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THE EFFECT UPON SIMPLE ANIMAL BEHAVIOR OF DIFFERENT FREQUENCIES OF REINFORCEMENT, PART II: SEPARATE CONTROL OF THE REINFORCEMENT OF DIFFERENT IRTs[1]

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Rats' responding was stabilized for over 35 days on 4-min variable-interval reinforcement. Reinforcements per hour for 4-sec wide classes of interresponse times were then separately controlled by adjusting those for each class to the variable-interval values that had just prevailed. This produced little or no change in interresponse times, indicating that the new procedure was substantially equivalent to a variable-interval schedule. The variable-interval schedule produced a high and stable conditional probability of interresponse times in the 0- to 4-sec class, associated with a peak in reinforcements per hour for this class. Reducing the reinforcements per hour for this class while raising that for another class (by 3.3 reinforcements per hour) significantly reduced the conditional probability of 0- to 4-sec interresponse times. Restoring the 3.3 reinforcements per hour to the 0- to 4-sec class significantly elevated the conditional probability of interresponse times in this class. Hence, it is concluded that the distribution of interresponse times produced by a subject during some variable-interval schedules is determined partly by the relative reinforcement of different interresponse times that the variable-interval schedule provided.

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COMMENT BY AUTHOR

Part I of the Surgeon General report, with minor modifications, was submitted as a Ph.D. thesis to Harvard University in 1955 and later published (Anger, 1956). Part II was not published, although mimeographed copies were distributed, and it was deposited in the American Documentation Institute (Library of Congress) as Document 7779. Since Part I is already published, it is not reprinted here, but it should be consulted for introductory material, usage of terms such as IRTs/Op, further apparatus details, and where reference is made to Part I. A few editorial changes should be obvious (e.g. *from* compartment *to* band). *Figure 4 of the Surgeon General report was dropped from the 1956 paper, so references in Part II to figures after Figure 4 in Part I should be reduced by one in consulting the 1956 article.*

Part II was not published in 1956 because better experiments were planned, but that research was interrupted. The invitation to reprint Part II at this time was accepted in spite of the shortcomings of the study, in the hope that it might bring attention to its methods, methods that still seem particularly appropriate to the problem. The current status of the complex problem involved in this study cannot be reviewed here, but it may be helpful to comment on several aspects of the problem that seem to have been misunderstood or overlooked.

It seems important to recognize that there are several hypotheses of differential reinforcement of IRTs. Two somewhat different proposals were discussed in Part I (Anger, 1956), though unfortunately they were not sharply distinguished there. The title, the first paragraph of the introduction, and the first paragraph of the summary discussed in general terms the possibility that during reinforcement schedules some measure of the relative frequency of reinforcement given to different IRTs plays an important role in de-

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termining the relative frequency of the different IRTs. That general proposal *of differential reinforcement of IRTs was based on Skinner's (1938, p. 284) earlier and more specific proposal that differences in the probability of reinforcement (reinforcements/IRT) of different IRTs influence the relative frequency of different IRTs. However, the general proposal was intended to lack any commitment concerning the measures of reinforcements or IRTs, or the possible effects of other differences between IRTs of different length, such as differences in the frequency of unreinforced IRTs.*

Most of the findings of Part I only supported the general proposal, but one finding favored a particular relation between reinforcements and IRTs. Exposing an animal to a VI schedule produced changes in the animal's IRTs/Op (conditional probability of response) curve that resulted in a closer resemblance between the IRTs/Op curve and the schedule reinforcements per hour curve than was present initially. Consequently, a more specific proposal was also made in Part I (paragraph 3 of the summary, and p. 154) that the change in shape of the IRTs/Op curve was controlled by the relative reinforcements per hour received by different IRTs. Let us call this more specific proposal the reinforcement-rate proposal *of differential reinforcement of IRTs. The difference between the two proposals is that the general proposal* only *says that at* least *relative reinforcements influence relative IRTs; the general proposal does not specify measures, other influences, and the nature of the relation. The reinforcement-rate proposal goes further (as did Skinner's proposal) by suggesting a particular measure of the reinforced-IRT distribution and a particular measure of the IRT distribution. Distinction between these different proposals seems desirable, because their present status seems to be different. Evidence has continued to accumulate for the general proposal, but little additional support has appeared for the reinforcement-rate proposal, and some reported evidence does not agree with it (Blough, 1966).*

The experiment of Part II was designed from the point of view of the reinforcement-rate proposal (i.e., *the rate of reinforcement of different IRTs was controlled). However, the major results of Part II support only the general proposal, because manipulating the rate of reinforcement also changes the reinforcements/IRT and other reinforcement measures. (Some incidental observations were mentioned that supported the reinforcement-rate proposal, but they were said to have "little weight"). Consequently, Part II does not appreciably favor the reinforcement-rate proposal as compared with alternative specific proposals.*

Some independent observations were reported in Part I that were relevant to the question of what mediates the reinforcement-IRT dependence. However, neither proposal was intended to be committed to any particular theory of what mediates the dependency (discrimination versus *response differentiation,* etc.) *or to any theory of what is responsible for the timing. All these proposals seem to be equally compatible with all the various mediation and timing theories.*

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PART II

The following experiment was designed to reveal whether rats are adjusting their production of IRTs to the relative reinforcement of different IRTs during a VI schedule. In Part I it was shown that such adjustment takes place under some conditions (*e.g.,* reinforcement of only > 40 sec IRTs); does this adjustment also take place during VI? In Part I it was found that VI provides more reinfs/hr for short IRTs than long, and this differential reinforcement matches the observed change

from equal IRTs/op at different IRTs to greater IRTs/op at short IRTs. The match suggested that adjustment of the IRTs to the reinforcement distribution generated by VI was taking place, but the possibility remained that some other factor might be responsible for the peak in the IRTs/op curve at short IRTs and the match between the IRTs/op and reinfs/hr curve might be fortuitous.

