

# The Opponent-Process Theory of Acquired Motivation

## *The Costs of Pleasure and the Benefits of Pain*

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**ABSTRACT:** When an unconditioned stimulus, a reinforcer, or an innate releaser is repeatedly presented to human or animal subjects, three major affective phenomena are often observed. First, one often sees affective or hedonic contrast. Second, frequent repetition of the unconditioned stimulus, reinforcer, or innate releaser often gives rise to affective or hedonic habituation (tolerance). Finally, after frequent repetition of these stimuli, a withdrawal or abstinence syndrome often emerges directly following stimulus termination. These affective dynamics of organismically important stimuli generate new motives, new opportunities for reinforcing and energizing operant behaviors, based on the hedonic attributes of withdrawal or abstinence syndromes. This article describes the opponent-process theory of such new or experientially produced motives and discusses recent research testing the theory. The theory attempts to account for such diverse acquired motives as drug addiction, love, affection and social attachment, and cravings for sensory and aesthetic experiences (cases in which the initial reinforcers are positive) and for such acquired motives as parachuting, jogging and "marathoning," sauna bathing, and a variety of self-administered, aversive stimuli like electric shocks (cases in which the initial reinforcers are negative). The empirical laws governing the establishment of these new motives are described. Crucial variables include the quality, intensity, and duration of each stimulus presentation and the time intervals between presenta-

tions (interreinforcement intervals). The theory also gives a plausible account of the development of addictive behaviors, whether initiated by pleasurable or by aversive events.

How strange would appear to be this thing that men call pleasure! And how curiously it is related to what is thought to be its opposite, pain! The two will never be found together in a man, and yet if you seek the one and obtain it, you are almost bound always to get the other as well, just as though they were both attached to one and the same head. . . . Wherever the one is found, the other follows up behind. So, in my case, since I had pain in my leg as a result of the fetters, pleasure seems to have come to follow it up.

—Plato, *Phaedo*

Acquired motives can be as powerful as innate ones. They can become the focus for the major behaviors of an organism, even at the expense of innate needs. A heroin addict, for example, may spend the better part of each day in drug-seeking behavior, may ignore food, liquid, and sexual incentives, and may abandon normal societal obligations. The heroin motive is acquired only because certain experiences have occurred; it is not innate. We tend to think of such addictions as pathological, but they are not. One thesis of this article is that most acquired motives, such as love, social attachments, food-taste cravings, thrill seeking, and needs for achievement, power, and affiliation, obey the empirical laws for the addictions. I develop a theory that explains why this is so and describe recent research testing the theory.

When Miller (1951) wrote his important essay on "learnable drives and rewards," the concept of acquired motivation was an *associative* one. By processes of Pavlovian conditioning, by means of

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contingencies between conditioned stimuli (CSs) and unconditioned stimuli (UCSs), previously neutral stimuli could acquire some of the attributes of UCSs. Some UCSs had drive properties, some had reward properties, and the CSs acquired the appropriate properties—either to motivate or energize behaviors or to reinforce specific behaviors selectively. The stimuli rendered no longer neutral by such conditioning were called either acquired drive stimuli or conditioned reinforcers (secondary reinforcers), depending on the attributes of their UCSs. In a real sense, no new drives or incentives were created, but new stimuli, once neutral, could now elicit drivelike effects or rewardlike effects. The related phenomena were sometimes called “derived motives” (Bolles & Moot, 1971; D’Amato, 1974). The derived motives were thus believed to be a consequence of associative or conditioning processes.

In a similar vein, Freud and the neo-Freudians considered acquired motives to be derived from innate needs, and associative processes were the focus of clinical attention. The search in this case was not for CSs but for symbols, though the underlying ideas were the same (Hall & Lindzey, 1957). People were driven by, or rewarded by, symbols that were derived from the dynamics of the libido, the innate needs of the organism.

The phenomenon of addiction does not easily fit the old associative-process theories about derived motives. My argument is that many new motives are of the addictive type, not necessarily of the derived type, and that associative processes, though often occurring in these cases, are neither necessary nor sufficient to produce the addictive behaviors. The clearest case is, of course, opiate addiction, in which one sees the emergence of a powerful new motive characterized by drug craving and abstinence agony. Such phenomena do not appear to require either Pavlovian conditioning or symbol formation for their establishment. Furthermore, I believe that the addictive type of acquired motive is representative of many, if not most, of the acquired motives that are the major features of human behavior in a social context. My arguments, therefore, pertain to social psychology and developmental psychology as well as to personality theory.

I start with some very dogmatic claims, but I believe they are right. In every case of acquired motivation, *affective* or *hedonic* processes are involved; whenever one identifies an acquired mo-

tive, one can, in every case I have found, describe or measure three affective or hedonic phenomena. These are (a) affective or hedonic *contrast*, (b) affective or hedonic *habituation* (tolerance), and (c) affective or hedonic *withdrawal* (abstinence) *syndromes*.

## Empirical Generalizations

### AFFECTIVE CONTRAST

Following is an example of affective, emotional, or hedonic contrast that occurs when a positive reinforcer is presented and removed. A powerful, species-specific unconditioned stimulus is presented to a laboratory animal. In this particular case, a moving mother duck is presented to a 5-hour-old duckling for the first time in its life. The duckling becomes excited, stumbles in the general direction of the mother, and moves its head quickly so that the mother is kept in sight. After 1 minute elapses, the mother is removed. Then, with a latency of 5–10 seconds, the duckling shows agitated head movements and energetic, seemingly random locomotor activity and emits a repetitive, high-pitched sound referred to as *distress calling*. These distress calls persist for several minutes and then cease.

One can show that the introduction of the moving mother duck functions as a positive reinforcer (see Hoffman, Searle, Toffey, & Kozma, 1966; Hoffman, Stratton, Newby, & Barrett, 1970) because the shaping of an arbitrary operant occurs if the presentation of the moving mother is contingent upon the occurrences of that operant. In opposite fashion, the removal of the mother negatively reinforces any already established operants, and, indeed, even species-specific, instinctive behavior chains can be weakened by this procedure (see Hoffman, Stratton, & Newby, 1969). Therefore, one can infer that the presentation of the reinforcer engenders a pleasant or desirable hedonic state but that the termination of the reinforcer results in an aversive or unpleasant hedonic state that finally ceases after several minutes of stimulus absence. This affect sequence (baseline state → State A → State B → baseline state) characterizes hedonic or affective contrast. State A and State B appear to be in a contrasting relation to each other with regard to their reinforcing properties.

Affective contrast engendered by positive reinforcers occurs in a variety of settings. It seems

to be the rule, not the exception. A few years ago, I became curious about the generality of affective contrast in humans. With the assistance of Reuben Kron, I carried out a brief, incomplete experiment in the baby nursery of Philadelphia General Hospital. I presented a nursing bottle to several sleeping babies who were about 12 hours old. Such babies usually are not hungry or thirsty because they are still digesting a large quantity of amniotic fluid. However, if one wiggles a nursing nipple into their mouths, they wake up and suck, ingesting some of the nutrient (especially if it is sweet). When I allowed them to suck for 1 minute and then withdrew the nipple, the obvious of course occurred: The babies started crying with a latency of 5–10 seconds, cried for several minutes, and then went back to sleep. The babies would not have cried at that time had I not introduced the nipple and withdrawn it. Affective contrast can therefore occur whether or not the positive reinforcer or UCS is “needed” at the time. In this experiment, one can infer that the babies went from baseline state → State A → State B → baseline state. Stimulus termination precipitated both the ducklings and the infants into an aversive state, one in which they would not have been had the positive reinforcer or UCS not been introduced and withdrawn. Indeed, it is possible that neither duckling nor infant could ever have experienced their particular affective State B during the presence of any known variety of direct stimulation.

Next is an example of affective or hedonic contrast that occurs when a negative reinforcer is presented and then removed. It comes from Epstein's (1967) report of work on the emotional reactions of military parachutists. During their first free-fall, before the parachute opens, military parachutists may experience terror: They may yell, pupils dilated, eyes bulging, bodies curled forward and stiff, heart racing and breathing irregular. After they land safely, they may walk around with a stunned and stony-faced expression for a few minutes, and then they usually smile, chatter, and gesticulate, being very socially active and appearing to be elated. Here again, one sees the affect sequence: baseline state → State A → State B → baseline state.

Or, consider the following example, quoted from an article on the effects of being struck by lightning:

My interest in this subject was aroused when my neighbor's son was struck by lightning as he was returning

from a golf course. He was thrown to the ground. His shorts were torn to shreds and he was burned across his thighs. When his companion sat him up, he screamed “I'm dead, I'm dead.” His legs were numb and blue and he could not move. By the time he reached the nearest hospital he was euphoric. (Tausig, 1969, p. 306)

Need I say more? Well, perhaps. It is possible that neither Epstein's nor Tausig's behavioral description is decisively convincing to the reader. Therefore, my next example is semiphysiological and may thus be, to some, more acceptable. We put laboratory dogs in a Pavlov harness and presented a frightening shock to their hind feet, measuring the unconditioned heart rate changes (Church, LoLordo, Overmier, Solomon, & Turner, 1966). The shock lasted 10 seconds.

Figure 1 shows the heart rate reaction during the onset, maintenance, and termination of the shock, as well as during the minutes following shock termination. The upper panel shows the large heart

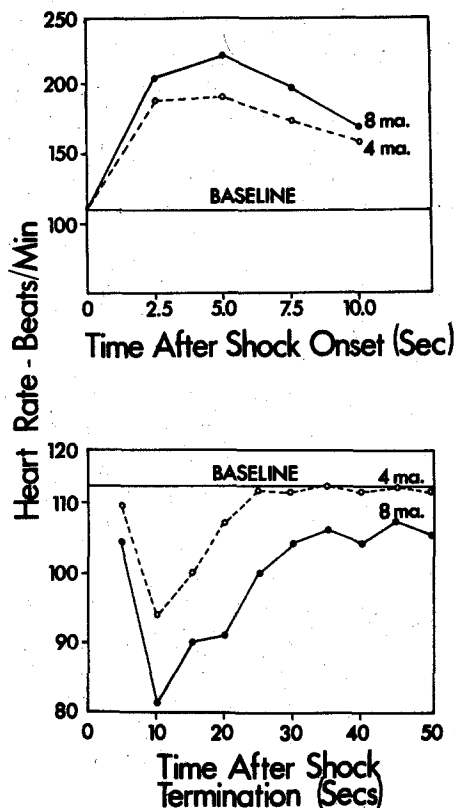


Figure 1. The unconditioned heart rate response in dogs during a 10-second shock (ma. = milliamperes) to the hind paws (upper panel) and after the shock is terminated (lower panel). (Note the change in scale for the ordinate in the lower panel. Adapted from Church, LoLordo, Overmier, Solomon, and Turner, 1966.)

rate increase that occurred when the shock went on. This increase reached a peak after about 5 seconds of shock, and the rate then declined, even while the shock was still on. The lower panel shows the heart rate changes that occurred directly after the 10-second shock was terminated. Heart rate first decreased to a level below the original baseline and then recovered slowly to the original baseline. The rate did not simply decline from its peak level directly back to the baseline. Assuming, of course, that heart rate changes measure affective changes, one can infer affective contrast in this example.

