Why Do People Have Painful Feelings? An Evolutionary Tale of Misery and Woe

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Introduction

There are many types of painful feelings that humans normally experience. There is the pain of heat, the pain of cold, the pain of flesh that has been crushed, the pain of flesh that has been cut, the pain of a chemical burn, the pain of electric shock, the pain of hunger, the pain of eating something distasteful, the pain of being sick with an infection, the pain of a noise that is too loud, the pain of fear, the pain of anger, the pain of disgust, the pain of jealousy, the pain of guilt, the pain of shame, the pain of anxiety, the pain of sadness and depression, and so on. It should be clear from this list that I am using the term "pain" in a rather broad sense to refer to any unpleasant, aversive feelings.

Why do we have painful feelings? Evolution has imbued our nervous system with the capacity to experience painful feelings to help us avoid problems or threats that—over evolutionary time—were associated with decreased reproductive success (fitness). These feelings are ancient—we share them with many other organisms, including fish and invertebrates. There are many different types of painful feelings because there are many different types of problems and threats that organisms have evolved to avoid.

In this chapter, I provide an overview of evolutionary accounts of painful feelings, including the pain that accompanies tissue injury and painful emotions or feelings that accompany social problems. I further demonstrate the utility of this perspective in the context of depression. I focus on this feeling state because it is usually considered pathological in the mental health fields. In other words, depression represents a feeling state where an evolutionary perspective provides novel insights. However, the principles are generalizable to other feelings of clinical interest, such as anxiety, which I also briefly discuss.

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The Evolution of Painful Feelings

In the field of evolutionary psychology, most researchers explicitly or implicitly propose that emotions have two related functions (Andrews & Thomson Jr, 2009; Buss, 2000; Nesse, 1990; Sznycer et al., 2016; Tooby & Cosmides, 1990; Tybur, Lieberman, Kurzban, & DeScioli, 2013). First, emotions have a motivational function in which the organism gives precedence to some problem or threat relative to other competing goals. Second, all emotions have a coordinating function. The body is composed of multiple systems (e.g., circulatory, musculoskeletal, pulmonary, integumentary, digestive, immune, and nervous systems), each of which perform crucial functions. To produce an adaptive response to environmental contingencies, these systems must be regulated in a coordinated fashion.

The motivational and coordination functions of emotions are really two sides of the same coin. To give priority to one problem or another in the environment requires that body systems be differentially regulated. For instance, fear will increase the priority given to avoiding a predator above other goals, such as foraging, mating, and immune defense, but this can only be accomplished by differentially regulating the digestive, musculoskeletal, and immune systems. The emotion of disgust motivates behavior involved in avoiding a potentially contaminated food source and upregulates immune defense, while downregulating the systems involved in foraging and mating (Tybur et al., 2013).

Researchers interested in whether fish and invertebrates have the capacity to feel pain have reached similar conclusions (Sneddon, 2015; Sneddon, Elwood, Adamo, & Leach, 2014). A normal pain response has two major features. First, painful feelings are necessarily aversive, and it is their aversiveness that gives them motivational power. "The key function appears to be that the aversive experience of pain creates a strong and lasting motivation that enables the animal to avoid getting into a similar situation in the future" (Sneddon et al., 2014, p. 202). Thus, the injured organism is motivated to engage in avoidant learning and avoidant behavior.

At this point, it is useful to distinguish between two uses of the term "avoidant" in psychology. Avoidant learning is typically viewed as an adaptive process by experimental psychologists who study how behavior is shaped by experience. In this research tradition, the emphasis is on how the organism learns to avoid some noxious situation or stimulus, which are external events. However, within clinical psychology, "avoidance" often refers to how individuals avoid aversive thoughts or feelings, which are internal states. In this context, avoidance is thought to be maladaptive because it inhibits the processes that allow negative moods to resolve, such as reappraisal and habituation (Clark, 1999; Foa & McLean, 2016; Hunt, 1998; Litz & Keane, 1989). In this chapter, I discuss both concepts. However, I will only use *avoidant behavior* and *avoidant learning* to refer to the adaptive avoidance of noxious external stimuli. To maintain conceptual clarity, I will use the term *analgesic behavior* to refer to distraction, thought suppression, rubbing of injured tissue, self-medication, or other behaviors that attempt to avoid or soothe aversive internal feelings. Of course, the aversive properties of painful feelings motivate

both avoidant behavior and analgesic behavior. However, the evolved function of painful feelings is to motivate avoidance of noxious external stimuli. Below, I show an example in which the administration of an analgesic can lead to maladaptive outcomes by impairing the organism's ability to avoid noxious external stimuli. Consequently, analgesic behavior is a property of a normally functioning pain mechanism (Sneddon, 2015; Sneddon et al., 2014), but it is not the evolved function of pain mechanisms. Analgesic behavior is more properly termed a *byproduct* or a *spandrel* (Andrews, Gangestad, & Matthews, 2002).

