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Spring 2021

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## Abstract

The hopelessness theory of depression, mainly contributed by Martin Seligman, theorizes that the cause of depression is due to a belief that one has no control over the outcomes of their life, leaving them hopeless. “Learned helplessness” or “hopelessness” is a key phenomenon that helped Martin Seligman develop the theory of hopelessness depression. The current literature review traces the history of learned helplessness, from the first findings of evidence of the phenomenon to the development of the hopelessness depression theory.



## Keywords

depression, theory, hopelessness depression, learned helplessness

## Disciplines

Psychology | Theory and Philosophy

## Comments

Written for PSYCH 341: History of Psychological Science



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# The History and Development of the Hopelessness Depression Theory

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**Abstract**

The hopelessness theory of depression, mainly contributed to by Martin Seligman, theorizes that the cause of depression is due to a belief that one has no control over the outcomes of their life, leaving them hopeless. “Learned helplessness” or “hopelessness” is a key phenomenon that helped Martin Seligman develop the theory of hopelessness depression. The current literature review traces the history of learned helplessness, from the first findings of evidence of the phenomenon to the development of the hopelessness depression theory.

Depression, formally known as Major Depressive Disorder (MDD), is a psychological disorder that affects one's mood, causing persistent feelings of deep sadness and loss of interest in activities that previously brought them joy. Many theories exist explaining why and how one succumbs to depression. The Diathesis-Stress model proposes that stressful life events such as the death of a loved one, divorce, or health and money issues can lead to depression. There are cognitive theories that have been proposed that argue negative thinking habits trigger depression; negative thoughts, interpretations, self-evaluations, and expectations result in a "cognitive vulnerability" (Gotlib & Joormann, 2010). One cognitive theory proposes that some people possess depressive schemas which render them more likely to have depression. Another, known as the rumination theory, explains that peoples' persistent dwelling on their depressive symptoms is the driving force behind their depression. Finally, the hopelessness theory of depression, proposed by Martin Seligman (1972), explains that people gain negative expectations of their life due to a "learned hopelessness", leaving them more vulnerable to depression. Seligman's theory was derived from tests conducted on dogs, whom he claimed formed a learned hopelessness or helplessness which in turn caused the dogs to be unable to help themselves out of traumatic situations. However, the history of the development of this theory begins 30 years before Seligman's theory, beginning with Walter B. Cannon's article on "voodoo death" (1942). By tracing the observations and research conducted on learned hopelessness and related ideas, it is demonstrated how the theory of hopelessness depression came to be.

Walter B. Cannon (1942) described instances in which black magic or sorcery had been reported to be the cause of death in "primitive" people native to lands such as Africa, South America, Australia, New Zealand, and islands of the Pacific. Cannon refers to this occurrence as "voodoo death". It is brought upon someone by the word of a tribe's medicine man or through a

cursed object. Cannon also explains how some from more civilized communities, such as Dr. S. M. Lambert from Western Pacific Health Service of the Rockefeller Foundation, have come to witness this voodoo death. Many instances described in this article are similar to the account of Dr. Lambert's, where a native convert who worked with Dr. Lambert at a missionary in Mona Mona, Australia, had a bone pointed at him by Nebo, a famous witch doctor. Within hours, Dr. Lambert's helper grew tremendously ill and weak, but showed no symptoms of fever or disease. Dr. Lambert sought out Nebo and forced him to reverse whatever had been done to his helper. Nebo complied and went to Dr. Lambert's helper, assuring him that his life was no longer in danger. Almost instantaneously, Dr. Lambert's helper was back to normal. In order to determine what causes voodoo death, Cannon describes what it is like for a native in these types of communities to be subjected to bone-pointing or this black magic. He explains that these communities are so superstitious and convinced of voodoo, black magic, evil spirits and such, that when they believe they have been cursed, "the gravest known extremity of fear" befalls the victim, filling them with "powerless misery". Within the community, the victim is outcast as all his kin and friends are under the belief that he will die and he is left with no support of his tribe. The victim is left in utter isolation, abandoned by the community. Cannon turns to a quote from William James to demonstrate the effect of being outcast by society. He describes the "social self" as a being that needs to be noticed by others, and that