Of the four instances I have given of affective contrast, the heart rate data (Church et al., 1966) will serve as our general guide. However, data from color vision (Hurvich & Jameson, 1974) provide an important analogy. Turn on a relatively pure red light and keep it on for 30 seconds. At first the red appears to the observer to be rich and saturated. As the seconds go by, however, the redness seems to decrease, as though one had mixed white light with the red. Now turn the red light off. The observer experiences a green afterimage that peaks in saturation immediately and then slowly dies away until the greenness is undetectable. Many data sources suggest that there is probably a similar or analogous standard pattern of affective dynamics for hedonic stimuli.

This standard pattern is idealized in Figure 2; it contains five distinctive features. First, when the stimulation begins, there is a rapid departure from baseline affect, which peaks within a few seconds (State A). Next, the affect intensity or magnitude

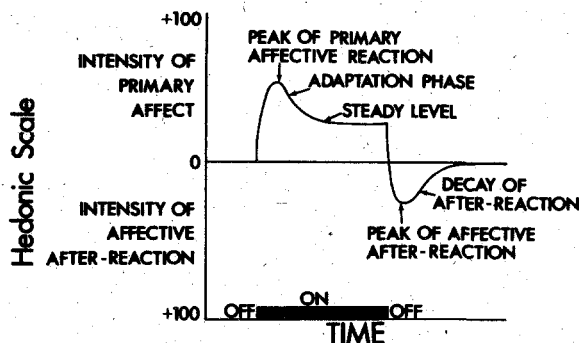


Figure 2. The standard pattern of affective dynamics. Note the five labeled distinctive features of the complex affective outcome produced by a simple square-wave stimulus input. (From "An Opponent-Process Theory of Motivation: I. Temporal Dynamics of Affect" by R. L. Solomon and J. D. Corbit, *Psychological Review*, 1974, 81, 119-145. Copyright 1974 by the American Psychological Association. Reprinted by permission.)

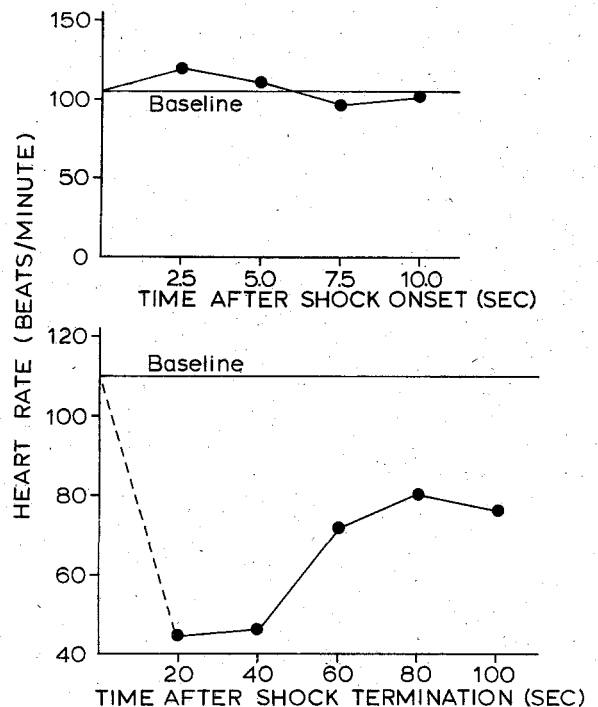


Figure 3. The heart rate response of a "veteran" laboratory dog to shocks to its hind feet. (Note the virtual absence of an acceleratory phase in response to shock onset and the exaggerated deceleratory response after shock is terminated. Adapted from Katcher et al., 1969.)

starts to decline, even while the precipitating stimulus is still present. The decreased State A affect then approaches a relatively steady level. When the stimulus event is terminated, there is a quick, phasic decrease in the affect level until the baseline is crossed, and then a new, contrasting affective state (State B) emerges, which quickly approaches a peak and then slowly decreases in magnitude until the original affective baseline is reestablished. There is no overshoot into State A after State B has slowly died away. This pattern is assumed to be a consequence of either pleasurable or aversive reinforcers, releasers, or UCSs.

#### AFFECTIVE HABITUATION

In the example I gave of dogs having their hind feet shocked and heart rates measured, it was easy to observe a contrast effect, and the habituation process was unambiguous. The heart rate slowed after its initial acceleration, even while the UCS was still there. Furthermore, as shown in Figure 1, the contrast effect was evidenced in the comparison between the 4-mA and 8-mA shock conditions. The peak of the *on* response to 4 mA was of a lower

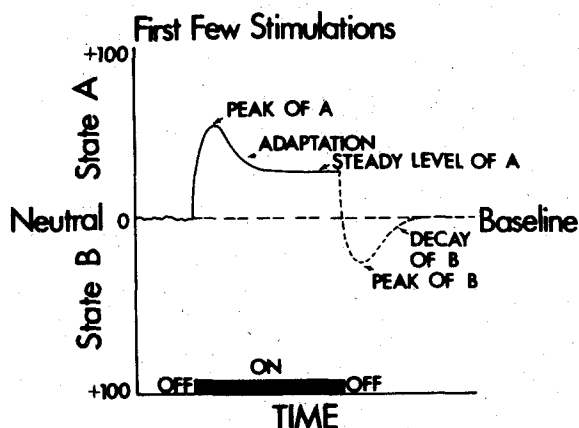


Figure 4. The standard pattern of affective dynamics produced by a relatively novel unconditioned stimulus.

amplitude than that for 8 mA. More important, the peak of the after-reaction showed a greater departure from baseline for 8 mA than for 4 mA. This is the symmetry one would expect for contrast phenomena. However, it turns out that this initial symmetry is misleading. It is characteristic only of the nonhabituated subject. The habituated subject not only fails to show a contrast effect but also shows an asymmetrical reverse. This is illustrated in Figure 3, which shows the heart rate of a "veteran" laboratory dog after it had received hundreds of shocks over a period of many days (see Katcher et al., 1969). The increase in heart rate at shock onset was very small, yet the decrease in heart rate after the shock was terminated was very large, and the time required for return to baseline heart rate was relatively long. As far as the heart was concerned, the dog had become "used to" or "tolerant of" shocks to its hind feet. From this one can infer that affective tolerance or habituation occurred to some extent.

In general, when a UCS of medium intensity is repeated many times within relatively short periods of time, the affective reaction to that UCS often diminishes (see Kimmel, 1971). This generalization is meant to apply to either positive or negative reinforcers, to UCSs or releasers of either an exteroceptive or an interoceptive (drugs, chemicals) sort. (For a review, see Randich & LoLordo, 1979.)

#### AFFECTIVE WITHDRAWAL SYNDROMES

In Figure 3 one can see that the veteran dog, which had received a large number of frightening shocks, no longer showed much cardiac arousal when shocks

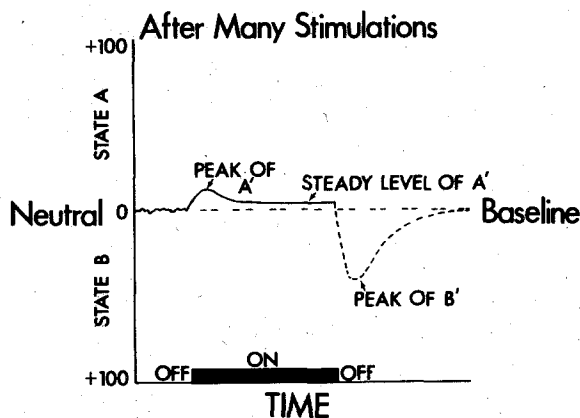


Figure 5. The standard pattern of affective dynamics produced by a familiar, frequently repeated unconditioned stimulus.

were present. Furthermore, the deceleration of heart rate below the baseline is of greater magnitude than it was during the first few shocks received. In addition, this after-UCS reaction is of quite long duration relative to that seen in Figure 2. The emergence of a long-lasting, high-amplitude affective after-reaction is one characteristic consequence of a frequently repeated UCS.

It is now possible to portray the standard patterns of affective dynamics for two conditions—one in which the UCS event is new and one in which the UCS event has been repeated many times. Figures 4 and 5 show the habituation effect occurring during a UCS and the enhancement or elaboration of the withdrawal syndrome occurring after UCS termination, both a consequence of repeated stimulations.

The phenomena illustrated in Figures 4 and 5 occur in a wide variety of situations for a very large number of UCSs. In Tables 1–4, I present but a few of the examples available from the experimental psychology laboratory, the psychological clinic, and the arena of everyday life. The students in my research seminar have over a period of years called to my attention over 50 such examples of the three highly correlated effects of an often repeated UCS: (a) The affective reaction to the onset and maintenance of the UCS will gradually decline; (b) the affective after-reaction will grow in intensity and duration; and (c) a distinctive affective quality of the after-reaction will often emerge, and it will appear to be hedonically opposite to that quality which was engendered by the onset and maintenance of the UCS during the first few presentations.

Tables 1-4 are arranged to contrast the affective events that take place during presentations of a reinforcer on early trials with those that take place after many repetitions of the reinforcer. They are also arranged to point out the typical affects that occur before, during, and after each presentation of a particular reinforcer.

