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POSITIVE AND NEGATIVE BEHAVIORAL CONTRAST IN THE RAT[1]

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Three groups of rats received either 8, 23, or 53 sessions of multiple variable-interval variable-interval baseline training before being shifted to a multiple extinction variable-interval schedule. The rate of responding during the unaltered component was higher for the groups shifted to multiple extinction variable-interval than for control groups remaining on multiple variable-interval variable-interval (positive contrast). Furthermore, when the multiple variable-interval variable-interval schedule was re-instated, stable negative contrast was found in the groups that had received 23 or 53 baseline sessions, but not for the group that had received only eight sessions. Positive and negative contrast were also demonstrated in the eight and 23-session groups when the multiple extinction variable-interval and multiple variable-interval variable-interval schedules were re-administered in further phases of the experiment. These results suggest that both positive and negative behavioral contrast can be obtained reliably in a species other than the pigeon.

Positive behavioral contrast occurs when the alteration in one component of a multiple schedule reduces the response rate in that component, and concurrently increases the response rate in the unaltered component. Reynolds (1961) demonstrated this effect using a three-phase procedure. Phase 1 consisted of baseline training on identical variable-interval (VI) schedules in a two-component multiple schedule (*mult* VI VI). Subsequently, one component was shifted to extinction (*mult* VI EXT) in Phase 2, and back to VI (*mult* VI VI) in Phase 3. The response rate in the unaltered component increased during Phase 2 (positive contrast), and returned toward baseline during Phase 3 (negative contrast). The re-instatement of the VI VI schedule in Phase 3 served as a control to ensure that the increased VI rate in Phase 2 was a function of the extinction treatment in the other component, and not of a tendency for VI rate to increase over time.

The VI VI-VI EXT manipulation of Phases 2 and 3 can be repeated in Phases 4 and 5 (Arnett, 1973; Bloomfield, 1967; Terrace, 1966) to ensure further that the increase in VI rate during VI EXT is reproducible. This was especially important in Bloomfield's (1967) study because he failed to obtain negative contrast in Phase 3. As a result, positive contrast occurred in Phase 4 relative to the VI VI rate in Phase 3, and negative contrast occurred in Phase 5 relative to the VI rate in Phase 4.

The experiments cited above were conducted with the pigeon. Experiments designed to generalize these results to the rat have been only partially successful. For example, Freeman (1971), using rats, reported a reduced response rate for both the extinction and VI components in Phase 2; *i.e.*, negative induction rather than positive contrast was found. Pear and Wilkie (1971) did obtain positive contrast during the *mult* VI EXT of Phase 2, but the VI VI rate in Phase 3 was higher than the VI rate in Phase 2; *i.e.*, positive induction was found in Phase 3. Furthermore, positive contrast was not convincingly re-established when VI EXT was re-administered in Phase 4. Thus, behavioral contrast in the rat may appear to be markedly different from that in the pigeon. However, a number of procedural differences could account for this apparent discrepancy.

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For example, in the pigeon experiments, two different visual cues were associated with the two components of the multiple schedules. In the rat experiments, however, the components were signalled by the presence or absence of either a visual or auditory stimulus. In the present experiment, a light (EXT) noise (VI) discrimination was used to determine whether positive and negative contrast could be obtained in the rat. In addition to the two control procedures described above, a third was used in which performance of subjects in Phases 2 and 3 was compared to the performance of subjects maintained on Phase 1 VI VI training. Furthermore, duration of baseline training was manipulated in this experiment because pilot data had suggested that this variable may be an important determinant of both positive and negative contrast.

METHOD

Subjects

Twenty-eight experimentally naive female hooded rats (90 to 100 days of age) were selected from the colony at the Experimental Psychology Laboratory, Syracuse University. Subjects were reduced to and maintained at approximately 80 to 85% of their free-feeding weights.