The second major feature of painful feelings is that they must involve a whole-body response, which is related to the idea that emotions have a coordination function. Put another way, the concept of a whole-body response implicitly recognizes that multiple systems in the body must be regulated in a coordinated fashion to produce adaptive motivational changes.

Pain in Fish and Invertebrates

Lynne Sneddon and her colleagues have thoroughly reviewed the evidence that fish and many invertebrates meet both the motivational and coordination criteria for a normal, evolved pain response to physical injury (Sneddon, 2015; Sneddon et al., 2014). In this section, I briefly review a few relevant findings to demonstrate the proposition that painful feelings motivate the organism to avoid noxious stimuli.

In an elegant experiment, Robyn Crook and her colleagues demonstrated how painful feelings help longfin inshore squid avoid predators (Crook, Dickson, Hanlon, & Walters, 2014). They randomly assigned the squid to either injury or no-injury conditions and crossed that with an anesthetic treatment or no anesthetic treatment in a 2x2 experimental design. In the injury conditions, they clipped the end of one of the squid's tentacles during surgery; some squid received a transient local anesthetic treatment during surgery whereas others did not. Then the squid were placed into a tank with a natural fish predator of the squid—black sea bass. Interestingly, the sea bass appeared to be able to detect the injuries, because they preferentially targeted the injured squid. Moreover, the injured squid were more likely to be caught by the fish. However, the squid that were at the greatest risk of capture were those that had been injured and received the anesthetic. The injured squid that had not received the anesthetic were less likely to be caught because they took evasive maneuvers sooner, taking greater efforts to maintain distance from the predator. In other words, the pain of the injury motivated the squid to take extra precautions that helped it to avoid predation by the sea bass. These extra precautions were not taken by the anesthetized squid.

Another set of experiments have shown that goldfish are capable of learning spatial and visual cues that are associated with electrical shock, that they use these cues to avoid the shock, and that a local anesthetic inhibits this learning (Dunlop, Millsopp, & Laming, 2006; Millsopp & Laming, 2008; Yoshida & Hirano, 2010). In one of these experiments, goldfish were trained to feed in a certain area of the tank

(Millsopp & Laming, 2008). Subsequently, the researchers introduced an electric shock when the fish came to the feeding area. The fish avoided the feeding area in a dose-dependent fashion—the more intense the shock, the more they avoided the feeding area. In a follow-up experiment, the shock in the feeding area was kept constant, but the researchers varied the amount of time that the fish were deprived of food. Again, they found dose-dependent effects—the longer the fish were deprived of food, the more time they spent in the feeding area and endured the shock.

These results effectively demonstrate the point of this chapter. The electric shock is aversive, and the fish are motivated to learn that the feeding area is associated with the shock. Consequently, they spend more time avoiding the feeding area when the shock is more intense. The motivational nature of aversive feelings is further highlighted by the follow-up experiment. In that experiment, the fish are put in a dilemma in which they are deprived of food, and they must endure a severe shock in order to feed. The fish integrate their need for food with the aversiveness of the shock, and they are motivated to avoid the more severe problem. When they have recently been fed, they avoid the shock and the feeding area, but when they have been deprived of food for several days, they endure the shock in order to feed.

The Painful Feeling Triggered by Inescapable Shock

The evolutionary view of painful feelings can provide novel insight into the depressive-like state that rats experience when they are exposed to repeated, inescapable shock (Maier & Seligman, 2016). In the typical inescapable shock experiment, animals (usually rats) are randomized to one of three conditions. In the first condition, the rats are simply restrained (R). In the second condition, the rats are exposed to shocks that they can learn to terminate by spinning a wheel or pressing a lever. This is often called the escapable shock (ES) condition, because the shock is under the rat's control. The third condition is called inescapable shock (IS). The IS condition involves tethering the rat to another rat in the ES condition such that they both receive shocks of the same intensity and duration at the same time. The difference between the IS and ES conditions is that the IS rat is unable to terminate the shock through its own efforts. Thus, the IS rat lacks the ability to control the shocks that the ES rat has.

