Incentive Theory III: Adaptive Clocks

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A number of parametric studies have revealed a very orderly process of acquisition for autoshaped responding, and a strong dependence of the rate of acquisition on the durations of the trial (CS) and of the intertrial interval (ITI). In this article I review a few principles of reconditioning, and propose a way of combining them to account for the acquisition data.

The first principle is that incentives excite animals, with the delivery of each incentive increasing the probability of a response, either instrumental (terminal), or adjunctive (interim). A simple model of the cumulation of arousal during conditioning (and its "decumlation" during extinction) is isomorphic with a model of how animals average^{2,3}:

$$A_{t} = (1 - \beta)A_{t-1} + \beta x. \tag{1}$$

This is an exponentially weighted moving average, with A_i the average at time t, β the currency parameter, A_{i-1} the average at the previous unit of time, and x_i the "input" at time t—unity during an incentive and zero at other times.

When do animals strike the average? There are several plausible models; I assume that they are continually updating their average. These instantiations of the averaging model give us the picture in FIGURE 1, showing how the average changes as a function of the presentation and removal of an incentive, for two different values of beta. (If we take t to be measured in seconds, the values of beta actually obtained with animals are an order of magnitude smaller than these). For larger values of beta, the averager is more responsive to current input, and the system is "faster." For smaller values, the averager integrates over longer periods of time, and the system is "slower" and more stable.

The second principle is that animals cannot respond faster than some ceiling rate; any theory of how animals translate arousal into action must take that into account. One way of accommodating that principle is to assume that the proper time base for measures of response strength is not real time, but the time available for a response. If each response requires delta seconds for its emission, an animal responding at P responses per second has available only $1 - \delta P$ seconds for the P + 1st response in that time unit. If that additional response occurs, it reflects a more than proportionate effort to emit it. Our measure of response strength is then:

$$S = kR/(1 - \delta R), \tag{2}$$

and the predicted response rate for a given strength is:

$$R = S/(k + \delta S). \tag{3}$$

This second principle asserts that response rate is a severely nonlinear measure of response strength, and the above equations are offered as a first-order correction.

The third principle is the partial reinforcement extinction effect (PREE): Animals

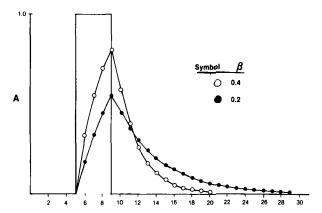


FIGURE 1. The response of an exponentially weighted moving average (EWMA) to an impulse occurring at t = 5, for two values of beta. (From Killeen. Reprinted by permission.)

trained on a schedule of intermittent reinforcement respond through periods of extinction with greater persistance than do animals trained on a schedule of continual reinforcement; in the parlance of foraging theory, "giving up time" is greater in the former case. It is as if training with intermittent reinforcement shifted an animal to a smaller currency parameter—in FIGURE 1 from a beta of 0.4 to a beta of 0.2. The currency parameter thus cannot be a "hard-wired" characteristic of the organism, but must itself be affected by the conditions of reinforcement.

The first two principles, and models that accommodate them, are adequate to characterize the time course of acquisition and extinction of behavior, but the third principle is necessary to derive the correct parameter values for those curves, as I demonstrate below.

Referring to Equation 1, in reinforcement is discontinued at t = 0 and the average

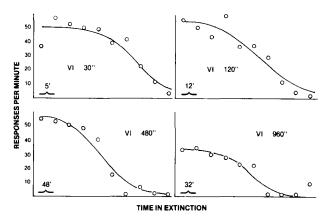


FIGURE 2. Response rates in extinction for one pigeon after exposure to various variable-interval schedules. The theoretical curves are from Equation 5. (From Killeen.² Reprinted by permission.)

then is A, at t = 1 it is $(1 - \beta)A$, at t = 2 it is $(1 - \beta)^2 A$, and at t = t it is $(1 - \beta)^4 A$. This may be written as:

$$A_1 = Ae^{-\beta t}, \quad \text{for } \beta \ll 1.$$

If we combine Equations 2, 3, and 4, and take A as the basis of response strength (S = A), we obtain predictions for the time course of extinction:

$$R = (\delta + e^{\beta t}/kA)^{-1}. \tag{5}$$

FIGURE 2 shows that this logistic equation accommodates extinction data. At the same time it illustrates the third principle, for the time scales on each of the graphs must change to accommodate the changes in persistence, reflecting the PREE. This is better seen in FIGURE 3, where the time constants (the reciprocal of the currency

