Chapter **7**

Acquired Drives and Rewards

In previous chapters, we have referred to the impact of learning on how, and whether, we express even basic biological motives. This chapter will focus on cases in which the motives themselves are learned.

These cases are clearly of great importance. Biologically based drives and incentives obviously account for only a minute fraction of human and animal behavior. We act, and act persistently and vigorously, when we are not hungry, thirsty, angry, or sexually aroused, and when no delicious food or attractive partner is in prospect. If biological motives fall far short of explaining here-and-now actions, then non-biological motives must fill the gap. Learned motives are prominent among these; and relatively simple learned motives, of the kind we consider here, might provide building blocks for more complex ones.

CONDITIONING

The study of **conditioning** has been of particular interest to psychological theorists, for two reasons. First, it was an early and influential theory about how learning in general might take place, so it was natural to apply it in this context. Second, it is a versatile idea that can be applied in many, many contexts. Here are examples.

Pavlov's *classical conditioning* experiments (pp. 46–47) had shown that salivation—a part of the body's preparation for food—could come to be elicited by an *arbitrary* external stimulus, the bell. Now salivation is a response. But so are feeding, and the cessation of feeding. Can these responses be conditioned?

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Conditioned Satiety

Consider what happens as a rat eats. It tastes, smells, chews, and swallows the food. Soon satiety sets in, and feeding is inhibited and shuts down. We have a sequence that resembles a conditioning experiment. Certain stimuli are present—the bell for Pavlov's dogs, taste and other oral sensations for the rat. A response is elicited—salivation for the dog, cessation of eating for the rat. Can cessation of eating be conditioned?

Quite a few experiments now suggest that it can.[1] In one, a hungry rat ate a 50 percent carbohydrate solution on some test days, a 5 percent carbohydrate solution on other days. Each solution was given an arbitrary odor as a label; call these Odor A for the high concentration and Odor B for the low one.

The concentrated solution satiates the rat faster than a dilute one does, so the rat will drink more of the dilute solution. Now if cessation of eating can be conditioned to an arbitrary stimulus, then the odor that is *paired with* the concentrated solution—Odor A—should come to elicit a more rapid cessation of feeding. And it does. To test this, we let the rat have an intermediate concentration of carbohydrate (30 percent). But it is labeled with either Odor A or Odor B. Sure enough, rats take in less of the *same* solution if it smells like the more concentrated one. The smell *associated with* rapid satiety can now *elicit* rapid satiety. An analogous experiment in humans gave similar results.[2]

This conditioning mechanism may be quite important in the calibration of ingestion, especially in humans. As we have seen (pp. 80–81), humans are not very sensitive to the caloric value of the food they eat as they eat it. Adjustment of energy intake to energy output in humans may require that we *learn* how much to eat.

Conditioned Feeding

Finally, what about the act of ingestion itself? Can it be brought under control of an external stimulus as a conditioned response? Harvey Weingarten showed that it can.

His experiment was conducted in two phases. First, his rats received six brief meals a day; the presentation of food was accompanied by an external stimulus, a light and a buzzer. Those brief meals were all the food the rats received, so they quickly learned to come running to the food cup and eat when the stimulus occurred.

In the second phase, food was available all the time; so the rats were no longer restricted as to when they could eat. Once a day on some days, the conditioned stimulus came on. And when it did, the rats would promptly rush to the food cup and begin to eat. They would eat as much

[1]See for example Le Magnen, 1971, 1981; Booth, 1972; Booth and Davis, 1973.
[2]Booth, 1977.

as 20 percent of their total daily intake in that one meal—much more than rats normally take in a single meal.[3]

We have here a simplified model of how *arbitrary external stimuli* can come to trigger ingestion as a result of learning. An obvious example in the human case is control of feeding by the clock. In this society, simple *time of day* influences our meal-taking behavior at least as much as anything else.[4]

ACQUIRED DRIVES AND REWARDS

These studies of conditioning showed its effects on the control of a behavior—feeding—that we already have. Now let us turn to cases in which the motives themselves are learned. We will look at acquired drives and acquired rewards.

As an example of an **acquired drive**, suppose you and I are afraid of hornets, because we learned about them the hard way. Then the sight of a hornet will evoke an unpleasant state that motivates action. We might call it *fear.* It can motivate an arbitrary response such as opening a car window to let the hornet out. It is true that we also have a goal or incentive—relief from fear, or avoidance of pain. But we didn't have to learn to seek freedom from fear and pain. What we learned is that creatures that look like *that* are to be feared, and so *that* stimulus evokes the drive of fear.

As an example of an **acquired reward**, consider a person drinking coffee. Now the odds are that that person did not like coffee at all when he first tasted it. Put a bit of coffee on an infant's tongue, and she will make a nasty face and reject it. It is a curious fact that *most* people, in cultures all over the world, learn to seek and enjoy commodities that at first are disliked.

Access to a cup of coffee, in this society, supports arbitrary responses that make it available: We make our way to the coffee shop, plunk down money, and so on. Yet there may be no internal state that motivates this action. We may not be thirsty at all; or if we are, we didn't have to learn to be. We did have to learn to seek coffee.

In summary, an acquired drive is an internal impetus to action—a push from within—that we have learned. An acquired reward is something we seek—a pull from without—because we have learned to seek it. This chapter will explore both of these cases.

AVOIDANCE LEARNING

Pain is a powerful motivator. So is the *threat* of pain; we may go to great lengths to escape that threat, as in taking any action available to get that hornet out of here.

[3]Weingarten, 1983.
[4]Rodin, 1981.

How do threats become motivators? Here we will follow our usual tactic of beginning with simpler cases. Then we will see how the resulting ideas may bear on more complex problems.

Avoidance Conditioning

Consider this experiment. A rat is placed in a long narrow box, divided in half by a partition. The floor of the box is a grid of steel rods that can be electrified to provide a painful shock (Figure 7-1A).

After a few seconds, a buzzer comes on. Now the rules of the experiment are this: The rat has, say, 5 seconds to cross the barrier over to the

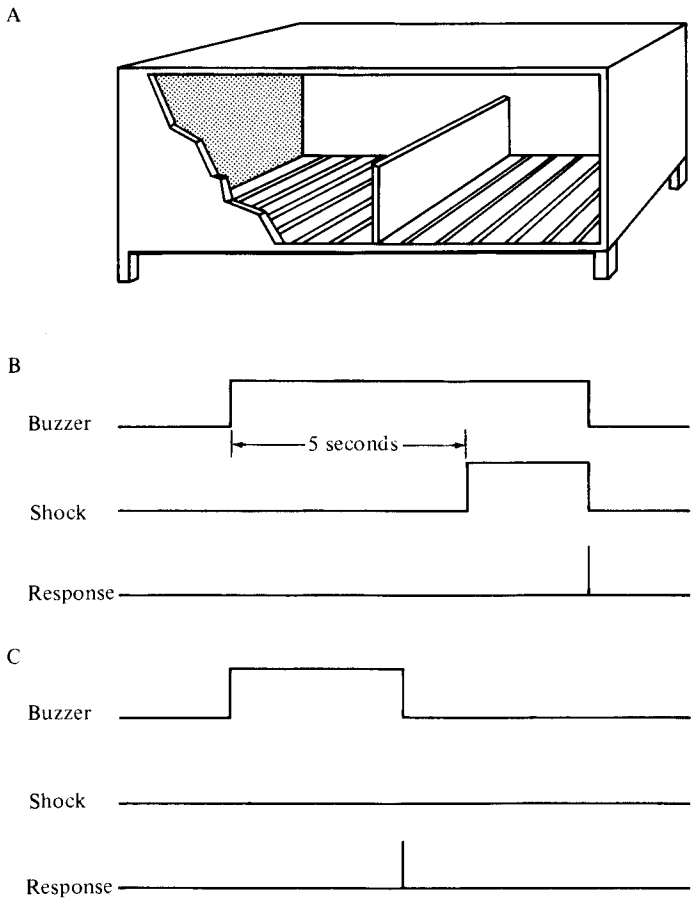

Figure 7-1.
(A) A shuttle box. The floor is made of metal rods that can be electrified. (B) An escape trial. The response terminates the buzzer and the shock. (C) An avoidance trial. A response made before the onset of shock turns off the buzzer and prevents the shock from occurring.

other side of the box. If the rat does this, the buzzer goes off and all is well; nothing else happens. But if the rat fails to cross in time, then shock comes on and continues until crossing does occur.

In either case, there will be an interval of time after the rat makes the crossing-over response; then the buzzer comes on again, and the rat must again cross the partition. Over a series of such trials, therefore, the rat must shuttle back and forth from one side of the box to the other, and so this apparatus is called a **shuttle-box.**

What happens in such an experiment? On the first few trials, the rat is likely to make no crossing response when the buzzer comes on. The shock comes on five seconds later, causing the rat to run and scramble and, before long, cross over the barrier. Crossing turns the shock off, and we say that the rat has *escaped* the painful shock (Figure 7-1B).

Over successive trials, we usually will see a progressively more prompt escape response. That is, the rat will freeze or crouch when the buzzer comes on, but after the shock begins the rat will quickly make the crossing-over response that turns it off. But eventually most rats will begin to make *avoidance* responses, crossing during the warning interval between buzzer onset and shock onset. That prevents shock from occurring (Figure 7-1C). Eventually, a well-trained animal may go on trial after trial, day after day, crossing over on signal and never receiving a shock.

Now a rat's escape response—running to turn off shock after it has come on—poses no particular problem and involves no acquired drive. We can see it as maintained by the law of effect: The shock is painful, running turns it off, and so running is rewarded and strengthened. Running, as we say, is *reinforced* by escape from pain. The problem comes when we ask what reinforces the *avoidance* response, which prevents the shock from coming on at all. Does the absence of shock keep responding going? But how can the *absence* of an event be a reinforcer? How can a non-event strengthen and maintain behavior?

One answer is the cognitive answer.[5] The rat expects shock if it does not make the response, and expects not to get shocked if it does make it. It prefers no shock to shock, understandably enough. And it makes the response that has the most preferred expected outcome.

Behaviorists will have none of that. We simply do not know, they point out, what if anything a rat expects. We know the response that occurs, so let us speak so that we know what we're talking about. Can we show that avoidance behavior is really a response to a stimulus, not to a non-stimulus, after all?

We can try. It is an instructive exercise in what early behavioristic theorizing was like. And it will show us the relations and the contrasts between two points of view—the behaviorist and the cognitive ones. So let us work through it.

[5]See for example Seligman and Johnston, 1973.

