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Lactation *See:* Oxytocin; Prolactin and Stress.

Learned Helplessness

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Glossary

Explanatory style	A person's characteristic ways of explaining the causes of the events that happen in his or her life.
Shuttle box	Experimental apparatus for animals with two compartments separated by a barrier that can be adjusted by the experimenter.
Uncontrollable reinforcer	A reinforcer whose probability is the same whether an emitted response is present or absent. In this situation, the responding organism cannot control the reinforcer.

Original Observations and Theory

Learned helplessness refers to the motivational, cognitive, and emotional deficits that may follow from an organism's exposure to uncontrollable stressors. The theory arose from the observation that, after experiencing inescapable shock over which they had no control, dogs in the laboratory displayed a variety of behavioral deficits. In the original experiments on the learned helplessness phenomenon, the following design was typically used. The dogs were first given unpredictable and inescapable electric shocks in a Pavlovian hammock. Then, 24 h later, the dogs were placed in an experimental shuttle box. In this shuttle box, there were two compartments, and the dog was given 10 trials of escape/avoidance training. Shock was given in both compartments, but the termination of the shock took place when the dog jumped from one compartment to the other.

Approximately two-thirds of the dogs placed in the shuttle box after experiencing inescapable shock did not learn the escape/avoidance behavior; that is, they never learned that they could end the shock simply by jumping to the other compartment. Instead, they struggled at first and then stopped attempting to take action that might terminate the shock. The vast majority of dogs who had not experienced inescapable shock prior to entering the shuttle box had no trouble learning the escape/avoidance procedure to terminate the shock. The deficit in many of the previously shocked dogs was psychological rather than physical because these same dogs were able to run out of the shuttle box when the exit was opened.

This learned helplessness effect observed in the previously shocked dogs (i.e., they passively accepted later shock and did not learn to avoid it) was caused by the uncontrollability of the shocks experienced in the Pavlovian hammock. This was demonstrated by Seligman and Maier in 1967 using a yoked experimental design with three groups of dogs. The first group could press a panel in the hammock to terminate the shock. The second group received the same duration of shock as the first group, but could not control the onset or termination of its shock in the hammock. The third group received no shock prior to being placed in the shuttle box. The yoked group,

which had received uncontrollable shock in the hammock, showed the strongest deficits in learning in the shuttle box. The dogs that had received the same amount of shock but could press to control the termination of the shock did not show these deficits.

The deficits observed in helpless dogs fall into three categories. First, motivational deficits exist because the dogs stop initiating voluntary behaviors, such as jumping from one compartment to the other. Cognitive deficits also appear in that the dogs do not learn that their responses have been effective even when they have indeed caused the desired effect. Finally, the dogs show transient helplessness effects that dissipate over time, suggesting that helplessness may be a passing emotional response. Interestingly, exposure to controllable shock in the shuttle box prior to experiencing uncontrollable shocks appears to immunize dogs against later helplessness deficits and forcibly exposing dogs to the appropriate response contingency (i.e., that jumping to the other compartment terminates the shock) can eliminate these deficits.

According to the researchers, the dogs who received uncontrollable, inescapable shock learned that outcomes were independent of their responses. When organisms experience uncontrollable outcomes, they may notice this contingency and learn that the outcomes are independent. They will then expect outcomes to be independent of their responses in the future. This expectation involves a cognitive representation of the contingency. The purpose of voluntary action is to cause certain outcomes, so a belief in response–outcome independence will reduce the organism's motivation to engage in voluntary responses. Because forming this expectation of independence is an act of learning, this cognitive representation will interfere proactively with future attempts to learn about response–outcome dependence. Finally, the fear that follows a traumatic event may be replaced with negative emotions when the organism realizes its lack of control in the situation. Thus, learned helplessness theory seeks to explain the three primary deficits observed through a cognitive pathway. In their 1976 review paper, Maier and Seligman reviewed alternative hypotheses for the observed deficits and argued that the helplessness account fits most parsimoniously with the available data.

Learned helplessness is not specific only to dogs in the shuttle box. Deficits have also been documented in cats, rats, and humans. These extensions of learned helplessness to animals other than dogs have been empirically and theoretically useful. One study found that helpless rats were less likely to reject cancerous tumors than nonhelpless rats, suggesting an important connection between helplessness and immune function. In humans, several studies used a yoked paradigm in which participants (college students) in the first group could turn off a loud noise by pressing a button. Students in the second group heard the same noise for the same duration of time as those in the first group, but the sound was not contingent on their pressing a button. A final control group did not hear any noise. In one study conducted by Hiroto, students then put their hand in a shuttle box; they were shocked but could escape by moving their hand to the other side. Just as in the dogs, the students in the uncontrollable group showed helplessness deficits; they did not learn the avoidance strategy and just took the shock. In another study by Miller and Seligman, students who heard the uncontrollable inescapable noise had more trouble solving difficult anagrams than did those who heard escapable noise and those who heard no noise at all.

Learned Helplessness and Psychopathology in Humans

Although these experiments demonstrate that learned helplessness deficits can be produced in humans, a large amount of research has investigated whether the learned helplessness model can be thought of as an analog for various psychological disorders in humans. The most work has been done on depression and posttraumatic stress disorder (PTSD), so we consider here how learned helplessness might be useful for the understanding and treatment of these two types of psychopathology. Although our focus in the remainder of this article is on links between learned helplessness and mental health, there is an additional, smaller body of work linking learned helplessness and physical health in humans.