Apparatus

Four Grason-Stadler operant chambers (29.5 by 24.0 by 19.5 cm) were mounted in sound-attenuating enclosures containing exhaust fans that ventilated the chambers and masked extraneous sounds. The chambers contained a single response lever located in the middle of the panel, 9.5 cm above the grid floor, protruding 1.5 cm into the chamber. Operation of the lever required approximately 16 to 18 g of force (0.16 to 0.18 N). Standard 45-mg Noyes pellets were delivered into magazines located 3.0 cm directly below the lever.

A two-component multiple schedule (*mult* S1 S2) was arranged by electromechanical relay equipment. The schedule consisted of 10 presentations of S1 and 10 presentations of S2 occurring in strict alternation, beginning with S1. The duration of each component was 3 min, and consecutive components were separated by brief (4 sec) blackout intervals. A houselight was always on in both components. Both the S1 cue and the houselight were 10-W bayonet lamps (G.E. #10C7/DC). The S1 lamp was attached to the outside of the Plexiglas chamber door, 8.0 cm above and 10.0 cm to the right of the lever. The houselight was encased in a red plastic dome 1.0 cm above and 7.0 cm to the left of the lever. The S2 cue was a 75-dB white noise (SPL) delivered through a speaker located on the left side of the front panel below the level of the grid floor. The S1 and S2 cues were present during each phase of training. During EXT VI phases, light (S1) was associated with EXT and noise (S2) with VI.

Procedure

All subjects received three sessions of lever-press training on a continuous reinforcement schedule, during which they received 100, 60, and 60 reinforcements, respectively. They were then placed directly on a *mult* VI 30-sec VI 30-sec schedule. After the eighth session of *mult* VI VI training, subjects were matched according to their baseline rates. These response rates were divided into class intervals of five responses per minute, ranging from 16 to 20 to 31 to 35 responses per minute. Two groups of 10 and 18 subjects were matched for mean and range of response rates. The group of 10 subjects was shifted to Phase 2 (*mult* EXT VI) conditions and constituted the short-baseline (SBL) group. The group of 18 subjects remained on Phase 1 (*mult* VI VI) conditions. The SBL group received 15 sessions in each subsequent phase: Phase 2 (EXT VI), Phase 3 (VI VI), Phase 4 (EXT VI), and Phase 5 (VI VI). The group of 18 subjects remained on *mult* VI VI for a total of 23 sessions, at which point they were divided into two groups of nine and seven subjects, respectively, which were matched for mean and range of response rates at Session 23. Two subjects, which were sole occupants of extremely high (56 to 60 responses per minute) and low (16 to 20 responses per minute) class intervals, were discarded. The group of nine subjects, constituting the intermediate-baseline (IBL) group, was then shifted to Phase 2 (*mult* EXT VI) conditions, and the remaining group of seven subjects, constituting the long-baseline (LBL) group, continued on Phase 1 (*mult* VI VI) conditions for 53 sessions. The IBL group received 15 sessions of Phase 2 (EXT VI), Phase 3 (VI VI), Phase 4 (EXT VI), and five sessions of Phase 5 (VI VI) conditions. Following their 53

Table 1

Summary of the Schedule Conditions Received by the SBL, IBL, and LBL Groups

	Consecutive Sessions					
Group	*1-8*	*9-23*	*24-38*	*39-53*	*54-68*	*69-73*
SBL	VI VI	EXT VI	VI VI	EXT VI	VI VI	–
IBL	VI VI	VI VI	EXT VI	VI VI	EXT VI	VI VI
LBL	VI VI	VI VI	VI VI	VI VI	EXT VI	VI VI

baseline sessions, the LBL group received 15 sessions of Phase 2 (EXT VI) and five sessions of Phase 3 (VI VI) conditions. These conditions are summarized in Table 1, which indicates that Phase 1 of IBL overlapped Phases 1 and 2 of SBL, and that Phase I of LBL overlapped Phases 1, 2, 3, and 4 of SBL, and Phases 1, 2, and 3 of IBL. A similar design was previously used by Mackintosh, Little, and Lord (1972) to compare the VI rates of rats receiving a treatment to those remaining on baseline. The present design also permitted between-group analyses of negative contrast effects.