One way to show the presence of this adjustment during VI is to show that small changes in the reinforcement distribution from that produced by VI result in small changes in the IRTs/op curve. The reinforcement changes should be large enough so that a significant change in the IRTs/op curve results, but they should be small enough so that the reinforcement distribution and IRTs/op curves differ little from those of VI, and hence the situation producing the adjustment is comparable with VI. The radical changes in the IRTs/op curve produced by reinforcement of only > 40 sec IRTs showed adjustment but the conditions producing the adjustment were so different from those of VI that some doubt remained whether VI also produced adjustment.

The results from Part I indicated that it is the relative reinfs/hr of different IRTs, the reinfs/hr curve, that influences *S*'s responding. But it is difficult to make small changes in the reinfs/hr curve since the reinfs/hr curve is not entirely under experimental control, it depends partly on *S*'s behavior. The reinfs/hr in any compartment equals the IRTs/hr in that compartment (determined by the rat) multiplied by the reinfs/IRT of that compartment (determined by the schedule). Hence if a small increase in the reinfs/hr of certain IRTs does increase the corresponding IRTs/op this IRTs/op increase will further increase the reinfs/hr. In addition the chance variation of the reinfs/hr in an individual compartment is great at 5-min VI since with 5-min VI there are only a total of 12 reinforcements for all compartments in an hour. Obviously this lack of experimental control over the reinfs/hr curve during VI raises a serious obstacle to the demonstration of significant IRTs/op changes due to small reinfs/hr changes. Consequently this experiment is best done by eliminating the dependence of the reinfs/hr curve on the *S*'s behavior and bringing it under experimental control. This was accomplished by controlling separately the reinforcements for the different 4-sec-wide compartments out to 28 sec. The reinforcements for 0-4 sec IRTs were programmed independently of other IRTs as were 4-8 sec IRTs, *etc.* Let us call this sort of reinforcement control *IRTs-separate reinforcement* or *IS*. Since control of the reinfs/hr curve was sought in this experiment, FI or VI schedules were always used for each compartment, though of course FR and VR schedules can be used also for different compartments in IS.

The use of IS requires demonstration that IS satisfactorily duplicates VI, so the following general plan will be used in this experiment. The *S*s will first be given 4-min VI. Four-min VI will be used instead of the 5-min VI used in Part I because the Part I conclusions indicated that a higher reinforcement rate should produce a more pronounced peak in the IRTs/op curve at short IRTs and should produce it faster. When the IRTs/op curve produced by 4-min VI is relatively stable, the reinforcement control will be changed to IS with the reinfs/hr in each 4-sec-wide compartment out to 28 sec set at the same values as those just prevailing on the 4-min VI. This might be called "synthetic 4-min VI" since it duplicates what appear to be the major variables (the reinfs/hr curve, the irregular temporal spacing of reinforcements, etc.) that control the responding during VI. Of course "synthetic VI" only duplicates the reinfs/hr curve that occurs at one point in the complex sequence of changes that seem to occur during VI, since IS does not duplicate the circular relation which obstructs the objective of this experiment. This "synthetic VI" will be given to determine whether IS duplicates adequately the effect on the rat of VI. Generalization from the results with IS to VI will only be permissible if IS does not produce any important change in the responding. If no important changes result from IS, then it will be worthwhile to move a few reinforcements from one compartment to another, and determine whether the IRTs/op curve shows a corresponding change.

Treatment 6 of Part I revealed a danger that is present when there is interval reinforcement of a restricted group of IRTs. When only < 28 sec IRTs were reinforced in Treatment 6, the rat reacted to the more frequent reinforcement of short IRTs following long IRTs than short IRTs following short IRTs. Since in IS short IRTs will be reinforced on a separate sched-

ule, short IRTs will be reinforced more following long IRTs, and the rat may react to the difference. If the rat does react to this difference between VI and IS, this difference in the relation between reinforcements and the second-back IRT, then different behavior will result from VI and synthetic VI, and results obtained with IS will not be fully applicable to VI. To eliminate this difference between IS and VI, arrangements were made so that after the timer tape assigned reinforcements to 0-4 sec IRTs, the reinforcement of a 0-4 sec IRT was delayed until a certain number of 0-4 sec IRTs had occurred. In this way the probability of reinforcement of 0-4 sec IRTs was made relatively independent of the IRT length preceding the IRT reinforced, as it is in VI. This arrangement was clearly unnecessary with long IRTs, such as 20-24 sec IRTs whose frequency was low enough so that IRTs of other lengths almost always occurred after the assignment of a reinforcement and before its delivery. Perhaps the arrangement was unnecessary with short IRTs too, since it turned out that the response rate was high, hence the differences were not large between the reinfs/hr following short-short IRT sequences and the reinfs/hr following long-short IRT sequences (see under Treatment 6, Part I). However, there was doubt whether this synthetic VI would satisfactorily duplicate VI reinforcement, so the first attempt was made under as favorable conditions as possible.

METHOD

Apparatus

Animal cages. Same as in Part I.

