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RECENT EXPERIMENTS TESTING AN OPPONENT-PROCESS THEORY OF ACQUIRED MOTIVATION

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Abstract. There are acquired motives of the addiction type which seem to be non-associative in nature. They all seem to involve affective phenomena caused by reinforcers, unconditioned stimuli or innate releasers. When such stimuli are repeatedly presented, at least three affective phenomena occur: (1) affective contrast effects, (2) affective habituation (tolerance), and (3) affective withdrawal syndromes. These phenomena can be precipitated either by pleasant or unpleasant events (positive or negative reinforcers). Whenever we see these three phenomena, we also see the development of an addictive cycle, a new motivational system. These phenomena are explained by an opponent-process theory of motivation which holds that there are affect control systems which oppose large departures from affective equilibrium. The control systems are strengthened by use and weakened by disuse. Current observations and experiments testing the theory are described for: (1) the growth of social attachment (imprinting) in ducklings; and (2) the growth of adjunctive behaviors. The findings so far support the theory.

The opponent-process theory of acquired motivation was suggested by the existence of three related phenomena found in many acquired motives:

1) hedonic contrast effects,
2) hedonic habituation or the growth of tolerance,
3) hedonic withdrawal syndromes or abstinence syndromes.

Hedonic contrast effects refer to the fact that the reinforcing properties of the presentation of, and maintenance of, many reinforcers contrast with the reinforcing effects of the removal of, or continued absence of, the same reinforcers. A mirror-image relation often exists. This relation is illustrated in Fig. 1. There we see, in the top panel, the dog's heart rate response to the onset, 10-s maintenance and cessation of shocks to the hind toe pads. At shock onset, the heart rate increases for about 5 s, then decreases even while the shock is still on. Then the shock is terminated after 10 s, and we see, in the bottom panel, a marked deceleration, *below* the resting baseline level, followed by a slow, sluggish recovery to baseline level. Note that the 4 mA. shocks produced less acceleration and less deceleration. The 8 mA. shocks produced more acceleration *and* more deceleration, with deceleration lasting longer than it did for the 4 mA. shocks. This is the *hedonic contrast* phenomenon. It has five distinctive features, idealized in Fig. 2, which portrays how we think the onsets and terminations of many reinforcers (unconditioned stimuli — UCSs, reinforcers — rfts, or innate releasers) affect hedonic, affective or emotional processes in the organism. This *standard pattern of affective dynamics* shows that the after-reaction in-

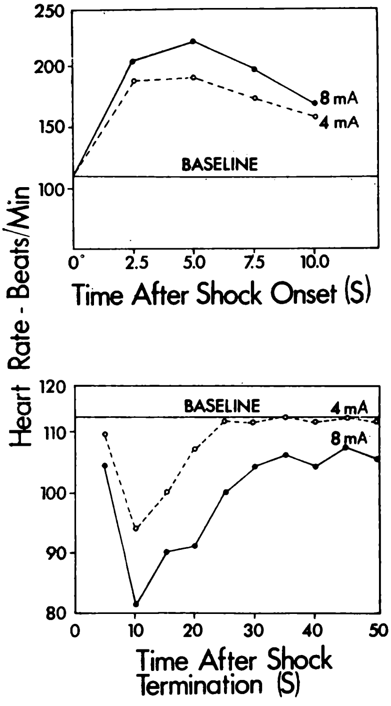

Fig. 1. The unconditioned heart-rate response in dogs. The upper panel, the response to the onset and 10-s duration of 4 mA and 8 mA shocks. The lower panel, an after-effect, after shock termination. The graphs represent the mean values from six dogs with randomized presentation of shock intensities.

volves a hedonic or affective state qualitatively different from that of the primary reaction to the presence of the reinforcer. Here, a square-wave input is transduced to a five-featured resultant. The same function can be drawn for color vision; the stimulus presentation can be red, and then the after-reaction will be green, the negative after-image. The hedonic contrast phenomenon is like a hedonic negative, affective (rather than color) after-image.

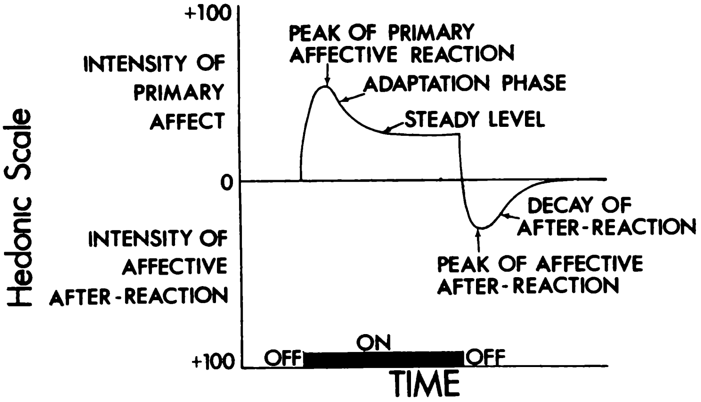

Fig. 2. An idealization of the standard pattern of affective dynamics. At reinforcer onset, there is a peak of arousal which then subsides while the reinforcer is maintained. Then, when the reinforcer is removed, there is a contrasting after-reaction which peaks quickly and then decays back to the original affective baseline.

