nonconsciously detect threats and control defense responses to them.

Thus, rather than saying that fear stimuli activate a fear system to produce fear responses, we should state that *threat stimuli* elicit *defense responses* via activation of a *defensive system*.75 Because "threat" and "defense" are not terms derived specifically from human subjective experiences, using them would go a long way toward making it easier to distinguish brain mechanisms underlying the conscious feeling of being afraid or anxious from mechanisms that detect and respond to actual or perceived danger. Similarly, what we now call fear conditioning can simply be called what it is: threat conditioning. So, in place of "fear CSs" and "fear CRs," we can refer instead to "threat CSs" and "defensive CRs" (Table 2.2).

Some think we should stay the course—that the value of our work will be diminished if we separate the feeling of fear or anxiety from the mechanisms that detect and respond to threats. But separating the processes does nothing to diminish research on their individual contributions and instead paves the way to a richer understanding of how fear and anxiety emerge from neural circuits. For example, if, as I suggest, anxious feelings arise from mechanisms that go well beyond those that control the behavioral and physiological symptoms that also trouble anxious people, more effective therapies are more likely to emerge if we acknowledge the separate mechanisms involved than if we disregard the differences. Recall from Chapter 1 that modern understanding of panic disorder began when Donald Klein found a difference in the ability of a drug treatment to affect the conscious feeling of being terrified of dying (a cognitive

interpretation) without changing physiological symptoms (which are direct consequences of survival circuit activation).

DEEP SURVIVAL

Problems with the way fear has been conceived become clear when we consider the widespread capacity to detect and respond to danger in the animal kingdom. This ability is necessary to survive and is present in every animal, whether it's a worm, slug, crayfish, bug, fish, frog, snake, bird, rat, ape, or human. Should we argue that crayfish, worms, and cockroaches escape from threats because they are driven by feelings of fear or anxiety? Or should we simply state that they possess mechanisms that enable them to detect and react to danger? Many are happy to agree to the latter characterization with respect to invertebrates, and even for fish and frogs; fewer are willing to do so where mammals are concerned. But if conscious feelings of fear are not required for a human to respond to danger, why should we resist the idea that defensive responses in other mammals reflect nonconscious processes as opposed to conscious feelings?

What we should be concluding, it seems to me, is not that humans have inherited fear per se from our animal ancestors, but rather that, through a long line of evolutionary history, we have inherited from them the capacity to detect and respond to danger. Problems result when it is assumed that this capacity depends on a feeling of fear intervening between threat stimuli and defense responses in human or nonhuman animals. This assumption compels the search for things that cannot be readily measured in nonhuman animals and forces researchers to bend the rules of evidence in order to conclude that such states exist. If, however, we accept that this capacity to detect and respond to danger does not require consciousness, then we will not be driven to search for elusive processes. We can, then, just study the specific topics that interest us without having to endlessly debate whether animals experience what we experience.

It is not my intention here to deny conscious feelings in animals. My aim, rather, is to highlight problems that hinder measurement of feelings in animals scientifically, to suggest a path forward that allows us to study those aspects of brain function that we know from objective evidence are shared between humans and other animals, and to focus on studies of humans for those functions that can be verified only in our species.

The lines of descent underlying threat detection may go much deeper than already discussed—as deep, in fact, as single-cell organisms, which also have to determine what's harmful and beneficial in their world. Viewed in this light, the capacity to detect and respond to threats is a deep survival mechanism, one that is as crucial to the life of an individual bacterial cell as it is for the numerous cells in a complex organism, whether in a fly, rat, or human. In animals, detecting and responding to danger is not only something that each cell in the body does on its own, it is also a function of a defense system in the brain, which enables the organism as a whole to defend itself. The evolutionary function of this ancient capacity is not to generate emotions like fear or anxiety, but simply to help ensure that the organism's life continues beyond the present.

We have, in short, been looking at the brain from a very human-centered point of view—as if our conscious introspections can tell us how ancient survival mechanisms that operate nonconsciously are organized in the brain. As I've noted, the conscious mind is compelled to explain what the brain does, even when it does not know.76 We think we react to danger because we feel fear, and it is this belief that leads scientists to search for the fear in the brains of animals by looking for circuits that control defense responses. But instead of trying to locate fear in the brains of animals, we should be trying to understand how processes that are similar in animals and humans—namely, nonconscious processes that detect and respond to threats—contribute to feelings of fear we experience.

SURVIVAL CIRCUITS AND GLOBAL ORGANISMIC STATES

The innate view of emotion is said to apply to states that are wired into ancient subcortical circuits that have

been inherited from our animal ancestors.77 We certainly do have circuits that control innate responses that are commonly associated with emotions in people. But these are not emotion circuits; they are not feeling circuits; they are *survival circuits*.78

I recently introduced the expression “defensive survival circuit” as a way to discuss brain mechanisms that are often labeled as fear circuits.79 To me, this term is preferable over “fear circuit” or “fear system” because it doesn’t imply that defensive behaviors are propelled by conscious feelings of fear. Thus, the amygdala circuitry that has been a subject of my research does not make fearful feelings; it detects threats and orchestrates defensive responses to help keep the organism alive and well.