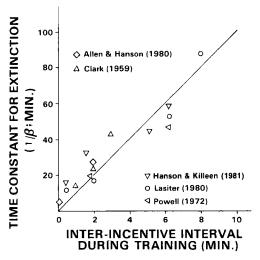


FIGURE 3. The value of the time constant $(\tau - 1/\beta)$ as a function of the interval between incentives during conditioning. (After Killeen.³)

parameters) of several extinction studies are plotted as a function of the interreinforcement interval during training.

The fourth principle is that an animal can attend to only one thing at a time, and what it is attending to at the time of reinforcement is what gets conditioned. What does an animal attend to? Signals of reinforcement. Stimuli become signals through conditioning, so that a positive feedback loop is implicated. We capture this fourth principle by letting the probability of attention to a stimulus (and therefore probability of conditioning on a trial) be proportional to the relative strengths of the signals present then.

We may now combine these principles for our theory of conditioning.

Upon the delivery of an incentive, we invoke Equation 1 to update the average arousal associated with the stimulus the animal had been attending to. Let us take the value of the reward (x) to be unity, and the probability that the animal had been

attending to the CS (rather than to the background) to be equal to its relative strength:

$$p_{cs} = A_{cs}/(A_{cs} + A_{bg}).$$
 (6)

After the delivery of the incentive, Equation 1 is iterated, and may be transformed to an exponential function as we did for Equation 4:

$$A_{cs}' = (A_{cs} + p_{cs})e^{-\beta t} \tag{7}$$

where A' is the new average, and t is the duration of the trial. This equation states that the old average is incremented by the value of the incentive (1) times the probability that the animal had been attending to the CS (p_{cs}), and this quantity is decremented by decay from the onset of the CS until the incentive is delivered (t).

A similar equation is written for A_{bg} , but with t set equal to the duration of the background stimuli, that is, the interreinforcement interval, and with p_{cs} being replaced by $p_{bg} = 1 - p_{cs}$. These measures of strength are compared at each trial. When the association for the CS exceeds that for the background by a threshold amount (DL), a response occurs to the CS.

One last step is necessary to make this a complete model. We have noted that the currency parameter is not constant, but approaches proportionality with the rate of reinforcement (FIGURE 3). We do not know how quickly or in what manner the animal rescales its clock, but rescale it it clearly does. Equation 1 is perhaps the simplest way of getting from one parameter value to another, so I invoke it as a model for changing the scale value of the clock:

$$\beta' = (1 - \gamma) \beta + \gamma c/T, \tag{8}$$

where c is the slope of the function relating the average interincentive interval to the asymptotic time constant (compare FIGURE 3), and gamma tells us how quickly the speed of the clock is updated. I labored with this model for some time before recognizing that it was fundamentally wrong. Equation 8 has the animal averaging rates of reinforcement, not interreinforcement intervals, and it generates an asymptotic clock speed proportional to the harmonic mean of the interreinforcement intervals. The data suggest, however, that animals must operate on and update not the currency parameter, but its reciprocal (the time constant, τ):

$$\tau' = (1 - \gamma) \tau + \gamma cT$$
, where $\tau = 1/\beta$. (9)

Note that we take T to be determined by the time between incentives, not the duration of the trial nor of the intertrial interval. The clock runs in real time. Its speed is adjusted during the receipt of reinforcement, and only then.

These three equations instantiate our theory of conditioning: on each trial Equations 6, 7 (for both CS and BG), and 9 are evaluated, and a response is predicted when the difference in strengths exceeds a threshold value. There are numerous parameters, but only two need to be modified to account for much of the autoshaping data: the threshold, DL, and the adaptive parameter, gamma. We may freeze c at a value of 16 for all experiments, and take the starting value for A_{bg} to be zero. Where there is explicit hopper training before conditioning trials, the three equations are iterated the proper number of times, with clock speed incrementing, but with only A_{bg} accruing strength. (Where the ITI during pretraining was unspecified, I assume it to be 15 sec). I start the time constant at zero. This rather arbitrary initial default has some intuitive appeal in that the animal starts the experiment with a very fast clock,