The Two-Factor

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The Two-Factor Theory of Avoidance Conditioning

The theory we now consider says that avoidance learning involves two stages, or **factors**. First, the rat acquires a conditioned response—fear—to the buzzer or other warning stimulus. Second, the avoidance response reduces the fear. What reinforces avoidance responding is not a nonevent—the absence of shock. It is an event—fear reduction—that is the reinforcer. Let us look more closely.*

Consider what happens on early trials. The buzzer comes on and, 5 seconds later, the painful shock. Now the shock will produce many responses: muscles tense, heart rate increases, breathing rate increases, the adrenal gland pours forth secretions, and so on. This happens in the presence of the buzzer, and so we have the situation under which *classical conditioning* occurs. These stress responses begin to occur in response to the buzzer, just as, in Pavlov's dogs, salivation occurred in response to the bell (Figure 7-2A). This is the first of the two factors in two-factor theory.

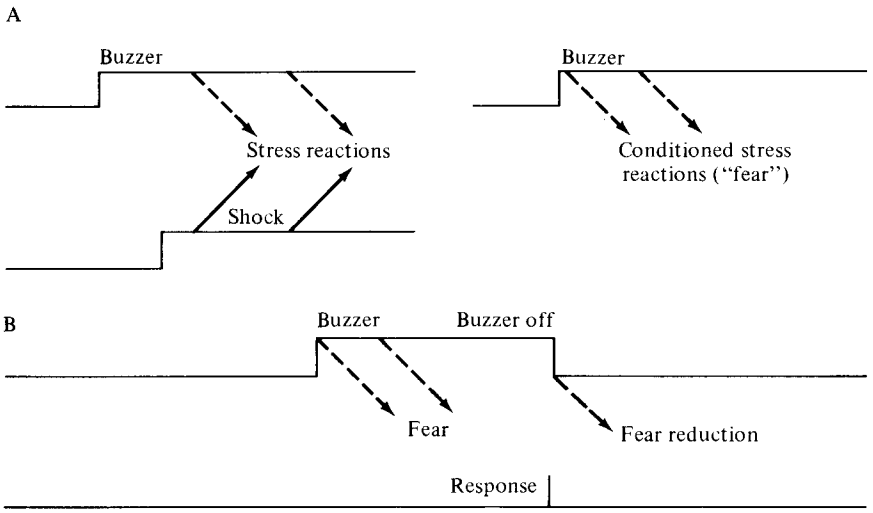


Figure 7-2.
The two-factor theory of avoidance learning. (A) Early in training, stress reactions are conditioned (dashed lines) to the buzzer or other warning signal. (B) Once that conditioning has taken place, any response that turns off the warning signal will be reinforced by reduction of fear.

Now let us assume that these responses, of muscle tension and the like, in turn produce stimuli and that these stimuli are unpleasant. We might think of them as comprising an emotional state, *fear. Fear, in this analysis, is the acquired drive.* It is not a mental state but an internal stimulus

*For fuller discussion see Mowrer, 1950.

situation. It is the set of internal stimuli produced by the conditioned stress responses. It is also unpleasant or, as we say, *aversive,* just as the shock itself is. Therefore, any response that terminates fear will be reinforced and strengthened (the law of effect). Well, if the rat crosses over before shock comes on, that turns the buzzer off; the stimulus for fear is taken away, and so the fear state ends. That is the reinforcement for the avoidance response (Figure 7-2B). The avoidance response is strengthened by reinforcement—fear reduction—and that is the second of the two factors.

Putting these two factors together, we have our familiar negative-feedback loop. A stimulus (the buzzer) produces fear, which in turn calls forth an action (running) that removes the fear by removing the stimulus that causes it. Fear reduction also reinforces the response, making it more likely to occur the next time.

The analysis is an ingenious one. It treats the avoidance response, not as a foresightful response to something that hasn't happened yet, but as a response to something that *has* happened—fear produced by the buzzer. And it treats the avoidance response as reinforced, not by something that doesn't happen, but by something that does—reduction of fear. No expectancies, forethoughts, or cognitions are required.

Two-Factor Theory and Human Fears

Despite the problems with two-factor theory (see below), it has been an enormously influential one—and not just for rats or dogs in shuttle-boxes. It has been extended in fascinating ways to explain some human behavior.

Many ineffective and even irrational forms of human behavior may be seen as avoidance responses. They may be motivated by fear, and maintained by fear reduction; and the fear itself may be traceable to certain experiences that created them, just as shock creates fear of the buzzer in the rat. And if this is so, what we learn about the principles of avoidance conditioning may suggest ways of dealing with troublesome fears.

Symptoms as Avoidance Behavior: Obsessions and Compulsions

As an example, perhaps the behaviors known as *compulsions* can be seen as habitual avoidance responses, reinforced by anxiety reduction.* Freud thought so.[6] So did many behaviorist writers.[7]

Compulsions are repeated, stereotyped, ritualized actions. The patient feels *compelled* to perform them, and unable to control the urge to do so. They often go along with **obsessions**, which we may think of as *compul-*

*Many writers would distinguish *fear* from *anxiety,* treating the first as realistic and evoked by some definite stimulus, the second as diffuse and less definite as to its object. The distinction is not critical for the argument here, however; for discussion of it in this context, see Schwartz, 1984.

[6]Freud, 1917.

[7]Dollard and Miller, 1950.

sive thoughts. These are thoughts that persist in coming into consciousness; often they are frightening or abhorrent, but the patient finds them difficult or impossible to dismiss.

The effect of these compulsive rituals is not trivial. They can make life an unending misery for patient and family. Listen:

> One of our patients . . . feared contamination by germs. As a result she engaged in prolonged and intensive washing and cleaning rituals. Her young child was restrained in one room of the four-bedroom house, as it was the only one that she could keep satisfactorily free from germs. Three of the rooms were kept permanently locked because she was incapable of ensuring that they were sufficiently sterile. . . . The patient's fear of contamination made her virtually housebound, and her child was not permitted to leave the house except on a very few essential occasions. On returning from work each day, her husband was obliged to go through a series of decontaminating-cleaning rituals. Their sexual relationship, never satisfactory, had been abandoned because of her fears of contamination. Their social life was damaged beyond repair, and they had lost all but one of their friends; even the members of their families could neither visit them nor be visited by them.[8]

All that cost, all that pain, because of intense and irrational fear of germs. The behavior seems as bizarre as—well, as the behavior of a rat compulsively shuttling back and forth in a two-compartment box. Of course, if we know the rat's conditioning history, its behavior makes sense. So might the patient's, if we knew enough about *her* conditioning history. So the behaviorist would say.

However, the suggestion that these rituals are avoidance responses came originally not from behaviorists, but from Freud! The obsessive thoughts and the actions that come from them are seen as defenses against really terrifying thoughts. Abhorrent wishes threaten to break into consciousness; this causes intense, though unconscious, fear. The patient unconsciously transforms his thoughts into ones that symbolize the forbidden wish but are less threatening. These are the obsessive thoughts. And so fear, though it may persist, is still reduced; it is much less intense than it would otherwise be.

There is evidence, from both the clinic and the laboratory, that fear or anxiety does underly these symptoms.[9] If a patient is prevented from performing the compulsive act, he may experience overwhelming anxiety. Moreover, some patients report that they do experience anxiety before performing the compulsive act, and lose it when the action is performed. It may be just this that makes obsessions and compulsions difficult to treat. They work! They reduce anxiety, just as the rat's avoidance response turns the buzzer off and reduces fear.

[8]Rachman and Hodgson, 1980, pp. 58–59.
[9]Rosenhan and Seligman, 1984.

The Treatment of Fear: Response Prevention in Behavior Therapy

As the theory of avoidance conditioning may help us understand where some problematic behaviors come from, so it may help us do something about them.

The class of treatment methods that incorporate behaviorist theories is known as **behavior therapy**.[10] It draws directly upon the laboratory analysis of behavior, especially learned behavior.

Behavior therapists believe that compulsive actions are avoidance responses. Think again about the well-trained rat, shuttling back and forth in the box. Now suppose that the shock apparatus has been turned off. Then crossing the barrier when the buzzer comes on is no longer necessary. The behavior no longer serves any purpose. But the rat, as long as it goes on responding to the buzzer, cannot learn that.

One way to bring the rat to reality is to *prevent the avoidance response from occurring.* Place a barrier between the sides of the boxes. Let the buzzer come on; and the animal, unable to make an avoidance response, must discover that shock no longer threatens. If that happens several times, the fear response will undergo extinction. Then, when the barrier is removed again, the animal should sit placidly when the buzzer comes on. And that is what happens.[11]

Exactly the same logic has been used in the clinic, where it is called **response prevention**. Let a patient, who fears contamination by dirt, touch a contaminated object, and prevent her from performing any cleaning or other rituals that otherwise would compulsively ensue. When this is done a number of times, and when nothing bad happens, the fear should weaken—or, as we say, it should undergo extinction—and the compulsive behavior it motivates should stop. This direct approach has been used to treat compulsions, with some success.[12] Some patients are unable or unwilling to tolerate the stress of such treatment; but when the patient can do it, it often helps.

THEORETICAL ISSUES IN AVOIDANCE CONDITIONING

Although the two-factor theory has been influential in both human and animal research, there are some problems with it.[13] It may tell part of the story of what happens in avoidance conditioning, but it does not tell it all.

Difficulties with the Two-Factor Theory

Everything in two-factor theory hinges on the idea that a **warning stimulus**, such as the buzzer, produces the fear that motivates the avoid-

[10]For an excellent introduction see Rimm and Masters, 1979.
[11]Solomon, Kamin, and Wynne, 1953.
[12]Reviewed by Rachman and Hodgson, 1980.
[13]Discussed by Rescorla and Solomon, 1967; Schwartz, 1984.

ance response. Running across the barrier turns the buzzer off; offset of the buzzer takes the fear away, and that reinforces avoidance behavior. So the theory goes.

Avoidance Conditioning without a Stimulus

In fact, however, one can see avoidance behavior maintained in a stimulus situation that *does not change when the response occurs.*[14] In one such method, the rat is in a box, with lever-pressing the avoidance response. At regular intervals—every 10 seconds, let us say—automatic apparatus delivers a brief pulse of shock through the box floor, unless the animal makes an avoidance response. The rat can avoid these short shocks by pressing the lever. Every lever-press *postpones* the delivery of the next shock for a period of time—say, 10 seconds. If the rat presses, and then waits a full 10 seconds, then shock will occur after 10 seconds. But if the rat presses the bar at 9.9 seconds, then that shock will not occur and the next shock will not come due for a full 10 seconds more; and it too can be postponed if the rat presses again. Thus, if the rat presses the bar at least once every 10 seconds, shock will never occur.

