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Memories and Habits: Two Neural Systems

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The term "global anterograde amnesia" implies the rapid forgetting of all new experiences, yet investigators agree that persons suffering from this syndrome readily retain new experiences of a certain type or in a certain way. Characterizing the essential difference between the lost and spared retention abilities has become one of the major goals of both clinical and animal amnesic research.

Beginning with Milner's (1962) dramatic demonstration of a nearly normal rate of improvement of mirror-drawing skill in the severely amnesic patient H. M., the steadily mounting evidence for the dissociation of retention processes in amnesia has generated numerous theoretical interpretations of this dissociation. Among the labels that have been applied to the lost versus spared abilities are recognition versus associative memory (Gaffan, 1974), episodic versus semantic memory (Kinsbourne & Wood, 1975), working versus reference memory (Olton, Becker, & Handelmann, 1979), vertical versus horizontal associative memory (Wickelgren, 1979), declarative versus procedural knowledge (Cohen & Squire, 1980), elaborative versus integrative processing (Graf, Mandler, & Haden, 1982), and automatic versus effortful encoding (Hirst, 1982). All of these distinctions and numerous others (Huppert & Piercy, 1976; Cermak & Butters, 1972; O'Keefe & Nadel, 1978; Cutting, 1978; Cormier, 1981; Stern, 1981; Warrington & Weiskrantz, 1982) postulate, in essence, that the retention of experience entails two widely differing processes, a more and a less cognitive one (or a more and a less flexible one), only the first of which is affected in global amnesia.

The present report is written from this same theoretical perspective but adopts still another set of labels for the two types of retention: "memories" versus "habits." This particular functional distinction, which is deliberately drawn more sharply than any of those listed above, is essentially the one that was advanced by Hirsh in 1974 and later elaborated by him (Hirsh, 1980). A

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quote helps give the flavor of his position. "The present theory views the hippocampus as the gateway to memory. . . . In the absence of the hippocampus . . . learning is a matter of habit formation. Readers familiar with learning theory will realize that the behavior of normal animals is treated in a neo-Tolmanian framework, while that of hippocampally ablated animals is held to be everything for which early S-R theorists could have wished" (Hirsh, 1974, p. 439). He later modified this view slightly, as follows: "The [S-R] associative system must have been present all along if it is free to express itself following hippocampal ablation" (Hirsh, 1980, p. 181). In short, according to Hirsh, both sides in the great debate between behaviorism and cognitivism must ultimately be declared the winners, since the evidence from the study of amnesia demonstrates that both types of processes must be constantly present in normal behavior.

If this radical resolution of that long and difficult debate proves correct, it will have enormous implications for both psychological and neuropsychological research. First, every piece of learning will have to be analyzed and reanalyzed carefully for contributions to it by not just one but two qualitatively different types of retention processes. And second, two different types of retention processes implies two different storage mechanisms, or even two entirely different neural systems. The data reported in this chapter appear to us to lend support to Hirsh's provocative proposal and therefore to encourage the further development of a two-systems theory of learning.

MEMORIES AND A CORTICO-LIMBIC SYSTEM

The first form of learning to be considered, the one here labeled memory formation, is the one that by nearly universal agreement has been attributed to the hippocampal system exclusively. This attribution is explicit in the quotes from Hirsh. The evidence from our research on the monkey, however, suggests that memory formation has a broader limbic substrate than this, one that includes the amygdaloid system as well (Mishkin, Spiegler, Saunders, & Malamut, 1982). The discovery that memory functions may be shared jointly by the amygdala and the hippocampus grew out of work on the role of cortico-limbic interaction in the mnemonic process of linking neutral sensory stimuli with rewards.