Electrical circuit. The electrical circuit used was the same as in Part I except for the reinforcement control circuit. Even the reinforcement control was the same as in Part I during Treatment I (4-min VI). However during Treatments 2 through 4 of Part II the circuit provided the following IS control: For each rat there were 8 punched tapes all driven by the same timer; one punched tape for each 4-sec-wide compartment out to 28 sec, and one for $>$ 28 sec IRTs. After assignment of a reinforcement by the tape for 16-20 sec IRTs, the next 16-20 sec IRT was reinforced. The same sort of arrangement, though independent ones, existed for 20-24, 24-28, and $>$ 28 sec IRTs. With $<$ 16 sec IRTs the circuit delayed the reinforcement for several IRTs after the assignment in order to eliminate the above described dependence of the reinforcement probability on the preceding IRT that otherwise occurs with VI of a restricted group of IRTs. After a reinforcement assignment by the tape for 12-16 sec IRTs, the third 12-16 sec IRT that occurred was reinforced. (Note especially the following aspects of this arrangement: (a) the third 12-16 sec IRT, not the third IRT is reinforced; (b) there is *no* requirement that the three 12-16 sec IRTs immediately follow one another, other IRTs usually intervened; (c) the delay is between assignment and reinforcement, not between response and food pellet, the food pellet is always delivered immediately after a bar response.) A similar independent arrangement controlled the 8-12 sec IRTs, and another independent one was used for 4-8 sec IRTs except that the reinforcement went to the fourth 4-8 sec IRT after an assignment by the tape for 4-8 sec IRTs. A similar independent arrangement was used for 0-4 sec IRTs except that the reinforcement went to the seventh 0-4 sec IRT after an assignment by the tape for 0-4 sec IRTs, and that one seventh of the assignments from the 0-4 sec tape resulted in the reinforcement of two successive 0-4 sec IRTs (other IRTs could intervene or could be absent). This last mentioned feature was arranged because past experience has shown that if a schedule does not provide two reinforcements separated by a very short time interval then pauses develop in the responding following reinforcements. The delay of reinforcement after assignment and lack of storage made the above arrangement necessary in order to provide enough successive reinforcements. (For this reason the reinfs/hr received by the 0-4 sec IRTs equalled 1 $\frac{1}{7}$ times the reinfs/hr assigned by the 0-4 sec schedules specified later.)

Only one assignment from a timer tape could be stored at a time. Hence if an assigned reinforcement was not delivered to *S* before another was assigned, the second was lost. Reinforcements were rarely lost due to the rate of responding encountered in this experiment and the timer schedules used.

Subjects

The *S*s were 3 experimentally naive male albino rats of the Wistar strain which will be

identified by the letters Z, Ab, Ac. Each *S* came from a different litter and was 151 ±17 days old when conditioned.

Procedure

The 3 *S*s were treated essentially the same throughout the experiment, and received all the treatments described below. Up to the time of conditioning, the treatment of *S*s was very similar to that in Part I. They were fed about 8 grams of Purina Chow daily for 14 days to lower their weight about 28% below their initial ad lib weight. They were then moved into the experimental cages and treated as in Part I (22 hr of each day in the same cage, 2 hr in a different type of cage during loading of magazines, etc.)

Feeding and habituation. As in Part I except for the following slight differences. The *S*s spent only 21 days in the experimental cages before conditioning. As before, the daily feeding was adjusted during this time, but with these *S*s a weight of 28% below the initial ad lib weight was sought (instead of 25% as in Part I). As in Part I the total amount of food fed each *S* daily was not changed after conditioning (amount ranged from 10.2 to 11.3 grams for these *S*s). All 3 *S*s displayed stable body weights throughout the bar pressing; the range of each was less than 18 grams or 6% of their initial weight. Most of this range resulted from a very slow drift over many days. With all *S*s the mean weight fell between 27 and 30% below the initial ad lib weight.

Magazine training (6 days). This training was the same as the magazine training in Part I except that 120 pellets were given, 20 on each of the 6 days preceding conditioning.

Treatment 1. Conditioning of bar pressing and 4-min VI (36-50 days). This treatment was essentially the same as Treatment I of Part I except for the use of 4-min VI instead of 5-min VI and the use of three 1-hr bar-sessions a day. The three bar-sessions were 3½ hr apart and the last ended 2 hr before the feeding period. The 2-hr period for loading the magazines, cleaning cages, etc. ended ½ hr before the start of the first daily bar-session of Z and Ab, and ended 2 hr before the first bar-session of Ac.

On the day of conditioning, after 3 magazine training pellets, the bar was introduced for the first time. The first bar depression was reinforced as were the next 15 depressions that followed pellet eating. After approximately 5 pellets on a 2-min VI schedule the 4-min VI began and continued for the remainder of Treatment 1. This conditioning took place about the time of the first of the three daily bar sessions, but this conditioning session was only 40-min long. The two other bar sessions on the day of conditioning and all later ones were 1 hr long. The 4-min VI schedule assigned reinforcements with the following spacings (in sec) and sequence: 6, 41, 123, 251, 651, 333, 181, 18, 427, 76, 533, 76, 181, 427, 18, 251, 533, 333, 6, 41, 123, 651. The duration of Treatment 1 was 36 days for Ab, 43 days for Ac, 50 days for Z (the success of Treatment 2 and the circuit used for it were explored with Ab before committing the other animals).