These "after-images" have reinforcing attributes. If the onset is a positive reinforcer, then termination will function as a negative reinforcer, and vice versa. When we observe such a phenomenon, then we usually will observe the next one.

Hedonic habituation or tolerance effects occur when the frequent repetition of a reinforcer (UCS, rft or innate releaser) results in the decreased capacity of the reinforcer to reinforce. The reinforcer then cannot as easily influence ongoing behaviors nor form associations with CSs or with operants. The clearest examples come from repeated drug use, where we use the term *tolerance*. But other than chemical reinforcers show the same characteristic *habituation* effect. We know that many reinforcers produce smaller UCRs when repeated frequently. This phenomenon is illustrated in Fig. 3, for heart rate reactions in a "veteran" laboratory dog who has received hundreds of shocks (10-s duration, 4 mA.) over a period of several weeks. At the end of this protracted treatment, the dog's heart shows very little acceleration in response to UCS onset (see top panel). Then, when a shock is terminated, we see the emergence, shown in the bottom panel, of the *withdrawal or abstinence syndrome.*

The hedonic withdrawal or abstinence syndrome is characterized by a large amplification of the after-reaction, so that it is very intense and lasts a long time. Of course, we are used to observing this in connection with repeated opiate or alcohol use, when the highly tolerant drug user also shows intense, long-lasting aversive affect (negative reinforcer) when the chemical treatment is terminated. Hedonic withdrawal syndromes can be either aversive or desirable depending on the hedonic quality of the reinforcer onset and maintenance. They aren't always aversive, and so the drug case is partially misleading. For example, when military parachutists become highly tolerant of free falls (are no longer afraid), they also experience a period of exhilaration following the jump session. Or a marathon runner may experience long-lasting exhilaration or mood elevation following a long run to which the runner has already become tolerant. These are positive reinforcing effects

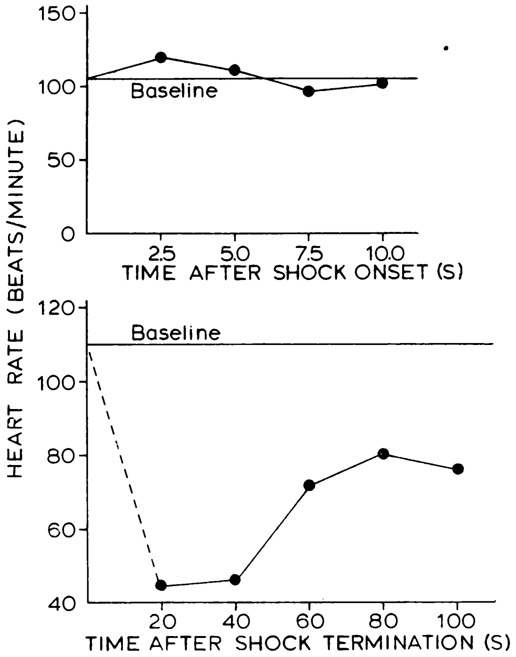

Fig. 3. The heart rate reactions of a "veteran" laboratory dog who has previously received hundreds of shocks over several weeks (means for 6 repeated shock presentations). The upper panel, response to shock onset and 10-s shock duration. The lower panel, after-effect, after shock termination. Note that the biphasic heart rate deceleration and recovery to baseline is of large amplitude and long duration relative to what it was in Fig. 1.

and are sought. They are derived from constant re-exposure to originally-aversive reinforcers.

The three hedonic or affective phenomena are characteristic of many acquired motives. They are all analogous to the phenomena of opiate addiction. In all cases, the power of the substance or stimulus to reinforce a CS or an operant via onset is *reduced* by repetition. But the power of the after-reaction to reinforce becomes *enhanced* by repetition. This asymmetry of course destroys the contrast phenomenon seen in Fig. 2 and replaces it with the result seen in Fig. 3. In addition, the hedonic sign of the withdrawal syndrome is functionally *opposite* to that of the onset effect.