Failures to learn this difficult task are not uncommon, but some rats do learn it, and work away at the lever *even though no stimulus change follows the lever-press.* There is no buzzer to go silent and take the fear away. How can *fear,* as a response to a warning stimulus, be reduced if there is no warning stimulus? And if fear is not reduced, what reinforces lever-pressing?

Is Fear Maintained?

An even worse problem for two-factor theory is this: If we measure the effects of fear in other ways, fear simply does not seem to have the properties the theory requires.

We can measure fear of a buzzer, for example, by seeing how much it suppresses some other, ongoing activity (p. 194). When we do so, we find that early in avoidance training, the buzzer does indeed provoke fear. Later, however, a well-trained rat may show little evidence of fear—but it continues to make the avoidance response when the buzzer sounds. It is as if the well-educated rat knows just what to do about buzzers, and does it placidly, without fear, when occasion arises.

Cognitive and Behaviorist Theories

That, of course, is the cognitivist view of avoidance behavior. The rat expects shock if it does not respond, no shock if it does respond; it prefers no shock to shock, and so it responds.[15]

Behaviorists still find this account unacceptable. We do not know what

[14]Sidman, 1953; see also Herrnstein and Hineline, 1966.
[15]Seligman and Johnston, 1973.

a rat expects or prefers; and if we did, the expectancies and preferences would just have to be explained in turn.

The modern behaviorist—the reinforcement theorist—would not speculate about what the rat believes or prefers. She would seek simply to relate the behavior to *environmental* conditions. If absence of shock, or reduction in shock frequency, can be shown to be important in maintaining avoidance behavior—then so be it. That is the reinforcer for avoidance behavior (see Chapter 8).[16]

We will not pursue the argument here.[17] There are problems with both these theories; they are too simple. In particular, neither one addresses the issue of biological factors in avoidance, which we take up in the next section. But now, rather than join the behaviorist-cognitivist debate, let us see if we can draw some useful ideas from *both* points of view.

An Attempt at Synthesis: Cognitive-Behavioral Theory

Out of the arguments between behaviorists and cognitive theorists have come some attempts to bridge the gap between them. The point of view called **cognitive-behavioral theory** is such an attempt. It tries to draw ideas from both, and in doing so, it asks each theory to give a bit. It asks cognitive theorists to admit that thoughts, wishes, and the like are *responses.* And it assumes, as a working hypothesis, that they follow the same laws as any other responses—in particular, the laws of conditioning. It asks behaviorists to admit, for their part, that such thoughts and wishes can be *causes* of behavior. This statesmanlike compromise seems to be gaining popularity, especially among clinicians. Let us see what it can do.

Desensitization Therapy

One product of this union is a behavior therapy technique. It is the method of **desensitization therapy**, often used to combat troublesome fears. Briefly, the patient is given practice at *imagining* feared situations —examinations perhaps, or spiders. The person forms a clear and vivid mental image of the feared situation, *while maintaining a state of deep-muscle relaxation.* Extensive practice at this is given. The idea is this: If one can establish relaxation as a conditioned response to the situation, then the relaxation will inhibit fear and the fear will be reduced. Notice that this is a conditioning situation, with this difference: The stimulus is *not in the environment but in the person's imagination.**

One therapist, for instance, has used this method to reduce fear of snakes in college students.[18] He found, first, that the method was highly

[16]Herrnstein, 1969.
[17]See Schwartz, 1984.
*Of course one may also try to condition relaxation to actual feared situations rather than imagined ones; but, interestingly enough, it is not clear that this method works any better than the one described, done in the patient's head.
[18]Davison, 1968.

successful; and second, that its success did depend on a specific conditioning procedure. Just learning to relax, and just getting used to thinking about snakes, did not help. The two had to be paired with each other, so that relaxation responses could become *conditioned* to the thought of snakes.

Covert Sensitization

Conversely, imagery can be used to *create* an avoidance response rather than to combat one; and here we can correct a certain one-sidedness in our discussion thus far. Not all fears are bad things. It is just as well if a child develops a fear of cars in the street, or light sockets, or deep water. And if there are things that an individual ought to stay away from, then conditioning an avoidance response is one way of helping her to do it.

Use of imagery for this purpose is known as **covert sensitization**. One is instructed to imagine the activity, and imagine aversive consequences associated with it. Since imagined aversive consequences are safe, however severe, one can safely make them very severe indeed. Listen:

> Now I want you to imagine that you've just had your supper and have just decided to have an after-dinner cigarette. . . . As you are about to reach for the pack, you get a funny feeling in the pit of your stomach. You start to feel queasy, nauseous and sick all over. As you pick up the pack, you can feel food particles inching up your throat. You're just about to vomit. As you pull a cigarette out of the pack, the food comes up into your mouth. . . . You bring the cigarette up to your mouth. As you're about to open your mouth . . . you vomit all over your hands, the cigarette, the pack. . . . You turn away from the cigarette pack and immediately you start to feel better. You run out of the room, and as you run out, you feel better and better. You wash and clean yourself up, and it feels wonderful.[19]

Extensive practice at imagining *that* is given. Here we have again a conditioning situation, but this time both the situation and the *response* —nausea and vomiting—are imagined. Clearly this is a cognitive procedure, aimed at affecting thoughts and feelings. But its debt to behaviorist ideas is also clear. The intent is to establish nausea as a *conditioned response* to cigarettes—even if the person knows intellectually that none of the misery he has imagined is going to happen. If treatment is effective, then reaching for a cigarette will produce an *acquired drive* of nausea, reduced by putting the cigarettes away.

And how did you feel, Reader, while reading that scenario? Do you think you would feel like smoking, if beginning to do so brought those images to mind—that is, evoked them as conditioned responses? If you

[19]Rimm and Masters, 1974, p. 400.

found it not a pleasant experience, you have seen the power of *imagery* in inducing motivational and emotional states.

AVOIDANCE, INSTINCT, AND LEARNING

To this point, we have considered the learning of motives as if it occurred against a blank background. Early discussions of the nature-nurture issue often were posed that way: Is this or that action innate *or* acquired? Is it provided by heredity *or* environment? We have come to see that the question cannot be asked that way. Rather, there are complex interactions between what we teach an actor and the untaught repertoire she or he already has available. We will explore three such cases: instinctive defensive reactions in avoidance learning; taste-aversion learning; and fear of strangers in human children.

Defensive Reactions and Avoidance Conditioning

In setting up an avoidance experiment, the response we choose as the "correct" one is arbitrary. For a rat it may be running, or lever-pressing, or rearing up on the hind legs. For a pigeon, it may be pecking an illuminated disk mounted on the wall, called a *pecking key;* or it may be wing-flapping, or jumping on a treadle to press it down. We can choose any response we like, and arrange matters so that it will escape, or avoid, something unpleasant.

We can choose any response we like. But the animal may or may not be able to learn it. Some responses are learned much more easily than others. These constraints were part of laboratory folklore, tricks of the trade of teaching animals to avoid shock, but they played no role in our theories until Robert Bolles[20] pointed out their great importance.

A pigeon, for example, readily learns to jump on a treadle to avoid shock, but it learns to peck a key only with great difficulty or not at all. That is not just because key-pecking itself is more difficult, for if the reinforcer is food rather than shock-avoidance, then the key-peck response is rapidly learned. Similarly, rats learn to run in avoidance situations much more easily than they learn to press a lever.

Why? Bolles suggests this: An animal comes to the experiment equipped with certain ways of responding to danger. These form a hierarchy of instinctive action patterns, analogous to the stickleback's hierarchy of sexual responses (pp. 101–104). But this time, the instinctive system is not reproductive, but *defensive.* Various action patterns are components of the system—freezing, attacking, running away, flying away if one can fly, or withdrawing into a shell if one has a shell. What actions are included in the system depends on the species. Therefore, Bolles calls these action patterns **species-specific defensive reactions**, or SSDRs (Figure 7–3).

[20]Bolles, 1970.

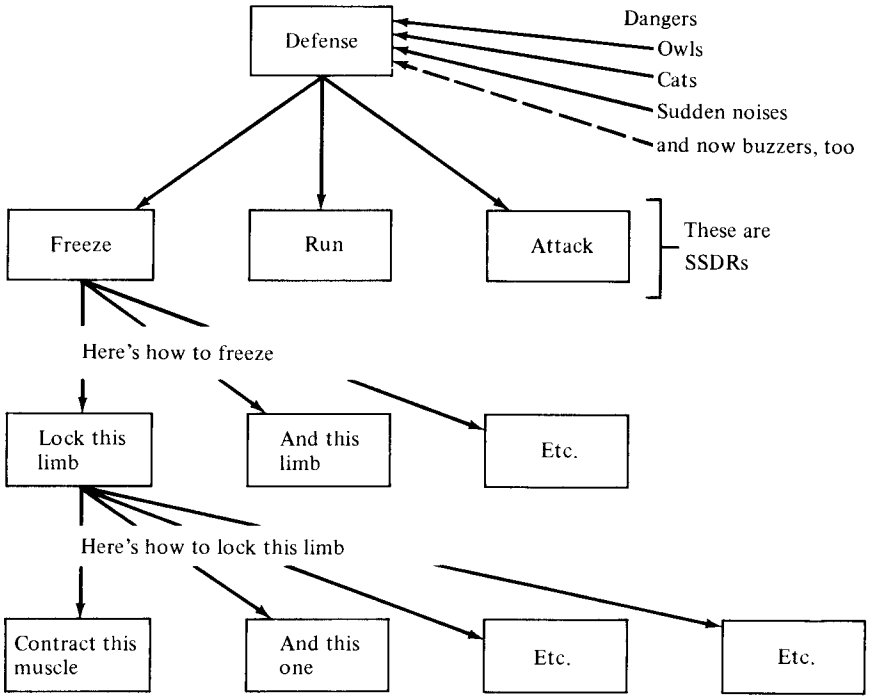


Figure 7-3.
The hierarchy of species-specific defensive reactions (SSDRs). An avoidance conditioning experiment does not provide new defensive responses, but a new situation in which to use defensive responses already available.

When the experiment begins, it is these instinctive responses that the animal uses. If one of them is effective, then that response will be learned quickly. If none of these SSDRs will work, then, and only then, will the animal try something that is not an SSDR. It will be learned slowly, laboriously, or not at all.