Recognition and Associative Memory

As detailed elsewhere (Jones & Mishkin, 1972; Mishkin & Aggleton, 1981), studies on discrimination learning in the monkey had led to the suggestion that each primary sensory area together with its modality-specific association areas form a hierarchical system devoted to processing the purely physical qualities of the stimuli in that modality. Consequently, the attachment of reward value, and of affective qualities generally, to a stimulus that would otherwise remain emotionally and motivationally neutral would require an additional neuronal step. The additional step was postulated to be activation, by the sensory modality's highest-order processing area, of an amygdalo-hypothalamic pathway. This two-stage neural model of stimulus-reward association received its

strongest support from a study that compared the effects of inferior temporal and amygdaloid lesions on the performance of two different visual-memory tests, each of which utilized easily discriminable trial-unique objects (Spiegler & Mishkin, 1981). One was a test of object recognition, that is, the ability to remember from a single trial whether or not an object had been seen before; the other was a test of object-reward association, that is, the ability to remember from a single trial whether or not an object had been baited before. On the recognition test, only the inferior temporal lesion produced marked impairment; on the association test, by contrast, both lesions produced marked impairment, and the two impairments in this case were equally severe. The pattern of results thus fit the scheme that stimulus-reward association is indeed a two-stage process entailing, first, a stimulus-recognition mechanism heavily dependent on inferior temporal cortex (viewed as the highest-order processing station in vision) and, second, a reward-attachment mechanism heavily dependent on the amygdala (viewed as a multimodal gateway to the hypothalamus).

But proof that the amygdala's role in stimulus-reward association depends directly on the visual input from inferior temporal cortex requires an additional demonstration, namely, that the association can be prevented by anatomical disconnection of the two structures. Before such a disconnection test was attempted, however, it seemed desirable to amplify the memory deficits if possible by enlargement of the two lesions to include, in the one case, more of the cortical visual system, and in the other, more of the temporal lobe limbic system. It was the latter extension of lesions that led to the discovery of global amnesia in the monkey (Mishkin *et al.*, 1982).

Whereas hippocampal removal alone had failed to yield a notable effect on either of the memory tests described above, its combination with an amygdaloid removal turned out to have a profound effect, not only on stimulus-reward association, which was the original goal, but on stimulus recognition as well (Mishkin, 1978); and not only on stimulus recognition in vision but also in touch (Murray & Mishkin, 1983). Furthermore, visual recognition was found to depend on the anatomical connections between the visual and limbic systems (Mishkin, 1982), on the further connections of the limbic system with the diencephalon (Bachevalier, Parkinson, Aggleton, & Mishkin, 1982), and, finally, on the medial thalamic portion of the diencephalon, specifically (Aggleton & Mishkin, 1983).

The Cortico-Limbo-Diencephalic System

At this point, it would be well to step back and consider briefly how this multitude of structures and interconnections could constitute a single system critical for memory formation. The model that has been proposed (Mishkin, 1982) views the storage of the neural representations of sensory stimuli as a fundamental ingredient of memory. The storage is conceived as taking place within the higher-order sensory processing areas of the cortex whenever stimulus activation of these areas triggers a cortico-limbo-thalamo-cortical circuit. Once triggered, this circuit is presumed to serve as an automatic rehearsal or imprinting mechanism, strengthening the cortical connections whose activation triggered the circuit in the first place. The strengthened cortical network of higher-order sensory neurons may be viewed as the stored representation of the

stimulus, which, whenever reactivated through the original sensory pathway, would result in stimulus recognition. In addition, through the interconnections that this stored representation would establish with the stored representations of other stimuli and events, it could evoke them or be evoked by them through the process of associative recall.