one that is maximally responsive to the new environment and minimally sensitive to previous conditioning histories. Other initial values, such as the 24-hour feeding cycle of the animals during deprivation conditions, were not generally as good. The initial value for p_{cs} is zero during hopper training, but must take a nonzero value when the CS is first introduced. Fortunately the model is robust over the initial value of this parameter, so that it may generally be assigned any value between .01 and .99 without affecting the goodness-of-fit. In the following analysis I employ a starting value for A_{cs} of 0.2, which starts p_{cs} somewhere in the middle of the above range, with the exact value depending on the amount of pretraining and the value of A_{bg} established by it. Of course, if a CS is thoroughly habituated before its introduction, the probability of attending to it may fall to such a level that conditioning becomes impossible.

TABLE 1. Parameters of the Adaptive Clock Model

Study	Gamma	DL	ω^2
Terrace et al.4			
Exp. 1	0.00135	4.49	.907
Exp. 2	0.00135	4.66	.899
Gibbon et al.5			
Exp. 1	0.00135	6.20	.789
Exp. 2	0.00135	5.75	.801
Gibbon et al.7			
Partial reinforcement	0.00135	8.00	.871
Downing and Neuringer ⁸			
Pretraining	0.00280	8.54	.998
Tomie9			
Pretraining	0.00315	9.65	.702
Balsam and Schwartz ¹¹			
Pretraining	0.00255	3.94	.812
Jenkins et al. 13			
Exp. 1-6	0.00085	2.17	.556
Exp. 7–8	0.00085	0.00	.876

Note: The parameter c was always 16, and the starting value for A_{cs} was 0.2 in all experiments except that of Downing and Neuringer, where it took a value of 0.0010. ω^2 is the proportion of variance accounted for by the model, using the logarithm of the acquisition scores. Medians of individual scores were used where possible. Unless otherwise noted in the text, if more than one of a group of animals failed to respond within a condition, and only a measure of central tendency was given that did not include those animals, those data were omitted from the analysis.

COMPARISON WITH DATA

Terrace, Gibbon, Farrell and Baldock4

Terrace and his associates kept the CS constant at 10 sec and varied the intertrial interval (ITI; the time from the end of one incentive to the beginning of the next CS) between values of 5 sec and 400 sec. In Experiment 1 the response key was dark between trials, and in Experiment 2 it was lit with a different color. Because only the ITI was varied, we may graph the number of trials before the first response as a function of the ITI. A straight line through these points misses the extremes, projecting a median of 141 trials for a 5-sec ITI (only one of four subjects responded in that condition, and that one on the 272nd trial). The adaptive clock model does better; the

parameters are given in TABLE 1. The improvement is greatest at short ITIs; for example, the model predicts 300 trials for a 5-sec ITI.

Gibbon, Baldock, Locurto, Gold, and Terrace5

Gibbon and his associates varied both the CS and the ITI over large ranges. In the first experiment, the durations of the ITIs were variable, and in the second they were fixed. Incentive theory accounts for the distribution of data better than a power function (see TABLE 1), although not as well as one might like. (The investigators excluded 5 data points from their analysis: three with trial durations under 4 sec, and

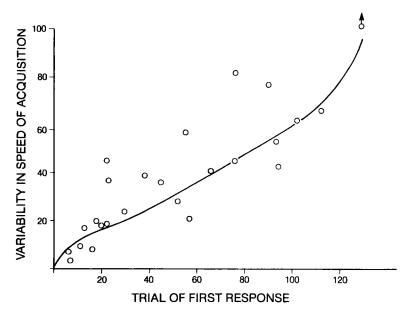


FIGURE 4. The standard deviation of the trial of the first response, plotted as a function of the predicted trial of the first response. (The data are from the first experiment reported in Gibbon *et al.*⁵; the curve is the predicted range of the trial numbers when the value of DL is varied by \pm 25%.)

two with extremely large ratios of ITI to CS. At very short trial values, the pigeons move directly to the hopper—they "goal track." I also exclude those points, but not the other two). Some of the variability in these data may be caused by the variable amount of pretraining that the subjects received (20 to 200 trials): in calculating the starting values I assumed an average of 50 trials for all.