Treatment 2. Synthetic 4-min VI, use of IS to duplicate the reinfs/hr curve of 4-min VI. The IS already described was used with the controlling tapes adjusted to give each *S* the same average reinfs/hr that he had received during the last 10 days of Treatment 1. These reinfs/hr values (in order from 0-4 to > 28 sec IRTs) were as follows for Ab: 7.4, 1.0, 1.4, 1.8, 1.1, .6, .4, 1.2; for Ac: 4.6, 1.8, 2.0, 1.9, 1.4, .7, .4, 2.0. The 24-28 sec reinfs/hr of Z were so low (.3) that a separate 24-28 sec compartment could not easily be used, so for Z the 24-28 sec and > 28 sec compartments were combined into a > 24 sec compartment. The reinfs/hr values for Z (order as above) were: 7.5, 1.6, 1.5, 1.2, .4, .5, 2.3. Certain other details of this treatment were described in connection with the electrical circuit.

The low reinfs/hr in most compartments together with apparatus limitations led to the use of FI schedules for all compartments except the 0-4 sec IRTs which had a VI schedule. The use of different FI schedules for the IRTs receiving few reinfs/hr did not reduce the variability of the overall reinforcement separations because the tapes for the different compartments had different lengths and hence the tapes "rotated" with respect to each other (*i.e.*, if at a certain time one tape assigned a reinforcement about 1 min after another tape, then the next assignments by these tapes would have a different separation, and only after many cycles would the same separation be repeated). The resulting variability in the time separation of reinforcement was ample to prevent the rats from developing a difference

in their responding at different times after the last reinforcement. Since the 0-4 sec IRTs received a large fraction of the reinforcements (about ½ for Ab and Z), VI schedules were used for the 0-4 sec IRTs. The reinforcement spacings (in sec) and sequence of these 0-4 sec schedules were for Z: 881, 160, 571, 789, 308, 686, 445; for Ab: 280, 540, 840, 64, 650, 910, 750, 156, 410, 950; and for Ac: 1330, 270, 980, 1230, 520, 1130, 780. The duration of Treatment 2 was 10 days for Ab, 12 days for Ac, and 14 days for Z.

Treatment 3. Reduction of the reinfs/hr of 0-4 sec IRTs by 3.3 reinfs/hr, and increase of the reinfs/hr of some other 4-sec-wide compartment by 3.3 reinfs/hr (16-25 days). The only change from IS used in Treatment 2 was the change of two reinforcement tapes for each S. After the reduction of the 0-4 sec reinfs/hr by 3.3, the reinfs/hr of the 0-4 sec IRTs were 4.2 for Z, 4.1 for Ab, and 1.3 for Ac. The reinforcement spacings (in sec) and sequence of the VI schedules employed were for Z: 1740, 250, 990, 1490, 500, 1240, 750; for Ab: 1820, 100, 1050, 1660, 1400, 350, 700; for Ac: 1630, 3260, 4890. With Ab and Ac the 3.3 reinfs/hr were added to the reinfs/hr of the 12-16 sec IRTs, which resulted in 5.1 reinfs/hr for Ab, and 5.2 for Ac. The VI schedules used for these IRTs were for Ab: 910, 285, 770, 890, 500, 820, 700, 860, 610; and for Ac: 895, 280, 755, 875, 475, 805, 685, 845, 595. With Z the 3.3 reinfs/hr were added to the reinfs/hr of the 8-12 sec IRTs during the first 13 days of Treatment 3. This raised the reinfs/hr to 4.8. The VI schedule employed was: 910, 280, 860, 900, 530, 880, 800, 890, 700. During the last 8 days of Treatment 3 of Z, the reinfs/hr of the 8-12 sec IRTs were returned to their value during Treatment 2 (1.5 reinfs/hr), and the 3.3 reinfs/hr were added to those of the 16-20 sec IRTs bringing their reinfs/hr up to 3.7. The VI schedule employed was: 1270, 300, 1090, 1240, 600, 1150, 990, 1200, 830. These first and last portions of the Treatment 3 given Z will be designated Treatment 3A and 3B respectively. Throughout both Treatment 3A and 3B, the reinfs/hr of all other IRTs were not changed and the total reinfs/hr of all *S*s were the same (15) throughout Treatment 3 as well as during all other treatments. The duration of Treatment 3 was 16 days for Ab, 21 days for Z, and 25 days for Ac.

Treatment 4. Return to the same conditions as Treatment 2 (12-16 days). The duration was 12 days for Z, 14 days for Ac, 16 days for Ab.

Results and Discussion

Treatment 1. Conditioning and 4-min VI. Figure 8 shows the results from this experiment. In this figure, as in Fig. 2 and 3, the IRTs/op values of different compartments are represented by different lines which show the changes in the IRTs/op for the compartments on successive days beginning with conditioning.

During the first bar session, and with some *S*s for several days thereafter, the IRTs/op values in different compartments were nearly the same, but further exposure to the 4-min VI caused the 0-4 sec IRTs/op to rise far above the other compartments. Thus the results from these 3 *S*s confirm the results of Part I where it was also found that VI produced a peak in the IRTs/op curve at short IRTs. This shape of curve was interpreted as indicating control by the reinfs/hr curve since the reinfs/hr curve has a peak at short IRTs.