Before I discuss particulars concerning the prototypes in Tables 1-4, the relationship of the generalizations (derived from these tables) about the dynamics of affect, on the one hand, to the generalizations about the motivation concept, on the other, needs to be mentioned. What do the laws of affect have to do with motivation? Put very simply, for a large set of acquired motives, the laws of affect are the laws of acquired motives. The reasons for this are fairly simple. Repetition of a reinforcer changes the hedonic or reinforcing potency of that reinforcer and results in the emergence of a new reinforcer, which occurs after the termination of the original reinforcer. The new reinforcer has a hedonic quality opposite to that of the original reinforcer's onset. So new or acquired motives arise from the dynamics of affect, as shown in Tables 1-4.

### *Some Prototypes of Acquired Motivation*

Table 1 outlines the affective dynamics in opiate use. The first few self-doses of an opiate (if the dosage is the right size) produce a potent pleasure called the "rush," followed by a less intense state of euphoria. This effect can be shown in people (Wikler, 1953), in monkeys (Deneau, Yanagita, & Seevers, 1969), and in rats (Kumar, Steinberg, & Stolerman, 1968), using various objective behavioral indices. The rush has been characterized by people as an intense sexual pleasure felt "all over the body, all at once." When the drug dose loses its effect

because of metabolic destruction, the user goes into a state of mild discomfort with both physiological and psychological aspects. The physiological aspect includes runny eyes and nose, abdominal pains, clammy skin, and muscular malaise. The psychological aspect is called craving and refers to an aversive state. Most organisms will perform an operant if it will get rid of an aversive state. Thus, drug users tend to redose because this is the surest and quickest way to get rid of the physiological and psychological aspects of withdrawal aversiveness. A slower way is merely to let time go by because the withdrawal aversiveness will slowly die away; but this method is less preferred.

The first few self-doses produce a pattern of motivational influences highly correlated with the standard pattern of affect dynamics shown in Figure 2. The onset and maintenance of the opiate first produce a peak of State A (the rush), followed by a decline in intensity (euphoria), the first sign of habituation. Drug-event onset functions as a positive reinforcer. Then, after the drug "wears off," State B, an aversive craving state called the withdrawal syndrome, emerges. Finally, the aversiveness disappears with the passage of time. In such a case, there are two motivational events capable of reinforcing operants: the onset of State A and the removal of State B.

If self-doses are frequently repeated, however, two correlated changes in affect then occur: (a) The rush is no longer experienced and euphoria is often absent (loss of euphoria); and (b) the withdrawal syndrome becomes much more intense, both physiologically and psychologically, and its duration lengthens dramatically. Thus, the positive reinforcer loses some of its power, but the negative reinforcer gains power and lasts longer. This effect is shown in the right-hand column of Table 1, under the heading After Many. Thus, the motivation in drug use changes gradually, with successive doses, from positive to aversive control. The user not only becomes drug tolerant but also becomes more intolerant of drug termination or absence.

In Table 1, the reinforcer is positive, and the acquired or new motivational state (State B) is aversive. The same generalization holds for Table 2.

Table 2 describes the behavior of ducklings when they are first exposed to a powerful innate reinforcer—a moving mother surrogate. They show a high level of excitement and increased locomotion, and their eye movements follow the moving object. When the reinforcer is removed, duration of distress calling is related to prior exposure duration, thus

TABLE 1  
*Changes in Affect Before, During, and After Each Stimulation (Self-Dosing With Opiates) for the First Few Experiences and After Many Experiences*

Period	First few	After many
Before	Resting state	Craving
During	Rush, euphoria	Contentment
After	Craving	Abstinence-agony
	Resting state	Craving

TABLE 2

*Changes in Affect Before, During, and After Each Stimulation (Social Attachment in Ducklings) for the First Few Experiences and After Many Experiences*

Period	First few	After many
Before	Contentment	Some distress
During	Excitement	Following
After	Distress	Intense distress
	Contentment	Some distress

demonstrating the contrast effect (Eiserer & Hoffman, 1973). After many exposures, the excitement level declines during an exposure period, but after stimulus termination, the intensity and duration of distress calling increase. Sometimes, after many repeated exposures to a mother surrogate, the duckling emits distress calls sporadically during several days of the mother's absence, though this behavior eventually ceases (see Hoffman, Eiserer, Ratner, & Pickering, 1974). Presentation of the mother surrogate has been shown to be a positive reinforcer (Hoffman & Ratner, 1973), and removal of the mother surrogate has been shown to function as a negative reinforcer. A new source of aversiveness is thus added to the ducklings' existence (Hoffman et al., 1969). This new motivational problem would never arise for the ducklings if they never saw a mother surrogate.

In Tables 1 and 2, the reinforcer is positive, and the acquired motivational state (State B) is aversive. In contrast, in Tables 3 and 4, the reinforcer is aversive, and the acquired motivational state (State B) is positive.

Table 3 describes the affective events that occur as a consequence of sauna bathing. The air is very hot, and during the first few repetitions the typical bather reports painful burning sensations and unpleasant affect, even fear. A short-lasting feeling of relief is reported after the session is

TABLE 3

*Changes in Affect Before, During, and After Each Stimulation (Sauna Bathing) for the First Few Experiences and After Many Experiences*

Period	First few	After many
Before	Resting state	Resting state
During	Pain, burning	Hot, exciting
After	Relief	Exhilaration
	Resting state	Resting state

over. Unless some extraneous motivation coming from social pressures, beliefs about health, and so on is provided, the bathing sessions are not likely to be repeated because their A state is aversive. However, if the behavior is repeated, two changes occur: The aversiveness of each session gradually declines, and a withdrawal syndrome emerges and intensifies. The withdrawal syndrome is characterized by sauna bathers as "exhilaration" and "a sense of well-being." This B state usually lasts from 1-2 hours in a well-habituated sauna bather. The sauna bather, by repeated exposures to a previously aversive stimulus pattern, now has a new source of pleasure; an acquired motive system has emerged.

Table 4 shows the pattern of affective changes for military parachutists (Epstein, 1967). During the first free-fall (before the parachute opens), even the bravest men show a fearful reaction. Their eyes may bulge, their lips retract, they may yell, their bodies curl forward, and their autonomic nervous systems are in a high state of excitation. There may even be involuntary urination. After they have landed safely, they go through a short period during which they look stunned and stony faced and do not talk much. Then, after a few minutes, they begin to chatter and enter into lively social interaction with peers. The after-reaction appears to last about 10 minutes. After many parachute jumps, the signs of affective habituation are clear, and the fearful reaction is usually undetectable. Instead, the parachutists look tense, eager, or excited, and during the free-fall they report a "thrill." After a safe landing, there is evidence of a withdrawal syndrome. The activity level is very high, with leaping, shouting, rapidly shifting social interactions, voluble talk, and general euphoria. This period, often described as exhilaration, decreases slowly in time, but often lasts for 2-3 hours. Indeed, I was once told by

TABLE 4

*Changes in Affect Before, During, and After Each Stimulation (Free-Fall in Military Parachuting) for the First Few Experiences and After Many Experiences*

Period	First few	After many
Before	Anxiety	Eagerness
During	Terror	Thrill
After	Relief	Exhilaration
	Resting state	Resting state

a sport parachutist (who was also a psychiatrist) that his "high" lasted 8 hours. A new, positive source of reinforcement is now available, one that could never have eventuated without repeated self-exposures to an initially frightening situation to which the subject then becomes accustomed.

The four prototypes described above are a fair sample of the different kinds of UCS that elicit affective, emotional, or hedonic reactions and that show the phenomena of (a) initial affective contrast; (b) affective habituation, or tolerance to the presence of the UCS; (c) the emergence of an affective withdrawal syndrome; and (d) a relation of affective oppositeness or reinforcer oppositeness between the states characteristic of the presence and absence of the UCS. A satisfactory theory is needed to account for these phenomena.

It is important to work out a useful theory because many instances of acquired motivation seem to fit the characteristics illustrated in Tables 1-4. First there are the drug UCSs, many of which produce an addictive cycle in which the drug user self-doses in order to terminate or prevent the aversive withdrawal syndrome. Such self-dosing then produces more tolerance to the drug and a more intense and longer lasting withdrawal or abstinence syndrome. Drugs such as opiates, alcohol, barbiturates, amphetamines, and bromides seem to reproduce well the four phenomena I have emphasized above. Then there are the aversive, nondrug exteroceptive and interoceptive UCSs. Many of these appear to be capable of producing tolerance, a pleasurable withdrawal syndrome, and new operant behaviors reinforced by the withdrawal syndrome affect (i.e., a new, acquired motive system). Some examples are "marathonning" (Milvy, 1977), jogging (Booth, Note 1), various feats of daring that are initially frightening, various disgusting stimuli (e.g., human anatomy class), various irritating stimuli such as tobacco smoke, and even some painful stimuli produced in sports. Certainly, hard work might be an example. A great deal of empirical investigation is needed to ascertain just which UCSs, reinforcers, or releasers, originally aversive and functioning as negative reinforcers, lead to the development of a new, positive reinforcer. Finally, there are the pleasurable UCSs, reinforcers, and releasers. Many of these seem to be subject to habituation or tolerance, and they can, after much repetition, generate an aversive withdrawal syndrome.

Such syndromes can then energize the develop-

ment of escape and avoidance behaviors and thus constitute an acquired motive system. I believe that many of the most pleasurable reinforcers in life fit the present description: love, social attachments, power and competence, achievement and recognition, and aesthetic pleasures. In everyday use are many terms for the withdrawal syndromes based on such reinforcers: loneliness, grief, sorrow, disappointment, and cravings for love, power, beauty, or sensory experiences (e.g., taste cravings). We do not yet know how many of these motivational influences demonstrate the phenomena I have pointed out. I suspect that most of them do, which leads to further interest in a theory that would, in principle, account for such acquired motives.