All three phenomena are usually found together. If you see one, you will usually see the others. All three are usually found in clear cases of *acquired,* in contrast to innate, motive systems. Acquired motives are those not built into the species without special experiences or external events. One does not have to be an opiate addict. One doesn't have to be a devotee of jogging. But these phenomena will develop if the right experiences are frequently repeated.

These hedonic phenomena are most easily explained by an opponent-process theory of motivation. The theory assumes that the brains of all mammals are, for some reason, 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* negative reinforcers. The opposing affective or hedonic processes are *automatically* set in motion by many of those stimuli which psychologists 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, rfts or innate releasers, are postulated to correlate closely in their magnitudes with the stimulus intensity, quality and duration of the reinforcer. These primary processes are phasic and sensitive to small stimulus changes. They may show some sensitization effects, but rarely do they show habituation. They are stable, unconditioned reactions. I call them *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* which functions to *oppose* and *suppress* the affective or hedonic state generated initially 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: (1) of sluggish latency (2) inertial, or slow to build to its asymptote and (3) 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 we shall see.

The affective or hedonic state of the organism at any moment is postulated to be the difference, without regard to sign, between the magnitudes of the a-process and b-process. The b-process has a negative sign because it opposes the a-process. The *state rule* is simple: (1) if /a–b/ shows a $>$ b, then the organism is in state A, and (2) 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.

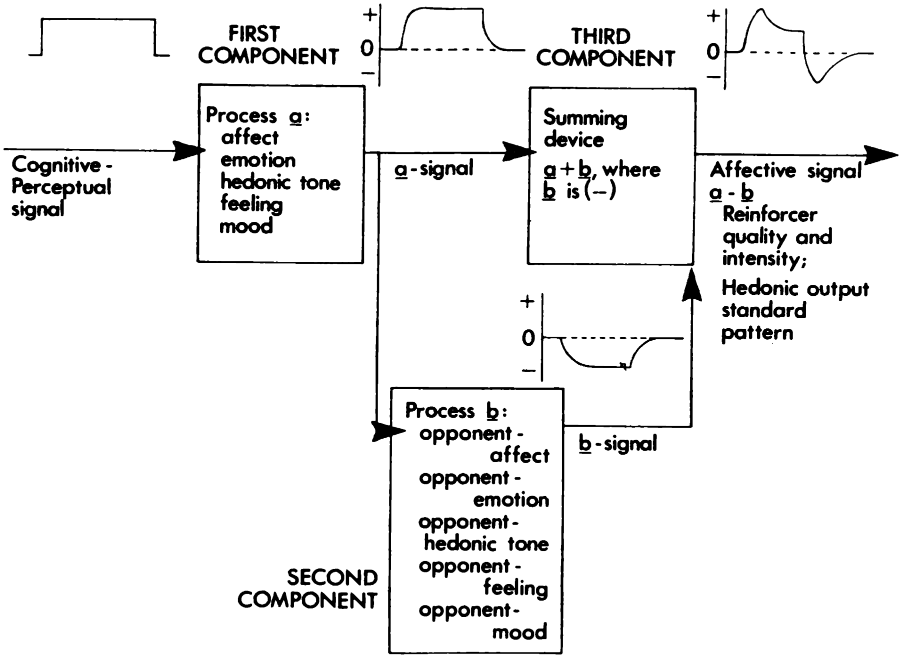

Fig. 4. The affect-processing system for the opponent-process model of acquired motivation. The presence of the reinforcer arouses the a-process, one side effect of which is to arouse the b-process, the affective opponent of the a-process. The summation of the a signal and b signal gives the current state of the organism. Because the b-process is of long latency, slow to grow and slow to decay, the sum (a–b) will produce the standard pattern of affective dynamics shown in Fig. 2.

The affect-processing system, reflecting the opponent-process assumptions made so far, is shown in Fig. 4. First, there is a cognitive-perceptual event representing the UCS, rft or innate releaser. For illustration, assume that the subject is a cat and 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 UCR is a fear reaction pattern. The occurrence of this reaction pattern will then result in arousal of a b-process, the opponent-process. It will have an affective or hedonic sign *opposite* in quality to that of the a-process. At this point, we can only guess at what the quality of the opponent really is. As we shall see, its quality will only be revealed when the categorical stimulus event is terminated.

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 build-up relative to that of the a-process. However, the slow build-up of the b-process will produce a gradual decrease in the amplitude of the A-state even while the UCS, the dog, is still present. The cat will look less fearful as time goes by. The cat will appear to be "accustomed" to the dog's presence.

