MAINTENANCE OF SELF-ATTRIBUTED AND DRUG-ATTRIBUTED BEHAVIOR CHANGE*

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It is proposed that behavior changes which are believed to be brought about by oneself will be maintained to a greater degree than behavior changes which are believed to be due to external forces or agents. Within the framework of psychoactive drug therapy, a change in overt behavior which is attributed to the subject's own efforts should be more persistent than a change in overt behavior which is attributed to a drug. An experimental analogue of drug therapy is reported in which subjects (a) underwent a pain threshold and shock tolerance test, (b) ingested a drug (really a placebo), and (c) repeated the test with the shock intensities surreptitiously halved. All subjects thus believed that a drug had changed their threshold performance. Half of the subjects were then told that they had actually received a placebo, whereas the other half continued to believe that they had received a true drug. It was found that subjects who attributed their behavior change to themselves (i.e., who believed they had ingested a placebo) subsequently perceived the shocks as less painful and tolerated significantly more than subjects who attributed their behavior change to the drug.

This research is concerned with a person's attributions about the causes of a change in his behavior. That is, given that a person is behaving in a particular manner, will the cognitive and behavioral consequences be different if the cause of it is attributed to himself than if it is attributed to an external force or agent? It will be hypothesized that behavior change which is attributed to oneself will persist or be maintained to a greater degree than behavior change which is attributed to an external agent such as a drug.

Although this research is restricted to overt behavior, much of it was stimulated by considerations of covert visceral behavior. If we define behavior as a perceptible physiological reaction, there is good evidence that different attributions about the source of this "internal behavior" will have different effects. Schachter and his associates (Nisbett & Schachter, 1966; Schachter & Singer, 1962) have clearly shown that if this internal behavior is attributed to oneself, the consequences will be different than if the behavior is attributed to an external agent such as a drug. When it is attributed to oneself, the person has some information which can lead to inferences about himself or about stimuli in the external environment. Thus, if I have reacted in this anger-inducing situation, then I must be angry. If I have reacted in this euphoric situation, then I must be happy. If I have reacted to these electric shocks, then they must be painful. The situation is quite different, however, if the physiological reactions or internal behaviors are attributed to an external agent: The person is less able to make inferences about the external situation or about himself if his internal behavior is drug-induced. Thus, subjects do not get angry or happy (Schachter & Singer, 1962) and do not consider shocks painful (Nisbett & Schachter, 1966) if they believe that their internal behavior is drug induced. In effect, a self-attributed visceral reaction makes us stop, look, and listen, and allows us to make certain inferences about ourselves and the stimulus situation. A drug-attributed visceral reaction provides us with little or no information about ourselves or the world.

These speculations about attribution are relevant to a psychiatric problem which is particularly serious but which unfortunately is far too often ignored. That is, how can psychiatric patients be weaned off tranquilizers? The remarkable effects that the intro-
The introduction of tranquilizers has had upon the treatment of psychiatric patients well-known effects. It has been estimated, for example, that the patient population of New York State, which for some time had been increasing by 2,000 or more every year, dropped by 500 through increased discharges during the first year in which chlorpromazine and reserpine were in general use (Waite, 1964). Within the hospitals still further consequences could be observed (for example, 75% fewer patients being placed on restraint or in seclusion).

Without denying the importance of these drugs, a curious problem has arisen in that it has become increasingly evident that drugs often cannot be withdrawn without causing patients to relapse (Kamano, 1965). Although drug produce significant and beneficial behavioral changes, these changes seem to be dependent upon the continued use of the drug. When the drug is terminated, the person’s behavior deteriorates. Psychiatrists have not adequately responded to this problem. The general practice is apparently to maintain patients on drugs indefinitely. Apart from the possibly adverse physiological effects of long-term drug usage, the problem is of interest for theoretical reasons. It seems to indicate that little if any relearning occurs on the part of people who take these drugs. That is, if a patient is chronically anxious or frightened by a number of things in his environment, he can be calmed down, perhaps indefinitely, by tranquilizers, but the situation that set him off originally will do so again once the drug is withdrawn. The patient has not learned that the situations are harmless.

A similar problem is evident in the animal literature. A number of studies, in particular those of Neal Miller and his associates (Miller, 1966), have found that drugs will reduce fear as evidenced by the reinstatement of a bar-pressing response which was previously associated with electric shock, but that once the drug is withdrawn, the animals stop the bar pressing. The fear response is evidently suppressed temporarily by the drug, but no relearning is apparent since it reappears once the drug is withdrawn. The animals evidently have not learned that bar pressing is no longer associated with shock. Although the animal literature is by no means consistent (e.g., Miller, Murphy, & Mirsky, 1957; Nelson, 1967), it can be summarized by saying that the transfer of a drug-produced change in behavior to the nondrug state is often difficult to fade.