In Part I it was predicted that raising the total reinfs/hr (such as was done by use of 4-min VI in Part II compared with 5-min VI in Part I) would make the peak of the IRTs/op curve develop faster, be more pronounced, and be located at shorter IRTs. The 3 *S*s of Part II on the average developed a peak faster, in all cases showed a far more pronounced peak, and showed it consistently at 0-4 sec IRTs (one *S* in Part I showed a peak at 4-12 sec IRTs for some time). Thus the results confirm the prediction, but the interpretation is complicated by the presence of other differences between Part II and Part I: (a) The use of three separate 1-hr bar sessions a day in Part II as compared with the one 2-hr bar session a day used in Part I. This probably speeded up somewhat the development of the peak. (b) The use of slightly higher deprivation, 28% below initial body weight instead of the 25% used in Part I. It was predicted in Part I that such an increase in deprivation would cause the IRTs/op peak to develop faster, be more pronounced and be located at shorter IRTs. Probably the three 1-hr sessions each day mainly affected the speed of peak development. The deprivation increase probably had less effect than the increased rein-

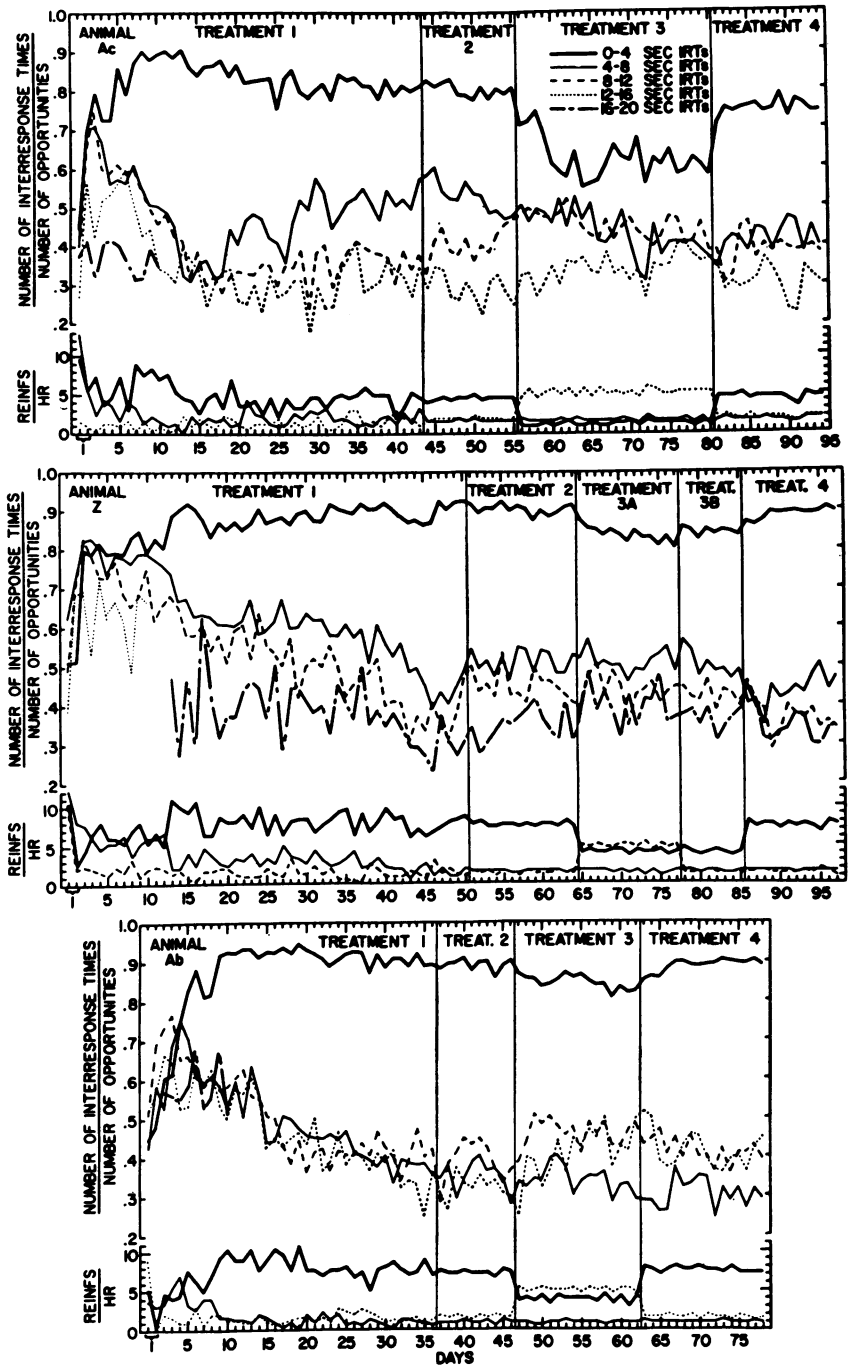

Fig. 8. The upper portion of each rectangle shows the change in the IRTs/op in different compartments on successive days after conditioning. For the first 10 days each of the first 5 compartments are shown by a different line, except that the lines are only drawn when they connect IRTs/op values based on at least 20 opportunities. After day 10 the 16-20 sec line was dropped for clarity, except for animal Z where the 12-16 sec line was dropped instead since with Z the 16-20 sec IRTs received special treatment. For each *S* the data for the first bar session on the day of conditioning was plotted separately from the other sessions of that day. The lower portion of each rectangle shows the reinfs/hr for three compartments.

forcement, but even if the deprivation change was important, its effect derives from the same argument as the effect of the total reinfs/hr, namely the view that the reinfs/hr curve controls the IRTs/op curve. Hence no matter whether the difference was due to the deprivation change or the total reinfs/hr change, the results lend some support to the hypothesis that the reinfs/hr curve controls the IRTs/op curve.