The foregoing is consistent with numerous anatomical and neurological facts and also satisfies some important theoretical considerations. For example, with regard to the anatomy, each sensory modality appears to be served by a hierarchically organized set of cortical areas and connections that are directed outward from its primary projection area toward the anterior temporo-insular region (Turner, Mishkin, & Knapp, 1980). This region, which encompasses the highest-order processing areas for each of the sensory modalities, is reciprocally connected with the amygdala directly (Turner *et al.*, 1980) and with the hippocampus indirectly via entorhinal cortex (Van Hoesen & Pandya, 1975). The amygdala and hippocampus are connected in turn, also often reciprocally, with various medial and midline thalamic nuclei, including the nucleus anterior medialis and ventralis, the magnocellular portion of the nucleus medialis dorsalis, and the nucleus paraventricularis, parataenialis, and reuniens. (For a recent review of the relevant anatomical literature, see Mishkin & Aggleton, 1981.)

As for the neurological evidence, the two subcortical regions comprising the proposed circuit—namely, the medial temporal and medial thalamic regions—are also the two major sites of neuropathology associated with global amnesia in humans (Milner, 1959; Victor, Adams, & Collins, 1971; McEntee, Biber, Perl, & Benson, 1976). Furthermore, as with our findings in animals, the severity of the amnesia in clinical cases appears to be correlated with the total amount of conjoint damage sustained by the amygdalo-thalamic and hippocampo-thalamic portions of the system (Mishkin *et al.*, 1982). In addition, it is the evidence from the clinical cases that has dictated the proposal contained in the neural model that the stored stimulus representations are located in the cerebral cortex, that is, distal to the site of the neuropathology, since memories that the patients formed prior to their limbo-diencephalic injury or disease onset are so often spared (Milner, 1970; Cohen & Squire, 1981).

Sequential Neural Processing

Finally, with regard to the theoretical considerations that the model attempts to satisfy, all relate to the notion of a sequential neural order in perception and memory. To deal with the issue of perception first, there is strong evidence to suggest that an integrated percept depends on the sequential processing of sensory information through the several tiers of cortical areas composing each sensory system (Turner *et al.*, 1980; Ungerleider & Mishkin, 1982). By the time the activity related to the stimulus has reached the modality's final, or highest-order, processing station in the anterior temporo-insular cortex, the various perceptual constancies are most likely to have been achieved (Gross & Mishkin, 1977; Mishkin, 1979). As a result, the neural activity representing the stimulus at that station would also remain relatively constant despite wide variations in such exposure conditions as intensity, background, proximity, and position on the receptor surface, variations that would necessarily evoke markedly differing

neural activity at earlier stations of the system. Once a relatively invariant neural representation of a stimulus has been achieved, it seems plausible to assume that this is the representation that would be stored, thereby providing for constancy of stimulus recognition under widely varying perceptual conditions.

By extension of the notion of a sequential neural order to the memory process, it also seems plausible that the stored stimulus representation is the neural entity that would be linked to subsequent neural events. In this way, not only stimulus perception and recognition but associative recall as well would benefit from the various stimulus-equivalence mechanisms that were present earlier in the sensory systems. The associative connection would thereby have to be formed only once, with a single invariant representation of the stimulus, rather than repeatedly with each of its numerous possible representations that would be evoked under varying perceptual conditions at earlier sensory stations. As already indicated, the subsequent neural events with which the stored stimulus representation could become connected would likewise be stored representations, although these would be not only of other stimuli but also of places in the environment, or behavioral acts, or, finally, affective states.

This last aspect of the model brings us back to the theoretical question that initiated our line of memory research, but with a new appreciation of the role of cortico-limbic interaction in stimulus-reward association—that is, the limbic system appears to participate in this specific memory process in at least two different ways. First, the amygdala contributes equally with the hippocampus in bringing about the cortical storage of the stimulus representation, thereby allowing for stimulus recognition. And second, the amygdala contributes on its own to the attachment of affective value to the recognized stimulus. As a result, in the absence of the hippocampus alone, there is little impairment in either one of these processes, since the amygdala can mediate both of them effectively; and in the absence of the amygdala alone, there is an impairment only in appreciating the significance of the stimulus, for although the hippocampus cannot mediate that function effectively, it can support stimulus recognition. But in the absence of both of these limbic structures, that is, when both of the alternative pathways for storing stimulus representations are destroyed, there is a profound impairment in recognizing the stimulus itself and, consequently, in acquiring any mnemonic association with it.