### *The Opponent-Process Theory of Acquired Motivation*

It turns out that the simplest, yet quite satisfactory, theoretical model for all of the affective or hedonic dynamics I have described is an affect-control system with a single negative feedback loop. It is an opponent-process theory, similar to many already described by physiologists and psychologists (see Hurvich & Jameson, 1974).<sup>1</sup>

The theory assumes that for some reason the brains of all mammals are organized to oppose or suppress many types of emotional arousals or hedonic processes, whether they are pleasurable or aversive, whether they have been generated by positive or by negative reinforcers. The opposing affective or hedonic processes are automatically set in motion by those stimulus patterns that psy-

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<sup>1</sup>The opponent-process theory is new, though it has been a central topic of my research seminar since 1970. It was described at the Psychonomic Society in 1972 and in several later articles (Hoffman & Solomon, 1974; Solomon, 1977a; Solomon, 1977b; Solomon & Corbit, 1973; Solomon & Corbit, 1974; Starr, 1978). The theory was elaborated and sharpened by John D. Corbit and Howard S. Hoffman and was reviewed by D'Amato (1974). It has recently been applied to job satisfaction (Landy, 1978), to fear conditioning (LaBarbera & Caul, 1976a, 1976b), to cigarette smoking (Pomerleau, 1979; Ternes, 1977), to fear-inhibitory associations (Maier, Rappaport, & Wheatley, 1976), to tonic immobility reactions (Berns & Bell, 1979), to tolerance to opiates (Siegel, Hinson, & Krank, 1978), to gastric ulcer formation (Desiderato, MacKinnon, & Hissom, 1974), to opiate addiction (Wikler, 1973), to sucrose preference (Cohen, Note 2), to eating (Cantor & Wilson, Note 3), to peer separations in monkeys (Suomi, Mineka, & Delizio, in press), to addiction to jogging (Booth, Note 1), and to habituation to test anxiety in college students (Craig & Siegel, 1980).

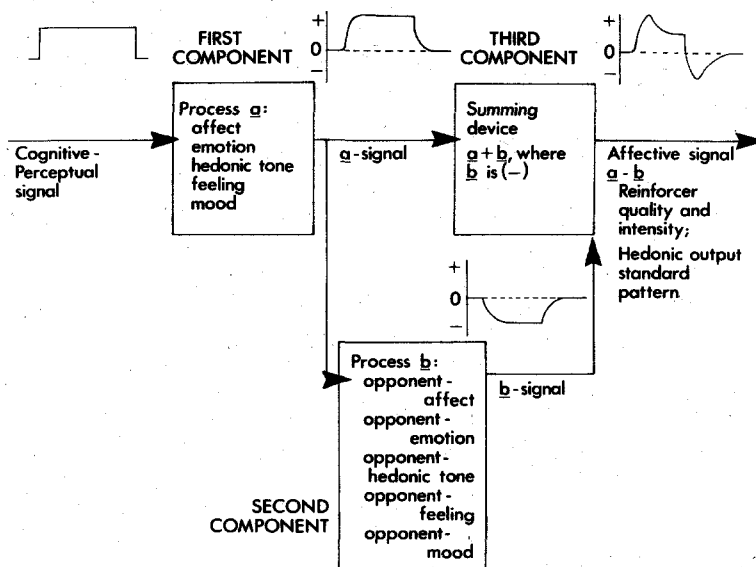


Figure 6. The affect-processing system for the opponent-process control of affect. (The arousal of primary process [*a* process] arouses an opponent process [*b* process], which subtracts from the resultant affect intensity through the action of an affect summator. Because the *b* process is of relatively long latency, slow buildup, and slow decay, the summator will yield the complex, standard pattern of affective dynamics shown in Figure 2.)

chologists or ethologists have shown, through defining experiments, to function as Pavlovian UCSs, operant reinforcers, or innate releasers.

All primary affective or hedonic processes elicited by UCSs, reinforcers, or innate releasers are postulated to correlate closely in magnitude with the stimulus intensity, quality, and duration of the reinforcer. These primary processes are phasic and are sensitive to small stimulus changes. They are stable, unconditioned reactions, which in the theory are called *a* processes. For example, a snake (UCS) elicits a reflex fear reaction (UCR) in a monkey. Or, the taste of chocolate syrup (UCS) elicits salivation (UCS) or excitement (UCR) and a pleasure state (UCR) in a child.

The primary process, the *a* process, in turn arouses a *b* process that functions to oppose and suppress the affective or hedonic state initially generated by the onset of the *a* process. The *b* process drags down the strength of an *A* state. The *b* process (the opponent process) is postulated to be (a) of sluggish latency, (b) inertial, or slow to build to its asymptote, and (c) slow to decay after the stimulus input (UCS) has been terminated and the *a* process (UCR) has stopped. Because the *b* process is an opponent process, its affective or hedonic quality must be opposite to that of the *a* process. The implications of such a simple assumption are far-reaching, as one will see.

The affective or hedonic state of the organism at any moment is postulated to be the difference, without regard to sign, between the magnitude of the *a* process and the magnitude of the *b* process. The *b* process has a negative sign because it opposes the *a* process. The *state rule* is simple: (a) If  $|a - b|$  shows  $a > b$ , then the organism is in State A, and (b) if  $|a - b|$  shows  $b > a$ , then the organism is in State B. Furthermore, if being in State A is positively reinforcing (pleasant, desirable), then being in State B will be negatively reinforcing (aversive, undesirable), and vice versa.

The affect-processing system, reflecting the opponent-process assumptions made so far, is shown in Figure 6. First, there is a cognitive-perceptual event representing the UCS, reinforcer, or innate releaser. For illustrative purposes, assume that the subject is a cat and that the incoming signal is categorical—a dog. The dog can be depicted as a square-wave input. One of its side effects is the arousal of an *a* process, a primary affective or hedonic process. In this case, the *a* process is a fear reaction pattern. The occurrence of this reaction pattern then results in arousal of a *b* process, the opponent process. It has an affective or hedonic sign opposite in quality to that of the *a* process. At this point, one can only guess at what the quality of the opponent really is. As one sees shortly, its quality is only revealed when

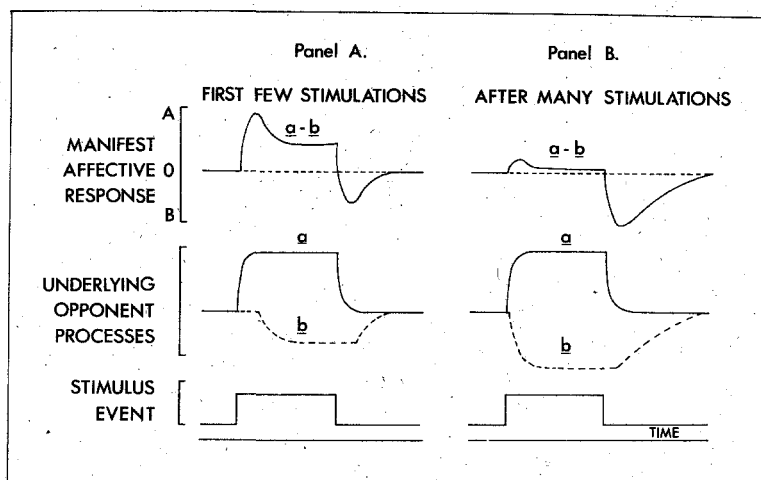


Figure 7. The comparison of the effects of  $b$  processes for relatively novel unconditioned stimuli and for unconditioned stimuli that are familiar and have frequently been repeated. (Note that the strengthening of the  $b$  process is assumed to shorten its latency, increase its asymptotic value, and lengthen its decay time. The standard patterns of affective dynamics shown in Figures 4 and 5 are produced by differing  $b$ -process strengths.)

the categorical stimulus event is terminated, just as in the case of a color stimulus whose opponent color is revealed when the stimulus is turned off.

The magnitudes and qualities of the  $a$  process and  $b$  process are fed to a summator that computes  $|a - b|$  for any moment. The summator determines whether the subject is in State A or State B as well as the quality and intensity of those states. At UCS onset, most  $a$  processes are more intense than their opposing  $b$  process, which as I have indicated, has a slow buildup relative to that of the  $a$  process. However, the slow buildup of the  $b$  process produces a gradual decrease in the amplitude of the A state even while the UCS, the dog, is still present. The cat looks less fearful as time goes by and appears to grow "accustomed" to the dog's presence.

When the dog goes away, there is no categorical stimulus to maintain the  $a$  process, and so it quickly subsides to zero. The  $b$  process, however, being sluggish and slow to decay, continues for some time. The peak of quality and intensity of the B state reveals itself directly after UCS termination, when the  $a$  process decreases to zero. The B state then slowly decays or subsides. The cat may look relieved or relaxed, may show a typical feline after-reaction of pleasure, and then slowly returns to equanimity.

The processing system deduces several features in Figures 1 and 2, the standard pattern of affective dynamics. However, an additional assumption is needed before the system can generate the

effects of many repeated presentations, as shown in Figures 3 and 5 and in the right-hand columns of Tables 1-4. The model must be able to produce the habituation effect as well as a new, strong withdrawal syndrome. It can do so if one postulates that  $b$  processes are strengthened by use and weakened by disuse. How this would work in the processing model is shown in Figure 7, which compares  $b$  processes and the resultant affective states during the first few UCS presentations and after many UCS presentations. The growth of the strength of the  $b$  process with repeated presentations of the UCS has two consequences: (a) The sum  $|a - b|$  during the onset and presence of the UCS is decreased, but (b) the sum  $|a - b|$  right after cessation of the UCS is increased. This fits the present empirical generalizations quite well.

### *New Experiments Pertinent to the Opponent-Process Theory*

It is one matter to organize empirical generalizations about the dynamics of affect into a coherent theory of acquired motivation. It is quite another matter to test new deductions from such a theory and to find new problems and questions inspired by that theory. Such a challenge has been exciting. In the past five years, members of my research seminar have pushed experimentation in several directions in an attempt to refute the theory, to see where revisions are needed, and to

explore the generality of the theory. At the same time, in other laboratories, several new findings have been used to test the validity of deductions from the theory. In addition, several other findings, although not initially intended to test the theory, have served this purpose. It appears that the concept of opponent process or compensatory process (see Siegel, 1977) is now in vogue.

The experiments can be classified as follows: (a) the growth of opponent processes in social attachment; (b) the modulation of ongoing aversive affect by introduction of events that produce either A states or B states (here one question is whether A states and B states are really opposites); (c) the conditioning of A states and B states; and (d) the role of endorphins in the opponent processes for aversive events. I start with our social attachment experiments because these have taught us a great deal about opponent processes.

