Neal E. Miller and His Research

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Neal Miller’s research contributions mainly concerned reward and learning mechanisms: (a) underlying thought processes and behaviors relevant to problem solving in psychotherapy and everyday life, (b) as mediated by the nervous system, and (c) involved in learning control over voluntary (conscious) skeletal muscle and autonomic (normally unconscious) internal-organ response systems for minimizing stress, treating disease, and promoting health. His career shows psychology’s evolution from a theory-driven but data-impoverished discipline in the 1930s to one integrating vast bodies of clinical, social, and physiological knowledge.

In 2002, the Monitor on Psychology newsletter (“Study Ranks,” 2002) of the American Psychology Association (APA) ranked Neal Elgar Miller (b. Milwaukee, Wisconsin, August 3, 1909; d. Hamden, Connecticut, March 23, 2002) among the 10 most eminent psychologists of the 20th century. Miller, who authored eight books and more than 276 articles, was highly influential as a learning theorist, neuroscientist, and science statesman and educator. Above all else, though, he was an incomparable exponent of the scientific method in research in illustrating to what deep principles of inquiry psychology as a mature science must hew (an article in this issue by Edward Taub elaborates on this theme). Among multitudinous honors, he was elected to the National Academy of Science (1958), was president of the APA (1960–1961), received the National Medal of Science (1964), chaired the founding of the Society for Neuroscience (1969), and served as president of that organization (1971–1972). He became a member of the Biofeedback Society of America (now the Association for Applied Psychophysiology and Biofeedback) in 1972 and its president in 1984.

Research at Yale University (1932–1965)

Foresight and the laws of learning. As a graduate student at Yale (1932–1935), Miller was influenced by Clark L. Hull, the leading learning theorist of his time. Hull aimed to show how the principles of classical conditioning could be applied to understanding trial-and-error learning, verbal learning, and higher mental processes such as purposeful, goal-oriented, and foresightful behavior. Toward the latter, Miller, in his PhD thesis (Miller, 1935, 1944), demonstrated in a human subject that after his saying the letter T and always getting shocked and the number 4 and never getting shocked, a learned change in his skin conductivity—now elicited by T but not by 4—would transfer to his merely thinking T. This showed that the mental acts of thinking are themselves responses that can serve as cues (response produced) to which other responses (such as skin conductance changes) can then be made; the acts of thinking are accordingly subject to the same laws of learning as are external responses and cues. These mental responses, unconstrained by the real-time sequencing of cues in the physical world, permit the playback of events in reverse of their actual happenings. Thereby, one can work backward in one’s mind’s eye from a hoped-for goal along a route that better illuminates how to reach it than groping blindly forward from the start. It is such use of response-produced cues that Miller identified as the basis for much foresightful behavior in problem solving.

Extending the quest to Freudian phenomena. Such insights emboldened Miller to also examine Freudian theory and practice in terms of the laws of learning. In 1935, Freud accepted Miller for a year’s postdoctorate work in the Psychoanalytic Institute in Vienna, where he underwent a didactic analysis with Heinz Hartmann. He long regretted

¹ In this article, Neal Miller’s research achievements that are described are largely stripped of their biographical contexts. For a fuller account of them, please consult the following references: Miller (1971), Evans (1976), Miller (1992), Coons (2002), and Koertge (2007).
turning down at least one session with Freud because an hourly $20 fee seemed more than he could afford.

Aggression as misplaced frustration. Soon after returning to Yale, Miller observed a dominant male monkey self-mutilate when prohibited from attacking a competitor, which had been given the first male’s harem. This suggested for him the Freudian concept of aggression turned inward and resulted, in 1939, in Miller’s book *Frustration and Aggression* (Dollard, Miller, Doob, Mowrer, & Sears, 1939), coauthored, among others, with sociologist John Dollard. Its major hypothesis was that when a segment of society is frustrated from attaining its goals, it tends to relieve that frustration by means of angryinations against an innocent, less powerful segment, as exemplified by Miller’s European encounters with anti-Semitism inflamed by the still-lingering economic privations from reparations required of Germany after World War I.