When the dog goes away, there is no categorical stimulus to maintain the a-process and so it will quickly subside to zero. However, the b-process, being sluggish and slow to decay, will perseverate for a while. The peak of quality and intensity of the B-state will thus reveal itself directly after UCS termination, when the a-process goes to zero. Then the B state will slowly decay or subside. The cat may look relieved or relaxed, may show a typical feline after-reaction of pleasure, and then will slowly return to equanimity.

The processing system deduces the major facts in Figs. 1 and 2, the standard pattern of affective dynamics. However, an additional assumption is needed before the system will generate the effects of many repeated presentations, as shown in Fig. 3. The model must be able to produce the habituation effect as well as the emergence of a new, strong withdrawal syndrome. It will do so if we postulate that the b-process is strengthened by use and weakened by disuse. How this would work in the processing model is shown in Fig. 5, which compares the b-processes and 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: (1) the sum /a–b/ during the onset and presence of the UCS is *decreased*, but the sum /a–b/ right after cessation of the UCS is *increased*. This fits our empirical generalizations quite well.

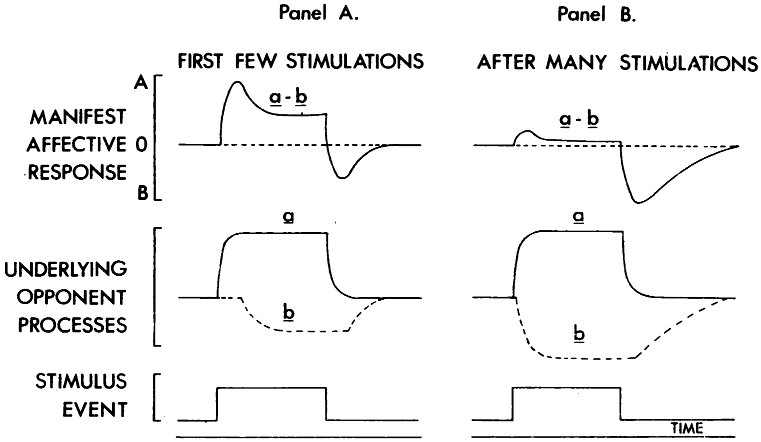

Fig. 5. A comparison of the standard patterns of affective dynamics for a relatively novel reinforcer and one that has been repeated many times. The repetition has strengthened the b-process, thus shortening its latency, augmenting its asymptote, and increasing its decay duration. These theoretical mechanisms explain the differences seen between Figs. 1 and 3. They account for habituation or tolerance, on the one hand, and the emergence of a distinct withdrawal syndrome, on the other.

New experiments pertinent to the opponent-process theory

It is one matter to organize the known 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 new findings, although not initially intended to test the theory, have served this purpose. It appears that the concept of the opponent-process or compensatory process (see 13) is "in the air" now.

Our experiments can be classified as follows: (1) the growth of opponent-processes in social attachment; (2) the growth of adjunctive behaviors as a function of strength of opponent-processes. I will start with the social attachment experiments, because these have taught us a great deal about opponent-processes.

The growth of social attachement (imprinting) in ducklings. Imprinting has been characterized as an all-or-nothing, innate learning event of surprising suddenness (9). It occurs when a newly-hatched, precocial bird first is 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 duckling develops more and more skilled locomotor behavior, resulting in its staying close to, or following after, the moving object.

A striking feature of imprinting is the affective reaction of the hatchling when the imprinting object is *suddenly removed.* The animal at first shows a "double-take", a perceptual startle with a very short latency. Then it becomes very active, appearing to be searching for the lost object and finally, after a 5–10 s latency, it emits high-pitched cries, or "distress calls". These distress calls can vary in the frequency with which they occur in time, and bursts of distress-calling will vary also in duration (4). 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 be discoverable:

First, the presentation and removal of an imprinting object should have opposite reinforcing effects. This is so. Hoffman et. al. (5) showed that arbitrary operants could be shaped by presentations of an imprinting object. Furthermore, Hoffman et. al. (7) showed that removal of the imprinting object functioned effectively in a punishment contingency to weaken an arbitrary operant.

Secondly, rather than imprinting being all-or-none, or "released", it should, instead, *develop gradually* in strength as the b-process is exercised by *use,* and it should *wane* in strength should the b-process be weakened by *disuse.*

After planning sessions with members of my research seminar, Hoffman's group at Bryn Mawr designed and conducted the first experiment on the growth of an aversive opponent-process in imprinting. They showed (6) that, with 1-min exposures alternated with 1-min removals of an imprinting stimulus, the amount of distress-calling per unit of time gradually increased. Their findings are shown in Fig. 6. We can infer that the opponent-process in imprinting is strengthened by use. We are then led, as others have been, to question the all-or-none characterization of the imprinting process but based on a different type of evidence (see 12, p. 198–200). Furthermore, we can now safely assume 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 up arbitrary operants. Indeed, so-called following behavior may function as an operant. Hoffman et. al. (8) 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.

