to criterion under a 5-hour delay procedure. These animals were subsequently tested according to a within-subject design in four different novel environments under the following conditions: (i) no-treatment, (ii) saline vehicle injection, (iii) naloxone (2 mg/kg) administered at the beginning of the 5-hour delay immediately after the first four choices on the maze; and (iv) naloxone (2 mg/kg) delayed for 2 hours after the initial four choices. The assignment of animals to treatments in each novel environment (a room) was approximately counterbalanced.

As there was no statistically significant difference between the no-treatment and saline-treatment conditions (Table 2), for each animal, mean trials to criterion and error scores were calculated. These mean control values were used in subsequent statistical analyses. As in the previous experiments, naloxone administered immediately after the first four choices significantly enhanced performance, as reflected in both trials and errors to criterion (*14*). The data from the naloxone-delay condition do not, however, differ significantly from either the control treatments or the naloxone treatment at no delay. These results indicate that naloxone does not produce an effect comparable to that of the no-delay condition when administration is delayed for 2 hours after training. As in the previous experiments, rotation of the maze on a test conducted for each animal after criterion performance indicated that performance was based on extra-maze cues provided in each environment (*13*).

The results indicate that when performance on a spatial learning task is less than optimal, administering an opiate antagonist after training can enhance memory. Thus, the memory-enhancing effect of opiate antagonists seems not to be restricted to memories acquired through aversive training or associated with noxious events. This view of a more general role for opioid peptides in memory is congruent with preliminary clinical studies that have recently indicated that naloxone improves memory functions in patients with Alzheimer's disease (*15*). Further research on the role of opioid peptides in memory may, therefore, have important implications for understanding the biological basis of both normal memory and its disorders.

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References and Notes

1. W. P. Russell and P. W. Nathan, *Brain* **69**, 280 (1946).
2. C. P. Duncan, *J. Comp. Physiol. Psychol.* **42**, 32 (1949); J. L. McGaugh, *Science* **153**, 1351 (1966).
3. I. Izquierdo *et al.*, in *Endogenous Peptides and Learning and Memory Processes*, J. L. Martinez, Jr., *et al.*, Eds. (Academic Press, New York, 1981), pp. 269–290; R. B. Messing *et al.*, *ibid.*, pp. 431–443.
4. C. Castellano, *Psychopharmacologia* **42**, 235 (1975); I. Izquierdo, *Psychopharmacology (Berlin)* **66**, 199 (1979); ——— and R. D. Diaz, *Neurosci. Lett.* **22**, 189 (1981); J. L. Martinez, Jr., and H. Rigter, *ibid.* **19**, 197 (1980).
5. M. Gallagher and B. S. Kapp, *Life Sci.* **23**, 1973 (1978); R. B. Messing *et al.*, *Behav. Neural Biol.* **27**, 266 (1979); M. Gallagher, *ibid.* **35**, 375 (1982); I. Izquierdo and M. Graudenz, *Psychopharmacology (Berlin)* **67**, 265 (1980).
6. J. Rossier, E. D. French, C. Rivier, N. Ling, R. Guillemin, F. Bloom, *Nature (London)* **270**, 618 (1977); H. Akil, S. J. Watson, J. D. Barchas, C. H. Li, *Life Sci.* **24**, 1659 (1979); I. Izquierdo *et al.*, *Psychopharmacology (Berlin)* **70**, 173 (1980).
7. M. Gallagher and B. S. Kapp, in *Endogenous Peptides and Learning and Memory Processes*, J. L. Martinez, Jr., *et al.*, Eds. (Academic Press, New York, 1981), pp. 445–461.
8. D. S. Olton and R. J. Samuelson, *J. Exp. Psychol.: Animal Behav. Process* **2**, 97 (1976).
9. J. O'Keefe and D. H. Conway, *Exp. Brain Res.* **31**, 573 (1978); S. Suzuki, G. Augerinos, A. H. Black, *Learn. Motiv.* **11**, 1 (1980).
10. Rats weighed approximately 250 g at the beginning of each experiment. All animals were maintained at 80 percent of free-feeding body weight by being fed a measured amount of laboratory chow each day after behavioral testing. Rats were housed on a 12-hour light-dark cycle (lights on between 0700 and 1900), and water was available at all times.
11. On each day of training and testing, animals were run on the maze until they had visited each arm once. Since an animal was free to re-enter arms already visited, it could accumulate many errors on a trial. Thus, because of a relatively large number of errors on early test trials, there can be more errors than trials to criterion.
12. Multivariate analysis of variance applied to trials and errors to criterion indicated a significant overall difference between treatment conditions [$F(2, 6) = 29.43$, $P < 0.001$]. Univariate analysis of variance revealed that naloxone treatment resulted in significantly fewer trials to criterion, [$F(1, 7) = 51.87$, $P < 0.0002$] and errors to criterion [$F(1, 7) = 21.44$, $P < 0.005$]. Multivariate analysis of variance revealed a significant overall difference between the saline and diprenorphine treatment conditions [$F(2, 8) = 7.36$, $P < 0.015$]. Diprenorphine treatment significantly reduced trials to criterion [$F(1, 9) = 15.06$, $P < 0.005$] and errors to criterion [$F(1, 9) = 14.49$, $P < 0.005$].
13. After four choices had been made, the maze was rotated 180° during the delay interval. The remaining food rewards were placed on arms that conformed to the correct location in the testing room. After maze rotation, the choice of arms during retention testing exhibited reliable accuracy based on food location in the extra maze environment.
14. Multivariate analysis of variance conducted on the control values and the combined data from the naloxone and naloxone-delay treatments revealed a significant treatment effect [$F(4, 5) = 11.44$, $P < 0.006$]. Subsequent specific statistical analyses indicated that, compared to the control conditions, naloxone administration immediately after the first four choices on the maze significantly decreased trials and errors to criterion from control values [$F(1, 9) = 22.96$, $P < 0.001$ and $F(1, 9) = 46.96$, $P < 0.0001$, respectively]; however, no significant differences were obtained between the control and the naloxone-delay condition.
15. B. Reisberg *et al.*, *Psychopharmacol. Bull.* **19**, 45 (1983); *N. Engl. J. Med.* **308**, 721 (1983).
16. Supported by NIMH grant MH35554, an Institutional Development Foundation Award, and NIMH research scientist development award K02 MH00406 to M.G. We thank Endo Pharmaceutical for their gift of naloxone and NIDA Research Technology Branch for providing diprenorphine.