The reinfs/hr given certain IRTs are also shown in Fig. 8 at the bottom of each rectangle. It can be seen that the changes in these curves during Treatment 1 are roughly similar in trend and often in minor fluctuations to the changes in the IRTs/op. As explained in Part I this similarity is partly due to the dependence of the reinfs/hr curve on the IRTs/op curve (due to schedule), and partly due to the dependence of the IRTs/op curve on the reinfs/hr curve (due to *S*). It is in the changes after conditioning that the two effects can be clearly distinguished. There is some indication in Fig. 8 that reinfs/hr changes precede slightly the fluctuations in the IRTs/op (*e.g.* the 0-4 sec IRTs of Z on days 13 to 20, the 0-4 sec IRTs of Ab on days 5 to 10), but this would be difficult to demonstrate rigorously.

Drift in IRTs/op values is quite conspicuous throughout most if not all of the 36 to 50 days of Treatment 1. It was pointed out in Part 1 that this is also to be expected if the reinfs/hr curve controls the IRTs/op curve since such control results in a circular relation which would take a long time to stabilize even approximately and might always show considerable variability and drift.

Probably little weight can be placed on the incidental observations in the above three paragraphs but at least they are quite intelligible in terms of the hypothesis that the reinfs/hr curve plays a major role in determining the IRTs/op curve.

Treatment 2. Synthetic 4-min VI, use of IS to duplicate the reinfs/hr curve of 4-min VI. The purpose of this treatment was to determine whether IS with the same reinfs/hr curve as 4-min VI is enough similar to 4-min VI that results obtained with IS can be generalized to VI. Figure 8 shows that although the 0-4 sec IRTs were quite stable by the end of Treatment 1, the IRTs/op in other compartments frequently were still changing at the end of 36 to 50 days. Chance fluctuation in reinfs/hr values may prolong this drifting indefinitely (note the decrease and then increase of the 4-8 sec IRTs of Ac). Consequently IS had to be initiated before the IRTs/op were as stable as desired, and some changes could be expected to continue into Treatment 2.

The first 10 days of Treatment 2 (IS) were compared with the last 10 days of Treatment 1 (4-min VI). The 0-4 sec IRTs/op were the most stable during Treatment 1 and are especially important in the later results of this experiment. These 0-4 sec IRTs show no sign of influence by the change to Treatment 2. For all *S*s the difference in mean IRTs/op before and after the change was less than the standard deviation both before and after the change (largest difference between means was .012). With the other compartments, Fig. 8 indicates that when the drift and variation of Treatment 1 is taken into account, there is no evidence of any effect from switching to Treatment 2. The *S* that was most stable before the change, Ab, shows the least sign of any effect. The Wilcoxon T test (1949) was used to test the differences between the last 10 days of Treatment 1 and the first 10 days of Treatment 2. All compartments with at least 20 opportunities each day were tested (except 0-4 sec IRTs already considered). With Ac none of the 7 comparisons were significant at the .05 level. With Ab 1 of 6 comparisons was significant at the .05 level (not significant at the .02 level). This was the 12-16 sec compartment, and, as Fig. 8 shows, this difference seems due to the drift before the change to Treatment 2. That this is the case is shown by the comparison of the last 5 days of Treatment 1 with the first 10 days of Treatment 2 which is far from significant at the .05 level (Mann and Whitney, 1947). With animal Z the variability before the change to Treatment 2 was greater, and significant differences were found for 5 of the 7 compartments (2 at the .05 level, 3 at the .01 level). Figure 8 indicates that these differences resulted from a temporary drop in several comparments just before the change to Treatment 2. This interpretation is supported by the fact that none of the differences are significant at the .05 level when we compare the last 20 days of Treatment 1 (which provides a more representative sample of the variability of Treatment 1) with the first 10 days of Treatment 2 (Mann and Whitney, 1947). Thus where the IRTs/op were stable

before the change, no significant differences were found; where significant differences were found they were closely related to variation during the last days of Treatment 1. Apparently IS duplicates fairly well the effects of VI on rats, though this question probably deserves further study. Certainly there is no evidence for any major changes in the responding, so if it can be shown that the reinfs/hr curve controls responding during VI, this finding should apply to VI also.

It may be useful to list the major differences that do exist between IS and VI and that could possibly affect *S*. (a) IS duplicates the reinfs/hr curve of VI but only within the limits set by the size of compartments employed, here 4-sec wide. Subdivision of the compartments would show that the different subdivided compartments do not have quite the same reinfs/hr curve as VI. This difference theoretically could be reduced to any desired degree by reduction in the size of compartments. (b) With IS the reinfs/hr curve is constant and is not affected by the responding of *S* so long as enough responses are made to obtain the assigned reinforcements. Consequently there is reduction in the drift and variability of the reinfs/hr in individual compartments (see reinfs/hr plots in Fig. 8). On the other hand in IS the reinfs/response curve is not constant as it is in VI. This difference is the reason for employing IS. (c) The overall temporal spacing of reinforcements (irrespective of the IRT they reinforce) is different. The 4-min VI schedule employed here repeated after 22 reinforcements; the cycle for IS was far more complex. This difference could be reduced by using a more complex VI schedule, or scheduling VI reinforcements from several tapes with different lengths. (d) The reinforcement of sequences of responses is not quite the same, but this difference can apparently be reduced to a quite small degree by the techniques described earlier.