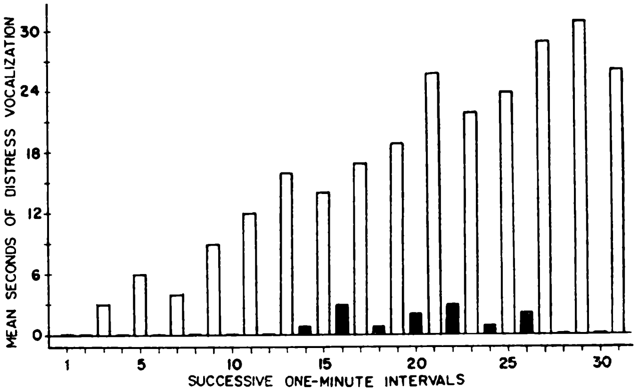

Fig. 6. The growth of magnitude of distress calling as a function of repeated 1-min presentations and removals of an imprinting stimulus. Adapted from Hoffman, et. al. (6). Imprinting, measured by distress calling, is not an all-or-none phenomenon.

At the time the Hoffman et al. (6) work was being planned, we did not know whether the opponent-process for imprinting could be weakened by disuse. Early claims suggested a negative answer. Lorenz had been impressed by the "irreversible" characteristics of imprinting. He thought it was quite different from ordinary learning in its irreversibility. In contrast, the opponent-process model deduces that the strength of social attachment, indexed by b-process magnitude, ought to decline with disuse. Starr (14) carried out the appropriate experiment to test the disuse postulate. He subjected four separate groups of ducklings to imprinting procedures. The groups were the same 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 12, 30-s exposures to the mother surrogate, with 1-min intervals between presentations. Group I-2 received 12, 30-s exposures with 2-min intervals between presentations. Group I-5 received 12, 30-s exposures with 5-min intervals between presentations. Finally, a control group I-0, received 6 min of continuous exposure (or 0 min between presentations).

The number of seconds of distress-calling, during a standard, 1-min observation period right after each removal of the mother surrogate, was recorded for each group. Figure 7 shows that the time interval

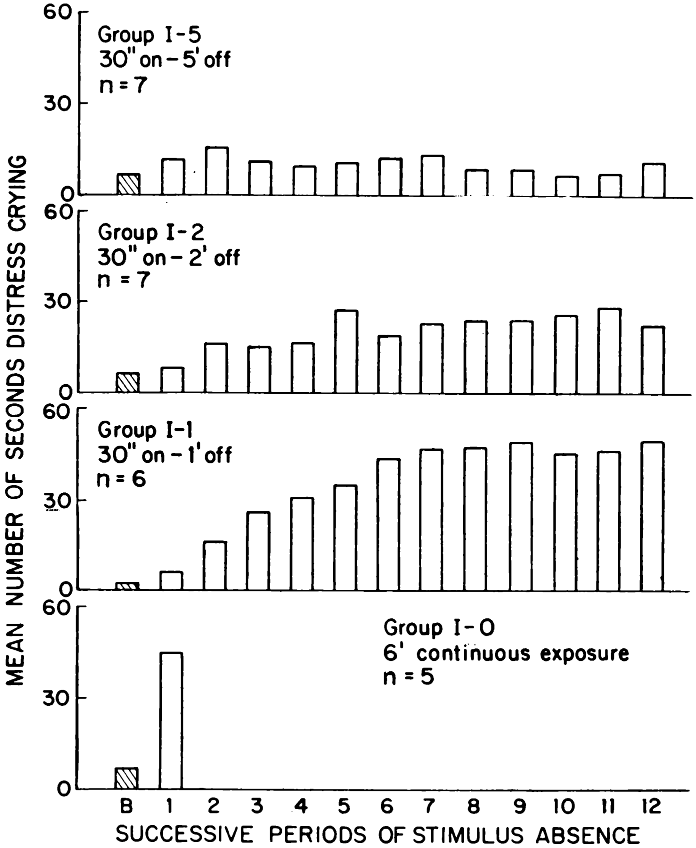

Fig. 7. The growth of magnitude of distress calling as a function of time between stimulations. Each stimulation is of 30-s duration. From the top panel, down the inter-stimulus interval is 5, 2 and 1 respectively. The bottom panel is a control condition for familiarity, a 6-min continuous exposure (zero inter-stimulus interval). Note that the distress calling magnitude stays unchanged when the inter-stimulus interval is long, but it grows when the interval is short. Adapted from Starr (14).

between presentations of the imprinting stimulus was a very powerful variable in determining the rate and amount of growth of distress-calling. The data for Group I-1 were quite similar to those recorded by

Hoffman and his colleagues at Bryn Mawr for 1-min exposures and 1-min intervals between exposures (Fig. 6). We see that distress-calling increased to an asymptote at which about 3/4 of the time was occupied by distress calls. 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 we see a crucial result. In this group, 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. The slope of the function was zero.