RESULTS

Response rate (responses per minute) for each session was calculated for each animal, and the group means of this measure are plotted in Figure 1 for the S2 (unaltered) component in all phases of the experiment (dotted functions), and for the S1 (altered) component for the EXT VI phases (dashed functions). Two subjects (one each from the SBL and IBL groups) developed middle-ear infections during training and were discarded. Thus, these data represent nine, eight, and seven subjects in the SBL, IBL, and LBL groups, respectively. The solid functions represent the control (VI VI) baseline rates of the S2 (unaltered) component from all available subjects. Because the duration of baseline training was manipulated, the number of subjects from which control data could be obtained decreased throughout the experiment. Thus, the control data for the SBL group (top panel) in Phases 1 and 2 were obtained from the Phase 1 VI VI training of the IBL and LBL groups ($N = 15$). The control data for Phases 3 and 4 of the SBL group (top panel) and for Phases 1, 2, and 3 of the IBL group (middle panel) were obtained from the Phase 1 VI VI training of the LBL group ($N = 7$). The bottom panel depicts the LBL group alone during Phases 1, 2, and 3.

Inspection of these data indicates no differences in S2 rates between experimental and control groups during Phase 1, and shows that positive and negative contrast occurred in each group during each applicable phase. However, the magnitude and stability of these effects differed among the three groups. For statistical analysis, the data from appropriate phases were divided into three blocks of five sessions each, and a 2 × 3 groups × blocks ANOVA was calculated. The S2 rates of the SBL group were compared to the combined control rates of the IBL and LBL groups in Phase 2, and to the control rates of the LBL group in Phases 3 and 4. The S2 rates of the IBL group were also compared to the control rates of the LBL group in Phases 2 and 3.

SBL group. The S2 rates of the SBL group were significantly higher than control rates during Phases 2 ($F\ 1,359 = 124$, $p < 0.01$) and 4 ($F\ 1,239 = 136$, $p < 0.01$), both of which were EXT VI phases. When the VI VI conditions were re-instated during Phases 3 and 5, the VI rate declined (negative contrast). In Phase 3, the response rate remained significantly higher than the control rate ($F\ 1,239 = 19.3$, $p < 0.01$). However, the Phase 3 level was recovered in Phase 5.

Six of the nine subjects in the SBL group shared response-rate patterns characteristic of the group curve, and one of these (Subject 12) is illustrated in Figure 2. The typical pattern was as follows: VI rate increased throughout Phase 2, but was relatively stable in Phase 4. Although the baseline rate decreased in Phase 3, the S2 rate of Phase 1 was not recovered, and the negative contrast effect was often transient. That is, in Phase 3, response rates in S2 declined during the initial sessions, but returned to or exceeded the Phase 2 VI rate in subsequent sessions. Similar effects occurred in Phase 5 for several individual subjects.