HABITS AND A CORTICO-STRIATAL SYSTEM

Little has been said yet about the level of learning that the memory system affords a normal monkey or, conversely, the degree of learning impairment that follows destruction of this system. Although research on one-trial visual discrimination in this species can be traced back at least to the early work of Harlow (1944), the monkey's truly remarkable visual memory capacity was uncovered only recently in studies by Gaffan (1974, 1979) and Sands and Wright (1982). Two of the animal's memory skills in particular are pertinent here: One is its ability to recognize among distractor items each of a long list of objects that were presented successively just once each; and the other is its ability to remember which objects in such a list were baited and which were not, also after they were presented successively just once each. Normal monkeys can perform both of these memory feats, that is, both recognition and associative

recall, with object lists of 10 or more items, at better than 90% accuracy. Monkeys with combined amygdalo-hippocampal removals, by contrast, fail almost completely either to recall which one of a single pair of objects had been baited just a few seconds ago (Mishkin *et al.*, 1982) or even to recognize which object in a pair had been presented only a minute or two earlier (Mishkin, 1978). Clearly, the impressive memory ability of the monkey is totally eliminated by extensive limbic lesions.

Spared Learning Abilities

Yet there is abundant evidence from another source that appears to contradict this conclusion. For example, it has been known for decades that monkeys with limbic lesions can learn a difficult visual-pattern discrimination presented with repeated trials, and that they can do so at about the normal rate (Mishkin, 1954; Zola-Morgan, Squire, & Mishkin, 1982). Furthermore, although monkeys with such lesions fail completely on tests with trial-unique objects, they are clearly able to learn repeated-trial object discriminations at a nearly normal rate, even though the successive trials are separated by the same 1- to 2-minute intervals that cause difficulty in the one-trial tests of recognition and associative recall (Orbach, Milner, & Rasmussen, 1960). These results in amnesic monkeys present the same paradox of normal learning in the face of rapid forgetting that is exhibited by amnesic patients. Indeed, the paradox in the case of the amnesic monkeys appears to be even sharper, since the materials, the rules, and even the responses are all essentially the same in the tasks that are failed as they are in the ones that are mastered.

A particularly dramatic example of this paradox was uncovered in a recent series of experiments that were designed to resolve it. The most obvious explanation for the amnesic monkeys' successful learning is that, despite their rapid forgetting on one-trial memory tests, they can still retain sufficient information after each trial that even a 1- to 2-minute intertrial interval is simply too short a separation to prevent the steady accumulation of information over trial repetitions. In an attempt to demonstrate that this was indeed the case, we trained monkeys with limbic lesions on object discriminations in which the successive trials were separated by intervals that would greatly exceed their putative memory span; and to be as certain as possible that the separation would exceed their span, we chose intervals of 24 hours.

The experimental design (Malamut, Saunders, & Mishkin, 1980) was as follows: A set of 20 different pairs of easily discriminable objects was presented for concurrent learning; but these 20 different pairs were presented just once a day, on successive days, until the animals attained the criterion of 90 correct responses in five 20-trial sessions, that is, in 100 trials distributed over 5 days. Within each pair, the baited (positive) object and the unbaited (negative) object remained constant across the daily sessions, as did the serial order of the pairs. The left-right position of the objects in the pair, however, was varied pseudorandomly from day to day. When the animals reached criterion on the first set, they were next trained in the same way on a second, completely different set, and then, once again, on a third set. Given all the evidence of their rapid forgetting, the results were a total surprise. Animals with the combined amygdalo-hippocampal lesions succeeded in learning the three object discrimi-

nation sets in about 10 sessions each, a period of training exactly the same as the period required by their normal controls.