After going through one of these three conditions, the rats are then given a task, which I will refer to as a *secondary task*, because it is secondary to the shock paradigm. In the original experiments, the secondary task involved learning to jump over a hurdle in a shuttle box in order to escape a shock (Maier & Seligman, 2016). While the rats in the R and ES conditions easily learned how to avoid the shocks by jumping over the hurdle, the IS rats had great difficulty learning this. Because the ES rats experienced just as many shocks as the IS rats, the slower learning by the IS rats must have been attributable to their inability to control their shocks. The learning deficits following inescapable shock (IS) have been demonstrated in a number of organisms, including dogs, cats, and fish (Abramson, Seligman, & Teasdale, 1978; Maier & Seligman, 2016; Seligman, 1975), but the paradigm has been most widely studied in rats.

The learning deficits following IS also generalize to a number of secondary tasks, a fact which was important in the development of the *learned helplessness* hypothesis (Maier & Seligman, 2016). The essence of the learned helplessness hypothesis is that exposure to IS leads the organism to believe that nothing it does matters (Maier & Seligman, 2016). The learned helplessness hypothesis requires that organisms have the capacity for developing the expectation that outcomes are uncontrollable, a proposition that—when applied to non-human animals—was quite radical in the 1960s when the hypothesis was first proposed (Maier & Seligman, 2016). Additionally, the learned helplessness hypothesis proposes that IS produces a motivational deficit. Specifically, that the organism loses the motivation to try to control its environment. Finally, the learned helplessness hypothesis proposes that IS triggers depressed affect. Animals exposed to IS exhibit a number of behavioral changes that correspond to the symptoms of depression in humans, and IS has become an important non-human animal model of depression (Maier & Seligman, 2016). Put simply, the learned helplessness hypothesis proposes that the IS rat experiences aversive feelings (i.e., depression), and it lacks motivation to avoid noxious stimuli.

The evolutionary account introduced above suggests that these two elements of the learned helplessness hypothesis cannot both be correct. Specifically, if the IS rat has truly lost motivation for avoiding noxious stimuli, then it should not feel any pain. The function of painful feelings is to motivate avoidance of noxious stimuli. Thus, if the IS rat does have painful feelings, it will be motivated to avoid noxious stimuli. In other words, the evolutionary account suggests that the learned helplessness hypothesis is wrong in one of two ways: (1) the IS rat does experience painful feelings, but it has not lost motivation to control its environment; or (2) the IS rat has lost motivation to control its environment, but it does not experience painful feelings.

In the rat, IS triggers behaviors that closely map onto many of the symptoms of depression in humans (Maier & Seligman, 2016). In humans, depression is aversive, but is it possible to tell whether the IS rat also experiences this state as aversive? IS rats exhibit an increased preference for analgesic substances, such as alcohol and morphine (Volpicelli, Ulm, & Hopson, 1990; Will, Watkins, & Maier, 1998). Moreover, the preference persists after the shocks have stopped, and it does not develop in ES or R rats (Volpicelli et al., 1990; Will et al., 1998). The preference for analgesia is an important attribute of organisms having normal pain responses (Sneddon et al., 2014), so the analgesic behavior of the IS rat strongly suggests that the depressive-like state is aversive.

Precisely because this state is aversive, it should have motivational effects. The fact that IS rats feel enough distress to seek out analgesia suggests that their pain does have motivational properties. What is their distress motivating them to avoid? Following IS, the rat shows an enhanced ability to learn which cues are associated with noxious stimuli, otherwise known as fear conditioning (Shors, 2004; Shors, Weiss, & Thompson, 1992). In other words, IS rats are motivated to identify cues associated with threats, which suggests that they may not have given up avoiding threats such as shocks after all.

We suggest that IS rats may simply have switched strategies for trying to avoid the electric shocks. In the literature on human cognition, it is generally recognized that there are two major information processing styles (Evans & Stanovich, 2013). Type 1 processing tends to be fast, associative, and automatic, while Type 2 processing is slower, attentionally demanding, rule-based, analytical, and controlled. Perhaps the defining characteristic of Type 2 processing is the use of working memory (Evans & Stanovich, 2013), which is a type of memory in which information is kept in an active state because it is used in ongoing processing (Baddeley, 2007). Moreover, there is some evidence that rats are capable of both processing styles (Beckers, Miller, De Houwer, & Urushihara, 2006; De Houwer, Hughes, & Barnes-Holmes, 2016). In the non-human animal literature, the rapid, simple processing style most like Type 1 is termed *associative*, while the slow, complex processing style most like the analytical Type 2 style is sometimes termed *propositional* (Boddez, De Houwer, & Beckers, 2017).