Thus pecking is *not* an SSDR for the pigeon. Jumping, however, is part of a flight or attack system of responses that pigeons instinctively use when threatened. For the rat, freezing is an SSDR. Faced with danger, a rat is likely to freeze. If freezing does not work, the running-away SSDR may be tried; and that will be effective in a shuttle-box situation, but not in a box where a lever-press is required. If running does not work either, biting the pain source may be tried; and a lever-press response is often made by biting the lever, thus depressing it by accident. But a lever-press response with the paws is not part of a rat's instinctive repertoire of reactions to danger. If that is made the correct response in an avoidance experiment, it will be learned very slowly, if at all.

This conception of avoidance is very different from the one we have dealt with up to now. It says this: The successful escape or avoidance of

shock does not *strengthen* a new response, such as running from one place to another. The rat already knows how to run; and more than that, it already is inclined to run if danger threatens. The learning that goes on teaches the rat, first, that *this* situation is dangerous; and second, that *this* member of the class of SSDRs is effective. It does not, we might say, teach the rat a new trick. It teaches the rat which of the tricks it already knows is the one to use.

Conditioned Taste Aversions

We have seen that the response one tries to reinforce, in an avoidance experiment, is not as arbitrary as we had supposed. A species-specific defense reaction is easy to condition; others may be very difficult or impossible to condition in this way.

Similar specificities, it turns out, are to be found on the stimulus side. Some stimuli make it easy to learn to avoid certain consequences; others make it very difficult indeed.

Demonstrations of Taste-Aversion Learning

Allow me a personal example here. When I was nine years old, I wrongfully ate an entire bag of jelly beans, and got violently sick while playing baseball on the lawn some time later. From that day to this, I have had an utter detestation for the sight, sound, smell, or mention of jelly beans. I am reluctant even to write about them!

Now why? I have no particular aversion to baseball, or to lawns, even though the sickness was most closely paired with baseball on the lawn, and the jelly beans had been eaten quite some time before. Moreover, the pairing of jelly beans with sickness happened only once, whereas avoidance conditioning is often a slow and gradual process requiring many trials, even in humans.

Apparently there is something special about *illness* as an aversive state that makes it particularly easy to associate with *tastes* and perhaps smells —very quickly, and across remarkably long time intervals. A clear demonstration of this is provided by the work of John Garcia and his colleagues.[21]

Briefly, rats were permitted to drink a novel and normally preferred saccharin solution. After that, they were exposed to X-irradiation, a treatment that produces nausea in humans and appears to do so in rats as well. In subsequent tests, the rats showed a clear avoidance of the novel taste, whereas rats that received the taste without the irradiation, or the irradiation without the taste, showed no such avoidance. And this was true after *one* taste-illness pairing, and even though the illness was separated from the taste by a full half-hour—and later experiments showed that the inter-

[21]Garcia, Ervin, and Koelling, 1966.

val could be much longer than that and still be effective. No rat would ever learn to avoid shock so quickly and with such a long delay.

This rapid learning seems to be specific to *taste paired with illness.* If a rat is shocked for drinking saccharin after a similar delay, it does not learn so rapidly to avoid saccharin. If it is made ill after hearing a buzzer and seeing bright flashing lights, it does not avoid those. In other words, neither taste nor illness by itself leads to especially rapid learning; but if we pair taste with illness, then learning is very fast indeed.

The phenomenon has also been demonstrated experimentally in humans.[22] The subjects were children who were undergoing radiation treatment for abdominal cancer, a treatment that often produces nausea. Briefly, subjects who were given a novel ice cream flavor (Maple Toff) before undergoing their treatment, liked that flavor less afterward (Figure 7-4). Control subjects, who received the treatment *or* the ice cream but not both, did not show this effect.

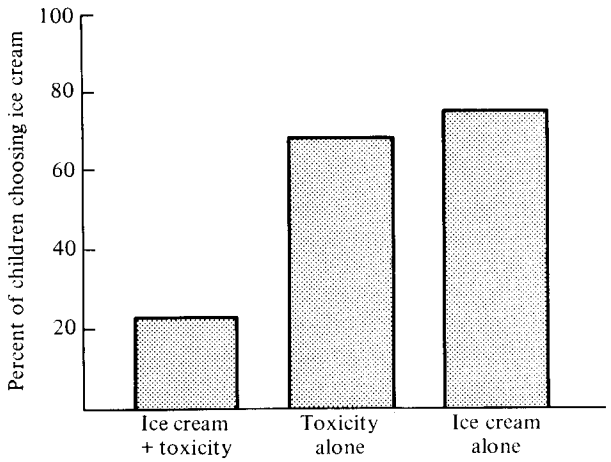


Figure 7-4.
Conditioned taste aversion in human children. Offered the choice between the new ice cream and a game, children were less likely to choose the ice cream if its taste had been paired with nausea. (Data from Bernstein, 1978.)

It is worth noting too that this avoidance of Maple Toff occurred even in the face of the subjects' cognitive apparatus. These children *knew perfectly well* that their nausea was caused by the treatment they were receiving, not the ice cream. But the ice cream was rejected anyway. Conscious knowledge, it seems, was no match for the biological "knowledge" that says to us: "If you feel sick, it's probably something you ate. Whatever you ate recently—avoid it."

[22]Bernstein, 1978.

And it is that biological knowledge that makes this case an example of the interaction between learning and instinct. Apparently, if they get sick, rats and children will avoid any new food that they have recently eaten. They need not learn to do that. What they learn in the experiment is that *this particular food*—a saccharin solution, or Maple Toff ice cream—is to be avoided.

Fear of Strangers

Let us turn to a quite different example in humans. Children at around nine to fifteen months of age, in many different cultures, go through a period in which the approach of a *strange adult* causes face aversion, crying, and other signs of intense fear. This is not just a response to novelty or surprise, for an unfamiliar child causes no such reaction.[23] It has to be an adult. And the fear response is an unmistakable action pattern, universal among human children, including the deaf and blind.[24]

The fear stage may develop and decay at different rates in different societies, so culture can influence its time course.[25] But why does it develop at all? Is it instinctive or acquired or both? It is both—it is an interaction of the two.

Suppose little Bill cries when his Aunt Sara—whom he has not seen since the day he was born, when he wasn't paying attention—approaches. Obviously Aunt Sara is not releasing stimulus; the brain did not evolve a recognition mechanism for Aunt Sara herself. Little Bill had to *learn* who is a familiar figure in his life. But little Bill probably has never been threatened or hurt by an adult; or if he has, it has probably been by a familiar one, not a strange one. Experience was needed to teach the child who is familiar and who is not. But *experience did not teach him to fear unfamiliar adults.* That fear response must be untaught.

A Look Backward: How Instinct and Learning Interact

What is common to all these cases—SSDRs, taste aversion, and fear of strangers—is this: In each of them, the learning experience teaches the animal what kind of situation it faces. But it does *not* teach it what to do about that situation. That part, it seems, is instinctive, or untaught. Thus we see that experience does not *teach* a child to fear strange adults, though it does teach the child which adults are familiar and which are not. Similarly, avoidance conditioning does not *teach* a rat to run when threatened. It does teach the rat that the experimental chamber, or the onset of the buzzer, is a threat. Rats and humans need not learn to avoid novel foods paired with illness. They do learn *what* foods to avoid on those grounds.

[23]Brooks and Lewis, 1976.
[24]Eibl-Eibesfeldt, 1972.
[25]Konner, 1972.

Instinctive Rules

We might say it this way: A human or animal brings to the learning situation certain *rules* about what to do in certain situations. The rules are ones that a human could express in words: "In danger, it's a good idea to freeze or run (for a rat) or to fly and jump (for a pigeon). Try it!" Or: "If you feel nauseous, it's probably because of something you ate. Remember it and avoid it!" Or: "If you see an adult who matches none of your memories, it's fearsome! Fear it!" We need not learn these rules. Experience teaches us when to apply a rule of this kind, but it does not give us the rules themselves. In some biological sense, we already know them.

The Concept of Preparedness

Now it is much easier to learn something if we already know something about it. Thus, if what we are to learn—these two stimuli belong together, or this is the response that the problem requires—fits in with rules we already have, the learning task is greatly facilitated. We are, in Martin Seligman's[26] words, already *prepared* to learn certain things about the world. We are prepared to learn to avoid foods whose ingestion is followed by illness, for example. Learning to avoid foods followed by shock is much harder; it is *unprepared.* We bring with us no rule that relates foods to external pain.

Does the notion of **preparedness** help us understand more complex human problems? It may. Here is an example.

Preparedness and Phobia

A **phobia** is a persistent fear reaction that is out of proportion to the reality of the danger. It is an irrational or unjustified fear. One speaks of claustrophobia, or a fear of being enclosed; agoraphobia, or a fear of open and exposed situations. Quite a few people are afraid of spiders or snakes, but some, *phobics,* are terrified of them, terrified even of photographs of them. And as anyone knows who has experienced severe anxiety at exam time or before speaking in public, even mild phobias can be truly debilitating.

Now we assume that these fears are learned. Yet the *content* of the fears does not suggest that an unfortunate experience with just any object can evoke fear of it. Certain objects are feared by a great many people who have never been hurt by them—spiders and snakes, for instance, or exposure before groups of people as in public speaking. Other objects are seldom the focus of a phobia; few people are afraid of lambs, shoes, or electric sockets, though the latter at least is more dangerous than any spider. Why?

Perhaps, Seligman has suggested, because we are *prepared* to learn to fear certain things and not others.[27] These may be things that, in fact,

[26]Seligman, 1970.
[27]Seligman, 1971.

were dangerous to our evolutionary ancestors: Small, crawly things can deliver painful bites; snakes can kill; speaking before a group could draw the attention of enemies or predators. Perhaps we have become prepared to associate these with negative consequences as we associate tastes with illness; and so it takes only minimal negative experience with certain stimuli to make us fear them, with an intensity that may be out of all proportion to the experience itself. It is as if we had a rule that says, "Crawly things are likely to be dangerous." Then we might quickly learn, with only one mild bad experience: "Yes, crawly things *are* dangerous!"*

There are some data that support this idea. The fears expressed by a large number of phobic patients were classified with rating scales, constructed independently, of "the extent to which the [feared] object or situation was . . . dangerous to pretechnological man."[28] Such things as predators, blood, thunderstorms, and darkness were rated high on this scale; such things as flowers were rated low.