> "No more fiendish punishment could be devised, were such a thing physically possible, than that one should be turned loose in society and remain absolutely unnoticed by all the members thereof. If no one turned round when we entered, answered when we spoke, or minded what we did, but if every person we met "cut us dead," and acted as if we were nonexisting things, a kind of rage and impotent despair would ere long well up in us,

> from which the cruellest bodily tortures would be a relief; for these would make us feel that, however bad might be our plight, we had not sunk to such a depth as to be unworthy of attention at all" (1890).

After the abandonment by the community, one's fear is only heightened. Cannon postulates that the cause of death in voodoo death is the sheer fear that one feels when subjected to bone-pointing or another form of sorcery that they believe in. Canon explains that fear, one of the most deeply rooted and powerful emotions, is associated with ingrained survival instincts: to attack or run away, commonly known as our "fight or flight" response. He continues that fear activates the sympathico-adrenal division within the nervous system. This division is responsible for maintaining constant blood flow throughout the body so that it is well equipped for a physical struggle. However, the results of a prolonged activation of the sympathico-adrenal division can have detrimental results. Cannon cites Philip Bard (1928) and Norman E. Freeman (1933) as having produced results from experiments on animals that a significant decrease in blood pressure is due to excessive activity of the sympathico-adrenal division. Cannon hypothesizes that a prolonged state of extreme fear resulting in a significant decrease in blood pressure is the cause of voodoo death. This would make sense for the accounts of voodoo death seen in native communities who seem to die after a couple of days since being "cursed"; multiple days is a long time to be constantly petrified.

Curt P. Richter (1957) examined a phenomenon of sudden death similar to voodoo death in an experimental setting with rats. Richter observed instances of rats who had had their whiskers trimmed off suddenly dying when being restrained in an experimenter's hand or being placed into a jar of water. Cannon's hypothesis was tested by using electrocardiographic records to observe the heart rates of the rats up until their deaths. The records demonstrated that, contrary

to Richter's expectations, the heart rates of the rats decreased until death, rather than increased. This suggests that the rats, "may have died a so-called vagus death, which is a result of overstimulation of the parasympathetic rather than of the sympathico-adrenal system" which Cannon hypothesized. Richter then explains that these observations suggest that the situation of the rats is not one of fight or flight, but instead of hopelessness; when the rats find themselves in a defenseless situation, they seem to just "give up" and do not attempt to resist by biting or squirming to free themselves. Once the rats are removed from the hopeless situation, they almost immediately return to normal behavior, which is strikingly similar to the behavior of natives that Cannon described who were on the brink of death before their curse had been reversed. Richter's observations provided a scientific foundation of learned hopelessness and is significant in demonstrating the phenomenon in an experimental setting. Although his observations provide evidence against Cannon's hypothesis, suggesting that the phenomenon which caused the rats to die was an emotional one of the parasympathetic system rather than the sympathico-adrenal system like Cannon believed, it is important to understand that Richter built off the ideas of Cannon. Similarly, the next stage in the development of the hopelessness theory of depression was built off of Richter.

Overmier & Seligman (1967) examined the effects of inescapable shocks on subsequent avoidance responses in dogs. Four groups of dogs were strapped into a harness and subjected to inescapable shocks produced from a platform on which they stood. Each group was presented with a different amount of shocks at differing durations and intervals, except one group which was given no shock presentation. 24 hours after shock presentations, all dogs were administered 10 escape-avoidance test trials where dogs were placed in a shuttle box that contained a barrier in the middle. For each trial, a shock was administered with the chance of escape for each dog by