The distinction between Type 1 and Type 2 processing styles can help explain some otherwise puzzling findings in the literature on IS. For instance, the shuttle box experiment is probably best solved with a quick, simple Type 1 processing style, since it only requires the animal to figure out that it needs to jump over a hurdle once to avoid the shock. Rats previously exposed to IS often solve the shuttle box task equally well as the ES and CR rats (Maier, Albin, & Testa, 1973; Maier & Testa, 1975). To elicit performance deficits in the shuttle box test by IS rats, the researchers must make the task more complicated by requiring the rat to cross the hurdle at least twice to stop the shock. However, the more complex the behavioral response required of the rat to stop the shock, the more likely it is that other extraneous events will occur (sounds, visual cues). Indeed, the IS rat is more likely to perceive multiple environmental cues that could potentially be associated with the termination of the shock (Minor, Jackson, & Maier, 1984; Shors, 2004). Thus, another potential explanation for the performance deficits on complicated versions of the shuttle box task is that IS rats—by virtue of increased Type 2 processing—consider and test more hypotheses about the causes of the termination of the shock.

Moreover, many of the learning deficits associated with IS—such as the slower learning in the shuttle box task—are due to the fact that IS rats are less physically active. When the second task does not require much physical activity to solve, IS rats tend to learn *more* rapidly than rats in the ES or R conditions (Glazer & Weiss, 1976). Because Type 2 processing is more effortful and attentionally demanding than Type 1, it may require organisms to be less physically active so that fewer attentional resources are devoted to navigating the environment (Andrews & Thomson Jr, 2009).

The Type 2 processing that rats are capable of has been demonstrated in a fear conditioning paradigm in which the rats are trained to associate a foot shock with auditory or light cues (Beckers et al., 2006). In this paradigm, the rats have been shown to engage in propositional reasoning about the causal relations between the cues and the shocks. In short, rats are capable of slow, analytical Type 2 reasoning about causal relationships if sufficiently motivated by a painful stimulus.

With this as background, I review an important set of experiments conducted by Thomas Minor in Steve Maier's lab (Minor et al., 1984). They involved the typical IS protocol discussed above followed by performance on a Y-maze task. In the Y-maze, rats start off at the base of the Y, and they receive foot shocks through the floor of the maze. The shock usually motivates the rats to move, but the shock continues as they move and come to the junction in the maze. If the rat goes down the correct arm of the maze (left or right) the shock terminates, and the shock continues if the rat goes down the incorrect arm. Previous research in Maier's lab had shown that IS rats, but not ES or R rats, were slower to learn the correct arm to terminate the shock (Jackson, Alexander, & Maier, 1980). However, other researchers who had employed a similar paradigm had failed to find a learning deficit in IS rats (Irwin, Suissa, & Anisman, 1980).

Minor et al. (1984) wanted to investigate reasons for the discrepancies, and they identified two possible reasons. First, Jackson et al. (1980) had used a variable delay in terminating the shock after the rat had entered the correct arm, while Irwin et al. (1980) had used a fixed delay. Minor et al. (1984) reasoned that the variable delay may have made it more difficult for the rats to determine that the cause of the shock's termination was their decision to go down one of the Y-maze's arms.

Second, Jackson et al. (1980) had placed an experimenter in the room when the rats were doing the Y-maze, whereas Irwin et al. (1980) had used an automated procedure that did not involve the presence of a researcher. Minor et al. (1984) considered the experimenter's presence to be an irrelevant task cue, and they suggested that perhaps the IS rats were more easily distracted by irrelevant cues. However, as discussed above, we now know that IS potentiates fear conditioning (Shors et al., 1992), and we also know that fear conditioning can promote Type 2 causal analysis in rats (Beckers et al., 2006; De Houwer et al., 2016). So, another possibility is that the IS rats may have entertained more hypotheses about the causes of the termination of the shock. After all, the rats were not privy to the design details of the experiment—they did not know that the experimenter's presence was an irrelevant cue. For IS rats—thinking more carefully about the causes of shock termination through Type 2 reasoning—the presence of the experimenter may have made the problem more complex because there were multiple causal hypotheses to consider for the termination of the shock (e.g., the rat's decision, the experimenter's behavior).