If some of the variance among subjects in the rate of acquisition is due to differences in the values of the threshold, we may select some arbitrary range for that difference and see what degree of variability the model predicts for the different experimental conditions. I evaluated the model for threshold values of \pm 25% of DL, and plotted the range of predicted trial of first peck (FIGURE 4, curve) as a function of the trial predicted using a threshold of exactly DL. The data are the standard

deviations of the acquisition scores for Experiment 1. The small sample size causes substantial scatter, but the predicted increase in variability with increases in the number of trials to acquisition is apparent. The reason for this increase in variability is that under extended training, the growth of strength for CS and for background becomes nearly parallel, so that any given change in threshold will be reflected in a larger range of acquisition scores. Another potential source of variability may be found in the variable schedules of intertrial intervals; several long intertrial intervals in a row might weaken background control enough to bring about an early autoshaped response. However, the parameters for gamma are very conservative, so that variability in A_{bg} is highly damped; in fact, the model predicts no greater variability in this condition, and the data show no evidence of it either.

Gibbon, Farrell, Locurto, Duncan and Terrace7

Gibbon and his associates omitted the food on a fraction of the trials, for a range of different intertrial intervals. This experiment permits us to resolve some fundamental issues concerning the assumptions of the theory: Do pigeons adjust their time constant on trials when reinforcement is omitted? Do they adjust it to the intertrial interval or to the interreinforcement interval? Does their average arousal change on nonreinforced trials? I evaluated the fit of the model to the data, using all permutations of these assumptions. Only two sets of assumptions permitted a good fit to the data, and they were equally good, both accounting for 87% of the data variance. The first construction was that nothing happened on nonreinforced trials, except that an additional count was added to the acquisition score. The other, and more plausible, construction was that the average arousal changed on every trial (decrementing on every trial, incrementing only on reinforced trials), but that the clock speed was adjusted only on reinforced trials, and then the adjustment was based on the time since the last reinforcement, not on the ITI. As in the previous studies, a variable number of training trials were administered before the autoshaping regimen began, and I have set that value at 50 (the model is robust over a plausible range of values). The parameters are shown in TABLE 1.

Downing and Neuringer⁸

These researchers explicitly studied the effect of hopper training trials on acquisition. They found that number of trials to acquisition was a U-shaped function of the number of training trials, with 1,10,100, and 1000 trials entailing 54, 43, 19, and 145 acquisition trials. Incentive theory can accommodate these data only by assuming a very small value for A_{cs} at its first introduction (that is, the model asserts that the probability of attention to the CS on the first trial is very small). This is the first study where the starting value of A_{cs} (and thus for p_{cs}) matters, and that is because of the nonmonotonic change in acquisition scores. According to the model, increasing the number of training trials slows the clock (increases τ); this is beneficial to conditioning because it speeds the cumulation of arousal, and since the CS is gaining strength at a faster rate than the BG, speeding the clock differentially benefits the CS. But increasing the number of pretraining trials also has a deleterious effect because it permits the BG a head-start on accruing strength, and concomitantly of capturing the attention of the animals. For the parameter values used to fit these data the first factor (modification of clock speed) outweighs the second for up to 100 pretraining trials. But as pretraining continues, the starting value for A_{bg} becomes so large relative to the starting value of A_{cs} that it requires many trials to shift attention to the CS. Because this is a probabilistic process, and just one trial of attention to the CS will have a

dramatic effect on the strength of the CS and on the attention subsequently given to it (with responding occurring soon thereafter), we expect a larger variance in the acquisition scores here than for any other condition. (In the other conditions, incremental strengthening dominates the processes, not probabilistic shifts in attention). This was found.

The fit of the model to the data was almost perfect, but that is not surprising, given three parameters and only four data points. Still, I know of no other models that have been elaborated enough to grapple with such data. The values for gamma and DL are relatively close to the values from other experiments. The small starting value for $A_{\rm es}$ (0.0010) may be idiosyncratic to these subjects, Cornish chickens 4 days old at the start of the experiment. The range of values acceptable for all of the other studies (0.01 to 1.0)—quite large, but definitely not including the value used in this study—suggests that the upturn at 1000 pretraining trials will not be found for mature pigeons.