As it happened, the majority of patients' fears *were* of things that once would have been dangerous. Not all of them were, but then the theory does not say that all phobias are prepared. It only says that prepared ones are most readily acquired and so ought to be most frequent—as, in fact, they are.[29]

In an even more dramatic demonstration, a fear response was directly conditioned in *nonphobic* human subjects by presenting certain stimuli along with electric shock. The stimuli were either snakes and spiders, or houses and faces. After conditioning, the experimenters told the subjects that no more shocks were coming; they even removed the shock electrodes. Fear of houses and faces disappeared at once; but fear of snakes and spiders persisted full-blown.[30]

Perhaps the irrationality of phobics is like the irrationality of the Maple Toff eaters. Those subjects *knew* that their illness had nothing to do with ice cream, just as phobic subjects *know* that a garter snake is harmless. But the biological rules that prepare us to fear certain stimuli may be among the many things that can override our intellects.[31]

CONDITIONED REINFORCEMENT

To this point, we have talked mostly about bad things—shocks and spiders and the like. In this section and the next, we turn to nicer things.

Many, probably most, of our actions are performed not only with no biological drive to motivate them, but also without any biologically relevant rewards to maintain them. Obviously, many of the things we work

*Or we may even learn from someone else's experience. Perhaps we are prepared to learn certain fears even by imitation, a possibility that needs study.

[28]deSilva, Rachman, and Seligman, 1977.

[29]For further discussion see Rachman and Hodgson, 1980.

[30]Hugdahl and Öhman, 1977.

[31]Rosenhan and Seligman, 1984.

for and strive for have value only because we have learned to value them. The acquisition of such value has been studied in the laboratory, in particular through the investigation of **conditioned reinforcement**. This is the process by which a stimulus that has no reinforcing power of its own, acquires such power through learning. The stimulus becomes an *acquired reward.*

Experimental Demonstrations

Actually, there are two kinds of experimental demonstrations that have been called conditioned (or secondary) reinforcement, and it is unfortunate that they have been called by the same name. They are quite different.

In one, a response is reinforced by something that has no value in itself, but is exchangeable for something that does. Chimpanzees, for instance, have been allowed to work for poker chips which, some time later, may be exchanged for grapes. Chimps are much attracted to grapes, whereas they show little interest in poker chips per se; but if the poker chips are a means to grapes, the animals will work avidly to obtain the chips (Figure 7-5).[32]

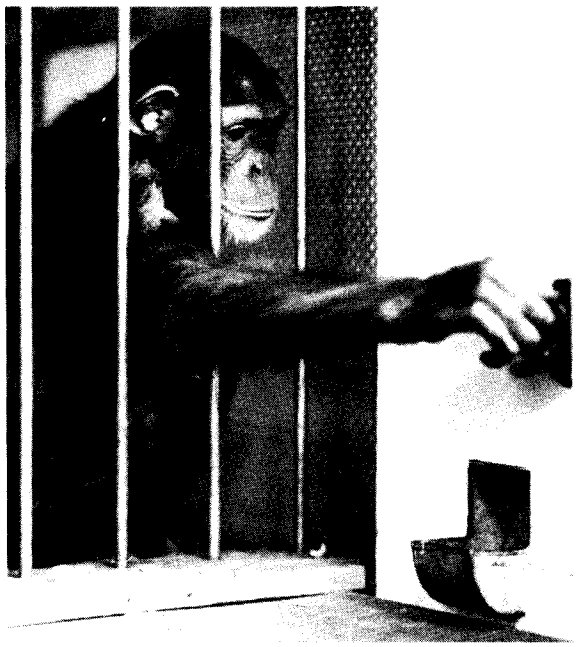


Figure 7-5.
Conditioned reinforcement. The chimp has been trained to work for poker chips that can be used to obtain food.

[32]Wolfe, 1936; Cowles, 1937.

Humans, of course, will go through some high-level cerebration to obtain poker chips, too—especially if the chips are exchangeable for green pieces of paper that are in turn exchangeable for worldly goods. Obviously, money itself can be a conditioned reinforcer in this sense.*

More puzzling is the second kind of conditioned reinforcer, in which the reinforcing stimulus is of no use to the subject whatever. It is not *exchangeable* for anything. Suppose, for instance, that a rat is trained to press a bar for food reinforcement, and that the delivery of a food pellet is accompanied by a loud click. Then the food-delivery apparatus is turned off; no food is delivered any more. For some of the rats, pressing the lever now produces nothing at all. For other rats, it produces no food, but it does produce the click of the pellet dispenser. The latter group will go on working at the lever much longer.[33] As someone has said in one of the worst puns on record, the rats press the lever "just for clicks."

Why? You can't eat a click, or even exchange it for food. Objectively it has no value, never had and never will. But it will maintain behavior for a while; and such a stimulus may even support the acquisition of a new response, for which it is the only reinforcer.

The Establishment of Conditioned Reinforcers

There have been several suggestions about what happens here. One is that the click becomes a reinforcer, not just because it is *paired with* a natural or primary reinforcer, but because it *predicts* the occurrence of one. Thus the click says to the rat: Food is coming soon.[34]

The other possibility is that a conditioned reinforcer need not *predict* a primary reinforcer, but need only be *paired with* one. It may be that a stimulus acquires reinforcing properties simply by occurring along with a stimulus that already has such properties. This view is close to the *associationism* of Hobbes (see Chapter 2). Hobbes might have said that the conditioned reinforcer acquires *pleasantness* through being associated with something already pleasant.

In the human case, there is evidence that stimuli acquire reinforcing power simply by being paired with reinforcers,[35] apart from what they predict or can be exchanged for—*if* we are willing to assume that reinforcing power and *pleasantness* are related.*

In one experiment,[36] human subjects were repeatedly exposed to two

*In fact, many textbooks simply make the flat assertion: Money is a conditioned reinforcer. A word of caution is appropriate here, for there has never been the slightest evidence that the child's *acquisition* of the desire for money occurs in this way rather than some other—for example, through imitation of, or identification with, adults. In older humans, of course, the value of money is maintained by its exchangeability for goods and services, as is demonstrated dramatically when money loses its value in an inflationary economy.

[33]Bugelski, 1938.

[34]For discussion see Fantino and Logan, 1979.

[35]See Martin and Levey, 1978.

*The definition of reinforcement does not require that assumption, as we will see in Chapter 8.

[36]Zellner, Rozin, and Kulish, 1983.

flavored solutions, one of which had table sugar as an added flavorant. Later on, the subjects tasted and then rated the pleasantness of both solutions, *without* the sugar. It turned out that the taste that had been associated with sweetness, even though it no longer was, was rated more pleasant than the other taste. Perhaps the pairing of new tastes with familiar and pleasant ones may be one way that humans develop likings for new tastes.

Functions of Conditioned Reinforcers . . .

Behavior theorists have assigned a variety of important functions to conditioned reinforcers, especially in complex human behavior. Here are some of the things conditioned reinforcers might do:

1. They can *provide information.* They tell the actor that he has done the right thing. The chimp earns tokens, the rat earns clicks, but only for appropriate responses.
2. They can *provide stimuli that guide subsequent behavior.* If the chimp has a token available, it can exchange the token for food.
3. They can *bridge gaps in time.* If conditioned reinforcers are important in human behavior, it is likely that this is their most vital function. If writing a book, or writing a paper, were reinforced only by its final payoff, the reward would probably be too long delayed to have any effect. The behavior would extinguish before the job was done. As it is, our telling ourselves, "One more page done!" might be a conditioned reinforcer, if one has been reinforced before for completing writing assignments. A chimp might never learn to make a response for food if thousands of responses were required for each morsel. But it may do so if it collects tokens along the way.
4. They may simply *become pleasant in their own right.* Chimps may come to *like* poker chips, as human subjects come to like a flavor that was paired with sweetness before.

. . . And a Note of Caution

The discovery of conditioned reinforcers was exciting to early behavior theorists. Such learned reinforcements could go a long way, it appeared, toward accounting for the variability and individuality of human behavior, and for the maintenance of behavior, such as studying or writing a book, that is far removed from any natural reinforcer such as food or warmth.

The key, once again, is the *arbitrariness* of the stimulus that acquires reinforcing properties. The rates pressed the bar just for clicks, but they could have pressed just for tones or flashing lights if the experiment had been set up that way. Chimps worked for chips, but marbles could just as well have been used instead. Presumably, *any* stimulus could become

a conditioned reinforcer—like the pieces of paper we call money, or the sound of praise in a familiar language, or marks on paper that constitute a high grade—depending on the actor's conditioning history, which has made those stimuli reinforcers for his actions.

There is a danger, however. Because the principle *might* explain so much, we may *assume* that it does when evidence is lacking. In discussing conditioned reinforcement, one writer[37] gives these examples:

> The student earns an "A" only for appropriate responses. . . . When the instructor says "Good backswing!" to her tennis pupil, the "good" provides feedback about having performed correctly . . . it may also make the pupil feel good.

Now these stimuli—an A, or praise from an instructor—are indeed reinforcers, if they lead us to repeat the actions that preceded them (the law of effect). And obviously their reinforcing value is learned. But was it *conditioned* in the way I've described? Are the stimuli reinforcers *because* they were paired with food or sex or even cuddles? There is no evidence that this is so, and it seems rather unlikely.

It seems much more likely that arbitrary stimuli can acquire reinforcing properties, and/or pleasantness, in other ways *besides* explicit conditioning or pairing. We turn to that topic next.

NON-ASSOCIATIVE MECHANISMS IN LEARNED MOTIVES

To this point, our discussion of acquired motives and goals has centered around the conditioning procedure. It builds on the work of Pavlov, and of Hobbes before him, in emphasizing the *pairing* of one thing with another. This is Pavlov's *conditioning* procedure; it is also what Hobbes saw as creating an *association* between one idea and another.

Thus in an avoidance experiment, the buzzer evokes an aversive state because it is related to something else—shock—that is already aversive. In a conditioned-reinforcement experiment, a click becomes a reinforcer because it is related to something else—food—that is already a reinforcer. Both cases depend on a relation or association between one stimulus and another.[38]

There are other cases of learned motives, however, that do not fit that framework. They have received much less study, perhaps because the concept of association has dominated our thinking about the matter. But we do know that they exist. Let us look at two examples.

[37]Schwartz, 1984, pp. 185–186.
[38]See Martin and Levey, 1978.

Imprinting

In certain species of birds, including domestic chickens and ducks, an intriguing and powerful form of *learned reward* can be demonstrated. Experimentally, let a very young, previously isolated chick or duckling encounter any large, moving object. That object then becomes the focus of approach behavior of a variety of kinds: The little bird will follow the object around, or make an arbitrary response to be brought into contact with it, and emit distress calls when it isn't there. This early and rapid learning is called **imprinting.**[39]

In the normal course of events, the large moving object will usually be the parent. Then all will be well; thereafter, the little bird will stick with its parent, and later approach other members of its species, as it should. But if it imprints upon a member of the wrong species, the consequences for its social life can be catastrophic. In later life it may prefer the company of the wrong species (Figure 7-6). It may even court the members of that wrong species. The ethologist Konrad Lorenz found that his adopted jackdaws made perfectly normal, jackdaw-like action patterns of courtship toward *him!*

Imprinting is most likely to occur during a certain sensitive period, early in life. Let the large object appear too late, and imprinting will not occur.