Minor et al. (1984) conducted several experiments to test these hypotheses, and they found that both the variable delay and the presence of the experimenter were required to produce the slower learning in the IS rats. It was known that IS is more likely to produce learning deficits on complex tasks (Maier & Testa, 1975), so the researchers suggested that both the variable delay and the presence of the experimenter may have contributed to the complexity of the Y-maze for the IS rats. Put another way, Type 1 processing is probably sufficient to solve the Y-maze—all the rat needs to do is figure out which arm to go down to stop the shocks. However, when a variable delay is employed and an experimenter is present, an IS rat that is primed to go through slow, careful, analytical Type 2 processing may be more likely to consider multiple causal hypotheses.

To investigate attention to external cues, Minor et al. (1984) conducted a follow-up experiment in which they first put the rats through the typical conditions of an IS experiment, then they tested them on a Y-maze where a light bulb had been added. The light bulb was either placed in the correct arm for avoiding the shock (C), the incorrect arm (IC), the same arm that the rat started in (S), or no light at all (NL). The rats were tested in five blocks, with 20 trials in each block.

In all light bulb conditions, the ES and R rats showed a pattern of declining errors across the five blocks, which means that over time they figured out which arm to go down to terminate the shocks. The IS rats also showed a declining pattern of errors for the IC, S, and NL light bulb conditions. However, the pattern for the C condition (when the bulb is placed over the correct arm to terminate the shock) showed a unique, inverted u-shaped pattern. In other words, in the first block, the rate of errors by the IS rats was low, which contrasted with the ES and R rats who had a high rate of errors. This suggested that the IS rats had learned more quickly than the ES and R rats in the first block. But the error rate increased (peaking at block 3) before declining again. This was a strange pattern, and the researchers suggested, "Perhaps yoked [inescapably shocked] subjects were more prone to test hypotheses concerning the relation of the light to correct choice responses" (Minor et al., 1984, p. 553).

Explaining why Minor et al. (1984) suggested that the IS rats "were more prone to test hypotheses" is easier if I use a bit of anthropomorphic language. The IS rats were paying attention to the light cue, and this resulted in a low error rate in the first block of the C condition. The pattern of results can be explained if the rats suspected that the light may have been helping them terminate the shock, but they were also entertaining other hypotheses. To prove whether the light was the key factor, they stopped using the light cue to guide their behavior in the second and third blocks. The increased rate of errors suggested that the light cue was in fact related to the termination of the shock, and so the rats reverted back to the use of the light cue in the fourth and fifth blocks, with a corresponding low rate of errors. In other words, when the rats used the light to guide their behavior, they had a low rate of errors; when they did not use the light as a guide, they had a high rate of errors; so the rats concluded that the light was causally related to the termination of the shock.

In summary, the evolutionary account of painful feelings suggests that the learned helplessness hypothesis for the effects of inescapable shock is inaccurate—particularly, the assumption that the IS rat lacks the motivation to avoid noxious stimuli. The depressive-like state triggered by IS is inherently motivational precisely because it is aversive. Only if the depressive-like state were not aversive would it lack motivational properties. Consistent with an altered motivational state, IS rats are more prone to analgesic behavior. What, then, is the function of this aversive, depressive-like state? What are the IS rats motivated to avoid? Most likely, they are motivated to avoid the shocks that triggered the depressive-like state. IS rats have not lost the motivation to learn cues that are associated with shocks (Shors et al., 1992), nor have they lost the motivation to avoid shocks (Glazer & Weiss, 1976). The IS rats appear to be motivated to adopt the slower, more methodical Type 2 processing style in which they consider a broader range of hypotheses about causal relations, which means they are slower to solve some tasks.

Depression in Humans

In psychiatry and clinical psychology, depression is considered an affective disorder characterized by a lack of motivation. The most important conceptual basis for this belief is the symptom of anhedonia (the loss of interest in activities that are normally pleasurable, such as sex, eating, humor, social companionship). According to the conventional narrative, the depressed person has given up the pursuit of adaptive goals—including sex and eating.