Depression

In his 1975 book *Helplessness*, Seligman asserted that the learned helplessness phenomenon appeared to be very much like human depression. Specifically, the motivational, cognitive, and emotional deficits that appeared in helpless dogs seemed to mimic the symptoms of reactive depression, in which a human gets depressed after a major life stressor. Certainly the apparent emotional withdrawal and passivity shown by helpless dogs and college students paralleled depression; in addition, decreased motivation and increased cognitive difficulty, also parts of the helplessness experience, are major symptoms of depression. Based on these and other similarities, Seligman hypothesized that both learned helplessness and reactive depressions result from the expectancy that responses and outcomes are independent. Thus, gaining a sense of control would serve as an important part of treatment for depression.

Although the expectancy of future response–outcome independence served as a helpful stimulant for research on the cognitive predictors of depression in humans, there were several flaws with this original learned helplessness conceptualization of depression. First, just as not every dog became helpless after uncontrollable shock, not every human becomes depressed after experiencing stressful events with uncontrollable outcomes. Second, the theory could not distinguish between a depressed person feeling that he or she personally could not effect outcomes and the feeling that the response–outcome independence was true universally.

Abramson, Seligman, and Teasdale therefore offered a reformulated learned helplessness model of depression in their 1978 paper. This cognitively oriented reformulation centered on the notion that, when a life stressor takes place, people do not simply notice the contingencies involved. Rather, they ask themselves why the event happened. People tend to have usual ways of answering this question, and this is called their explanatory style. Causes for events generated by people tend to fall along three separate dimensions: (1) people may feel the cause is due to themselves or to other people or luck, (2) they will either attribute the cause to factors that are stable in time or temporary, and (3) the cause may affect many life domains or just the ones involved most specifically with the stressor. The theory follows a diathesis-stress model of depression onset – it claims that people who have a pessimistic explanatory style, in which negative events are explained with internal, stable, and global causes, will be especially vulnerable to depression when faced with uncontrollable life stressors.

There has been a large amount of empirical support for this model, using both questionnaires (primarily the Attributional Style Questionnaire) and content analysis of verbatim data and a wide range of experimental methodologies. A usual study followed a population prospectively over time to determine whether pessimists were more likely to get depressed after experiencing negative life events. In one series of studies, pessimistic college students experienced the most enduring depressive symptoms after doing poorly on a midterm. A meta-analysis supported the conclusion that a pessimistic explanatory style relates to depression, especially with regard to explanations for negative events. However, the vast majority of studies used either college students or adult psychiatric patients; there is some indication that the relationship between pessimism and depression may be slightly different in older adults. Nonetheless, prevention programs based on teaching pessimistic children and college students to dispute their automatic pessimistic thoughts and to become more realistically optimistic have produced lower rates of significant depressive symptoms in participants at high risk for depression. In addition, there is some evidence that successful cognitive-behavioral psychotherapy can cause a shift in explanatory style such that clients become more optimistic.

Post Traumatic Stress Disorder

Despite the reformulated learned helplessness model's prominence in research on cognitive approaches to human depression, it is the original learned helplessness model that has primarily (although not exclusively) influenced research on PTSD in humans. The behaviors that follow inescapable shock in animals appear to mimic many of the symptoms of PTSD in humans. As already noted, animals exposed to inescapable shock show cognitive, motivational, and emotional deficits. Similarly, humans exposed to traumatic stressors may show a variety of sequelae, including anxiety and hyperreactivity, isolation, and anhedonia, that are similar to the sequelae observed in helpless animals. After inescapable shock, animals experience a depletion of catecholamine in their central nervous system. Van der Kolk and his colleagues posit that catecholamine depletion, especially norepinephrine (NE) depletion, is also involved in the negative symptomatology of PTSD, such as lack of motivation. They also posit that an augmentation of locus coeruleus pathways causes intrusive PTSD symptoms, such as nightmares and flashbacks.

More important to van der Kolk and his colleagues' work is the analgesia response observed in helpless animals, such that they do not appear to experience pain in response to subsequent stressors presented within a short period of time after the administration of inescapable shock. This response is due to endogenous opioids in the body. Interestingly, there are many parallels between the symptoms of opiate withdrawal and PTSD, leading van der Kolk and colleagues (1985: 320) to hypothesize that "both are, at least in part, due to central noradrenergic hyperactivity associated with a relative decrease in brain opioid receptor binding." This hypothesis is supported by evidence that trauma survivors often put themselves in situations similar to the traumatic event, perhaps in an attempt to evoke endogenous opioid release and thus a sense of calm and control. However, this sense of calm is followed by symptoms of opiate withdrawal, such as hyperreactivity, thus immersing PTSD sufferers in a cycle of reexposure and withdrawal.

Other research on helpless animals finds that the NE depletion may be the outcome of a more immediate increase in NE when a helpless animal is exposed to a mild stressor. The hippocampus of the helpless

animal appears to be conditioned by the inescapable, helplessness-inducing trauma to respond with increased NE to later lesser stressors. This increase in NE secretion immediately after later stressors could be part of the same general catecholamine dysregulation following traumatic stress as the NE depletion reported by van der Kolk.

In addition to the neurochemical and behavioral evidence for a learned helplessness approach to PTSD based on uncontrollable stress, evidence also exists supporting a relationship between helpless explanatory style and PSTD symptoms. McCormick and colleagues studied explanatory style among patients addicted to alcohol and/or gambling and found that patients with PTSD had a more pessimistic explanatory style than those who did not have PTSD. Although correlational, these results suggest that the cognitive reformulation of learned helplessness may also contribute to an understanding of the phenomenology of and potential treatments for PTSD. Moreover, recent work suggests that learning experiences can immunize animals against becoming helpless in the face of stress and that posttrauma therapeutic interventions centered on learning to predict and control stressors can be helpful in the treatment of PTSD in humans. This work suggests that the learned helplessness framework may be useful for both prevention and treatment of stress-related psychological disorders.

See Also the Following Articles

Cytokines, Stress, and Depression; Depression Models.

Further Reading

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