*Growth of social attachment (imprinting) in ducklings.* Imprinting has been characterized as an all-or-nothing, irreversible, innate learning event of surprising suddenness (Lorenz, 1935). It occurs when a newly hatched precocial bird such as the chick or duckling is first exposed to a moving object (or mother surrogate or mother). The hatchling becomes excited, looking at the moving object and often staggering toward it. Thereafter, the hatchling develops more and more skilled locomotor behavior and stays close to, or follows after, the moving object.

A striking feature of imprinting is the affective reaction of a duckling when the imprinting object is suddenly removed. The animal at first exhibits a double take, a perceptual startle with a very short latency. It then becomes very active, appearing to be searching for the lost object. Finally, after a 5–10-second latency, it emits high-pitched cries, or distress calls. These distress calls can vary in the frequency with which they occur in time and in duration (Hoffman et al., 1974). They have been used as an index of degree of social attachment, much in the same way that severity and duration of opiate withdrawal symptoms have been used to index the degree of physiological and psychological dependence on heroin or morphine. If one assumes that distress calls are an index of a *b* process, an opponent caused by the presentation of a highly reinforcing or innate releasing stimulus, then certain phenomena should occur.

First, the presentation and removal of an imprinting object should have opposite reinforcing effects. This is so. Hoffman et al. (1966) showed that arbitrary operants could be shaped by presentations of an imprinting object. Furthermore, Hoffman et al. (1969) showed that removal of the imprinting object functioned effectively in a punishment contingency to weaken an arbitrary operant. Second, rather than being all-or-none, or “released,” imprinting should instead develop gradually in strength as the *b* process is exercised by use; and it should wane in strength as the *b* process is weakened by disuse.

After planning sessions with members of my seminar, the research group at Bryn Mawr (Hoffman et al., 1974) designed and conducted the first experiment on the growth of an aversive opponent process in imprinting. They showed that with 1-minute exposures alternated with 1-minute removals of an imprinting stimulus, the amount of distress calling per unit of time gradually increased. The findings presented in Figure 8 demonstrate that the opponent process in imprinting is strengthened by use. One is thus led, as others have been, to question the all-or-none characterization of the imprinting process based on the distress-calling measure (see Scott, 1972, pp. 198–200). Furthermore, one can now safely assume

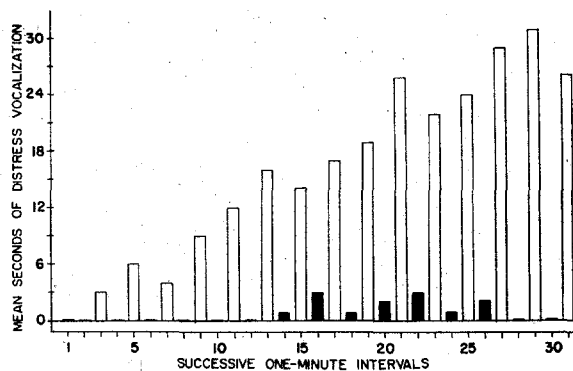


Figure 8. The growth of the opponent process (indexed by intensity of distress calling) for the action of an imprinting unconditioned stimulus. This growth function was produced by alternating 1-minute presentations of the unconditioned stimulus with 1-minute absences of it. (From “Development of Distress Vocalization During Withdrawal of an Imprinting Stimulus” by H. S. Hoffman, L. A. Eiserer, A. M. Ratner, and V. L. Pickering, *Journal of Comparative and Physiological Psychology*, 1974, 86, 563–568. Copyright 1974 by the American Psychological Association. Reprinted by permission.)

that "following" behavior is not what is "released" in the imprinting process. Instead, the released behavior is an affective reaction, an innate *a* process with positive reinforcement attributes. One can use this *a* process to shape arbitrary operants. Indeed, so-called following behavior itself may function as an operant. Hoffman et al. (1970) actually taught ducklings to go away from the imprinting object in order to bring about presentations of the imprinting object. If following behavior were released, this would not have been easy to do.

At the time the Hoffman et al. (1974) work was being planned, we did not know whether the opponent process for imprinting could be weakened by disuse. Lorenz was impressed by the "irreversible" characteristics of imprinting and therefore thought it was quite different from ordinary learning. However, the opponent-process model deduces that the strength of social attachment,

indexed by *b*-process magnitude, ought to decline with disuse. Starr (1978) carried out in our laboratory the appropriate experiment to test the disuse postulate. He subjected four separate groups of ducklings to imprinting procedures. The groups were equal in their total familiarity with the imprinting object: At the end of the experiment every animal had been in the presence of a mother surrogate for a total of 6 minutes. However, the groups differed in their time intervals between exposures (their *disuse* time). Group I-1 received twelve 30-second exposures to the mother surrogate with 1-minute intervals between presentations. Group I-2 received twelve 30-second exposures with 2-minute intervals between presentations. Group I-5 received twelve 30-second exposures with 5-minute intervals between presentations. Finally, a control group, I-0, received 6 minutes of continuous exposure (or 0 minutes between presentations).

The number of seconds of distress calling during a standard 1-minute observation period occurring right after each removal of the mother surrogate was recorded for each group. Figure 9 shows that the time interval between presentations of the imprinting stimulus was a very powerful variable in determining the rate and asymptote of the growth of distress calling. The data for Starr's Group I-1 were quite similar to those recorded by Hoffman and his colleagues at Bryn Mawr for 1-minute exposures and 1-minute intervals between exposures (Figure 8). Their distress calling increased to an asymptote at which about three fourths of the time was occupied by distress calls. Starr's Group I-2 showed some growth of distress calling, but its asymptote was significantly lower than that for Group I-1. It is in Group I-5 that one discovers a crucial result: The repetition of exposures to the mother surrogate produced no growth of distress calling over and above that level seen after the first exposure and separation.

It could be argued that the interstimulus interval had an associative function. The repetition of "imprinting stimulus present" → "imprinting stimulus gone" may have increased the magnitude of distress calling by making the short presence of the imprinting object a signal for its subsequent removal. Or it could be argued that distress calling functioned as an operant during the separation interval and was reinforced by presentation of the imprinting object on 11 occasions. Furthermore, following this line of reasoning, the 5-

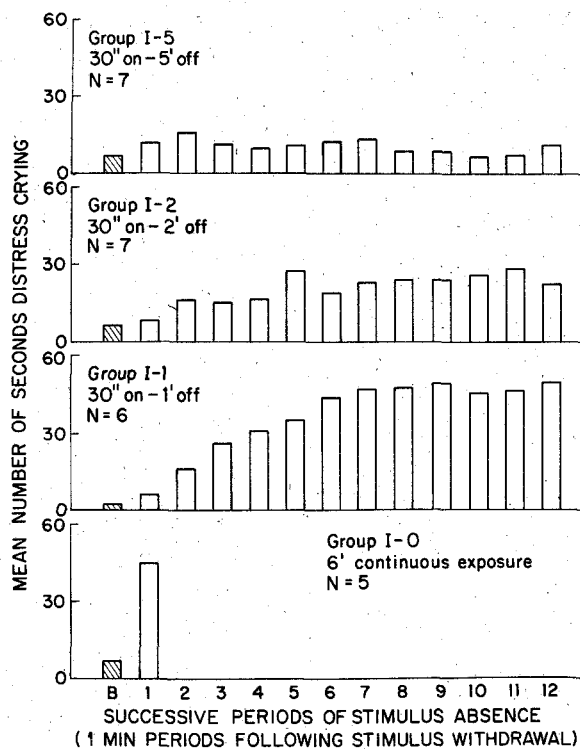


Figure 9. The growth of the opponent process (indexed by intensity of distress calling) for the action of an imprinting unconditioned stimulus (UCS) as a function of the temporal pattern of presentations and absences of the imprinting UCS. (Note that the total familiarity of all four groups with the UCS was the same [6 minutes], although the groups differed in their time intervals between exposures [see text]. Adapted from Starr, 1978.)

minute interstimulus interval group (I-5) suffered longer delays of reinforcement for distress calling than did Group I-2 or Group I-1. Association theory has a vast capacity to adapt itself to new settings! (Moreover, because I am a typical association theorist, it is hard for me to abandon these nice old habits of thought.)

However, such interpretations cannot account for the results obtained for the control group (I-0), which received 6 minutes of continuous exposure to the imprinting object before the object was removed for the first time. This group showed the same magnitude of distress calling (about three fourths of the 1-minute observation interval was occupied by calls) during the first disappearance of the imprinting stimulus as did Group I-1 after 12 repeated presentations and disappearances. The operant contingency, though possible in Group I-1, was precluded in Group I-0, and yet the distress-calling index of attachment was the same for these two groups. The conditioning argument seems weak here. Thus, my conclusion is that the interstimulus interval is a critical variable in the strengthening of the *b* process. Disuse, or prolonged absence of the UCS or releaser, weakens the *b* process *between* stimulations. During the stimulations themselves, the *b* process is strengthened.

From the above experiment, Starr (1978) induced the concept of the *critical decay duration* of the opponent process. The critical decay duration is that disuse time just adequate to allow the weakening of the opponent process to its original, innate reaction level. If reinforcing stimuli are presented at interstimulus intervals greater than the critical decay duration, then the opponent process will fail to grow. In Starr's experiment, the critical decay duration must have been between 2 and 5 minutes.

Starr discovered something else: a savings effect in the already strengthened opponent process. When he separated ducklings from their imprinting stimulus for several days, so that distress calling ceased, he found that the restrengthening of the opponent process by repeated exposures to the imprinting object took less time and fewer exposures than did the original exposures. A similar phenomenon, called *savings*, is often found in the reconditioning of reflexes and emotional reactions. Evidently, even though an opponent-process system has been weakened by disuse, some unique residues, or traces of past exercise of the opponent

process, remain and facilitate the restrengthening of the temporarily dormant system. Such a phenomenon is not unexpected. In alcohol addiction, for example, the abstainer is warned that one drink may be disastrous, and the reason is the savings principle. The reexercise of alcohol's opponent-process system strengthens the withdrawal syndrome very rapidly and sets up the special conditions for resumption of the addictive cycle. Cigarette smokers report the same phenomenon: Readdiction to nicotine takes place much more rapidly than does the initial addiction. The laws of social attachment may be identical to those for drug addiction. This was hinted at in the comparisons among Tables 1-4. However, the similarities are now extended to the fine, parametric details of opponent-process functioning.