My analysis of IS, widely considered a model of human depression, suggests instead that the rat exposed to IS is simply in an altered motivational state in which escaping the shock is prioritized over other goals, including mating and foraging. If IS is in fact a good model of human depression, then the evidence should lead to similar conclusions about the motivational and cognitive effects of depression in humans. Specifically, human depression should motivate people to avoid some problem or threat in their lives, and we might expect it to be associated with Type 2 processing.

Complicating the issue is the fact that the term "depression" is a catchall phrase that encompasses multiple phenotypes (Insel & Charney, 2003). These phenotypes share in common the symptoms of sadness and anhedonia (loss of interest in activities that are normally pleasurable), but they differ in other symptoms, causes, and neurological mechanisms (Andrews, Bharwani, Lee, Fox, & Thomson Jr, 2015; Andrews & Durisko, 2017).

Elsewhere (Andrews et al., 2015; Andrews & Durisko, 2017), my colleagues and I have reviewed evidence that the symptoms produced by IS closely correspond to the symptoms associated with the melancholic type of depression, as it is usually described (Taylor & Fink, 2008). In addition to anhedonia, both IS and melancholia are characterized by decreased time spent sleeping, but a greater proportion spent in rapid eye movement (REM) sleep; there is a decrease in eating, but a relative preference for carbohydrate over protein; and both conditions are associated with chronic activation of the hypothalamic-pituitary-adrenal (HPA) axis.

Melancholia is also associated with *rumination*, which refers to distraction-resistant thoughts about the circumstances surrounding the episode (Gold, 2005; Taylor & Fink, 2008). There is some evidence that rumination involves Type 2 processing, which is an interesting correspondence with the literature on IS in rats. For instance, evidence of Type 2 processing has been shown in mood induction experiments, studies of subclinical depression, and even in some studies of clinical depression (Ambady & Gray, 2002;Andrews & Durisko, 2017 ; Andrews & Thomson Jr, 2009).

The symptoms of melancholia appear to be organized in a way that supports rumination (Andrews & Durisko, 2017; Andrews & Thomson Jr, 2009). For instance, anhedonia reduces interest in normally pleasurable activities that could disrupt the slow, methodical processing characteristic of Type 2 processing. Similarly, a loss of sleep is associated with rumination (Guastella & Moulds, 2007), which allows the individual to spend more awake time processing information.

Moreover, when the individual does sleep, the increased time spent in REM sleep should help consolidate information learned during waking hours (Rasch & Born, 2013). Finally, many studies have shown that blood cortisol levels are positively associated with rumination (Zoccola & Dickerson, 2012), and it has been argued that cortisol helps release glucose into the bloodstream that the brain uses as fuel for rumination (Gold, 2015). In short, melancholia appears to satisfy the requirement of a coordinated, whole-body response.

But what, precisely, is melancholia a response to? If we look to the IS literature as a guide, it is clear that it is the loss of control over the shock—not the shock itself—that triggers depressive-like symptoms in the rat (Maier & Seligman, 2016). The literature on human depression suggests that loss of control over the environment is a contributing factor to depression (Abramson et al., 1978). However, people do not get depressed over problems or stressors they believe were impossible to avoid (Abramson & Sackheim, 1977; Garber & Hollon, 1980). Paralleling my analysis of IS, to become depressed, people must experience a problem or stressor that they believe they could have avoided. Painful feelings motivate avoidant learning and behavior. If there is nothing that could have been done to avoid a problem, there is no adaptive value in producing aversive feelings.

Is there any evidence that melancholia motivates avoidant learning and behavior? Melancholia can be triggered by a variety of stressors (Taylor & Fink, 2008). In principle, this could cause variability in the precise way in which avoidant learning and behavior is instantiated. For this reason, it is important to control for the type of stressor that triggers the episode. In this context, a strong case can be made that physicians who get depressed after making a serious medical error exhibit avoidant learning and behavior.

Physicians commonly agonize over medical errors they have made, and the reasons are understandable. Medical errors cause harm to their patients, and they can put the physician in a position where they are concerned for their reputations and careers. Because errors are often avoidable, it is not surprising that physicians commonly experience depressive symptoms after making a serious medical error. In a study of 114 residents in internal medicine, 81% reported feelings of remorse after making a serious medical mistake, 79% felt angry at themselves, 72% felt guilty, and 60% felt inadequate (Wu, Folkman, McPhee, & Lo, 1991). Two large studies have found that making a medical error put residents at substantial risk of clinical levels of depression (Sen et al., 2010; West et al., 2006). In one of these studies, 63% of residents who reported having made a major medical error in the last 3 months screened positive for clinical depression, compared to 33% who did not report any errors (West et al., 2006).