Now, it could be argued that the interstimulus interval had an associative function. The repetition of "imprinting stimulus present"→"imprinting stimulus gone" might 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 eleven occasions. Furthermore, following this line of reasoning, the 5-min 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! (Furthermore, I am a typical association theorist, and it is hard for me to abandon those nice old habits of thought). However, such interpretations *cannot* account for the results obtained for the control group (I-0) which received 6 min 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 3/4 of the 1-min observation interval 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. I have concluded 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. The stimulations strengthen the b-process.

From this experiment, Starr 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 duration, then the opponent-process will fail to grow. In Starr's experiment, the critical decay duration must have been between 2 and 5 min.

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 had ceased, he found that the re-strengthening of the opponent-process by repeated exposures to the imprinting object took less time and fewer exposures than had the original exposures. This phenomenon is often found in the relearning of verbal materials by human subjects. It has been called *savings.* 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. For example, in alcohol addiction the abstainer is warned that one drink may be disastrous, and the reason is the savings principle. The re-exercise 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: re-addiction 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. However we can now see the similarities extended to the fine parametric details of the opponent-process functioning.

Finally, Starr found that an enhancement of the *quality* of stimulation produced an increase in the critical decay duration of the b-process. When ducklings were exposed to an imprinting object which made honking noises, the opponent-process was strengthened rapidly, even with inter-stimulus intervals longer than the 5 min used in Starr's first experiment. A general law for the strengthening of b-processes, derived from Starr's imprinting experiments, will have to be something like this: *Opponent-processes are strengthened by use, approaching asymptotes having values directly proportional to the quality, intensity and duration of each exposure and inversely proportional to the inter-stimulus interval.*

We now have a developing science of opponent-process augmentation and weakening. 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 of acquired motivation. His ideas have considerable analytical power. We now understand some of the conditions leading either to the strengthening or weakening of opponent-processes of all types, and, consequently, to the strengthening and weakening of many experientially-acquired, new motivation systems.

It is now a matter of empirical verification to ascertain whether, in fact, the general law for strengthening b-processes does apply to many cases. Steven Seaman, working in my laboratory, has now started a series of experiments to test the application of this law to the growth of tolerance and the magnitude of the abstinence syndrome for morphine in rats. He will try to quantify the critical decay duration of the b-process for varying dose sizes 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 (14) 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 (i.e., heat, cold, sight of an enemy predator, long-distance running, weight-lifting, shocks, free falls, etc.).

The growth of adjunctive behaviors. In order to stretch the boundaries of applicability of the opponent-process theory of acquired motivation, we have entered territory quite new to us, the study of adjunctive behaviors. Adjunctive behaviors are fascinating because they appear to be a reflection of the almost-senseless generalization of one acquired motivation system to a motivation system that appears to be irrelevant. The most-studied case is experientially-induced polydipsia in the white rat, usually called "schedule-induced polydipsia". When rats are hungry and put on a fixed-time or fixed-interval feeding schedule, they have lots of free time between the spaced eatings of the tiny food pellets they are given. During this free time, if sessions are frequently repeated, the adjunctive behavior of drinking will *gradually emerge* and grow in magnitude, provided, of course, that a drinking tube is available. This adjunctive drinking becomes *polydipsic*: rats will sometimes consume 5 to 10 times their normal prandial water intake during the inter-pellet intervals. Furthermore, the bouts of adjunctive drinking tend to concentrate in the early seconds of the inter-pellet interval rather than the later seconds of the interval. Thus, one reasonable view is that the adjunctive drinking is related to attributes of the pellet *just eaten* and is *not* necessarily a reflection of a learned anticipation or expectancy of the *next pellet to come.* This type of adjunctive behavior distribution, in the between-pellet free time, can be seen in other kinds of behaviors, such as running in a running wheel, if, of course, a running wheel rather than a water spout is made available. The development of adjunctive behaviors in a function of many parameters (see 3 for a fine review). However, the opponent-process theory points to parameters that have largely been ignored. It will turn out that these parameters will cast light on the essential nature of the polydipsia phenomenon.