To be certain that the operated animals had not somehow compensated for their memory loss, we subsequently tested them on both a one-trial recognition test involving the principle of delayed matching-to-sample (Gaffan, 1974) and a one-trial object-reward association test involving the principle of win-stay, lose-shift (Gaffan, 1979). A variant of the recognition task will be described later, but a description of the association task is particularly pertinent here. In the latter task, a baited and an unbaited object were presented successively in a central position, with a 10-second interval between them. Ten seconds later the two objects were presented again, but this time simultaneously, with one on the left and one on the right. The animal found the reward only if it chose the previously baited object (hence, win-stay, lose-shift). The same procedure was repeated with a new pair of objects on every trial, the order of the positive and negative stimuli in the acquisition phase of the trial as well as their left-right positions in the choice phase having been determined pseudorandomly. Twenty such trials were presented each day until the animals achieved the criterion of 90 correct choices in 100 trials. The results on these tests with trial-unique objects (Malamut *et al.*, 1980; Malamut & Mishkin, 1981) confirmed our earlier findings in detail. On both measures, the animals with limbic lesions were profoundly impaired, requiring double presentation of the samples to learn the recognition task at short delays of a few seconds, falling sharply in performance at longer delays of a minute or two, and then failing completely in object-reward association learning even at the short delay.

The latter failure is particularly instructive, since the object-reward association task is nearly identical to the concurrent discrimination task with 24-hour intertrial intervals, on which the operated animals were completely unimpaired. That is, both tests utilize 20 pairs of easily discriminable objects each day; both employ reward contingencies that call for learning the response strategy of win-stay, lose-shift; and both require the animal to make its choice between two simultaneously presented stimuli. Yet if this choice must be made on the basis of a single acquisition trial, even though this trial was presented just a few seconds earlier, the operated animal, unlike the normal, fails; conversely, if the choice can be made on the basis of at least a few acquisition trials, even though these were separated from each other by 24-hour intervals, the operated animal is just as successful as the normal.

Slow versus Rapid Learning

We had initially assumed that if monkeys with limbic lesions have not only forgotten an object's reward value but have even forgotten the object itself after a minute or two, then either they should be unable to learn at all across 24-hour intervals, or if they do learn, they should be so seriously handicapped in comparison with control animals that their retardation would be obvious. Neither of these results was obtained. There appear to be two possible solutions to this remarkable puzzle. The first is that all of the products of memory that we enumerated earlier can be formed either through a rapid-learning system, which makes use of single experiences, or through a slow-learning system, which requires repetition, and that only the former type of learning is dependent on

the limbo-thalamic circuit. According to this view, the two systems are distinct, but their final products are not. Consequently, in the absence of the limbo-thalamic circuit, the remaining, or slow-learning, system can yield essentially the same memories as the other; the slow-learning system simply requires several repetitions of the experience to form the memories. In short, once animals with limbic lesions have had a few trials, as in the 24-hour concurrent-learning task, they can both recognize all the objects in the test and recall each object's reward value, just as normal animals can. There is a major difficulty with this interpretation, however, in that humans who have become profoundly amnesic as a result of medial temporal or medial diencephalic damage apparently cannot learn to remember in a cognitive sense even with many repetitions of an experience. Rather, as was indicated at the outset, the learning ability that is spared in amnesic humans appears to most investigators to be qualitatively different from the learning ability that has been lost. If so, then the final products of the two learning systems cannot be the same.