After making an error, physicians commonly make *constructive changes* to their practice that probably reduce their chances of making a similar error again in the future. In a classic study, 82% of internal medicine residents reported paying greater attention to detail in response to their most significant medical mistake, 72% reported that they were more likely to personally confirm patient data, 62% reported seeking more advice, 54% reported reading more medical literature, and 49% reported trusting the judgment of others less (Wu et al., 1991).

The emotional distress that physicians feel is crucial to motivating constructive changes to their clinical practice. In one study, if internal medicine residents felt responsible for the error, they felt more distress and made more constructive changes to their practice (Wu et al., 1991). In another study involving nurses who made a medication error, the nurses reported making more constructive changes to their practice if they felt more anger at themselves, more guilt, or more inadequate (Meurier, Vincent, & Parmar, 1997).

A very similar response unfolds when psychiatrists have a patient who commits suicide. Patient suicide is common enough that it has been called "the most significant event in the training of a psychiatrist" (Sacks, Kibel, Cohen, Keats, & Turnquist, 1987, p. 218). Indeed, it is common for psychiatrists who have had a patient commit suicide to report feelings of depression, grief, rumination, anger, shame, guilt, and self-blame (Hendin, Haas, Maltsberger, Szanto, & Rabinowicz, 2004; Sacks et al., 1987). Again, the symptoms often reach clinical levels. In a study of psychiatric residents reporting on their emotional response to their first patient who committed suicide, 24% had clinical levels of emotional disturbance, with symptoms of depression being prominent (e.g., anxiety, loss of self-worth, intrusive thoughts) (Ruskin, Sakinofsky, Bagby, Dickens, & Sousa, 2004).

How do distressed psychiatric residents behave after a patient's suicide? (Sacks et al., 1987, pp. 218–219).

> Clinically, the resident becomes preoccupied with insuring that another suicide does not occur. On inpatient units, the possibility of another suicide seems imminent. Passes are cancelled, and more patients are placed on suicide observations. Worry is understandable since suicides have been [known] to cluster so that increased concern is appropriate, but the distinction between appropriate and excessive caution is [for] the moment blurred. In outpatient settings, patients who would otherwise be sent home are admitted. It is as if every clinical interaction with a patient is burdened by the fearful question of whether it indicates a need for suicide precautions.

Psychiatrists who have had a patient commit suicide report a number of other changes to their practice: being more vigilant for signs of suicidal thinking, more careful documentation, more detailed communication about patient records, greater use of formal suicide assessment tools to try to improve the ability to evaluate suicide risk, seeking out more medical knowledge about suicide risk, increasing personal availability to patients so they have someone to turn to when they are distressed, consulting with colleagues or team members about patients' suicide risk, and increasing efforts to understand patients' situations and feelings (Alexander, Klein, Gray, Dewar, & Eagles, 2000; Rothes, Scheerder, Audenhove, & Henriques, 2013).

These are excellent examples of avoidant behavior. Current suicide assessment tools are unable to predict suicidal behavior with any real accuracy (Large et al., 2016; Runeson et al., 2017). The distressed resident who has had a patient commit suicide realizes this brutal fact with stark clarity and errs on the side of caution, implementing changes that reduce the risk of any other patients committing suicide. From a clinical standpoint, the resident does not always make decisions that are best for the patient. But their behavior is clearly interpretable as an attempt to predict and prevent further patient suicides.

Again, the emotional distress that psychiatrists feel motivates such changes. In one study, psychiatrists who were highly distressed by a patient's suicide were more likely to reconsider how they had treated the patient and were more likely to be cautious in their approach with other patients in the future (Wurst et al., 2011). In another study, mental health workers who reported making the greatest changes to their practice (learning more about suicide, greater hospitalization of suicidal patients, greater consultation with colleagues, more attention to legal matters) also reported the highest rates of needing and seeking therapeutic support to cope with the suicide (Gulfi, Castelli Dransart, Heeb, & Gutjahr, 2010).

There are further questions that are worth investigating. For instance, what are the precise thoughts that depressed physicians have after having made a serious medical error? Do these thoughts provide evidence that the depressed physician is trying to understand how to make constructive changes to their practice?