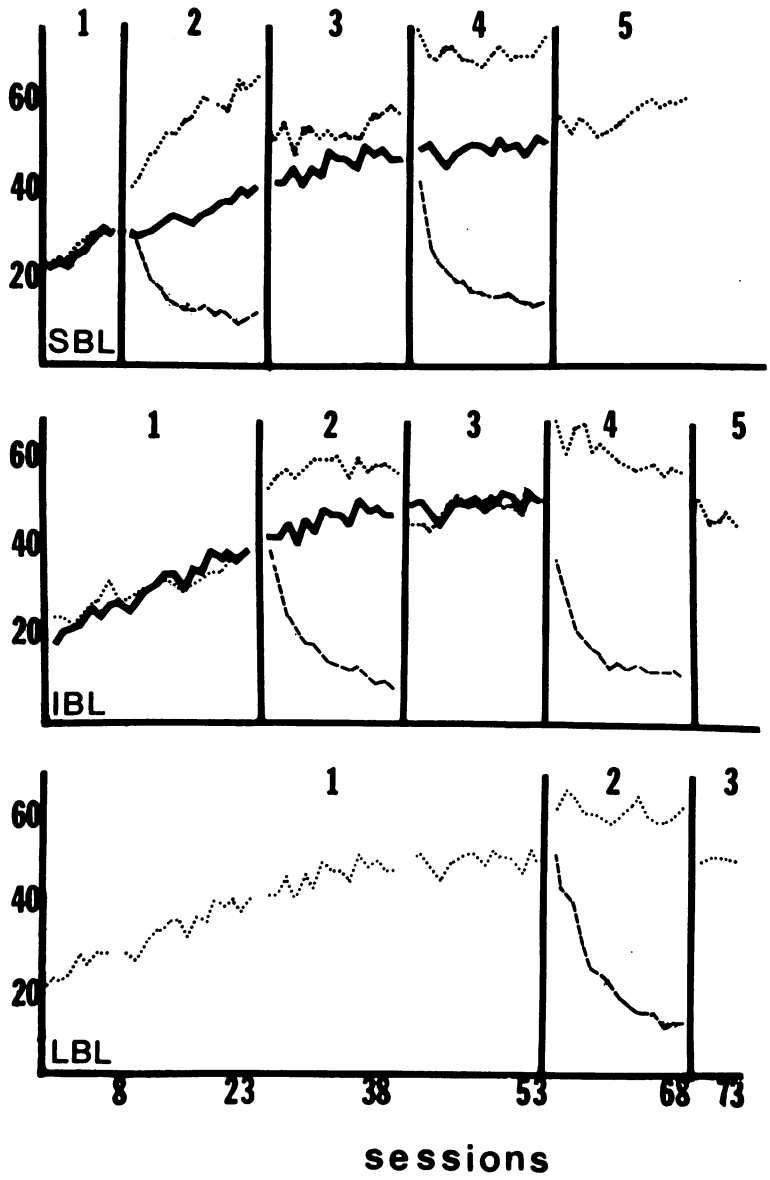

Fig. 1. Group mean data for the SBL (top panel), IBL (middle panel), and LBL (bottom panel) groups. Data for the S2 (unaltered) components are plotted in each phase of the experiment (dotted functions), whereas data for the S1 (altered) components are plotted only for the EXT VI phases (dashed functions). Control baseline data are superimposed on the SBL and IBL function (solid lines) in the top and middle panels.

Three subjects deviated from this pattern, and each deviated in a unique way. One such subject (51) is also illustrated in Figure 2, and showed stable positive and negative contrast effects, and failed to show the typical increase in overall VI rate.

IBL group. The IBL and control groups did not differ during Phase 1, but the S2 rate of the IBL group was higher than control during Phase 2 ($F\ 1,234 = 12.3$, $p < 0.01$). In subsequent phases, baseline was recovered during Phases 3 and 5 when the VI VI schedule was re-instated (negative contrast). In Phase 4, the VI rate tended to exceed that of Phase 2, and to decline to the Phase 2 rate during Phase 4.

Five of eight subjects in the IBL group shared response-rate patterns characteristic of the group curve, and one of these (Subject 121)