Tomie9

Tomic replicated part of Downing and Neuringer's study to see whether changing the background stimuli after pretraining would minimize its deleterious effects. Our predictions are straightforward: removing the stimuli that had accrued strength during pretraining should greatly facilitate training by minimizing competition with the CS. This effect should be greatest for the group that received extensive overtraining, and less for the group that received only basic hopper training (the "original learning control" or OLC group; both that group and the experimental group received 50 hopper training trials. I have assumed a 45-sec ITI during this condition, the value used in other studies by this author¹⁰). Tomie plotted the average number of trials with one or more responses in each block of ten trials during autoshaping. This way of reporting results provides many more data than the trial of the first response, and is therefore a more efficient use of the data. In fact, we might invoke Equation 5 to fit the complete acquisition curves. Here, however, I have merely interpolated for the estimated trial of the first response. The obtained (and predicted) values for the same/different background are: OLC, 20/10, (25/14); overtraining: 65/17 (87/10). I assumed a starting value of 0.2 for A_{bg} , the same as in all other studies using pigeons; because of the large number of pretraining trials in this study, however, the range of acceptable values is smaller, needing to be somewhere between 0.1 and 0.5.

The two effects of pretraining—the beneficial effect of speeding the clock and the deleterious effect of increasing $A_{\rm bg}$ —are thus separable. Note that we predict fewer trials to acquisition in the different condition for overtraining than for OLC. That prediction was not born out here, but it was in another study by Tomie¹⁰ that involved four groups: an OLC group and three groups with an additional 600 pretraining trials. Two of these latter groups then underwent two sessions of extinction, one in the training context, and one in a novel context. Remembering that extinction does not affect the speed of the clock, we predict fastest conditioning for the group extinguished in the same context, next for the OLC group, and slowest for the groups extinguished in the novel context and the group not extinguished (with differences there depending on the similarity of the novel context and the standard one). His FIGURE 2 shows that this is just what he found.

Balsam and Schwartz¹¹

This is another study of the effects of pretraining. Pigeons were given 150 hopper training trials in a chamber lined with cardboard and then 4, 8, 64, 128 or 256

pretraining trials in the unlined chamber, followed by autoshaping in the unlined chamber. We predict that hopper training should speed up the clock, but, since the BG was changed for the subsequent conditions, it should have no effect on A_{bg} . Pretraining should affect both A_{bg} and clockspeed (although the latter should be a substantial distance toward asymptote). The net effect of pretraining should thus be to hinder conditioning. Exact predictions are made complex by the investigators' tactic of varying hopper duration during pretraining to keep total access to food constant. The effects of hopper duration on conditioning are strongly concave, with little additional benefit derived from durations longer than 3 or 4 sec; three 3-sec hopper presentations are much more effective than one 9-sec presentation. As a first pass, we may assume that the effects of all hopper durations were equal. Optimal fits of the model under that assumption account for 75% of the data variance. Alternatively, we may invoke a model for the effects of hopper duration derived for the study of choice behavior. 12 That model takes the incentive value of D seconds of access to grain to be equal to $1 - e^{-\lambda D}$, and placed the value of lambda at about 0.7. Using that same "utility" function improves the accuracy of the predictions to 81%.

Jenkins, Barnes, and Barrera¹³

These investigators studied a range of experimental conditions that permitted them to address the question of why autoshaping depends on trial spacing. If the answer given by incentive theory and the associated model of adaptive clocks is correct, we should be able to predict, at least qualitatively, their results. However, it is difficult to make only quantitative predictions with our theory, because the complex interactions of clock speed and differential accumulation of arousal makes a number of different qualitative predictions possible for any situation. It is necessary to pin down the model by giving it explicit experimental parameters and then requiring explicit numerical predictions.

In analyzing the data of Jenkins and his associates, there are too few data points per experimental condition to find optimal parameter values in each case. Therefore I have used the same values for most of the experiments. The authors employed 16 sessions (640 trials) of "preliminary tray training" in the first study, and I assumed 5 "in other experiments [where] far fewer preliminary sessions were used."