Nevertheless, the fact that distressed physicians are more likely to make constructive changes to their practice after making a serious medical error demonstrates the utility of an evolutionary perspective. The conventional narrative recognizes the aversive nature of depression, but it inexplicably proposes that depression is a state in which motivation is lacking. This perspective is inaccurate—at least when it comes to depressed physicians who have made a serious medical error.

Anxiety-Related Conditions

The evolutionary perspective on painful feelings has also been generalized to anxiety and anxiety-related conditions, such as phobias, which I briefly review (Bateson, Brilot, & Nettle, 2011; Nesse, 2005; Ohman & Mineka, 2001; Russell, Maslej, & Andrews, 2015).

The primary symptom of anxiety is worry—distressing thoughts about an impending or anticipated problem. Put another way, anxiety is a future-oriented emotion where there is concern about a potential threat. From a clinical perspective, one of the most puzzling aspects of anxiety-related conditions is that they can be triggered by a threat that is highly unlikely to occur (e.g., "If I go to the party, everyone will laugh at me") or by something in the environment that does not seem to pose any real threat (e.g., a phobia of garden hoses). This makes anxiety-related conditions seem excessive or irrational.

However, from the evolutionary perspective I have reviewed, anxiety ostensibly evolved to avoid the threats that trigger the anxious feeling. The essence of a threat is that it is a feared problem or event that may be anticipated or impending, but it has not yet occurred. To prevent a threat from occurring is an interesting problem, because if one waits until the event occurs, it is too late to take corrective action. Thus, people must take corrective action on the basis of cues that are inherently imperfect indicators of the threat (Bateson et al., 2011; Nesse, 2005). For instance, when one suddenly comes across a long, slender object lying in the grass, it may be better to quickly jump aside before closely examining it to determine whether it is a

garden hose or a deadly snake. In situations of such uncertainty, there are two possible errors that can be made. A false positive error involves taking corrective action when the threat is not actually present, and a false negative error involves not taking corrective action when the threat really is present. But the two errors have different consequences. A false positive (jumping aside when the object is a garden hose) involves an expenditure of effort that turns out to have been unnecessary, while a false negative (not jumping aside when the object is a deadly snake) can be lethal. In such a situation, it may be better to adopt a better-safe-than-sorry approach.

The evolutionary perspective on anxiety is that it motivates a strategy of erring on the side of caution. Imagine that anxiety is controlled by a dial that goes from 0 to 10. As one turns up the dial from 0, anxiety goes from a wispy worry to an intense foreboding or trepidation. As one turns the dial up, the types of errors that one makes also change. When one has no anxiety at all (the dial is set at "0"), one will never make a false positive error (one will never confuse a garden hose for a deadly snake), but one will always make false negative errors (one will never take corrective action when the snake is actually present). And when the dial is turned all the way up to "10," one will never make a false negative error (one will always identify and avoid the snake), but one will be highly susceptible to false positive errors (one will take corrective action even when the snake is not present).

If you view the symptoms of anxiety as being controlled in this way, it is obvious that there is no point on the dial where one can definitively say that the symptoms are so severe that they must be disordered. Does anxiety become a disorder when the severity dial reaches 6? When it reaches 8 or 10? Most evolved emotional responses are regulated in a dial-like way because the brain must carefully match the emotional response to the demands of the situation. Put another way, the reason the anxiety dial goes up to 10 is because there are some situations where one simply cannot afford to miss a threat. Other threats, if missed, may only cause a slight inconvenience, so the whole range of the dial is required.

Conceptualizing anxiety as being regulated by a dial helps us to see that people will differ in where they set their dial, and these differences will often be adaptive. For instance, people who are exposed to more threats (e.g., they live in dangerous environments) or are more vulnerable to threats (e.g., they are injured or weaker) will probably have their anxiety dial set to a higher value (Bateson et al., 2011). A person who seems to have an excessive or irrational degree of worry may have good reason to err more on the side of caution.

Conclusion

The evolutionary theory reviewed in this chapter proposes that painful feelings evolved to motivate organisms to avoid problems or stressors in their environments. The reason why there are so many different kinds of painful feelings is because they evolved to motivate the organism to avoid different types of problems. Moreover, to effectively avoid a problem, each aversive feeling recruits and coordinates a different

whole-body response. I have focused on how this perspective can be employed to yield interesting and novel insights into feelings of depression and anxiety. But it has also been usefully applied to the study of disgust, jealousy, and shame (Buss, 2000; Sznycer et al., 2016; Tybur et al., 2013).

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