Single versus Repeated Experiences

This consideration leads to the second possible solution to our puzzle of normal discrimination learning in the face of abnormally rapid forgetting. According to this second alternative, both the learning process and its product are divisible into two qualitatively distinct forms. One is the memory process that has already been described and for which the limbo-thalamic circuit is critical; the process subsumes both recognition memory and associative recall, an example of which is object-reward association. Object-reward association is viewed as being built on recognition memory and as involving the rapid formation of an association between a recognized but affectively neutral cue object and a recognized as well as affectively potent food object. The product of this first learning process is a new piece of information regarding the reward value of a previously neutral object. The second learning process, on the other hand, is completely independent not only of the limbo-thalamic circuit but also of recognition and associative memory; it is viewed as involving instead the more gradual development of a connection between an unconditioned stimulus object and an approach response, as an automatic consequence of reinforcement by food. The product of this process is not cognitive information but a non-cognitive stimulus-response bond, that is, not a memory but a habit. Finally, what is stored in the habit-formation system is not the neural representations of such items as objects, places, acts, emotions, and the learned connections between them but simply the changing probability that a given stimulus will evoke a specific response due to the reinforcement contingencies operating at that time (Mishkin & Petri, 1984).

If the foregoing solution to our puzzle is correct, that is, if discrimination learning does involve both a memory system and a habit system, then these two systems must have properties differing in numerous ways other than in learning rate. In particular, the memory system, although it can yield discrimination mastery in a single trial, appears to provide no advantage to monkeys when they are required to remember long lists of objects over 24-hour intervals. Otherwise, the normal animals, with an intact limbic memory system, should have far surpassed the amnesic monkeys on the 24-hour concurrent-learning

task. The finding that they did not surpass them indicates, conversely, that the nonlimbic habit system is an especially powerful one, which does permit the simultaneous acquisition of a long list of stimulus-response bonds despite 24-hour intertrial intervals. The contrast implies a trade-off between short-term flexibility afforded by the memory system and long-term reliability afforded by the habit system.

Habits: A Primitive Process

Earlier we pointed out that if both habits and memories are constantly being formed by experience in normal animals, then the great debate between behaviorists and cognitivists will have finally been resolved in favor of both positions. There is one area, however, in which the behaviorist position will always remain unchallenged, and this is in its applicability across the entire phyletic scale. Even animals with the simplest nervous systems are capable of response adaptation; the acquisition of information or knowledge, by contrast, may require the evolution of a system analogous to the cortico-limbo-thalamic pathway of mammals. The supposition that the memory system is a more recent development than the habit system phylogenetically raises the related question of how these two systems compare ontogenetically.

To examine that question, we tested monkeys of different ages—3 months, 6 months, and 12 months—for both memory formation and habit formation, using our tasks of visual recognition and 24-hour concurrent learning (Bachevalier & Mishkin, 1983). The particular recognition task we chose was delayed non-matching-to-sample with trial-unique objects, since among all the one-trial memory tasks that we have tried, we found this one to be the easiest for normal animals to learn (Mishkin & Delacour, 1975). In this task, a single object of a pair is presented as the sample in a central position, and the animal displaces it for food reward. Ten seconds later, the sample and the novel object in the pair are presented simultaneously in lateral positions, and the animal finds the reward if it displaces the novel object. The same procedure is then repeated at 30-second intertrial intervals, with a new pair of objects presented on every trial. As in the tasks described earlier, the animals are trained at the rate of 20 trials a day to a criterion of 90 correct responses in 100 trials.

It seems that the task is quickly learned for at least two reasons. First, monkeys are naturally curious and so prefer to investigate the novel object. And second, the design is such that the animal is rewarded for responding to novelty on every occasion, that is, both on the sample presentation as well as on the choice test. Once the animals have mastered the principle of delayed nonmatching, thereby demonstrating their ability to recognize the sample on the basis of a single familiarization experience, their memory can be taxed further by prolongation of the delays between familiarization and test and by extension of the sample list to more than one object (Gaffan, 1974). In the latter case, all the samples in the list are first presented one at a time, and then each is paired with a novel object in a series of choice tests.

Adult monkeys that are experimentally naive can learn the delayed non-matching principle in fewer than 100 trials, and as indicated earlier, they can then perform at better than 90% accuracy on delays of up to two minutes and on lists of up to 10 objects. Despite the apparent simplicity of the test, however,

infant monkeys could not learn the basic principle until they were 4 to 5 months of age; and further, once they did learn, they could not achieve adult levels of proficiency on the performance test with longer delays and lists until they were close to two years of age. In sharp contrast, the data on the 24-hour concurrent learning task revealed that even 3-month-old infants were as proficient as fully mature animals in learning long lists of discrimination problems. These lists the infants mastered at the same rate as adults despite the 24-hour intertrial intervals.