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Optimization: A Result or a Mechanism?

Mazur reported "evidence against optimization as the basic mechanism underlying choice behavior" (*1*, p. 823). But in so doing, he missed an important point: optimality accounts are explanations in terms of final causes, not descriptions of a mechanism. It is a fundamental error to identify optimization theory with any particular mechanism by which optimal results are achieved (*2*, *3*). Consequently, an experiment ruling out a particular optimizing mechanism has little bearing on the general usefulness of optimality accounts.

Mazur described a possible optimizing mechanism that involves variation (properties unspecified) in the distribution of responses between two choices and comparison (across unspecified time periods) of the overall payoff rates so obtained. His results rule out such a process. However, many other processes, sufficient to yield maximizing under the usual conditions that yield matching of response and reinforcement ratios, are not ruled out. For example, if the animal always chooses the alternative with highest probability of payoff (hill-climbing), where the probabilities are based on its past history of choices, results similar to Mazur's are to be expected (*4*). Hence his experiment shows only that overall payoff rate (averaged over periods of an hour or so) is not used by pigeons as a guide to choice proportion. Few optimality theorists, however, assume either that overall reward rate is directly assessed by animals or that pigeons are capable of selecting a particular choice proportion, as opposed to simply making one or the other choice.

Optimality theory in general is not testable, since any experimental result can be expressed as the optimal solution to some problem; what is testable is constrained optimization, the idea that animals behave optimally subject to specified constraints (*5*). Mazur wrote, "One might argue that the pigeons in this study failed to optimize because they did not 'understand' the complex contingencies in effect. An animal's 'understanding' is not relevant to optimization theory, however" (*1*, p. 825). Actually, though, the animal's "understanding" defines the constraints within which it can be expected to optimize. For example, optimal behavior on variable-interval schedules with a finite sequence of interreinforcement intervals (the usual situation) is to remember the sequence of times and respond only when reinforcement is available. No theorist takes this position, because all recognize limitations on pigeon memory.

Similar, but unrecognized, constraints seem to hold in Mazur's experiment. It is not clear that pigeons can detect the difference between independent schedules and the interdependent schedules that Mazur used; nor do we know whether pigeons are capable of learning to adjust their proportion of responses directly, rather than arriving at a given response proportion as the indirect outcome of a sequence of particular choices. Yet both of these capacities must be assumed if Mazur's result is to refute an optimality analysis. One function of optimality analysis is to allow better understanding of the constraints within which optimization occurs; these constraints, in turn, provide clues to the processes that allow animals to behave optimally under normal conditions (*6*).

We must also disagree with Mazur's conclusion that "The results indicate that animals will approximate matching behavior even at the expense of a substantial loss of reinforcement" (*1*, p. 825), which implies that adherence to matching is the process that determines choice. This cannot be correct. Pigeons do not always match—matching depends both on the pigeon and on the feedback functions for the two schedules, and the relative importance of these two components has not been fully determined (*5*); attempts to demonstrate matching as a dynamic determiner of choice have failed (*7*). The matching relation by itself does not uniquely determine responding, since it is a relation between two derived measures—the ratio of reinforcements obtained (not scheduled) and the ratio of responses made. Hence matching, when it occurs, is something that requires an explanation at the level of individual choices, not itself a potential explanation for individual choice.