Treatment 3. Reduction of the reinfs/hr of 0-4 sec IRTs by 3.3 reinfs/hr, and increase of the reinfs/hr of some other 4-sec-wide compartment by 3.3 reinfs/hr. During 4-min VI, the major feature of the IRTs/op curve of each *S* was the high and stable peak at 0-4 sec IRTs. The IRTs/op in other compartments differed little among themselves. Since the purpose of this experiment is to determine whether this IRTs/op curve produced by VI is affected by the reinfs/hr curve, the major question is whether this 0-4 sec IRTs/op peak resulted from and depends on the high reinfs/hr given 0-4 sec IRTs. Consequently it was the 0-4 sec compartment whose reinfs/hr were changed. The reinfs/hr were reduced because the 0-4 sec IRTs/op of Ab and Z could increase but little.

Figure 8 shows the effect of the shift of reinforcements. With all *S*s there was a significant decrease in the 0-4 sec IRTs/op (.01 level[2]). (These, and other comparisons below, employed the last 10 days of Treatment 2 and the last 10 days of Treatment 3; with animal Z the last 10 days of Treatment 2 were significantly greater than both the last 10 days of 3A and last 8 days of 3B—8 days because there were only 8 days of 3B.) (.01 level) The 3.3 reinfs/hr were added to the 12-16 sec IRTs with animals Ac and Ab. With Ab there was a clear and significant increase in the 12-16 sec IRTs/op (.01 level). With Ac there was also a significant increase but it developed more slowly and when the earlier drifting of the 12-16 sec IRTs/op is considered, it does not seem safe to conclude that the increase was due to the reinfs/hr change. With animal Z the reinforcements were not added to the 12-16 sec IRTs because that compartment was less stable than the other compartments (12-16 sec IRTs/op were not plotted on Fig. 8 because they would have obscured the more important 8-12 and 16-20 sec IRTs/op). For this reason the 3.3 reinfs/hr were added to the 8-12 sec compartment. With this *S* there was no sign of any effect on the 8-12 sec IRTs/op during the 13 days of Treatment 3A (the means differed by only .017, with the mean of 3A the lower). To check that this result was not due to the particular compartment chosen, the 3.3 reinfs/hr were moved to the 16-20 sec compartment for 8 days, but there was no effect there either (decrease of .0013 in 8-day means).

Thus the major feature of the IRTs/op curve of each *S* was influenced by change in the reinfs/hr. Only 3.3 reinfs/hr out of 15 total reinfs/hr were shifted, and the responding never differed very much from 4-min VI responding. Consequently this demonstration of the effect of the reinfs/hr curve on the

[2]This and later tests of significance all employed the Wilcoxon T test (1949).

IRTs/op curve would seem to apply to VI responding also. Apparently the above average reinfs/hr given to 0-4 sec IRTs at least contributes to the 0-4 sec peak in the IRTs/op curve.

The failure of Z and perhaps Ac to show a clear rise in the IRTs that were given additional reinforcement indicates that there are limitations to the adjustment of the IRTs/op curve to the reinfs/hr curve. Many factors may contribute to this lack of adjustment: (a) The long duration of reinforcement before Treatment 3 (Z had 192 hr). Earlier initiation of Treatment 3 probably would have greatly increased its effectiveness. (b) The brevity of Treatment 3. Longer exposure might have produced a peak since the 12-16 sec IRTs/op of Ab, and perhaps Ac, were still increasing at the end of Treatment 3. Treatment 3 was kept short to maximize the likelihood that return to the conditions of Treatment 2 would restore the 0-4 IRTs/op to nearly their original value. (c) The IRTs involved. Compared with the 0-4 sec IRTs, longer IRTs may need a higher or broader reinfs/hr peak before an IRTs/op peak develops. During Treatment 3 of Part I, more reinfs/hr were given to 40-48 sec IRTs than were given to 0-4 sec IRTs during Treatment 1, but the IRTs/op peak of each *S* was significantly lower for the 40-48 sec IRTs (Fig. 6[3]). (.01 level) The difference ranged between .24 and .34.

The VI reinfs/hr curve seems to begin its action very soon after the start of bar pressing, and produces a peak at 0-4 sec IRTs (or perhaps a broad peak at longer IRTs). Consequently the lack of reaction of Z and perhaps Ac to a narrow reinfs/hr peak near 12-16 sec is not inconsistent with the view that the VI reinfs/hr curve produces the 0-4 sec peak in the IRTs/op curve of VI.

The fact that the change in the 0-4 sec IRTs/op of Z and Ab was not larger than it was may also be due to the long preceding reinforcement of 0-4 sec IRTs and the brief duration of Treatment 3. The 0-4 sec IRTs/op of both Z and Ac seemed to be still decreasing at the end of Treatment 2, and Part I showed that an especially long time is needed for extinction of 0-4 sec IRTs. The much bigger effect on Ac from the same reinfs/hr change was probably due to the lower initial value of the 0-4 sec reinfs/hr of Z. This implies a nonlinear relation between reinfs/hr and IRTs/op which is obviously necessary at some point since the IRTs/op cannot exceed 1.0.

There were some signs of generalization to neighboring compartments. The 8-12 sec IRTs/op of Ab, just short of the 12-16 sec IRTs that received the reinfs/hr increment, increased significantly (.01 level). With Ac the 16-20 sec IRTs/op, just beyond the compartment with the increment, showed an increase significant at the .02 level by the Wilcoxon *T* test. No other compartments showed an increase significant at the .05 level by that test.