Assume that each tasting and swallowing of a food pellet is a positive reinforcement engendering a pleasurable a-process. The termination of this a-process should make manifest its opponent-process, an *aversive* b-process. This should occur early in the inter-pellet interval, and should reach its peak early in the interval. Given that the rate of decline of the a-process stimulus after eating is a bit slow, the peak should not be right at the start of the inter-pellet interval, but ought to occur, say, between 5–15 s after the pellet has been delivered (it takes time for the pellet and its flavor to disappear). We will call this peaking b-process *taste-craving.* It is analogous to the withdrawal syndrome for opiates or to the occurrence of separation distress in ducklings. Such an aversive b-process may be quite general in its capacity to energize behaviors. Furthermore, it may be non-specific enough so that some classes of a-processes, somewhat unrelated to the b-process in question, may, nevertheless, reduce the intensity of a B-state via the summing mechanism, /a–b/. Thus, some adjunctive behaviors might be selected because of their special capacity to participate in the /a–b/ mechanism.

In commonsense terms, the rat on an FT 2′ schedule is "bugged" by, or "aroused" by, a vague taste-craving experience after each pellet is tasted and swallowed. This aversive state primes an array of selected behavior classes, and if one of these happens to result in an a-process which reduces the sum /a–b/ by combining with the peaking b-process, then that behavior will be *selected* out for future action. However, the *motivation* for that behavior will depend solely on the existence of the aversive b-process, in this case, taste-craving. If that b-process = 0, then there will be *no* adjunctive behavior at that moment.

It takes less than great logical leaps to see the relevance of adjunctive behaviors to the opponent-process theory as elucidated in Starr's (14) work. There should be critical decay durations for the b-processes which motivate adjunctive behaviors. Each critical decay duration should be a function of the quality, intensity and duration of prior stimulation. If the critical decay duration is exceeded, there should be *no* development of adjunctive behaviors, even though stimulation is repeatedly experienced. Different adjunctive behaviors should yield differing /a–b/ summations, if we hold the b-process quality and intensity constant, and they should therefore emerge and grow at different rates. These are all matters open to empirical test. Orderly results would tend to make quite rational an area of research now clouded by mystery or fanciful, unconvincing theories (see 3). We could then think of all adjunctive behaviors as special cases of acquired motivation engendered by strengthened b-processes.

Robert Rosellini, working in my laboratory, has made a good start in exploring in the rat those aspects of experimentially-induced polydipsia of interest to an opponent-process theorist. First, he had reconfirmed the fact (see 2) that the growth of polydipsic drinking is sensitively controlled by the inter-pellet interval. There is a critical decay duration for the b-process in FT pellet schedules. Rosellini has found that, holding pellet quality and size constant, one can *prevent* the development of polydipsic drinking during the inter-pellet interval by exceeding the critical decay duration. In his research, about 200 s was the critical decay duration, using 0.45 mg pellets of chow.

The opponent-process theory requires that the quality, intensity and duration of the reinforcer must control the growth of magnitude and duration of the b-process with repetition of the reinforcer. On early trials, there should be a small contrast effect, with the b-process often being weak. On later trials, the magnitude and duration of the peak of the B-state should be greater for qualities of greater hedonic intensity. Thus, a food flavor very high in the preference hierarchy should be more potent than one low in the hierarchy in: (1) energizing a b-process, and (2) producing a longer critical decay duration of that b-process. Rosellini and Lashley (11) have tested this deduction. With a standard, fixed time, food delivery schedule known to produce polydipsia in the hungry rat (1, 2, 3) Rosellini and Lashley (11) compared the rates of development of inter-pellet drinking as a function of the flavor preference value of the particular pellets being delivered. The effects of flavor were large, indeed. In Fig. 8 are shown the adjunctive behavior growth curves for but three of many flavors Rosellini and Lashley studied: quinine-adulterated pellets, unadulterated standard pellets, and sucrose-adulterated pellets, in ascending order of taste preference, respectively. The most-preferred taste of the sucrose-adulterated pellets produced a much higher rate of development of polydipsic drinking than did the other two flavors. In addition, the asymptotic level of polydipsia was ordered by flavor preference order. Such findings are in agreement with deductions from the opponent-process model.