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References and Notes

1. J. E. Mazur, *Science* **214**, 823 (1981).
2. J. R. Krebs and N. B. Davies, *An Introduction to Behavioural Ecology* (Sinauer, Sunderland, Mass., 1978); D. McFarland and A. Houston, *Quantitative Ethology: The State-Space Approach* (Pitman, London, 1981).
3. J. E. R. Staddon, Ed., *Limits to Action: The Allocation of Individual Behavior* (Academic Press, New York, 1980).
4. J. M. Hinson and J. E. R. Staddon, *J. Exp. Anal. Behav.* **39**, 25 (1983); C. P. Shimp, *ibid.* **9**, 443 (1966); J. E. R. Staddon, J. M. Hinson, R. Kram, *ibid.* **35**, 397 (1981); A. Silberberg, B. Hamilton, J. M. Ziriax, J. Casey, *J. Exp. Psychol.: Anim. Behav. Proc.* **4**, 368 (1978).
5. J. Maynard Smith, *Ann. Rev. Ecol. Syst.* **9**, 31 (1978).
6. J. E. R. Staddon, in (*3*).
7. R. J. Herrnstein and W. Vaughan, in (*3*).
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Staddon and Hinson agree with me that optimization is not a mechanism of choice behavior, but imply that no one ever said that it was. Rachlin and his colleagues (*1, 2*) have suggested that matching behavior is a product of a supposedly more fundamental process of reinforcement maximization. That is, Rachlin has maintained that when matching behavior occurs, it is because this manner of responding maximizes the rate of reinforcement. If Rachlin and his colleagues mean only that optimization is a more fundamental or more universal end result than matching behavior, we are all agreed that optimization is not a mechanism of choice behavior.

In that case, my experiment can be viewed as a test of whether optimization or matching is a more fundamental end result. The experiment favored matching, but Staddon and Hinson do not find this result surprising. They say that most optimization theorists believe neither (i) that animals are sensitive to the overall rate of reinforcement, nor (ii) that animals are "capable of selecting a particular choice proportion, as opposed to simply making one or the other choice." However, Rachlin *et al.* made these two assumptions explicitly: "With two alternatives, behavior A and behavior B, each point represents a certain amount of time spent performing behavior A and a certain amount performing behavior B. . . . Maximization theory assumes that animals always choose the available point with the highest numerical value" (*2*, p. 371). To avoid further confusion, this version of optimization theory might be called "literal optimization," for it predicts that animals will choose the response proportion that maximizes value. It was this version of optimization theory my experiment tested and failed to support.

On the other hand, Staddon and Hinson endorse "constrained optimization." This theory states that if we take into account all the constraints imposed upon a creature (including any limitations of memory and decision-making abilities, any inflexibility in response selection, and so forth), the creature will always do the best it can. I agree that my experiment did not test this theory of constrained optimization, but I suggest that this theory strains both the usual meaning and the testability of the term "optimization." Consider a frog that is starving to death in a room full of dead flies (since the frog eats only flies it catches in midair). According to this theory, the frog is behaving optimally, because it is doing the best that it can, given the inflexibility of its food-seeking habits. I think few people other than optimization theorists would call the frog's behavior optimal. Of course, optimization theorists can ignore common usage and define "optimization" any way they wish. But since any outcome can be defined as optimal after the fact (as Staddon and Hinson acknowledge), the concept of constrained optimization becomes a tautology, or more accurately a pretheoretical assumption not subject to experimental test.

Finally, I agree that just as optimization is not a mechanism of choice behavior, neither is matching. Although it would be logically possible for matching to be such a mechanism, empirical results (*3*) argue against this possibility. The point of my experiment was that whatever the dynamic mechanisms of choice behavior may be, they must be ones that can sometimes produce matching as an end result even when this behavior is far from optimal.

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References

1. H. Rachlin, *J. Exp. Anal. Behav.* **30**, 345 (1978); ——— and B. Burkhard, *Psychol. Rev.* **85**, 22 (1978); H. Rachlin, L. Green, J. H. Kagel, R. C. Battalio, in *The Psychology of Learning and Motivation*, G. H. Bower, Ed. (Academic Press, New York, 1976), vol. 10, pp. 129–154.
2. H. Rachlin, R. Battalio, J. Kagel, L. Green, *Behav. Brain Sci.* **4**, 371 (1981).
3. R. J. Herrnstein and W. Vaughan, in *Limits to Action: The Allocation of Individual Behavior*, J. E. R. Staddon, Ed. (Academic Press, New York, 1980).

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