The first study examined ITIs of 30 sec and 300 sec, and found acquisition in 55 and 10 trials, respectively. The model predicts 80 and 12 trials. (These investigators report medians of subjects that were conditioned; medians over all subjects would be larger, especially when many subjects failed to become conditioned. If we assume that all remaining subjects would have become conditioned immediately after the experiment was terminated for them, and we calculate the weighted geometric mean of their scores and the reported scores, the estimates increase to 90 and 14). In the second study the authors scheduled four extra feedings during the 300-sec ITI. They found that the location of the extra feedings within the ITI did not matter, and that on the average the animals required 22 trials before initiating a keypeck. The model is applied by iterating Equation 7 for A_{be} four additional times within each trial, which leads to a prediction of 16 trials. The third study involved very long trials, with a feeding immediately before the CS in one condition, and no prior feeding in the other. Half the animals reached criterion by 5 trials (first condition) and by 8 trials. The model predicts 5 trials for both conditions. In the fourth study an unpaired CS was presented on three occasions during the ITI. The location of the CS did not matter, and the average number of trials to criterion was 32. In the model, we iterate the extinction of A_{cs} (Equation 4, with t =CS) an additional three times during each trial, and predict 6 trials to acquisition. This is the one prediction that is in serious error. I discuss this problem below. In the fifth

experiment, the animals were trained on ITIs of 30 or 300 sec. Half of the subjects began their first trial after waiting 30 sec (W30), and half after waiting 300 sec (W300). For the 30-sec group the wait manipulation had no effect, with both groups responding on about the 32nd trial. The model predicts 25. For the 300-sec group, the median number of trials to criterion was 6 for the W30 and 2 for the W300. The model predicts 6 and 6. In Experiment 6, the investigators employed a type of variable ITI scheduling, with an average ITI of 300 sec, but with one 22-min interval and five 30-sec intervals. The model predicts autoshaping by the ninth trial, while the data show that it occurred by the eighth trial.

The next two experiments employed only a single trial per session, and demonstrated much faster acquisition than the previous studies. It is necessary to reduce the threshold parameter to a value of zero in order to capture the data. In Experiment 7, the sessions lasted for 15 min, with the chamber dark during the first 7 min, the last 7 min, both, or neither. I treat the first two conditions as the same (excluding the BO from calculation of T), and predict 3 trials to acquisition; the authors measured 5 trials and 2 trials. With no blackout, they found acquisition by the fourth trial; the model predicts 2. With two blackouts they found acquisition by the 18th trial; taking the functional duration of BG to be T=38 sec, the model predicts 20 trials. (The BG remained on for an additional 22 sec after the feeding; since no other feedings were possible after the single one, it is reasonable to presume that trace inhibition from the feeding caused it to function as a different stimulus. Had the 22 sec been included, the prediction would drop to 16 trials).

In Experiment 8 Jenkins and associates paired the CS with a feeding only once in the middle of a session, and preceded and followed that trial with 15 unpaired feedings coming once every 30 sec, once every 300 sec, or with 7.5 min with no additional feedings. They found that only one of five pigeons were conditioned by the end of the experiment (30 sessions) in the first condition; the model predicts they should have been conditioned, on the average, by the 55th session. The authors found acquisition by the sixth trial in the low-density feeding condition, and by the third trial in the no-additional-feeding condition; the model predicts 6 and 2 trials.

The quality of these predictions across experimental conditions, and with few changes in theoretical parameters, is generally very good, and certainly the predictions are within the standard error of the mean of the data in almost all cases. The exception is Experiment 4, where the predictions were grossly in error. I do not know why they were. A similar experiment was conducted by Gibbon and Balsam¹⁴ who compared groups with a 10-sec CS, a 5-sec CS, and a 5-sec CS with an additional 5-sec stimulus at the beginning or middle of the interval. The latter two groups did not differ in trials to acquisition (both required about 58). The first group required 40 trials, and the second 23. Using the same parameters as in the first study by Gibbon et al. (but with DL slightly higher: 6.6) generates reasonable predictions of 49, 49, and 23. The map between theory and this experimental manipulation thus remains uncertain.

DISCUSSION

There are three sources of error in the map between data and theory. The first is the large variability intrinsic to the data. According to the theory, much of this variability is due to the slow convergence of two nearly asymptotic curves, with small variations in attention or threshold having a magnified effect on responding, in much the same way that the movement of nearly parallel lines closer by a millimeter can move their point of intersection by many meters. This type of error is greatest when acquisition is slow, and the curves are more nearly parallel; note in FIGURE 4 that when acquisition occurs

on the 100th trial, the standard deviation is about 65; if this estimate was based on 5 subjects, there is only a 50% chance that the population mean lies between 80 and 120. This can be improved with very large Ns (with 10 subjects, the probable error in estimating the mean reduces to \pm 14 trials), or with a theory that lets one pool information across different conditions (such as a regression model; of course, the linear assumptions that involves fail in the extremes. Regression's utility as an exploratory tool does not make it a substitute for grounded theory).