Instrumental learning of social behavior. In 1941, Miller and Dollard wrote *Social Learning and Imitation* to show how a wide range of human behavior can be understood by knowing a few important principles of learning plus the social conditions or contexts in which the learning takes place. They listed four fundamentals necessary for instrumental learning, that is, for remembering which behaviors (responses)—guided by what signposts (cues)—have proven, for future reference, to be instrumental for successes (rewards) in achieving one’s goals (reducing one’s drives). Impressed with the strong influence of groups on individuals’ behavior, particularly as evidenced in imitation, he and Dollard conducted experiments on rats and young children, showing that the tendency to imitate could, itself, be learned under appropriate rewards and broadly generalized to many situations. Further, the children could be trained to note the degree of difference between their own behavior and that of the model they were trying to copy and use the degree of discrepancy as a motivational cue for how to minimize the discrepancy so as to improve the match. This learned use of negative feedback was an early example of what is now called a cybernetic analysis.

The drive-reduction hypothesis. Hull originally hypothesized that a response is reinforced (strengthened) if immediately followed by a reduction in a need. However, the assumption was that all needs drive (motivate) responses to reduce them. But some needs do not because they cannot be detected (e.g., the need to escape carbon monoxide). Thus, also in *Social Learning and Imitation*, Hull’s hypothesis was restated by Miller and Dollard as the “drive-reduction theory [hypothesis] of reward,” (p. 35[n]) which now dealt only with detected needs, henceforth defined as drives. In its strong form, the drive-reduction hypothesis asserts that the only events that can act as reinforcers of a response are those that immediately follow it and are themselves soon followed by a reduction in the drive motivating it or are highly associated with that drive’s later reduction. Assessing this strong form’s validity was the concern of much of Miller’s later research.

From reaction formation to approach-avoidance conflict. As a result of observations made while testing in rats the Freudian idea of reaction formation, Miller in 1944 published his famous theoretical-experimental analyses of approach-avoidance conflict behavior (Miller, 1944). These stated that if a goal is something an organism both wants and fears, there is an approach tendency to it, called an approach gradient, that grows stronger the nearer one gets to it, but there is also an avoidance gradient that does the same. However, the avoidance gradient increases more rapidly with nearness than does the approach gradient. The gradients often will cross each other; at that intersection, the organism’s approach will stop. It is there that “the rat will stand with reluctant feet where the gradients of approach and avoidance meet,” but just how close to the goal will depend on the relative strengths of the approach gradient versus the avoidance one.

Fear as a learnable drive. In 1948, Miller rigorously tested a hypothesis by Hobart Mowrer (proposed in 1939) that fear is an acquirable (i.e., learnable) drive (Miller, 1948a). He first shocked rats in the white side of a two-compartment box until they learned to run rapidly through a door into the black side to escape shock. Afterward, when put in the white side with the door closed but without shock administered, they defecated and showed other signs of having learned to fear the cues to which shock had become associated. If, by trial and error, they then rotated a small wheel by the door, which opened the door and allowed them to escape the white side’s cues and thus the fear itself, they quickly learned to rotate the wheel for escape on subsequent nonshock trials. This confirmed Mowrer’s hypothesis.

Displacement. In 1948, Miller published an article “Theory and Experiment Relating Psychoanalytic Displacement to Stimulus-Response Generalization” (Miller, 1948b), in which, harkening back to his “Experimental Studies of Conflict Behavior,” he posited the following: When the approach to a stimulus is inhibited by conflict with an avoidance of that same stimulus, responses tend to displace to other stimuli that are still similar enough to motivate the prospect of a successful approach but are dissimilar enough to minimize the interfering avoidance. For example, given the Freudian Oedipal conflicts between
a young son’s erotic love of his mother and retaliatory fear of his father, one can understand the displacement implied in the old vaudeville song, “I want a girl just like the girl [but not the same one, God forbid] that married dear old dad.’” This and other predictions were borne out by three other studies on displacement, all in the same year (Miller & Kraeling, 1952; Miller & Murray, 1952a, 1952b).

Principles of learning underlying thought processes, repression, and psychotherapy. Dollard and Miller (1950) published Personality and Psychotherapy: An Analysis in Terms of Learning, Thinking, and Culture. It was immensely influential in training the first post–World War II generation of clinical psychologists in the treatment of the neuroses and was, for years, widely used as a text in learning theory. It also fostered the behavior therapy movements of the 1960s and 1970s. Its thesis was that neuroses are learned bad habits and, therefore, through the same principles of learning, can be extinguished and supplanted with more adaptive behaviors through the practice of psychotherapy. Harkening back to Miller’s PhD thesis, the book showed how in therapy, the appropriate use of response-produced cues, particularly verbal ones, can facilitate generalizations between similarities that should be perceived in one’s life but maladaptively are not and distinctions between differences that, likewise, should be perceived but, again, are not. Also drawing upon the finding in rats that escape from the cues to which fear has been conditioned can reinforce the learning of the behaviors successfully involved, Dollard and Miller pointed out that repression can similarly arise by the automatic reinforcing of the suppression of taboo thoughts because it removes the anxiety (analogous to fear) that these forbidden thoughts arouse.