Now the stimulus upon which the animal imprints must have certain

Figure 7-6.
Imprinted ducklings follow a member of the wrong species—Konrad Lorenz.

[39]Lorenz, 1965.

characteristics: it must be fairly large, and it must move. Beyond that, it may be perfectly arbitrary. A chick can be imprinted upon a human being or, for that matter, upon a block of Styrofoam. Such a useless object would be rejected with contempt by a chick or duckling that had not been exposed to it during the sensitive period. Thus the imprinting phenomenon is related to some ideas we are familiar with, but it also is different, and may provide us with a new principle to consider.

The object—a Styrofoam block, or a green wooden cube, or whatever—does become a reinforcer, for the little bird will make an arbitrary response to obtain commerce with it.[40] It acquires that reinforcing power through learning. But it is *not* a conditioned reinforcer, because conditioned reinforcers acquire their rewarding power through being paired with, or predicting, a natural or *primary* reinforcer such as food. In imprinting, there is no primary reinforcer.

This is an important difference. We might think of conditioned reinforcement as a *transferring* of reinforcing power from a stimulus that already had it (such as food), to a stimulus that did not (a click or a poker chip). Similarly, we can think of the stress reactions to electric shock as *transferred* to a buzzer paired with shock, by the conditioning process.

But imprinting is not like that. Value is not transferred *to* the Styrofoam block *from* something else. There is no *something else.* The Styrofoam block does not offer the little bird anything of value—and it does not predict, and is not paired with, anything else that does. Apparently the bird does not so much transfer, as *assign,* positive value to the imprinted stimulus when it appears. That stimulus becomes a reinforcer, not because it is paired with a natural reinforcer, but simply because it appears at the right time—during the sensitive period—and has the right properties—it is large and it moves.

How many other such *assignments* of reinforcing power are there? The imprinting phenomenon itself may turn out to have considerable generality as a component of parent-to-young attachment in a variety of species, humans included.[41] But the process of assignment, as opposed to transfer, of reinforcing power has not been much discussed in other contexts. It seems unlikely that it is restricted to parent-child attachment situations.

Mere Exposure

Another case of non-associative learning in motivation is the effect of *mere exposure* to a stimulus complex. Both small children[42] and big college students[43] like a flavor better if they have experienced it before than if they haven't. Robert Zajonc,[44] one of the few who have studied the

[40]Hoffman and Ratner, 1973.
[41]Bowlby, 1969; Hoffman and Ratner, 1973.
[42]Birch et al., 1982.
[43]Pliner, 1981.
[44]Zajonc, 1968, 1980 (pronounced *Zah*-yonss).

matter in detail, finds that Chinese characters, nonsense syllables, and photographs of faces, all become more positive as subjects are repeatedly exposed to them. He summarizes other data showing that paintings or music in unfamiliar styles become more pleasing with mere exposure of the material, not paired with anything.

Why? We are not sure. The effect does not seem to depend on conscious recognition; it occurs even if subjects do not recognize the material as having been presented before.* Zajonc suggests that it may reflect the disappearance of an initial tense reaction to anything new—a kind of cognitive SSDR that says, "Is it dangerous?" As the stimulus is repeated and nothing bad happens, that reaction drops away.

The mere-exposure effect may be of great importance in, for example, the socialization process (Chapter 13). It is too bad that we know so little for sure about how it works.

THE OPPONENT-PROCESS THEORY

Now we are ready to look at a more complex process in the acquisition of motives. This theory deals with both nice things and nasty things, and it includes both associative and non-associative mechanisms. Let us begin with an example, and look again at imprinting.

When hungry, cold, or otherwise stressed, baby ducks emit characteristic high-pitched distress calls, over and over. Figure 7-7 shows the frequency of these distress calls over blocks of time in a warm, unstressed duckling, in a comfortable environment. At the beginning of the session

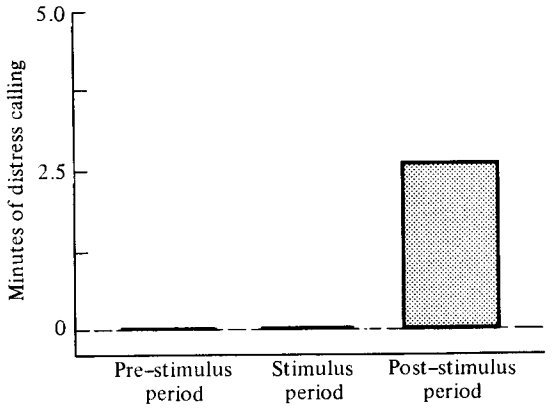


Figure 7-7.
Distress calling by ducklings, before an imprinted object is introduced (pre-stimulus period), while it is present (stimulus period), and after it is withdrawn (post-stimulus period). (From Hoffman and Ratner, 1973.)

*There is some dispute about that, however; see Birnbaum and Mellers, 1979; Moreland and Zajonc, 1979.

there are no distress calls (left panel). An imprinted object—a Styrofoam cube—is introduced; still no distress calls (middle panel). The cube is removed; and *then* distress calls begin (right panel). If we take distress calling as an indication of a duckling's misery, then we conclude that the *withdrawal* of the object evokes substantial misery when its simple *absence* did not.

We empathize with this reaction. We would react the same way if something nice were taken away. But why should it be that way? Objectively, the situation at the end of the session is the same as it was in the beginning: no Styrofoam cube. Why is the duckling's misery so much greater after the object goes away?

Richard Solomon and John Corbit[45] have developed a theory about the *time course* of positive and negative feelings that may shed light on the psychology of disappointment—and on much else besides. The basic idea is: If a situation generates a state of liking or disliking, of happiness or misery, then that state will in turn trigger its opposite—the **opponent process**. Happiness generates its opposite. So does unhappiness.

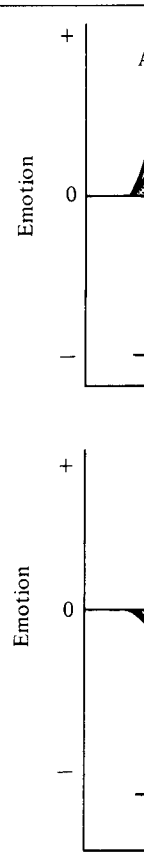
The theory is diagrammed in Figure 7-8. A situation has as its first component (Process A) a hedonic reaction; enjoyment or pleasure, let us say. That evokes the opponent process B, a state of displeasure or lack of enjoyment. Since B is opposite to A, it subtracts from it, and the resulting enjoyment levels off at some value below the original one.

Suppose now that the happiness-evoking situation goes away. Since B is evoked by A, not by the situation itself, it lags behind A. As a result, there will be a rebound—a swing toward negative feelings, because of the unopposed action of State B.

This theory is, in a sense, a *homeostatic* theory of emotion or mood. We see our familiar negative-feedback homeostatic system as developed in Chapter 3: An input (Process A) triggers an action (Process B) that reduces the input. The effect is that neither pleasure nor displeasure is allowed to be very intense for very long. That obviously brings good news and bad news; our moments of intense misery will be short-lived, but so will our moments of intense joy. And we can expect a rebound, either way. Solomon[46] calls it a Puritan's theory: Joy must be paid for with grief later, but pain now promises better to come.

We can see, too, that there are certain advantages to such a system. If distress is always moderated by its opposite, this may allow organisms to keep going in the face of pain, or disappointment, that could otherwise be immobilizing. On the positive side, we can *speculate* that even here there may be advantages to moderation. It might not be to our advantage to be too ecstatic for too long. In the wild where we evolved, our ancestors had many tasks and faced many dangers, and needed to keep their wits

[45]Solomon and Corbit, 1974.
[46]Solomon, 1980.



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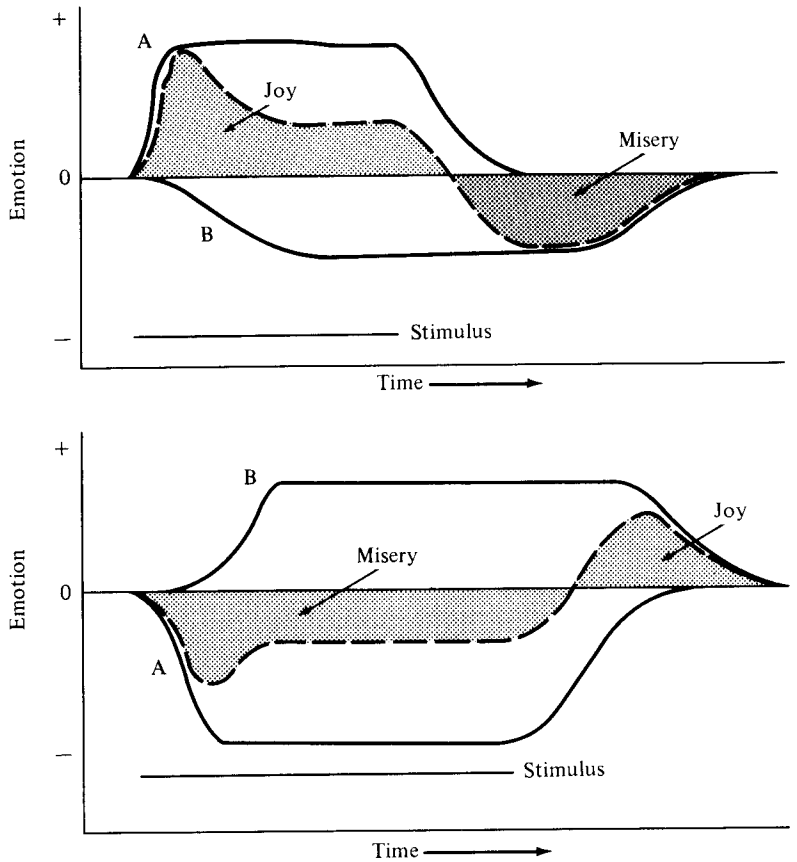

Figure 7-8.
The opponent-process theory. The shaded area represents the *difference* between the primary A process and the opponent B process. Top panel: Joy (A) produced by a stimulus is quickly reduced by the opponent process (B), which also produces a rebound toward misery when the stimulus ends. Bottom panel: The converse case; misery (A) is alleviated by the opponent process (B), which produces relief when the unpleasant stimulus ends.

about them even in moments of joy. Perhaps a joy-moderating system evolved out of that necessity.