There are some ways of reducing this source of error other than increasing the number of subjects. The use of an acquisition criterion other than the first response provides more reliable estimates of the point at which responding goes suprathreshold. This may be carried a step further in the following technique: Plot the cumulative number of trials on which a response has occurred against trial number, and interpolate for the trial of the first response. This assumes linearity, but over such a small range that more good will be done in attaining a reliable estimate than harm will occur from the introduction of bias. The technique assumes that there are not important sources of curvilinearity, such as a positive acceleration due to instrumental conditioning. Incentive theory does not invoke the hypothesis of instrumental conditioning, and from its viewpoint one would be surprised to see a positive acceleration.

Finally, we must admit that the trial of the first response is a very inefficient dependent variable; weeks of housing, establishment of ad libitum weights, reduction to 80%, pretraining, and final autoshaping bring forth a single datum, and not a very reliable one at that. The problem is in the end not an empirical one—it is a problem with the theory that must insist upon this very expensive input. The trial of the first response is merely the first point on an acquisition function; a theory that treats the function as a whole will have available a hundred-fold more data from a single additional session. Although we often hear the cry for more data, here it is more theory that is needed. I have outlined one appropriate theory in this paper, but have not yet begun to test it against the available data.

The second source of error lies in ambiguity about some of the experimental parameters. The intertrial interval during pretraining and the number of pretraining trials are often unspecified. If they are kept constant across subjects, the major effect will be on the particular values of the optimal theoretical parameters for the model. If these experimental parameters vary between subjects or conditions, however, they will be a source of variance that is not reducible, unless their values are reported and can thus be included in the model.

The third source of error lies in the nature of the theory, and its adequacy in representing the data; the theory could be wrong. Incentive theory and its model of adaptive clocks have gone through many revisions. Some were mere errors, others were plausible but incorrect instantiations (for example, Equation 8). In some cases, I had to revise provisional assumptions that subtly lost their provisional status. (For example, I kept the value of c constant at the value found in FIGURE 3, 10.0, for this provided an excellent fit to most of the data. However, it also required different values for gamma in situations where gamma should not have changed [such as between conditions in the first two studies listed in TABLE 1]. A larger value for c removed that irregularity. Optimization across experiments can only come after the major variables are identified and accounted for within studies: Theory construction is an iterative process.)

Some parts of the model are not essential for the majority of the parametric data. This is the case with the attention assumption, and the consequent requirement that the starting value of A_{cs} be set somewhere greater than zero. This is a reasonable modification, but it introduces a new parameter that comes into play only in a few conditions where there are many pretraining trials, and A_{bg} becomes large enough to have a chance of competing with A_{cs} for the animals' continued attention. I developed

this part of the model after Herb Jenkins impressed upon me the "path dependence" of autoshaping, with conditioning being difficult or impossible if the CS is thoroughly habituated. But I believe that the attentional mechanism is an important addition, for it may help to make sense out of a number of different types of studies, such as Williams's experimental analysis of the blocking of a conditional discrimination by preexposure to one of the stimuli, and the interaction of that effect with the locus of the stimulus, ¹⁵ and Roberts and Kraemer's ¹⁶ observations of the dependence of accuracy on the ITI in matching to sample experiments. In a subsequent article I modify Equation 7 so that extinction of a stimulus occurs only while an animal is attending to it. For a CS, t is replaced by p_{bg}^* CS. For the background, t is replaced by BG-CS + p_{bp}^* CS, which assumes complete attention to the background before the onset of the CS, and partial (or probabilistic) attention during the CS. The only effect of these modifications on the analysis of autoshaping data is to increase the values of gamma by 10%.

A provocative consequence of the theory is that the speed of the animal's clock is always changing as a function of its rate of reinforcement. Although this variation will cancel out of some psychophysical measures,¹⁷ it will not always cancel. The implications are profound: Animals drift through a relativistic world in which they have no access to absolute measures of time other than gross circadian rhythmicity. Yet, some data support this relativistic implication. Treisman noted that "facilitation of the pacemaker by the specific arousal center affects [increases] the rate at which pulses are produced." Unless there are multiple clocks available to animals, incentive theory predicts that their estimates of time will vary as a function of intertrial intervals and as a function of exposure to conditioning and extinction extraneous to the events being timed. This implication will soon be tested.