Into the gut and brain to test the strong form of the drive-reduction hypothesis of reward. Around 1950–1952, Miller turned to physiological interventions because they offered unique opportunities to test the strong form of the drive-reduction hypothesis of reinforcement against competing possibilities. For example, instead of the reinforcing value of food for a hungry animal residing in the food’s ability to reduce hunger, might it instead be either the pleasures of taste or of the swallowing of the food that is reinforcing? But if one could reward behavior by reducing hunger while bypassing both taste and swallowing, that would clearly support the drive-reduction hypothesis. Indeed, delivery of food to a hungry rat via a tube directly into its stomach (Miller & Kessen, 1952) rewarded the learning of correct choices in a T-maze for that delivery. This supported the drive-reduction hypothesis of reward but did not discount that taste and swallowing could also be rewarding.

More tests suggest the need for a variety of measures. Another plan for testing the drive-reduction hypothesis was to lesion (destroy) the ventromedial nucleus in a rat’s hypothalamus that then causes overeating and obesity. If this overeating had all the aspects of normal hunger, electrical stimulation of the intact nucleus should be satiating to a normally hungry rat and therefore should also reward the learning of correct choices in a T-maze. But contrary to hunger motivation, these lesioned rats, although eating a larger amount of highly palatable foods than normal rats, worked less hard for food and were less tolerant of less palatable foods (Miller, Bailey, & Stevenson, 1950). This result spoiled Miller’s plan but taught him the importance of taking a variety of measures before inferring the nature of an underlying state, a lesson that he strongly communicated in print in 1961 as a cautionary tale for psychologists working in the brain (Miller, 1961).

A trip into the brain regarding the learnability of fear yields unexpected rewards. The salience of the brain approach was heightened by two dramatic findings in the mid-1950s. One was the discovery by Olds and Milner (1954) of sites in the lateral hypothalamus that rats find rewarding to self-stimulate with volleys of brief electrical pulses by pressing a lever. The other was a reverse discovery by Delgado, Roberts, and Miller (1954) of sites where electrical stimulation would motivate cats to learn a response to escape or avoid the stimulation. But it was puzzling that at some sites, cats would learn a response to terminate stimulation but not a response to avoid it—an observation leading to the discovery of the reward-escape effect in which Gordon Bower was involved. Implanted rats showing this effect cycled repeatedly between pressing a lever to turn on the stimulation and rotating a wheel to turn it off (Bower & Miller, 1958). In 1957, Miller seized this opportunity for an unusual test of some drugs. He showed that methamphetamine enhanced and chlorpromazine reduced the rewarding aspects of the cycle while leaving the punishing aspects unaffected (Miller, 1957a, 1957b). This was a first evidence of what later was recognized as the involvement of the neurotransmitter dopamine in promoting reward. Miller presented these data and others with Herbert Barry to drug companies to advertise the potential benefits of behaviorally evaluating pharmacological agents (Miller & Barry, 1960). With his encouragement, this approach was to become the field of behavioral psychopharmacology.

The drive-reduction hypothesis problematically sustained. Another advantage of implanting electrodes in the lateral hypothalamus was to search in rats for where in cats von Hess (1954) had found stimulation causing them to eat.
But as Miller reported in his 1992 autobiographical article, the search took 2 years before Coons (Coons, 1964; Miller, 1957a) discovered the site where even thoroughly satiated rats would eat ravenously while stimulated there but stop immediately when the current was turned off. Behavioral tests showed this electrically elicited eating had all the earmarks of normally motivated hunger. Then why, contrary to the drive-reduction hypothesis, would these animals not press a lever to turn this hunger stimulation off but would do so to turn it on? In answer, Miller (1960) reported Coons’s finding that amphetamine raises the threshold to elicit this “hunger (as evidenced in feeding)” and lowers that to sustain self-stimulation, showing that a single system does not subserve both self-stimulation and this hunger. Also, Coons later found (Coons & Cruce, 1968) that at the lowest current required to elicit this hunger, rats would not press a lever for it unless food was available to eat while the current was on, just as the drive-reduction hypothesis would predict.