jumping over the barrier onto the other side of the shuttle box. In two of the groups that received inescapable shocks prior to the escape-avoidance test, the number of failures to escape was significantly higher than the group that did not receive inescapable shocks. Overmier & Seligman proposed that the interference with escape-avoidance behavior is a "learned helplessness" or "hopelessness", the same cause of the unusual deaths that Richter found in his mice. It was explained that because the dogs received shocks in a situation where they are completely incapable of eliminating the trauma of the shocks, they "learn" to become hopeless when experiencing shocks in the future, causing them to "give up" and fail to escape. This interpretation of results suggests that the level of control the dogs have over their exposure to shock is an important variable in their learning to become hopeless. It was suggested that high levels of control over the shock presentation may "immunize" interference with escape-avoidance behavior and should be investigated in future research.

The hypothesis that the degree of control over shock presentation eliminates interference with escape-avoidance behavior in dogs was tested by Seligman & Maier (1967). Two groups of dogs were presented with shocks while in a harness. However, the "escape" group was also placed between two panels of which could be activated once touched by a dog's head. Upon activation of the panels, the presentation of shock would be terminated. This variable allowing the dogs to terminate the shock presentation was intended to teach the dogs a degree of control that they had over their shock presentations. The "yoked" group was presented with inescapable shock presentations. All dogs were then administered the same 10 avoidance-escape test trials as used in experiment Overmier & Seligman (1967) 24 hours after the shock presentations. It was found that the number of failures to escape the shock for the "yoked" group was significantly higher than the failures to escape for the "escape" group, suggesting that the control that dogs

have over their initial shock presentation determines whether or not inference with escape-avoidance behavior occurs. The results support the idea that the dogs acquire a "learned helplessness" after learning that they are unable to control the shock presentations, which hinders them from trying to escape shocks in the future.

The work and observations of Cannon (1942), Richter (1957), Overmier & Seligman (1967), and Seligman & Maier (1967) demonstrate the development of the idea of "learned helplessness" which is the backbone of Martin Selgiman's helplessness theory of depression. Seligman (1972) writes an article providing an overview of his findings in his studies of dogs and learned helplessness, explaining his results and introducing his ideas of effects, cures, and preventions surrounding learned helplessness. It is emphasized that uncontrollable trauma is what causes a learned helplessness, not trauma itself. Seligman continues with an explanation on how learned helplessness may play a role in depression in humans, claiming that there are a number of parallels between the learned helplessness found in dogs and depression in humans. Learned helplessness, like depression, "is characterized by reduced response initiation as well as a 'negative cognitive set'", meaning that one finds difficulty in believing that they have the control to succeed through their own actions, even when they do have control. A factor analysis of depression demonstrates that "feelings of hopelessness, helplessness, and worthlessness have been characterized as the essence of depression". Furthermore, the norepinephrine depletion caused by uncontrollable trauma parallels the catecholamine hypothesis, which postulates that norepinephrine depletion is the cause of depression in humans. Finally, successful treatment of depression in humans where one comes to believe that their actions can produce desirable outcomes in their life parallels a finding that Seligman produced in another study with dogs. Seligman (1968) demonstrated that dogs who previously acquired a learned helplessness and

failed to show avoidance-escape behaviors could later learn to escape shocks by being forcibly shown that their actions can cause the shocks to be terminated. Seligman emphasized the point that only animals have been used in tests so only speculations can be made until tests on humans have been conducted.