SUMMARY

Incentive theory is extended to address the phenomenon of autoshaping. To do so, it is necessary to permit the speed of the animal's internal clock to vary with rates of reinforcement; clock speed is the basis for the animal's calculations of reinforcement densities. This notion of an "adaptive clock" is consistent with other effects, such as the partial-reinforcement extinction effect, and permits us to deal with the various experimental manipulations that are found in autoshaping experiments from a unified perspective.

REFERENCES

- KILLEEN, P. R. 1979. Arousal: Its genesis, modulation and extinction. In Reinforcement and the Organization of Behavior. M. D. Zeiler & P. Harzem, Eds.: 31-78. Wiley. New York, NY.
- KILLEEN, P. R. 1981. Averaging theory. In Quantification of Steady-State Operant Behaviour. C. M. Bradshaw, E. Szabadi & C. F. Lowe, Eds.: 21-34. Elsevier. New York, NY.
- KILLEEN, P. R. 1982. Incentive theory. In Nebraska Symposium on Motivation, 1981. D. J. Bernstein, Ed.: 169-216. University of Nebraska Press. Lincoln, Nebraska.
- TERRACE, H. S., J. GIBBON, L. FARRELL & M. D. BALDOCK. 1975. Temporal factors influencing the acquisition and maintenance of an autoshaped keypeck. Anim. Learn. Behav. 3: 53-62.
- GIBBON, J., M. D. BALDOCK, C. LOCURTO, L. GOLD & H. S. TERRACE. 1977. Trial and intertrial durations in autoshaping. J. Exp. Psychol. Anim. Behav. Processes 3: 264-284.

- LUCAS, G. A. & E. A. WASSERMAN. 1982. US duration and local trial spacing affect autoshaped responding. Anim. Learn. Behav. 10: 490-498.
- 7. GIBBON, J., L. FARRELL, C. M. LOCURTO, H. J. DUNCAN & H. S. TERRACE. 1980. Partial reinforcement in autoshaping with pigeons. Anim. Learn. Behav. 8: 45-59.
- DOWNING K. & A. NEURINGER. 1976. Autoshaping as a function of prior food presentations. J. Exp. Anal. Behav. 26: 463-469.
- TOMIE, A. 1981. Effect of unpredictable food on the subsequent acquisition of autoshaping: Analysis of the context-blocking hypothesis. In Autoshaping and Conditioning Theory. C. M. Locurto, H. S. Terrace & J. Gibbon, Eds.: 181-215. Academic Press. New York, NY.
- TOMIE, A. 1976. Interference with autoshaping by prior context conditioning. J. Exp. Psychol. Anim. Behav Process. 2: 323-334.
- BALSAM, P. D. & A. SCHWARTZ. 1981. Rapid contextual conditioning in autoshaping. J. Exp. Psychol. Anim. Behav. Processes 7: 382-393.
- KILLEEN, P. R. 1984. Incentive theory and amount of reinforcement. J. Exp. Anal. Behav. In press.
- JENKINS, H. S., R. A. BARNES & F. J. BARRERA. 1981. Why autoshaping depends on trial spacing. In Autoshaping and Conditioning Theory. C. M. Locurto, H. S. Terrace & J. Gibbon, Eds.: 255-284. Academic Press. New York, NY.
- GIBBON, J. & P. BALSAM. 1981. Spreading association in time. In Autoshaping and Conditioning Theory. C. M. Locurto, H. S. Terrace & J. Gibbon, Eds.: 219-253. Academic Press. New York, NY.
- WILLIAMS, B. A. 1982. On the failure and facilitation of conditional discrimination. J. Exp. Anal. Behav. 28: 265-280.
- ROBERTS, W. A. & P. J. KRAEMER. 1982. Some observations of the effects of intertrial interval and delay on delayed matching to sample in pigeons. J. Exp. Psychol. 8: 342– 353.
- 17. GIBBON, J. 1981. Two kinds of ambiguity in the study of psychological time. *In Quantitative Analysis of Behavior: Discriminative Properties of Reinforcement Schedules. M. L. Commons & J. A. Nevin, Eds.:* 157–189. Ballinger. Cambridge, MA.
- 18. TREISMAN, M. 1963. Temporal discrimination and the indifference interval: Implications for a model of the "internal clock". Psychol. Monogr. 77(13): 1-31.