Treatment 4. Return to the same conditioning as Treatment 2. The 0-4 sec IRTs/op of Z and Ab returned to the values they had during Treatment 2, and Ac returned 75% of the way back to Treatment 2 value. These results confirm that the decrease of the 0-4 sec IRTs/op observed during Treatment 3 was due to the reinfs/hr change. The difference between the means of Treatments 2 and 4 were tested by the Wilcoxon *T* test (1949). The last 10 days of Ab and Ac were compared, but only the last 9 days of Z, because Z was only observed for 9 days of Treatment 4 after stabilization. The differences for Ab and Z were not significant at the .05 level; the difference was only .008 for Z and .004 for Ab. This contrasts with Treatment 4 of Part I where the 0-4 sec IRTs/op of no *S* returned to the former value (Fig. 6), but there the reinfs/hr of 0-4 sec IRTs were not restored to their former value. The 0-4 sec IRTs/op of Ac during the last 10 days of Treatment 4 were significantly less than during the last 10 days of Treatment 2, and seemed to be stable (.01 level). This irreversibility of Ac may be related to the fact that the decrease in the 0-4 sec IRTs/op of Ac during Treatment 3 was much greater than the decrease of the other two *S*s. The 0-4 sec IRTs/op of all four *S*s were significantly greater than during Treatment 3 (.01 level, comparison of last 10-day periods).

The 12-16 sec IRTs of both Ab and Ac decreased significantly when their reinfs/hr were reduced in Treatment 4. The decrease of Ac was significant at the .01 level, that of Ab only between the .02 and .05 level. The decrease of Z was not significant at the .05 level. (Wilcoxon *T* test, comparison of last-8-day periods).

[3]Figure 5 in the published version.

The basic problem dealt with in this experiment and the approach used to solve it are probably of wide application in experimental psychology. Many problems involve the determination of whether a certain variable, say a stimulus, is controlling behavior in a certain situation. A major change in this stimulus or the elimination of other controlling stimuli may show the dependence of behavior on this stimulus without showing that the stimulus was controlling the behavior before experimental modification, because the modification so grossly changes the situation from the natural one. What is needed is the demonstration of the influence on the behavior of small changes in the stimulus that do not appreciably modify the situation from the natural one. However this method seems to require a high precision of measurement in order to detect the small changes in behavior.

This experiment shows the practicability of the IS technique. IS eliminates the circular relation that seems to be present in most schedules and both stabilizes and brings under experimental control what appears to be the important variable for the rat, the reinfs/hr curve. Consequently IS should be quite useful in further experimentation aimed at revealing more clearly the relation between the reinfs/hr curve and the IRTs/op curve. IS makes possible the maintenance of a reinfs/hr curve with equal values at different IRTs out to IRTs which are moderately frequent. Will this eliminate or greatly reduce the development of a IRTs/op peak? IS should be useful for studying other questions raised by this study: the question of the relative importance of the reinfs/hr and reinfs/IRT curve, the question of irreversibility, *etc.* The evidence suggests that IS should stabilize faster, and be more stable than other schedules. The interpretation of changes in response rate may be simpler too. Hence IS may provide a highly useful baseline in the investigation of the effect of various other variables on responding.

SUMMARY OF PART II

Evidence was presented in Part I which indicated that during a VI schedule rats adjust their production of IRTs according to the relative reinforcements/hr of different IRTs. However in the portion of Part I concerned with the experimental manipulation of reinforcements/hr, a large change in responding was produced. This result showed the ability of rats to adjust their IRT production according to the relative reinforcement of different IRTs, but left doubt about whether this adjustment was taking place during VI. The objective of Part II was to determine whether small experimental modifications in the features of the IRTs/op curve that appear due to VI change the IRTs/op curve appropriately. If such sensitivity of the IRTs/op curve to the relative reinforcements/hr is found while keeping the reinforcement and IRT distribution quite similar to VI, it should strengthen the conclusion that the relative reinforcement of different IRTs arranged by VI is at least partly responsible for the IRTs/op curve of VI.

Evidence presented in Part I indicated that the responding depends upon the relative reinforcements/hr of different IRTs. But during VI the relative reinforcements/hr depend to a great extent upon the responding of the animal. Hence in order to perform the just described experiment it was necessary to bring the reinforcements/hr of different IRTs under experimental control. This was accomplished by using separate schedules to control the reinforcements/hr of the 0-4 sec IRTs, the 4-8 sec IRTs, and so on out to 28 sec. This reinforcement control will be called *IRTs-separate reinforcement* or *IS*.

Three *S*s were given 4-min VI for over 35 days to allow the responding to stabilize, and then were changed to IS with the reinforcements/hr for each group of IRTs adjusted to the values that had just prevailed with VI. There resulted little or no change in the IRTs that had stabilized during VI. This result indicated that IS was substantially equivalent to a VI schedule. The major feature of the IRTs/op curve produced by the 4-min VI was a high and stable peak at 0-4 sec IRTs, which was associated with a 0-4 sec peak in the reinforcements/hr curve. These reinforcements/hr of 0-4 sec IRTs were reduced by 3.3 reinforcements/hr, and another group of IRTs was increased by 3.3 reinforcements/hr. This shift produced a significant reduction in the IRTs/op peak at 0-4 sec. Restoration of the 3.3 reinforcements/hr to the 0-4 sec IRTs produced a significant elevation in the IRTs/op of the 0-4 sec IRTs. Hence it is concluded that

during some VI schedules the distribution of IRTs produced by *S* is determined partly by the relative reinforcement of different IRTs that the VI schedule provides.

REFERENCES FOR PART II

(Omits references only in Part I)

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