Unlike fear, hunger proves not learnable; parsimony fails its test. The phenomenon of stimulation-elicited hunger lent itself to yet another question. Is hunger parsimoniously a learnable drive like fear? If so, would not a satiated animal then eat in the presence of a cue to which the experience of being hungry had in the past been closely associated? Earlier attempts by Myers and Miller (1954) to demonstrate such learning had proved a failure, but that could be because the slowness of onsets and offsets of normal hunger attenuated its association with the cue too much. The rapidity, however, with which stimulation-elicited hunger could be turned on whenever a designated cue was presented made for a much better test. However, as Miller (1964) reported, the predicted unstimulated eating in satiated animals was never observed to occur in the presence of the cue, even after numerous trials of pairing stimulation-elicited hunger with that cue.

Chemical coding of behavior—more unexpected rewards. The phenomena of electrically elicited hunger (evidenced by eating) and self-stimulation reward garnered much attention and focused scientists on the possibility of further findings to be gained by pursuing the brain’s role in behavior. In 1960, profound confirmation of this possibility came with Grossman’s finding in Miller’s lab that an agent mimicking the neurotransmitter acetylcholine elicited drinking when introduced into the hypothalamic feeding site via an indwelling cannula, whereas down the same cannula, an agent mimicking the neurotransmitter noradrenaline elicited eating. The excitement generated by this chemical coding of different consummatory behaviors from the same region jump-started the inquiry into neurotransmitter regulation of behavioral mechanisms in Miller’s lab (Miller, 1965) and elsewhere. Physiological psychologists could now feel that they were having the same scientific legitimacy and footing as neurophysiologists and chemists.

Research at Rockefeller (1966–1988)

Is voluntary control of autonomic functions learnable? In 1957, Gantt’s translation of Bykov’s book, The Cerebral Cortex and the Internal Organs (Bykov, 1942/1957), reported that autonomic responses in a wide variety of internal organ (visceral) systems, when elicited by their innately triggering stimuli, can then become elicitable (classically conditioned) by other stimuli that routinely closely precede and thus strongly predict these triggers. The book stimulated Miller to pursue his earlier hunch (Miller, 1951), entertained against popular opinion, that autonomic responses are not limited to becoming learned reactions to stimuli but, if properly rewarded, can be trained—like ordinary voluntary responses—to become intended behaviors to obtain those rewards. If so, the medical benefits of such conscious controllability would be enormous and would fulfill Hull’s and his overarching hope to show an underlying relatedness of all laws of learning, spanning across voluntary, cognitive, clinical, and—now—autonomic domains of behavior.

Indeed, in 1967, Miller and Carmona published evidence that thirsty dogs were able to increase or to decrease their autonomic response of salivation to obtain water rewards but, puzzlingly, displayed different postures during increases compared with decreases. Maybe just the postures were learned but somehow triggered the salivations (Miller & Carmona, 1967). To rule this out, rats were treated with curare, which completely paralyzed their voluntary muscle systems but left their autonomically controlled visceral muscle systems unaffected. Then, the autonomic response of increasing (or, alternately, decreasing) the rats’ heart rates was designated the specific response basis for their obtaining very rewarding brain stimulation.

As predicted and reported in two companion articles (Miller & DiCara, 1967; Trowill, 1967) to the Miller and Carmona (1967) study, the autonomic changes in heart-rate responding required for rewards dramatically occurred. Over many studies from 1965 to 1972, even the general public, vis-à-vis The New Yorker (Jonas, 1973) and other media, became aware of the medical benefits this promised. But then Miller and Dworkin in his laboratory began finding that these results were mysteriously diminishing until they could no longer be replicated (Miller & Dworkin, 1974) even after repeated and varied attempts (Dworkin &
Miller, 1986). When finally convinced of failure, Miller, though heartbroken, took great pains to publicize it widely.