Because of this early success in generating and testing deductions from the opponent-process theory, I am emboldened to add a tentative hypothesis: *all adjunctive behaviors are a consequence of opponent-processes*: such behaviors are energized by the emergence of B-states correlated with sudden *termination* of a-processes, *not* the onset and maintenance of a-processes. If this is the case, then *any* clear case of adjunctive behavior should operate according to *all* of the laws of opponent-processes, to the extent that we have so far been able to discover them. Recently, Osborne (10) has shown that the magnitude of general, restless activity during the early seconds of an inter-pellet interval is directly controlled by the magnitude of the preceding food reinforcement. The principle was the same for pigeons and for rats. Figure 9 is adapted from Osborne's data. It shows the restless-activity magnitude produced in pigeons when the food was available for 1.5, 4.5 and 9.0 s respectively. The inter-food time interval was 2 min during the testing period which produced the data in Fig. 9. The amount of activity rose abruptly after each occurrence of food reinforcement had terminated. The peak of post-reinforcement activity occurred early in the inter-food interval rather than late in the interval. Finally, the peak of activity was greater for the 9.0-s reinforcement duration than it was for the 4.5-s reinforcement duration; and both of these peak activity levels were greater than that following the 1.5-s duration of reinforcement.

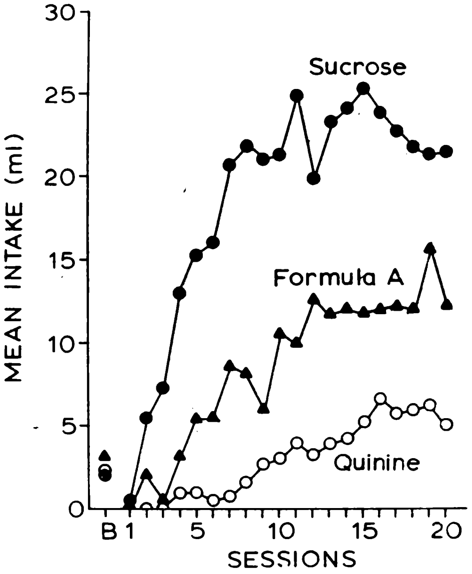

Fig. 8. The growth of adjunctive drinking as a function of the preference value of the food reinforcer. The graphs represent the mean values from 5 subjects. The greater the preference value, the more rapidly does the adjunctive drinking develop and the greater is the asymptote of polydipsia. Adapted from Rosellini and Lashley (11).

One feature of Osborne's data is especially relevant for an opponent-process theory of motivation. Note that in Fig. 9 the peak of activity was slow in growing during the inter-food interval. Furthermore, the reaching of the peak was more delayed the greater was the peak in magnitude. Such a phenomenon could be due to the particular rate of decline of the a-process as food is ingested and its flavor stimulation *slowly* terminated. The opponent-process model ideal, as shown in Fig. 2, of course assumes a *sudden* termination of the a-process stimulus. In practice, in an eating experiment, such suddenness will rarely occur. There is, however, an alternative interpretation. A fixed *rate* of emergence of the B-state might reflect some special feature of all B-state emergences, not some property of the a-process, and if so, would require a modification of the model. The strengthening of the b-process has been assumed to result in a B-state that emerges more quickly, rises to a higher asymptote, and lasts longer (see Fig. 5). The Osborne data might be reason to question this assumption, but only if we first assume that the gradual decline of taste stimulation after each reinforcement was irrelevant to the results. This possibility should be tested in appropriate experiments.

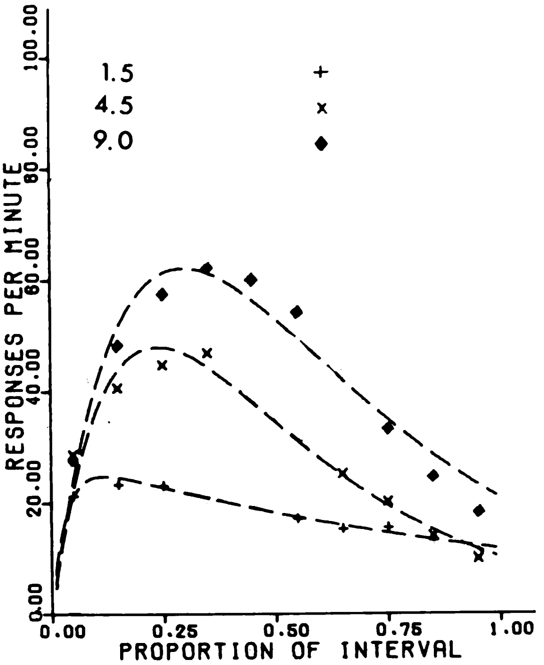

Fig. 9. The frequencies of occurrence of restless activity during different portions of a 2-min inter-pellet interval, as a function of the magnitude of food reinforcement. The 1,5; 4.5; 9.0 are the feeding times. Note that the maxima occur during the early portions of the inter-pellet interval. Note that the activity level is higher, the greater is the magnitude of each feeding. This figure is a modification from Osborne (10) p. 302.

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