Defensive survival circuits are one of several classes of survival circuits that are common to most animals. Others include circuits for acquiring nutrients and energy sources, balancing fluids, thermoregulation, and reproduction.80 The circuits involved in these functions are conserved within and between mammalian species, and to some this is true across vertebrates. The nervous systems of invertebrates are organized differently—for example, although they don’t have an amygdala or any other brain areas that are present in vertebrates, they do have circuits that perform survival functions that are similar to, and likely precursors of, comparable functions in vertebrates.81 Because similar functions are also present even in single-cell organisms lacking nervous systems, these functions predate, evolutionarily speaking, neurons, synapses, and circuits82 and as such are primitive precursors of survival functions in more complex organisms with nervous systems.83 Survival circuits do not exist to make emotions (feelings). They instead manage interactions with the environment as part of the daily quest to survive.

Survival circuits are activated in situations in which well-being is potentially challenged or enhanced. The overall response of the brain and body that results is a *global organismic state*.84 For example, activation of a defensive survival circuit results in a *defensive motivational state*.85 Such states involve the whole organism (that is, body as well as brain) as part of the task of managing resources and maximizing chances of survival in situations where challenges or opportunities exist.86 Global organismic states in mammals and other vertebrates,87 like the survival circuits that initiate them, are elaborations of similar states in invertebrates.88

When a defensive survival circuit detects a threat, as I pointed out in Chapter 1, it not only triggers defensive reactions; it also activates brain areas that control the widespread release of chemical signals, including neuromodulators and hormones.89 As a result, the organism becomes highly aroused and vigilant—attuned to the sensory environment, focusing on the clear and present danger, but also being on the alert for other potential sources of harm. The threshold for the expression of additional defensive responses is lowered, whereas other motivated behaviors, such as eating, drinking, sex, or sleep, are suppressed. This global defensive motivational state reflects the wholesale mobilization of brain and body resources for the purpose of staying alive and helps ensure that the subsequent actions that are performed in an effort to cope with danger in more complex ways guided by past instrumental learning are suited to the external circumstances—escape or avoidance when in danger. In other motivational circumstances, global organismic states function similarly: for example, helping to guide the approach to food or drink when energy supplies or fluids are low, etc.

Figure 2.7: The Survival Circuit View of Fear and Defensive Motivation.

Because my traditional view of the fear system (Figure 2.6) was often misconstrued as implying that the amygdala is the seat of fear in the brain, I have revised my terminology. In the current model the term fear is no longer used to describe functions of the amygdala. I now describe the amygdala circuit that detects and responds to threats as a defensive survival circuit. One consequence of survival circuit activation is the

establishment of a defensive motivational state throughout the brain. This state is not the neural instantiation of a feeling of fear. The state (or neural components of it) provides neural ingredients that when cognitively interpreted give rise to a feeling of fear. This view differs from the commonsense approach in that fear does not cause defense responses. It differs from the central state view in that both defense responses and the central state are consequences of activation of the survival circuit. While the defensive motivational state does not cause innate defensive reactions and changes in body physiology that accompany them, it does contribute to motivation of instrumental behaviors that allow the organism to act, rather than simply react, in the face of danger.

The idea of global organismic states is closely related to that of central states (see earlier discussion). And the idea of defensive motivational states has also been around for some time.90 But earlier views treated defensive motivational states as the cause of defensive responses. My view, in contrast, is that the defensive motivational state is a global consequence of activating a defensive survival circuit (Figure 2.7). Defensive responses in the present conception are thus not caused by, but instead actually contribute to, defensive motivational states. But as just noted, once a global motivational state is present, it helps guide instrumental behaviors in the effort to survive and thrive.

Although defensive motivational states can occur in both simple and complex organisms, only animals that have the ability to be consciously aware of their own brain's activities can experience the state we commonly refer to as fear. I propose that defensive motivational states, or at least components of such states, are ingredients that, along with other factors such as perceptions and memories, contribute to conscious feelings. Thus, when a defensive survival circuit has been activated in your brain and its consequences linked to the present stimulus and to your memories regarding it and similar stimuli, all in relation to your awareness that the event is happening to YOU, a feeling of fear arises.

Ultimately, feelings like fear require that we somehow have the *concept* of fear, based on words and their extended meanings, in our minds.91 We learn such concepts because they are important for our well-being and account for significant experiences in our lives. We also learn to associate these concepts and relevant words with the consequences of defensive survival circuit activity. Every culture ends up having these concepts and corresponding words because every human brain has built-in defensive survival circuits that produce similar kinds of innate reactions and supporting changes in brain and body physiology. But the feeling of fear is not a direct product of a survival circuit. It is a cognitive interpretation, which is, in canonical instances, based on the consequences of survival circuit activation. And because survival circuits have an innate foundation within a species, they provide at least some universal signals that are the basis for cognitive interpretation, and thus help make fear feel like a familiar experience when in danger, and also make self-reports of fear across individuals have similar content as well. Animals obviously cannot label and interpret survival circuit activity in the ways made possible by human language. They may experience something, but it is incorrect, in my opinion, to assume that their experience is the same as, or even similar to, what a human often experiences when a defensive survival circuit is active in his or her brain.

In sum, fear is not something that is unleashed from an innate circuit. Instead, it is, as I and some others argue, a conscious state that emerges when certain kinds of nonconscious ingredients coalesce and are cognitively interpreted.92 If so, then the search for innate circuits that unleash fearful feelings is the wrong approach for understanding fearful feelings. Innate circuits are important to survival but are not feeling circuits.

THE MENTAL LIFE OF ANIMALS

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