Hiroto (1974) examined learned helplessness in humans using loud and aversive tones as the inescapable trauma, and a human analogous to Seligman's shuttle box as a test for helplessness. College students were divided into three groups: an escape group, an inescapable group, and a control group. The escape and inescapable groups were given pretreatments, where they were taken to a room with a button presented before them. They were given instructions that a loud and unpleasant tone will sound and there was something they could do to stop it. For the escape group, by pressing the button before them, the loud tone was terminated. For the inescapable group, the button did nothing. This manipulation of the inescapable group intended to "teach" the participants that they had no control. The control group was given no pretreatment. For the test for helplessness, all participants were presented with an apparatus with a knob that slid left and right, and were instructed that they will be given 18 trials in which a loud tone will sound and that there is something they can do to stop it. For all participants, the correct escape-avoidance behavior was sliding the knob one way on the apparatus when the tone sounded and then sliding it to the other side when it sounded again. Correct escape-avoidance behaviors resulted in a termination of the tone until it began again for the next trial. The latency of response was recorded as the measurement of helplessness. Results indicated that the inescapable group had significantly higher latency scores than the other groups, suggesting that the inescapable group had learned to be helpless. These results demonstrate that the learned helplessness found in Seligman's dogs can be experimentally induced in humans. These results

provide evidence that a perceived lack of control leads to a learned helplessness in humans, disabling them from effectively escaping undesirable situations even when they do have control.

With evidence that learned helplessness is a phenomenon that can occur in humans, Hiroto & Seligman (1975) produced results demonstrating the generality of learned helplessness in humans using both physical and cognitive pretreatments, as well as physical, like Hiroto's (1974) sound-shuttle box, and cognitive tests of helplessness. It was suggested that because it was generalized across motivations and tasks, learned helplessness acts as more of a "trait" rather than a "state".

The idea of learned helplessness in humans gained significant popularity during the time Seligman was proposing his ideas and using experiments to demonstrate it occurs in humans, not just animals. However, before a formal theory of depression was proposed, there were critiques to Seligman's work in learned helplessness which are addressed and resolved by Abramson et al. (1978). The first of which addresses the issue of universal vs. personal helplessness, explaining that there is no differentiation between: a person believing there is nothing anyone can do to solve the issue (universal), and when there may be a solution but only he is unable to solve it for himself (personal). The second critique addresses generality and chronicity, explaining that there is no consensus on when and where a person who expects outcomes to be uncontrollable will show deficits. In the reformulation, where a distinction between universal and personal helplessness is made, and an explanation of the generality and chronicity is given, a clearer picture of learned helplessness in humans is presented. The issues solved now allow for development in the theory of learned helplessness and depression in humans.

Abramson et al. (1989) proposed a formal theory of depression based on learned helplessness in humans, called *hopelessness depression*, and described hypothesized causes,

symptoms, course, therapy, and prevention. The cause of symptoms of hopelessness depression was defined as, " an expectation that highly desired outcomes will not occur or that highly aversive outcomes will occur and that no response in one's repertoire will change the likelihood of occurrence of these outcomes". Evidently, what causes depression in the hopelessness theory is, not surprisingly, hopelessness. The years of research preceding the development of this theory provide evidence that one becomes hopeless and helpless in the face of aversive situations due to the belief that what may transpire is out of their control, and therefore the person has no desire to try to influence the outcome with their actions. Two emphasized symptoms of hopelessness depression are retardated initiation of voluntary response and sad affect. Other symptoms hypothesized to be associated with hopelessness depression are suicidal ideation, lack of energy, apathy, disturbed sleep, and difficulty in concentration. Although no specific treatments were given, most likely due to insufficient research, treatment of hopelessness depression was proposed to be any therapeutic strategy that undermines hopelessness and reinstates hopefulness; "hopelessness should be attacked directly". To prevent the onset, relapse, and recurrence of hopelessness depression, it was proposed that building "nondepressive" cognitive styles should be prioritized, meaning positive thinking habits and styles should be learned, rather than negative ones.

The development of the hopelessness depression theory is largely due to the research conducted by Martin Seligman on learned helplessness in dogs and his ideas on its application to humans. However, the phenomenon can be traced to scientists and scholars that came before Seligman, and by beginning with those who first witnessed the phenomenon, a fuller picture of the development of the theory of hopelessness depression is revealed. His initial theory of learned helplessness eventually progressed into a formal theory of depression, hopelessness

depression, which is still a predominant theory behind the cause of depression and is still a researched topic today.

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