Finally, Starr (1978) found that an enhancement of the quality of stimulation could produce an increase in the critical decay duration of the *b* process. When ducklings were exposed to an imprinting object that made honking noises, the opponent process was strengthened rapidly, even with interstimulus intervals longer than the 5 minutes used in Starr's first experiment. A general law for the strengthening of *b* processes, derived from Starr's imprinting experiments, might be stated as follows: Opponent processes are strengthened by use and approach asymptotes having values that are a direct function of the quality, intensity, and duration of each exposure and an inverse function of the interstimulus interval. It is, of course, possible that this law governs most nonassociative behavioral changes.

A science of opponent-process augmentation and weakening is now developing. It takes little imagination to see how Starr's data and concepts can be applied to drug dose frequency, quality, and size or, for that matter, to any of the phenomena listed in Tables 1-4. His ideas have considerable analytical power, and it is now possible to understand some of the conditions leading to either the strengthening or the weakening of opponent processes of all types and, consequently, to the strengthening and weakening of many experientially acquired, new motivation systems.

Whether the general law for strengthening *b* processes does in fact apply to all cases exemplified in Tables 1-4 will have to be empirically verified. Steven Seaman, working in my laboratory, has started a series of experiments to test the application of this law to the growth of toler-

ance and the magnitude of the abstinence syndrome for morphine in rats. He is trying to quantify the critical decay duration of the  $b$  process for varying dosages and durations. In principle, it should be possible to quantify the critical decay duration for any opponent-process system as a function of prior stimulation parameters. In such experiments, the phenomena of habituation (in opponent-process terms,  $|a - b|$ ) and withdrawal syndrome intensity and duration (the  $B$  state) would be the two major dependent variables. The analysis would be equally applicable to positive and negative reinforcers. Although Starr's (1978) work concerned a powerful positive reinforcer, in principle it should be just as feasible to assess the strengthening of the  $b$  process for a negative reinforcer (e.g., heat, cold, sight of an enemy predator, long-distance running, weight lifting, shocks, free-falls, etc.).

*Modulation of ongoing affect by the precipitation of A states and B states: The oppositeness concept in opponent-process theory.* In none of our research reported so far have we quantitatively demonstrated that  $a$  processes and  $b$  processes are motivational, hedonic, affective, or emotional opposites, although oppositeness has been inferred in Tables 1-4. Two undergraduates, Peter Berns and Laura Bell, working with Starr and me, developed a test of oppositeness by using chicks. The respondent, affective behavior baseline they used was the "tonic immobility" reaction (Gallup, 1977). This reaction—a chick remaining motionless for relatively long periods of time—is induced by seizing the chick, holding it firmly until it stops struggling, and then placing it on a flat surface on its side or back. The reaction duration is partly controlled by fear-evoking stimuli and fear-suppressing stimuli. The reaction is part of the prey-predator affect system of the young chick, and Gallup has presented convincing evidence that fear is the major mediator of the immobility reaction. Pretreatments with shocks, Pavlovian fear CSs, and models of natural predators, for example, all result in longer durations of tonic immobility. On the other hand, tranquilizers like Pacitran result in significantly shortened tonic immobility reactions. Finally, adrenalin injections prolong the tonic immobility reaction, while chlorpromazine shortens it. It seems safe to conclude, as did Gallup, that the duration of an induced tonic immobility reaction in the young chick is a reasonable correlate of fear intensity or, at

least, an index of the intensity of some relatively long-lasting and aversive affective or emotional state.

The strategy of Berns and Bell (1979) was simple. First, they induced tonic immobility in large numbers of chicks to obtain an index of fear derived from their particular way of handling the chicks in our laboratory setting. Mean immobility durations for several groups of chicks varied from 37 seconds to 45 seconds, a reasonably narrow range. Next, Berns and Bell measured the duration of the tonic immobility reaction when immobility was induced in the presence of an imprinting object (mother surrogate) that had previously been present for 70 minutes. Finally, the experimenters induced the tonic immobility reaction right after the familiar imprinting object had been removed.

The results are shown in Figure 10. The presence of the imprinting object resulted in a significantly shortened tonic immobility reaction, whereas the removal of the imprinting object lengthened the duration of the reaction. If one assumes that the baseline affect for the immobility reaction is an aversive state, then one can con-

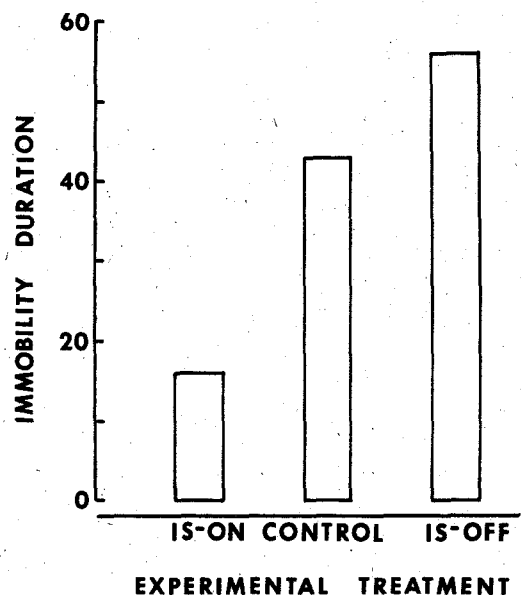


Figure 10. The duration of the tonic immobility reaction in chicks as a function of the presence of and removal of the imprinting unconditioned stimulus (UCS). (IS ON means the imprinting UCS is present; IS OFF means the imprinting UCS has just been removed. The control condition is the immobility duration for nonimprinted chicks. Duration is in seconds. Adapted from Berns and Bell, 1979.)

clude that presence of the imprinting object decreased the aversiveness and that removal of the imprinting object enhanced the aversiveness. Presentation of an innate reinforcer or releaser has thus been shown to produce an affective result opposite to that produced by the removal of the reinforcer, and vice versa. This is the sense of oppositeness that is inherent in the opponent-process theory of motivation.

The paradigm used by Berns and Bell is patterned after the typical conditioned emotional response (CER) experiment, but includes a respondent, species-specific reaction as the baseline rather than the usual free-operant baseline. In principle, by using this technique it should be possible to quantify all five major distinctive features of the standard pattern of affective dynamics (see Figure 2) for any given reinforcer against a respondent baseline.

The Berns and Bell technique is not the only one available to us, however. There is the standard CER paradigm that usually measures the effectiveness of a UCS, a CS<sup>+</sup>, or a CS<sup>-</sup> in modulating an ongoing, reliable, and steady rate of operant responding. This method can be used to test many deductions from the opponent-process model. According to the present theoretical argument, there should be a period directly following the termination of an aversive event (as long as the subject has been habituated to that event) during which the affective after-reaction has positive reinforcement properties. This positive reinforcement effect, like any *b* process, should quickly peak and then decay in time. It should be measurable by its effect on either aversively controlled or appetitively controlled operant behavior.

Using the CER technique, LaBarbera and Caul (1976a, 1976b) have carried out a program of research designed to assess opponent processes engendered by aversive stimuli. Their findings are encouraging. In one experiment, food-deprived rats pressed a lever to obtain food. During each 1-hour session, four test trials were run. Each trial consisted of pairing a CS<sup>+</sup> with a shock of .75-mA intensity and .5-second duration. However, on three of these four trials the CS<sup>+</sup> was preceded by a .5-second shock of .25, .50, or .75 mA. The four trials were at least 14 minutes apart. The rate of appetitive responding to obtain food was greater in the presence of the CS<sup>+</sup> when it had been preceded by shock, and the highest rate was produced by a .75-mA preshock. Furthermore,

there was a pronounced habituation effect over the days of testing, with the .75-mA shock producing the most rapid habituation.

LaBarbera and Caul (1976a) interpreted their findings to mean that a postshock *b* process of a pleausurably reinforcing type dilutes the suppressing power (fear-eliciting power) of the CS<sup>+</sup> presentation, thus allowing more appetitive behavior to occur. In addition, they assumed that the *b* process was strengthened by exercise. To confirm such an interpretation, however, one would have to carry out their experiment with spaced versus massed shock trials and a between-groups design.

In another experiment, LaBarbera and Caul (1976b) showed a decrement in what they called distress induced by an aversive event when that event occurred during the period directly following the termination of another aversive event that had repeatedly been experienced. Rats licked for water, and the CER technique was used. The rats showed less suppression of drinking in response to a given CS<sup>+</sup> for shock if that CS<sup>+</sup> was preceded by another CS<sup>+</sup> for shock. The authors stated, "The results are compatible with the opponent-process theory and suggest the presence of a positive hedonic afterreaction to an aversive event which reduced distress to a following aversive event" (LaBarbera & Caul, 1976a, p. 485).

This result suggests that *b* processes, though interacting with *a*-process arousers, retain their integrity in time and are not destroyed, discharged, or terminated when *a* processes are superimposed on them in time. This is, of course, what one would expect based on the observations of military parachutists, who experience a long period of exhilaration after a free-fall is terminated. The exhilaration has been reported to persist (Epstein, 1967), even through subsequent odious tasks. The inverse of this would be the persistence of opiate craving during opiate withdrawal; the craving seems to permeate all concurrent activities and hedonic events. Therefore, *b* processes, if they are derived from aversive *a* processes, can provide a relatively enduring source of positive hedonic tone following the removal of the aversive reinforcer. Fear thus has its positive consequences.

The opponent-process theory maintains (1) that the reinforcing properties of a given *b* process must be correlated with the magnitude of an A state because the state rule is  $|a - b|$ ; (2) that changes in the magnitude of State A must be correlated with changes in the magnitude of State B;

and (3) that such changes are nonassociative in nature, coming about only because of the repetition of a reinforcer. Starr's (1978) experiment with ducklings demonstrated the changes in the B state with repetition of the reinforcer, but there was no A-state measure during the presence of the reinforcer. Distress calling indexed only the B state.