Now let us apply the theory to the duckling case. When the cube arrived, let us assume that it was greeted with a swing toward pleasure. That pleasurable reaction may have decayed with time—no measure of that is provided here—as a result of the modulating action of Process B. Then when the cube went away, Process B was unopposed, and the duckling went into a rebound mood of misery and distress. The resulting disappointment was expressed long and loud.

The Generality of Opponent Processes

What this theory does is to bring motivational/emotional states into line with what seems to be a general principle of nervous system function: Push the nervous system in one direction, and it will push back in the other. One example is the effect of drugs. Amphetamine is a nervous system *excitant.* The symptoms of amphetamine withdrawal, after prolonged and heavy use, are the symptoms of nervous system *depression:* lassitude, slowness, and somnolence. Conversely, alcohol is a nervous system *depressant.* And the symptoms of alcohol withdrawal, after prolonged and heavy use, are the symptoms of nervous system *hyperexcitability:* exaggerated reflexes, irritability, and (in extreme cases) convulsions and hallucinations. In each case, removal of a drug that causes one set of effects triggers the opposite effects.

The Dynamics of the Opponent Process

Thus far, States A and B can be considered more or less fixed processes underlying our hedonic states. Complications come in with Solomon's further suggestions about the properties of the opponent process B. It changes over time, and in two ways. First is a non-associative change: Process B is *strengthened by use.* Second, there is associative change: Process B is *conditionable.* Let us look at these complications in that order.

The Growth of Opponent Processes

The opponent process B, but not the initial state A, is assumed to become faster and bigger if it is evoked repeatedly and often. In this respect it is like fatigue. Lift a weight once a second, and we will quickly get tired; lift the same weight once a week and we won't. In a similar way, if Process A is evoked often enough, with only short times between, the B process will increase over successive episodes. Since the A process doesn't change, this means that (1) the difference between them will diminish—the first emotional reaction will get smaller and smaller—and (2) the rebound swing to the opposite mood will become more and more severe.

Here is an example. Parachutists, during their first jump, often experience utter terror. Their hearts race, their breathing is spasmotic, their bodies are curled and stiff. After landing safely, they may appear stunned for a few minutes, stony-faced and silent. Then they go into a period of elation, with smiling and rapid excited talk. A mild euphoria has replaced the misery of fear.

After a number of jumps, matters are different. The fear is much reduced, and the parachutists may look forward to their jump with excitement. After landing, activity level is very high, and there may be leaping, shouting, and back slapping. Their success at jumping, we might say, has taught them that they can jump safely; perhaps that is why the fear is

lessened. But why should the euphoria after the jump become even greater than before?

It makes sense if one assumes that the opponent process leading to euphoria has been strengthened by use (Figure 7-9). We assume that Process A, the fear response, doesn't change; but that the opponent process, B, gets bigger and bigger with repeated jumps. Thus, on the first few jumps, the opponent process mitigates fear somewhat and overshoots to produce mild euphoria. But after many jumps, Process B is greater in magnitude. It neutralizes fear (Process A) almost entirely; moreover, when danger is over, it rebounds into an even greater euphoria than it did originally.

On the negative side, it appears that a duckling's misery is also strengthened by repeated evocations. When the Styrofoam cube is introduced and

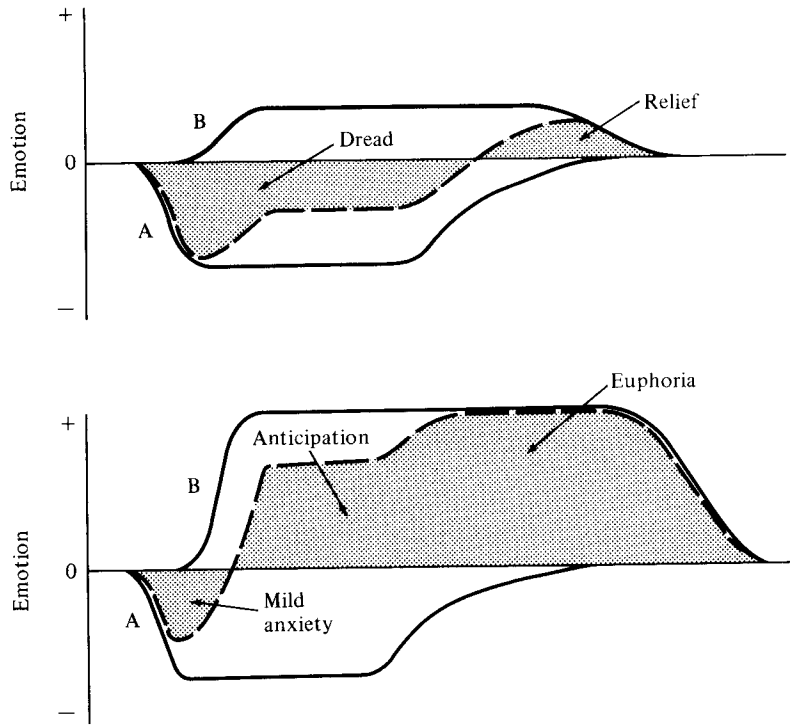

Figure 7-9.
Effects of strengthening the opponent process by repeated use. Top panel: Over the first few experiences, the weak B process alleviates parachutists' dread only slightly and leads to only a mild rebound after the jump. Bottom panel: On later experiences, the strengthened B process may quickly overcome the A process, producing anticipation instead of dread, and a large rebound into elation and euphoria after the jump.

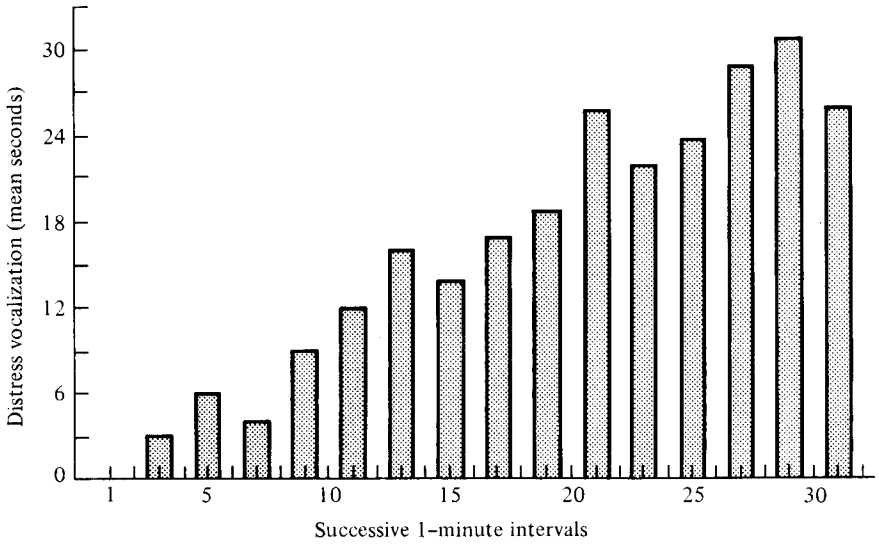

Figure 7-10.
Strengthening of disappointment by repeatedly evoking it. As an imprinted object was repeatedly presented and withdrawn, the distress calls which followed the withdrawal became more and more prolonged. (From Hoffman et al. 1974.)

then withdrawn the first time, distress calls are evoked, as we have seen. As such episodes are repeated, distress calling becomes more and more prolonged (Figure 7-10). A new source of misery is thus added to the duckling's existence. The first disappointment is bad enough. Repeated ones get worse and worse.

Finally, let us see how the idea applies to an acquired motive that can dominate an individual's existence, causing severe disruption of both the individual and the community. Consider *addiction.*[47]

The self-administration of heroin is followed by a rush of pleasure, which some people compare to orgasmic feelings (Process A). This initial thrill soon settles down into a state of pleasant euphoria (pleasure partly neutralized by Process B).

After frequent and repeated use, however, the picture changes. Even if Process A stays the same, the rebound process, B, gets bigger and bigger. This has two consequences. First, the pleasure produced by the drug is much reduced, and higher doses are required even to approach it. This is known as **tolerance**. Second, the discontinuance of the drug results in distressing physical symptoms, known as **withdrawal symptoms**. At this stage, the person, now physically dependent on the drug, may seek it, not

[47]Solomon, 1980.

for pleasure but for relief of the discomfort of withdrawal. A positively-reinforced response has now become an escape or avoidance response, motivated by withdrawal symptoms. One seeks to prevent their onset (avoidance), or end them if they do occur (escape).

How tolerance and withdrawal work at the cellular level is not known. But many of our theories, including the opponent-process theory, see the two as reflecting a single underlying principle. It is the principle we saw earlier: Push the nervous system and it will push back. Thus tolerance may occur in part because the opponent process B, strengthened by repeated elicitations, neutralizes the pleasurable process A. And withdrawal may occur in part because the strengthened process B results in progressively greater rebound as the drug leaves the body.

Conditioning of Opponent Processes

We have seen that the opponent process, B, begins as a response to the initial process, A. However, Solomon and his colleagues suggest that Process B also can be conditioned, so that *external* stimuli can cause it to occur.*

In the drug case, for example, perhaps Process B—and its consequences, such as the withdrawal syndrome—can come to be triggered by the external situation in which the sequence has occurred before. Recovering heroin users are urged to avoid the settings in which drug use has occurred. There may be good reason for this, as shown by this case study:

> The patient was a 28 year old man with a 10-year history of narcotic addiction. He [was arrested and] reported experiencing severe withdrawal during the first 4 or 5 days in custody, but later . . . felt like a new man, and decided he was finished with drugs. . . . On the way home after release from prison, he began thinking of drugs and feeling nauseated. As the subway approached his stop, he began sweating, tearing from his eyes and gagging. [These are characteristic withdrawal symptoms.] This was an area where he had frequently experienced narcotic withdrawal symptoms while trying to acquire drugs. As he got off the subway, he vomited onto the tracks. He soon bought drugs, and was relieved. . . . The cycle repeated itself over the next few days and soon he became readdicted.[48]

Quite possibly, the occurrence of withdrawal symptoms, as *conditioned responses* to a setting in which drug use has occurred, may lead the recovering addict back into drug use in order to escape those symptoms. A similar process has been suggested for relapses by recovering alcoholics.[49]

*Opponent-process conditioning has been demonstrated experimentally in the effects of a familiar drug—caffeine. See Rozin et al., 1984.

[48]O'Brien, 1976.

[49]Ludwig and Wikler, 1973.