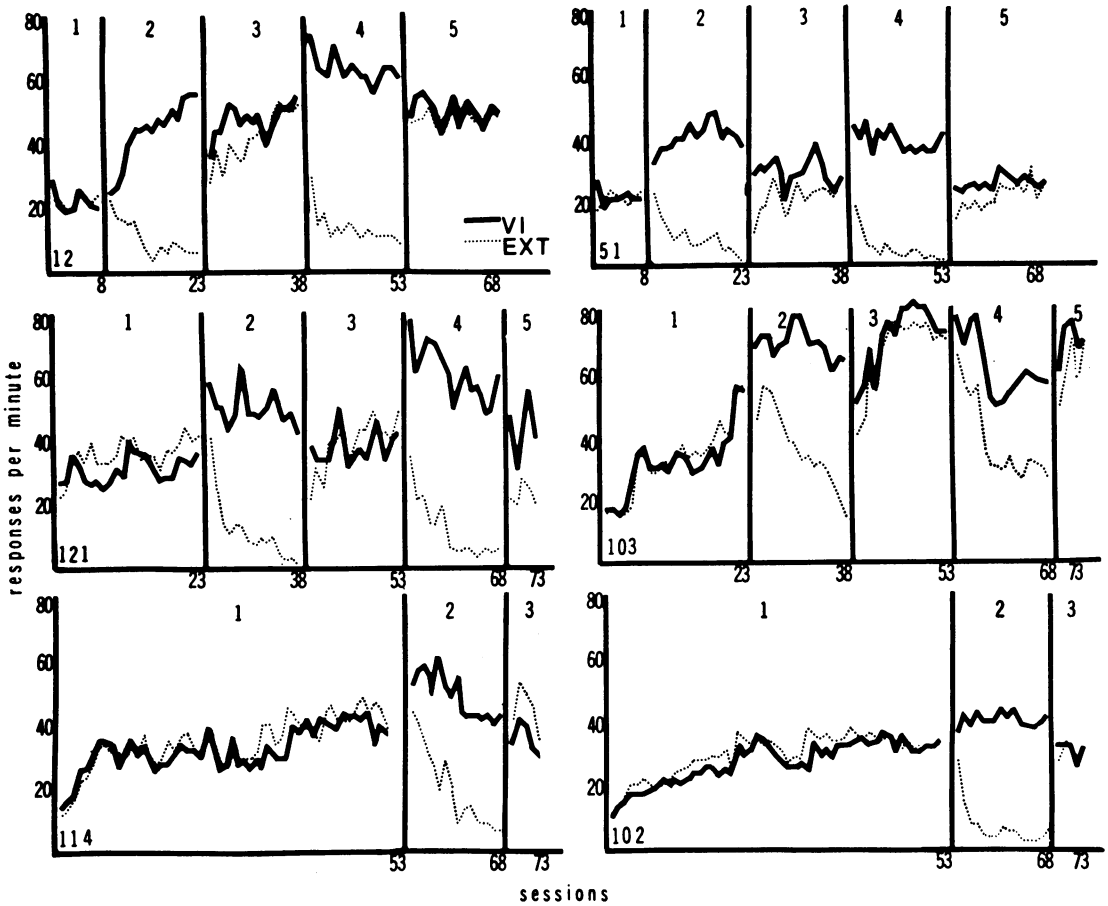

Fig. 2. Individual subject data representing the SBL (top panels), IBL (middle panels), and LBL (bottom panels) groups. The data for Subjects 12 and 121 characterize the Figure 1 curves of the SBL and IBL groups, respectively, and are representative of most subjects in these two groups. Subjects 51 and 103 represent sample deviations from these patterns. A single pattern characterizing the majority of subjects was not found with the LBL treatment. Rat 102 illustrates the LBL group curve of Figure 1, and Rat 114 shows an unstable positive contrast effect in Phase 2.

is illustrated in Figure 2. The most deviant subject in the IBL group (Subject 103) is also depicted in Figure 2, showing transient negative contrast in Phase 3 followed by negative induction in Phase 4.

LBL group. The S2 response rate in Phase 2 shows a small but stable positive contrast effect, and recovery of baseline in Phase 3. However, a single pattern did not emerge for the majority of subjects. Two subjects are illustrated in Figure 2. Subject 102 typified the group data, and Subject 114 illustrates an instance of unstable positive contrast in Phase 2.