A recent series of experiments by Overmier, Payne, Brackbill, Linder, and Lawry (1979), however, offers powerful support for the three points listed above. Overmier et al. used the CER paradigm and measured the effects of a CS<sup>+</sup> for shock on a free-operant, shock-avoidance response baseline, using dogs as subjects. In Experiment 1, they showed the decline in magnitude of the CER when 300, as compared with 18, reinforced trials were run. Then, in Experiment 2, they showed this decline in the potency of the CS<sup>+</sup>, of the UCS, or of both to be nonassociative in nature. Their evidence is striking. All subjects received 300 reinforcements, but each of three groups received the reinforcements in different sequences: (a) 18 CS<sup>+</sup>-UCS pairings, followed by 282 UCS-alone trials and then a test for the CER power of the CS<sup>+</sup>; (b) 282 UCS-alone trials, followed by 18 CS<sup>+</sup>-UCS trials and then a test for the CER properties of the CS<sup>+</sup>; and (c) 282 UCS-alone trials randomly intermixed with 18 CS-UCS trials, fol-

lowed by the CS<sup>+</sup> test. The experimenters had previously determined, with another group of dogs, that 18 CS-UCS trials produced a sizable CER: The avoidance operant rate increased (more fear) 75% when the CS<sup>+</sup> was tested during the operant behavior. However, in all three of the experimental treatments with 300 presentations of the reinforcer, the CER-eliciting power of the CS<sup>+</sup> was negligible. The results of Experiment 2 are shown in Figure 11. Also illustrated in Figure 11 is the finding from Experiment 1 that 300 CS-UCS pairings produced no greater CER-elicitation power of the CS<sup>+</sup> than did the three tested combinations of 282 UCS-alone trials plus 18 CS-UCS trials. One can therefore conclude, as did Overmier et al., that the habituation to the UCS was a consequence of nonassociative processes and that the habituation effect significantly lowered the CER power of the CS<sup>+</sup>. Presentations of the UCS alone were sufficient to bring this about.

With Experiments 1 and 2, Overmier et al. showed that A-state magnitude is considerably smaller after 300 reinforcements than it is after 18 reinforcements and that this difference is due to nonassociative processes not dependent on pairings or contingencies. However, even though Point 3 was substantiated, Points 1 and 2 still remained untested.

In their third experiment, Overmier et al. (1979) used a backward-conditioning paradigm to establish a CS<sup>-</sup> for their CER test. In the backward-conditioning paradigm, the CS<sup>-</sup> is presented on each conditioning trial immediately after the UCS is terminated (after the shock goes off). Presumably, if the CS<sup>-</sup> occurs only a few seconds after UCS termination, each CS<sup>-</sup> presentation then coincides in time with the peak of the B state. In the present case, the experimenters used a 10-second CS that began just as the UCS terminated, thus "blanketing" the time interval during which the peak of the B state is inferred to occur. They compared two modes of establishing a CS<sup>-</sup> with 300 trials: one in which the CS<sup>-</sup> was presented only on the first 40 trials, followed by 260 trials of UCS alone, and one in which the UCSs alone were given on the first 260 trials, followed by 40 trials on which the CS<sup>-</sup> was presented.

The CER data are given in Figure 12. The CS<sup>-</sup> for the first mode showed a slightly excitatory property, causing an avoidance response rate increase of about 15%. The CS<sup>-</sup> for the second mode, established on the 40 trials after 260 UCS-

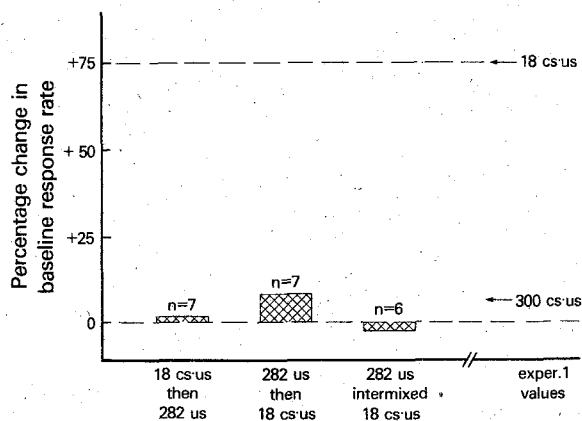


Figure 11. The fear-arousing power of a CS<sup>+</sup> (conditioned stimulus) measured by percentage of change in avoidance response rate. (Data are from Overmier, Payne, Brackbill, Linder, and Lawry, 1979. Shown here is the fact that the CS<sup>+</sup> is most potent when paired only 18 times with the US [unconditioned stimulus]. With 300 US presentations, the CS<sup>+</sup> becomes significantly weaker. This is so whether the USs occur as part of pairings or as US-alone events. Therefore, the habituation effect is most likely a nonassociative one.)

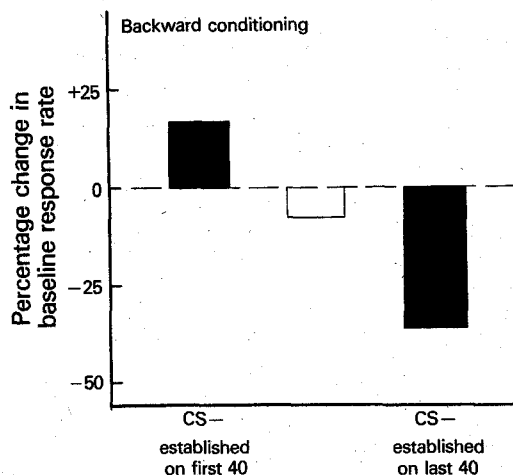


Figure 12. Data from Overmier, Payne, Brackbill, Linder, and Lawry, 1979. (The fear-inhibiting power of a backward-conditioned CS- [conditioned stimulus] is much greater when it has been paired with the last 40 terminations of shocks in a 300-trial sequence than it is when it has been paired with the first 40 shock terminations. Indeed, the first 40 shocks produce a slightly excitatory CS-. Therefore, it is likely that the establishment of the fear-inhibitory powers of a backward CS- depends on habituation to the unconditioned stimulus already having occurred.)

alone trials had already occurred, produced a significant decrease of about 40%. I am most interested in the comparison of the two modes because they test Points 1 and 2 above, which relate the magnitude of State A to the magnitude of State B. These experimental findings show that 260 UCS-alone presentations occurring prior to 40 backward conditionings of the CS- make that CS- an avoidance response suppressor or a fear inhibitor. In contrast, the 40 backward-conditioning trials yielded an excitatory CS-. One can conclude here that a backward CS- is dependent on a strengthened *b* process for its fear-inhibiting effect. Furthermore, such a fear-inhibiting CS-, depending in theory on a strengthened *b* process, can only be established after habituation to the UCS has occurred. In Overmier et al.'s Experiment 2, the 300 presentations of the UCS were shown to produce much habituation. Points 1 and 2 have therefore been substantiated.

The demonstrated relationship between the decline in A-state amplitude and the augmentation of B-state amplitude gives firm support to the tentative impressions drawn from Tables 1-4. How general such opponent-process laws may be is not known. If they are general, then military

parachutists should not be able to experience exhilaration after a jump *until* they are habituated to the jump itself; and opiate users should not be able to experience strong craving and a withdrawal syndrome *until* there is some development of affective tolerance to the opiate. A wide variety of empirical tests on positive and negative reinforcers are needed before one can safely conclude that the *b* process is unitary and that the state rule is really  $|a - b|$ .

*The conditioning of A states and B states.* Although the growth of the *b* process has been postulated to be a nonassociative process, this does not rule out the action of Pavlovian associative processes in producing conditioned A states and B states whenever the proper requirements for those processes have been met. The conditioning of A states is now not problematical. For example, one can condition fear reactions by presenting a neutral CS a few seconds prior to the presentation of a fear-evoking UCS. The CS acquires aversive, affect-arousing properties. The conditioning of B states is not as certain, but the evidence is accumulating that such does occur. The Overmier et al. (1979) findings are certainly suggestive of B-state conditioning. Their backward-conditioning paradigm is appropriate for the investigation of conditioned B states because it places the occurrence of the CS near the postulated peak of the B state occurring right after the termination of the UCS.

Moscovitch and LoLordo (1968) have shown that a backward CS in a fear-conditioning experiment can acquire fear-inhibiting properties. It can subtract from a baseline fear level and so appears to elicit a conditioned opponent to conditioned fear. More recently, Maier, Rappaport, and Wheatley (1976) have shown that the temporal placement of the backward CS is important in determining how powerful a fear-inhibiting effect can be governed by the backward CS-. They presented tone CSs to rats either 3 seconds or 30 seconds following terminations of the shock UCSs. The tone at 3 seconds showed a fear-inhibiting property, but the tone at 30 seconds did not. This result suggests that backward CSs need to be near the peak of the B state to acquire conditioned control over that B state. It also suggests that CSs do not become conditioned to B states if such states are dying out or weak.

The backward-conditioning paradigm is not the only one capable of producing putative condi-

tioned B states. Siegel (1975, 1976, 1977; Siegel, Hinson, & Krank, 1978) has been able to show conditioning of tolerance for doses of morphine in rats to specific signals for drug administration. Tolerance to morphine was measured by rats' reactions to a hot plate. When the rats were exposed to the hot plate in an environment like that existing when they had received their morphine shots, they were more tolerant to morphine than when they were tested in a dissimilar environment. Siegel inferred that in such a case a "compensatory process" has been conditioned to environmental cues. Siegel's compensatory process obviously shares attributes with a *b* process for the affect aroused by morphine.

Yet this conclusion raises both theoretical and methodological problems. Why should the *b* process in Siegel's experiments have been more strongly conditioned than the *a* process? After all, the environmental cues preceded and overlapped with the morphine injection, so they should have elicited conditional *a*-process elements. The "needle-freak" phenomenon in opiate users strongly suggests that *a* processes can become conditioned to environmental cues. Why didn't Siegel's rats become "needle freaks" and show conditioned opiate-like reactions when they were tested with a saline injection? They showed a conditioned "opiate-opponent" reaction instead and were hyperalgesic on the hot plate rather than analgesic. Was there some special feature of Siegel's (1977) procedure that gave precedence to *b*-process conditioning? Some temporal relationship in the experiment, for example? The dosage of morphine? We do not know, but we are now starting to experiment with morphine tolerance with these questions in mind. We would like to tease apart the associative and nonassociative components of tolerance.

The puzzling aspects of Siegel's findings are further highlighted by other findings. For example, infants born of opiate-addicted or opiate-maintained mothers show a high degree of tolerance to paregoric, and they go through a devastating withdrawal syndrome. Siegel's conditioning principle strongly suggests that because the postnatal world is so different from the prenatal world, the newborn should be nontolerant to paregoric. These questions about tolerance remain unanswered, but they must be answered if the generality of the opponent-process theory is to be tested adequately.