As in the case of mature monkeys rendered amnesic by limbic lesions, the success of the normal infant monkeys in 24-hour concurrent learning demonstrates that their failure in one-trial learning cannot be attributed to any inadequacies of perception, attention, motivation, or general learning ability. The simplest interpretation is to appeal again to the distinction between habits and memories. According to this interpretation, whereas infants can readily acquire habits, they are seriously deficient in forming memories, presumably because the cortico-limbo-thalamic circuit that constitutes the memory system undergoes a relatively slow ontogenetic development.

The Cortico-Striatal System

The question to which these arguments lead, of course, is what are the neural structures that constitute the postulated habit system. Here the evidence is still relatively sparse, and so our proposal must remain tentative. Nevertheless, data from a number of sources point to the possibility that habit formation in primates and other mammals depends in large part on the second major cortico-subcortical system of the forebrain, namely, the cortico-striatal system. The striatal complex or basal ganglia is an obvious candidate from an evolutionary standpoint in that it antedates both the cerebral cortex and the limbic system in phylogenesis (MacLean, 1977). Consequently, it seems reasonable to suppose that the striatal complex precedes the others in ontogenesis, too, and there is evidence from ablation studies in infant monkeys to support this notion (Goldman & Rosvold, 1972). A second reason to look to the cortico-striatal system as a major participant in habit formation is its neuroanatomical organization. The caudate and putamen together receive a heavy and, to some extent, topographically organized input from most of the cerebral cortex, and the two striatal nuclei project in turn to the globus pallidus and its associated structures within the extrapyramidal system (Grofova, 1979; Kemp & Powell, 1970; Johnson, Rosvold, & Mishkin, 1968; Turner *et al.*, 1980; Van Hoesen, Yeterian, & Lavizzo-Morey, 1981). This system of projections therefore provides a mechanism through which cortically processed sensory inputs could become associated with motor outputs generated in the pallidum and so yield the stimulus-response bonds that constitute habits.

Whether or not the cortico-striatal system actually serves such a function is still unknown, since critical tests of the proposal have not been made. The data that are available, however, are clearly consistent with the proposal. For example, visual-pattern-discrimination habits, which are unaffected by limbic lesions, are markedly affected by damage along the cortico-striatal pathway. Thus, pattern-discrimination learning and retention can be impaired by lesions either of the inferior temporal cortex (Mishkin, 1954) or of some of the striatal

regions to which this cortex projects, including the tail of the caudate nucleus (Divac, Rosvold, & Szwarcbart, 1967) and the ventral portion of the putamen (Buerger, Gross, & Rocha-Miranda, 1974). Furthermore, the same impairment can be reproduced by transections of the white matter of the temporal stem that presumably interrupt this cortico-striatal pathway (Horel, 1978; Zola-Morgan *et al.*, 1982).

The role of sensory inputs to the striatum has not received much emphasis before. As a result, the proposal that the striatum serves as an essential link in the formation of stimulus-response connections may seem highly improbable. Yet recent results obtained with the 2-deoxyglucose technique (Macko *et al.*, 1982) have revealed that, at least in the visual modality, sensory input is surprisingly effective in activating widespread portions of the striatum. This new finding, together with the developmental, connectional, and behavioral evidence just cited, indicate that the hypothesis of a cortico-striatal habit system existing alongside a cortico-limbic memory system may be sufficiently plausible to merit direct testing. If the hypothesis should hold up, the intriguing new questions will be how the learning process is actually shared by the two systems (Mishkin & Petri, 1984) and how the two systems might both cooperate and conflict.

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