Biofeedback in the health-directed control of homeostasis and stress. Despite disappointments in this line of research, it led to advances in technology to measure otherwise impossible-to-detect subtle changes in heart rate and other physiological responses. After finding that paralyzed rats failed to learn autonomic control, Miller shifted this technology to seeing whether people who had been paralyzed by gunshot wounds that severed their spinal cords could gain that control. They differed from the rats in being better candidates in terms of Miller and Dollard’s (1941) four fundamentals necessary for effective instrumental learning: These patients had a high drive to try gaining control because their blood pressure was so low that whenever they sat or stood up, they fainted. Unlike the rats, they were shown their own amplified heart rate and blood pressure readings, thus providing them biofeedback informational cues about their own performance, as Miller (1973) was invited to report. To this biofeedback information, the response they reported using—to try to change their readings—was to think emotional, often sexy, thoughts to which the desired blood pressure changes are normally reflexively connected. As these paralyzed patients became successful, they were gradually able to command these changes directly (Miller & Brucker, 1979; Pickering et al., 1977). Whenever there was a desired response, even if too small an increment initially to be clinically relevant, the mere detectable fact of it was a reward, given the paralytics’ high achievement motivation. Slowly, the summing of such increments mounted to clinically significant levels, and as they did, the rewards became enormous because the paralytics could now sit up without fainting and, as a result, they could now also attend plays and ball games. But how the patients achieved voluntary control of blood pressure via the autonomic nervous system was puzzling; with a severed spinal cord, the usual route of elevating blood pressure via the sympathetic component of the autonomic nervous system is also cut off. Parasympathetic (vagal) or blood-borne humoral factors are suspected of perhaps some subtle respiratory mediation still surviving paralysis, as reported in 1976 regarding vasomotor responses (Lynch, Hama, Kohn, & Miller, 1976).

This and other studies using biofeedback also convincingly suggested the ability to bring autonomic responses under voluntary control, whether directly or indirectly. And as Miller loved to point out, toilet training, particularly the learning of control over the autonomic bladder sphincters, is a well known—and rewarded—universal fact of life. Certainly, by 1985, the application of biofeedback method-ology promoted by Miller and his associates had proved highly beneficial medically in treating a wide variety of problems, such as idiopathic scoliosis, enuresis, and migraine, problems involving both voluntary and autonomic response systems (Dworkin et al., 1985).

From the mid-1970s until a few years before his death in 2002, Miller’s inquiries into biofeedback and learned behavior took on a new emphasis, that of their use to maintain homeostasis (Miller & Dworkin, 1980) and minimize stress. Coordinating with this approach were a series of studies on fear, stress, and coping being conducted in Miller’s lab at Rockefeller under the direction of Jay M. Weiss and Bruce S. McEwen (Miller, 1976). These studies used a combination of behavioral, physiological, and biochemical techniques to examine the effects of predictable versus unpredictable shock, conflict, and toughening on measures of behavioral depression, physical symptoms of stress, catecholamine depletion in the brainstem, and glucocorticoid uptake in and effects on the hippocampus and the memory functions it serves. Miller’s attention, thereby, to the mechanisms by which the nervous system and body interact with each other contributed substantially to the establishment not only of biofeedback as a discipline but also the fields of behavioral medicine (Miller, 1975, 1983a) and health psychology (Miller, 1983b; Stone et al., 1987), all of which consider Miller a founding father. Meanwhile, vis-à-vis his long-term interest in feeding behavior, he continued to support research at Rockefeller done in the behavioral neurobiology laboratory of his former postdoc, Sarah Leibowitz (coauthor of this article), on eating behavior, alcohol consumption, and the mechanisms by which they relate to addiction and obesity.²

Among Miller’s last research contributions, 1993–1994, were his collaborations with Dr. Patricia Cowings (Cowings, Toscano, Miller, & Reynoso, 1994) and with Dr. Edward Taub (Taub, Crago, et al., 1994; Taub, Miller, et al., 1993). In particular, he took great pleasure in working with Taub in the development of constraint-induced movement therapy.

² There were a number of other research ventures in Miller’s lab that do not fit nicely into the trajectory of his life characterizing this portrait but which he strongly encouraged, supported, and included in his Selected Papers (Miller, 1973). Recommended readings are his collaborations, among others, with (a) David Egger on findings premonitory of the Rescorla-Wagner model that now dominates studies in learning, (b) David Quartermain concerning memory consolidation as growing out of a study by Coons and Miller, and (c) studies devoted to understanding the signals for thirst with Don Novin, salt appetite with George Wolf and Edward Stricker, and hunger with John D. Davis, Stan Tenen, David Booth, Eric Stone (Steinbaum), and Jonathon Winson. Among others whose research in his lab he fostered were Eleanor Adair, Gordon Ball, Derek Hendry, Frank Krasne, E. E. Kriechhaus, and Robert P. Vertes.
therapy, a very effective treatment to rehabilitate stroke victims with motor impairment by overcoming their learned nonuse, which in turn also promotes neuroplastic changes in the brain that further enhance motor recovery. As a result, a fitting research epitaph to his entire career, devoted to understanding to what the laws of learning apply, can be the following statement: “The brain controls learned behavior but in turn learned behavior also controls the brain—a biofeedback cooperation.”

References