There may be more dangers than readdiction here. The hedonic opponent process B, as it affects our pleasure or displeasure, is surely only one component of a general compensatory reaction by the body to the effects of a drug. In a word, the body attempts to compensate for what the drug is doing, and thus reduce the drug's effects. Obviously that process could be life-saving at high doses of dangerous drugs.

But suppose the compensatory reaction is also conditioned as a response to certain environmental cues. Then it will be weakened if those cues are changed. Suppose, for instance, that a heroin addict injects the drug at a dosage she has learned is safe—but does it in a novel environment. Then the body's compensation could be less than usual, because environmental conditioned stimuli for that compensation are lacking. A fatal overdose could result. There is evidence that this can and does happen in drug users. And it has been shown by direct experiment that it can happen in animals: A dose of morphine that was safe for addicted rats, if given in an environment associated with its use, was life-threatening in an environment where it had not been given before.[50]

Before moving on, however, we ought to note an unresolved difficulty here. This theory of drug tolerance appeals to the conditioning of a process that *opposes* the effects of the drug itself. Such conditioning has been demonstrated.[51] But we also know of cases where the conditioned process *mimics* a drug effect, rather than opposing it.[52] When does conditioning produce a drug-like effect, and when does it produce a drug-opposing effect? We don't know.

The Status of the Opponent-Process Theory

What are we to make of the opponent-process idea? Certainly it has made sense of data obtained in a variety of species and settings—orphaned ducklings, parachutists, and human drug users.

It is obviously a very flexible theory, and that is what makes some writers, including me, uncomfortable with it. Solomon himself has expressed some misgivings about its flexibility. It could easily be tailored so as to explain any possible finding. For example, why does Dad's apple pie never lose its attraction? If it is because we don't eat it that often—often enough to build up a strong opponent process B—well, how often is *that often?*

The theory's account of addiction leaves similar loose ends. Morphine or heroin *can* produce intense pleasure on first usage. But many users find the first few experiences very unpleasant. For them, heroin is an acquired taste. Are we to say that an opponent process leads from displeasure to pleasure early on, and *another* opponent process later swings the other way to produce tolerance and withdrawal? Obviously, if we are

[50]Siegel, et al.,1982.
[51]Rozin et al., 1984; Siegel, 1975.
[52]Siegel, 1972.

allowed to stack opponent processes end to end like that, we could explain anything whatever.

None of this is intended to dismiss the opponent-process theory. It only says that we are not yet sure how to evaluate it. We need to test its many implications, and to work out the laws that the processes can be expected to follow—how strong is strong enough, and how often is often enough, and whether the occurrence of separate early and late opponent processes can be demonstrated with independent evidence. Then we will be able to judge whether the theory is on the right track, or only ingenious.

A LOOK BACKWARD: THE PROBLEM OF INTERNALIZATION

It would be wonderful if we could end this chapter with a short and complete list of principles governing even the simpler cases of learned motives. We cannot. Any list we drew up would not be short and it would not be complete.

It would not be short because there are so many different mechanisms that can come into play. Neutral things can become awful things through being related to already-awful things, as in conditioned fear. They can become nice through being related to already-nice things, as in conditioned reinforcers. But also, just by occurring under the right conditions, they can be nice, as with imprinted objects, or awful, as when an adult face is not recognized by a child. And those *right conditions* are likely to be very different from species to species and case to case. Finally, things can be awful just because they were nice before, or conversely—the opponent-process theory.

And the list is almost certainly not complete. There are powerful acquired motives that the ideas we have surveyed do not adequately account for. As an example, consider the problem of *acquired tastes.*

The vast majority of adult humans, all over the world, eat or drink one or more of the following: alcohol, bitter substances like caffeine, or irritants such as black pepper or chili pepper. Now most babies detest coffee or alcohol or chili peppers. And yet many human adults come to like these initially negative tastes; they will seek them out, and pay money to obtain them. Why?

We can see some obvious possibilities. Maybe the obnoxious tastes are paired with nicer ones, or with some external reward, and so *become* nice (conditioned reinforcement). Mere exposure might make them nicer. Or maybe the noxiousness of the initial taste produces a rebound toward niceness (opponent process).*

However, there is a problem with all these simple ideas. If they were right, then it ought to be easy to teach acquired taste to animals. In fact, it is almost impossible. An acquired taste for peppers has been demon-

*In the case of chili peppers, however, experimental tests of the possibility have not confirmed it; see Rozin, Ebert, and Schull, 1982.

strated, to date, in two chimpanzees.[53] But in rats, no amount of exposure or conditioning has succeeded in producing a preference for a taste that is initially disliked.[54]

If the acquired-taste phenomenon is *almost* uniquely human, maybe it is responsive to characteristically human motives. We may eat or drink nasty substances in response to peer pressure, or a desire to be like the big folks. But that only accounts for our eating or drinking them; it doesn't explain how we come to *like* them.

Yet clearly we do. "Tens of millions of little chili haters become chili lovers every year."[55] The same could be said of little coffee, whiskey, and tobacco haters. Our culture affects not only what we eat, but also what we like and don't like. Somehow, the likes and dislikes of our culture become *internalized*—they become our own likes and dislikes—so that, for us, certain innately nasty tastes become nice ones instead.

How this works, we do not understand. It is not hard to see how parents and peers could teach us—by rewards, through imitation, and the like—to *eat* certain foods. But how do we learn to *like* what our society likes? Conversely, this society can easily teach us to *say* that ants for breakfast is a disgusting idea—but how do we come to *be disgusted* by it?

The same problem arises in sexual motivation. In Chapter 4, we saw that the culture we grow up in has a great deal to do with whom we find sexually attractive. Our upbringing exposes us to the culture's standards of sexual attractiveness; it labels persons with certain features as attractive; and clearly it maintains pressure on us to *behave* in accordance with its standards. But how are those standards *internalized?* Conventional learning experiences might teach us to *call* people "sexually attractive" if they have such-and-such characteristics. But how do we come to mean what we say, so that we are genuinely attracted to such people? We do not know.

SUMMARY

Conditioning can modulate even simple forms of motivated behavior. For instance, both the starting and the stopping of feeding can be conditioned.

Acquired drives are internal states that motivate action, and that occur as a result of learning. Acquired rewards are stimuli or situations that become incentives as a result of learning. Since biological drives account for only a fraction of behavior in humans and animals, we look to acquired motives to explain the rest.

A case of acquired drive is *avoidance learning,* in which an animal learns to prevent the occurrence of a painful stimulus such as shock. Since the response prevents the stimulus from occurring, there is the

[53]Rozin and Kennel, 1983.
[54]Rozin, Gruss, and Berk, 1979.
[55]Rozin and Schiller, 1980, p. 100

question of what maintains it—how can a non-event reinforce responding? The *two-factor theory* is an early behavioristic theory designed to answer that question. It proposes that an animal first learns to be afraid of a warning stimulus, such as a buzzer or tone. The conditioning of that *acquired drive* is the first of the two factors. Second, a successful avoidance response turns off the warning signal and reduces the fear. That is the second factor: Fear reduction reinforces avoidance responses. This theory has been applied in the clinic—for example, in understanding and treating compulsive behavior through response prevention.

But there are problems with the theory, for avoidance responding can be maintained with no warning stimulus and no stimulus change following the response. And there may be no evidence of fear, especially in well-trained rats. To some writers, such data argue for a cognitive interpretation of avoidance conditioning: We must consider not only what the rat's environment is like but what it expects and perceives. Behaviorist writers disagree, preferring simply to relate the rate of avoidance behavior to conditions in the environment. The theories draw together in *cognitive-behavioral theory,* which attempts to draw the best from both. For example, it applies conditioning procedures, borrowed from behavior research, to cognitive responses such as imagining scenes, as in desensitization therapy and covert sensitization.

There are instinctive constraints on avoidance learning. Animals and humans come to a learning experience already *prepared* to try certain responses to threat (Bolles's *species-specific defense reactions,* or SSDRs); to associate certain events with others, as mammals quickly associate taste with illness; and, in human children, to fear unfamiliar adult faces. In such cases, learning experiences tell the animal when to apply its biological rules—that *this* situation is a threatening one, or that *this* taste goes with illness, or that *this* face is not a familiar one. The rules themselves are untaught. And we apply them even when we know better. Perhaps the prevalence of certain kinds of phobias reflects a preparedness to fear certain kinds of objects.

Like acquired drives such as fear, acquired rewards were first studied as instances of conditioning. *Conditioned reinforcers* can be established in two ways: (1) by making some neutral object, like a poker chip, *exchangeable* for a natural or *primary* reinforcer such as food and (2) by simply presenting a neutral stimulus, such as a click, along with the primary reinforcer. Often in such cases, the neutral stimulus must predict the occurrence of the primary reinforcer; but there also is evidence that simple pairing will sometimes do, at least in humans. Such conditioned reinforcers can provide information, telling the actor that the right response has been made; they can guide subsequent behavior; they can bridge gaps in time, maintaining behavior even when the reinforcement is long delayed; and they may become pleasurable in their own right. However, there is the danger of *assuming* that a conditioning process is operative in all cases of learned rewards. This is unlikely.

Some acquired rewards clearly do not involve conditioning in the usual sense. They are not stimuli that are paired with, or that predict, natural rewards. Rather, they seem to be *assigned* positive value if they occur under certain conditions. In the case of *imprinting,* which occurs in some animals including ducks and geese, an arbitrary object becomes a reinforcer if it appears during a sensitive period when the animal is young. Or a stimulus may be assigned higher value simply because it has occurred before, the *mere-exposure effect.*

The *opponent-process theory* combines many of these ideas. A swing toward pleasure or displeasure (the A process) causes another process (the B process) that opposes it and outlasts it. Therefore, either happiness or unhappiness will be moderated by the opponent process; and when the mood-arousing circumstances are removed, a rebound will follow. It is assumed that the B process, but not the initial A process, is strengthened by repeated use, and can be conditioned as a response to environmental cues.

Similar opponent processes may operate in the nervous system. Addictive drugs have less effectiveness with repeated use; this is *tolerance.* When the drug is discontinued, the rebound reaction may be intense, producing *withdrawal symptoms.* Both tolerance and withdrawal could occur because the opponent process, which opposes the effect of the drug, is strengthened with repeated, frequent drug use. Moreover, tolerance may be in part a conditioned drug-opposed response to the situation, making drugs more potent and potentially dangerous in an unfamiliar environment.

Thus the mechanisms by which motives and goals are acquired form a very diverse list, and there probably are still more that we have not yet identified. Even with all these ideas to draw on, the problem of *acquired tastes* eludes a full explanation.