*Role of endorphins in the opponent processes for aversive UCSs.* The relatively recent discovery that the brain synthesizes and secretes opiate-like substances gives added meaning to the concept of *b* process for aversive stimuli. These substances, called endorphins, are secreted when certain environmental stresses are imposed on animals. Perhaps, in special cases, they are the major substrate for a *b* process, and if so their presence would be responsible for the development of affective tolerance to selected aversive events. In the sum  $|a - b|$ , *b* would then be endorphin amount. The opponent-process theory generates several important deductions about the action of endorphins when they are secreted as a consequence of an aversive UCS: (a) The injection of the opiate antagonist, naloxone, should be able to reduce the *b* process to zero. (b) Reducing the *b* process to zero should destroy tolerance to an aversive UCS. (c) Reducing the *b* process to zero and keeping it at zero should prevent the growth of the *b* process with repeated UCS presentations. (d) Reducing the *b* process to zero following UCS termination should suddenly terminate a B state.

These deductions are empirically testable. There are, even now, hints in reported research that some are correct. In my laboratory, Ehrman, Josephson, Schull, and Sparich (Note 4) have shown that the power of an aversive UCS to establish a conditioned reaction to a CS<sup>+</sup> can be strongly affected by an injection of naloxone. As Overmier et al. (1979) have shown, the constant repetition of an aversive UCS will result in a decrease in the effectiveness of that UCS (tolerance or habituation) in reinforcing an association between it and a CS<sup>+</sup>. However, if one administers naloxone after many UCS presentations, subsequent pairings of a CS with that UCS will be highly effective in strengthening the CS-UCS association, according to the findings of Ehrman et al. Furthermore, one can use naloxone to heighten the rat's reaction to heat applied to the tail (Bernston & Walker, 1977). A similar process occurs in human reaction to shocks (Buchsbaum, Davis, & Bunney, 1977).

There are, however, many negative findings. For example, formalin-induced pain is not enhanced by injections of naloxone (North, 1978). Furthermore, naloxone does not alter human reports of pain when the cold pressor test is used to produce the pain. Some comments are in order here. According to the opponent-process model, naloxone should be most dramatic in causing changes only

when the  $b$  process is already very large because of repetition (when tolerance is strong). In this case, the quantity  $|a - b|$  could theoretically be restored to  $|a - b| = a$ , from  $|a - b|$  of some small value, if naloxone were administered and if the  $b$  process were, indeed, an endorphin process. Therefore, an experiment on pain thresholds may not address this point. The affective reactions of human subjects (to the cold pressor test) would only be enhanced by naloxone if they were already well habituated to the cold pressor test. The opponent-process model tells one how to do such an experiment in a more decisive way: Use highly tolerant subjects, *then* administer naloxone. Schull (Note 5) is carrying out such an experiment for his dissertation in our laboratory.

The deductions I have listed suggest some experiments that might be dramatic in their outcomes. For example, suppose a sauna bather (see Table 3) is thoroughly affectively habituated to the heat. Suppose that the  $b$  process includes a great amount of endorphin secretion. Naloxone should then do three things: (a) It should "break" the habituation, throwing the bather back into his or her affectively intolerant condition; (b) it should prevent exhilaration from being experienced when the bath is over; and (c) if the naloxone is injected during the exhilaration period, it should suddenly terminate the exhilaration. These expectations could be tested, and analogous expectations could be tested in parachutists or in marathon runners. By now, the reader can well deduce the outcomes of many naloxone experiments, but how would they actually come out?

## Discussion

*Prediction of experimental outcomes.* How has the theory fared in predicting experimental outcomes? Almost too well. Well enough at least to make me very suspicious! Every experiment generated by the model has failed to refute the model, even though the experiments have been designed to be capable of doing so. To me, this is quite threatening. We are now living through an era of behavior theory in which very few modest explanatory schemes are being generated to account for rather limited experiments. The minimodel is preferred to the grand, all-encompassing models of the past, partly because those grand models ultimately become impossible to refute. The opponent-process theory is quite encompassing. Although it does not claim to account for all phenomena of

acquired motivation, it certainly does try to account for many. Perhaps it overexplains. I am not sure. But there are advantages to the theory. In teaching the subject of acquired motivation to both undergraduates and graduates, the existence of the opponent-process theory saves me time in exposition, orders large arrays of data in a tight and unambiguous fashion, and generates many new questions. A few of those questions have been answered. However, much is left to be done. It would make my life simpler were the theory to be unequivocally refuted by a fine experiment. Until that time arrives, however, I think the theory is seminal enough to warrant an intensive effort at empirical testing. The early stages of that program are under way.

*Social philosophy.* Another feature of the opponent-process theory warrants comment. It is obviously a puritan's theory. It argues for the existence of psychological mechanisms for the automatic or autonomic control of affect, such that repeated pleasures lose a lot of their pleasantness and make one potentially capable of new sources of suffering; in the same vein, repeated aversive events lose a lot of their unpleasantness and make one potentially capable of new sources of pleasure. The philosophical implications of such a theory should be obvious.

*Psychosomatic medicine.* Furthermore, the theory implies some striking things about pathology. If one assumes that the phasic  $a$  processes are usually negated by their  $b$  processes, then the chronic, long-lasting effects of repeated reinforcers, whether pleasant or aversive, should be the attributes of enduring  $b$  processes. Therefore, if a  $b$  process has physiological "costs," like any defensive system or immunological system, such costs should be equally characteristic of pleasantness and unpleasantness. Thus, if the opponent processes can wear out, or become exhausted (a feature of Selye's, 1950, stress adaptation theory), some unfortunate physiological dislocations may ensue.

We have been taught to think of aversion and trauma as the only affective sources of physiological stress. The opponent-process model implies that often repeated pleasures are just as fertile a source of physiological stress. Indeed, the model suggests that two major classes of physiological pathology are derived from repeated reinforcers—one class for aversive events and the other for pleasurable events. Pleasure as stressful has somehow been overlooked in our treatises on "the effects of stress on \_\_\_\_\_." The stress hormone system of the

pituitary-adrenal axis has somehow become identified with aversiveness and trauma. The opponent-process theory suggests a fresh look. Some physiological pathologies may be generated by overworked *b* processes for pleasurable *a* processes. It might be worthwhile to look for them.

The opponent-process model strongly implies that physiological pathology precipitated by frequently repeated reinforcements is caused by the *b* process for a particular reinforcer. Therefore, one would expect the pathology to develop much more rapidly following the termination of the reinforcer, when the B state is "pure," than during the presence of the reinforcer, when the quantity  $|a - b|$  may be very small. Desiderato, MacKinnon, and Hisson (1974) reported findings strongly in agreement with this expectation. They subjected rats to aversive stress by putting them in an avoidance-avoidance conflict, wherein one aversive stimulus (shock) was intermittent while the other (shock) was continuous. Each rat participated in a continuous 8-hour session of this sort (a very exhausting experience). The stomachs of one group of rats were examined for ulcerative lesions immediately after an 8-hour stress period, and the stomachs of a second group were examined following a 2-hour rest-and-safety period that followed a 6-hour stress period. Rats in a third group, a control for aversiveness per se without a conflict feature, received 6 hours of continuous shock and were then given a 2-hour rest-and-safety period, after which their stomachs were examined. The results were quite clear. Conflict plus a rest period yielded a mean of over nine lesions per stomach, whereas conflict without a rest yielded a mean of only a little over three lesions per stomach, even though the total number of hours (8) allowed for ulcer development was the same for both groups. This is exactly what one would expect if the *b* process was the pathogenic agent. The opponent-process model tells us to look at event terminations, as well as event onsets, for clues to the development of experientially induced, physiological dislocations or malfunctions.

*The reinforcement concept.* Similarly, the opponent-process theory tells us that the reinforcement concept probably is inadequate. When psychologists write about their Pavlovian conditioning procedures, UCSs are either presented or not. When operant conditioning procedures are described, reinforcers are either presented or not, are presented in some scheduled way, or are presented according to complex schedule sequences. When

behavior therapists use their reinforcers, they are either presented or not. The opponent-process model tells us that reinforcers, in addition to their presence or absence and in addition to their schedule, have (a) an onset, (b) a maintenance or duration interval, and (c) a posttermination interval. None of these three aspects is psychologically equivalent to any of the other two. Each of them can be orthogonally manipulated. Furthermore, each of the three aspects changes dynamically with frequent repetition. Thus, a reinforcer is not just a reinforcer. It is a *triad* of affective influences that can change with repetition. Simple operationism is not sufficient for a fruitful reinforcement concept. Theory is needed.

### Summary

I have presented the postulates of a new theory of acquired motivation. These postulates can be summarized as follows:

1. Primary affect, or the hedonic attributes of reinforcers, usually arouses affective opponent processes, the action of which is (a) the reduction of the hedonic potency of the reinforcer and (b) the occurrence of opponent hedonic aftereffect.

2. Primary affect, or the hedonic attributes of reinforcers, closely tracks the stimulus intensity properties of the reinforcer over time. These are called *a* processes. In contrast, the opponent hedonic process does not track accurately. Instead, the opponent process has a long latency, a sluggish course of increase, and a sluggish course of decay after the reinforcer is terminated. The opponent processes have a high inertia. They are called *b* processes.

3. The primary hedonic process aroused by onset of a reinforcer remains unchanged when the reinforcer is repeatedly presented. In contrast, the opponent hedonic process is strengthened by use and weakened by disuse.

4. Opponent hedonic processes are strengthened by use if and only if they are aroused at inter-reinforcer time intervals less than the time interval required to allow the opponent process to decay to some near-neutral baseline value. This time interval is called the critical decay duration of the opponent process or *b* process.

I have detailed some of the sources of data that led to the formulation of the present theory. I have described some current research instigated by the theory or highly relevant to it: (a) the growth of social attachment in ducklings and chicks; (b)

the modulation of ongoing fear by the occurrence of primary hedonic processes (*a* processes) and opponent processes (*b* processes); (*c*) the conditioning of primary affective states (*A* states) and opponent affective states (*B* states); and (*d*) the role of the endorphins in the opponent processes for aversive stimuli. Finally, I have discussed some of the systematic implications of the theory, its adequacies and inadequacies, and its role as a generator of new questions.

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