DISCUSSION

On the basis of the control conditions used in this experiment, the positive contrast reported cannot be attributed to factors other than the extinction treatment. Not only was negative contrast found in Phase 3, but positive and negative contrast were replicated in Phases 4 and 5. Bloomfield (1967) noted that omission of the Phase 3 control condition in contrast experiments makes the Phase 2 effect difficult to interpret. The present findings support this contention. Since baseline rate did rise for many sessions, a positive contrast effect in Phase 2 may merely represent a continued increase of this rate, instead of an effect due to the treatment.

With respect to the manipulation of duration of baseline training, 23 days of baseline proved optimal because it produced stable negative contrast after significant and stable positive contrast. The SBL treatment did not pro-

duce stable negative contrast, and the LBL treatment did not produce stable positive contrast. Judging from the baseline trends, the introduction of Phase 2 after only eight sessions occurred when the baseline rate was increasing. This correlates with the increasing VI rate found for most SBL subjects during Phase 2. On the other hand, baseline rate was typically more stable after 23 sessions, and this correlates with the relatively stable VI rates found for most IBL subjects during Phase 2. After 53 baseline sessions, response rates were variable between subjects, but were relatively stable within subjects, and this correlates with the small or transient positive contrast seen in Phase 2 for LBL subjects.

Both Bloomfield (1967) using pigeons, and Pear and Wilkie (1971) using rats, failed to obtain negative contrast in Phase 3 after positive contrast in Phase 2. However, Bloomfield established both effects in subsequent phases, whereas Pear and Wilkie did not. The present results are more characteristic of Bloomfield's than of Pear and Wilkie's.

Bloomfield attributed the failure to find negative contrast in Phase 3 to a response topography change in Phase 2. Specifically, the pigeons were observed to stand closer to the key in Phase 2 than in Phase 1. This topography did not change in subsequent phases. Since negative contrast did not occur until Phase 5, it is possible that a stable response topography is necessary before discrimination training in order for negative contrast to occur subsequently. This is consistent with the present results, since longer baseline training would predict a greater likelihood of such stabilization.

However, a stable baseline response rate (and/or response topography) does not appear to be a sufficient condition for negative contrast. Pear and Wilkie's subjects received either 14 or 25 baseline sessions, a length comparable to the IBL (23-session) treatment of the present experiment, which resulted in stable negative contrast. Pear and Wilkie's subjects showed positive induction in Phase 3, whereas even in the SBL (eight-session) group of this experiment, most of the subjects showed at least transient negative contrast.

These results may support Pear and Wilkie's contention that stimulus control of not responding in Phase 2 extinction is necessary for the occurrence of negative contrast in Phase 3. In their experiment, light signalled the VI and darkness signalled the extinction component of the *mult* VI EXT schedule of Phase 2. They hypothesized that darkness failed to acquire inhibitory stimulus control of not responding in Phase 2, and hence promoted positive induction, rather than negative contrast in Phase 3. This interpretation applied to the present finding suggests that the S1 cue (light) gained control of not responding in Phase 2 extinction, and its presence in Phase 3 facilitated the occurrence of negative contrast. This hypothesis would predict that negative contrast would not occur if the stimulus controlling not responding in Phase 2 was absent in Phase 3.

The different results obtained from the SBL and IBL groups are consistent with this interpretation. It is possible that a stimulus associated with extinction gains stronger control over not responding after longer baseline training. For example, Weisman and Ramsden (1973) shifted a *mult* VI VI baseline in Phase 1 to a *mult* VI variable time (VT) in Phase 2. Subjects received either three or 20 baseline sessions in Phase 1 before the shift. Generalization gradients taken around the stimulus associated with VT were flat for the three-session group, but were U-shaped for the 20-session group.

Thus, it is possible that the major procedural difference between Pear and Wilkie's conditions and the present ones were the stimuli associated with VI and extinction, and that the empirical differences were amplified by the effect of the baseline variable in a discrimination that established control over not responding in extinction light (EXT) noise (VI) *versus* light (VI) dark (EXT). Unfortunately, no direct measures of inhibitory control were made in either the present experiment or in Pear and Wilkie's.

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