Granted that the problem exists and that it warrants further attention, how can we get a drug-induced change in behavior to persist into the nondrug state? Considering the experiments dealing with the labeling of visceral behavior, the answer may be quite simple. What would happen if we made someone attribute his changed behavior to himself rather than to the drug? Imagine a patient whose persistent fears have diminished due to a tranquilizer that he has been taking for some months. After this period let us convince the patient that we have substituted an inert placebo for the real drug throughout the past month. What should he conclude from this? We believe that the patient would now have to accept the responsibility for his changed behavior and because of this might make three inferences: (a) "The world can’t be that frightening after all" (he will reevaluate the stimulus situation); (b) "I have succeeded and am competent" (he will feel happy and proud of himself); (c) "I will subsequently be able to behave differently when in stressful situations" (his expectations and aspirations for improved behavior will be higher). These kinds of inferences would seem to facilitate the maintenance of the behavior change once we have actually terminated the drug. Such subjects should maintain their improved behavior to a greater degree than those who have a drug-attribution for their behavior change.

These attribution-notions lead to the following hypothesis: Subjects who are disabused of the cognition that a drug has produced certain significant changes in their behavior will be more likely to maintain these behavior changes than subjects who continue to believe that their changed behavior was due to a drug. A direct way of testing this hypothesis would be to use drugs, but for several reasons we chose not to: they require extensive pretesting; they are relatively unpredictable; and the supervision of a physician would be
necessary. Additional difficulties are posed by the necessity to establish a dosage which would be strong enough to change behavior yet weak enough to convince a subject that he had "actually" received a placebo. While all these problems do not appear insurmountable, our primary interest in attribution per se rather than in drugs led to an analogical test of the hypothesis. The experiment was therefore conducted by (a) obtaining a pre-measure of pain and tolerance thresholds using fingertip-administered electric shocks; (b) administering a placebo capsule which was presumably a drug; (c) surreptitiously lowering the overall intensities of the shocks during a second threshold procedure so that all subjects believed that they could take more electric shocks before pain was experienced and before they desired to stop than they did during the first threshold procedure; (d) telling half the subjects that they had received a placebo; and (e) assessing their reactions during a third threshold procedure during which the subjects received no pill. It was predicted that those subjects who were led to attribute their behavior during Threshold II to themselves (because they had received a placebo) would subsequently raise their thresholds more than subjects who attributed their behavior during Threshold II to a drug.

**STUDY**

**Subjects.** Subjects were male undergraduates enrolled in introductory psychology courses. As part of the course requirements, they were offered participation in two separate 3-hour experiments to be rubbed back-to-back. The first was described as an experiment in skin sensitivity and the second as an experiment in intersensory stimulation. Subjects had no expectations about the use of electric shock or drugs.

Apparatus. The shock generator delivered 60 cycle shocks, 500 milliseconds in duration. The intensities for the first and third threshold tests began with 0 microamperes and increased by 200 microamperes at each step. The intensities for the second threshold test began with 0 microampere and increased by 100 microamperes at each step. The electrodes were Grout silver-plated cup-type, 0.5 inch in diameter, taped with electrode paste to the first and third fingertips of the subject's non-dominant hand.

**Procedure.** Structured to the subject as two independent experiments, the procedure actually entailed three shock-threshold tests, the first two administered by one experimenter and the third by another. The procedures were identical for all subjects except where noted below. The design is summarized in Table 1.

After the usual introductions, the experimenter explained that he was studying the possible effects of a vitamin compound, "parataxin," upon skin sensitivity. In order to test this, he would determine, by means of a specially constructed shock apparatus, when the subject first perceived a shock, test felt that it was "painful or unpleasant," and finally perceived it as "so painful or unpleasant that you cannot tolerate any more." A parataxin capsule would then be administered and a second threshold taken a few minutes after ingestion of the "fast-acting drug." The subject was assured that both the pill and the shocks were quite harmless. At this point each subject was asked to sign a "Subject Consent Form," which indicated that a full explanation of the experiment would be given later and that the subject could withdraw at any time. No subject withdrew from the experiment.

After attaching the electrodes the experimenter gave the subject a score sheet on which to check the shocks as they were administered; it was intended thereby to make the manipulated changes at Threshold I all the more salient. One hundred shock intensities were listed, and the subject was to circle those corresponding to the three threshold points described above. The first threshold test was then administered. Following this test, the subject was given the parataxin capsule and kit alone "while it takes effect." After 2 minutes the experimenter returned and repeated the threshold test, except for a surreptitious manipulation of the actual shock intensities designed to make the subject believe that the capsule had changed his threshold

<table>
<thead>
<tr>
<th>Grout</th>
<th>Procedure</th>
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<tbody>
<tr>
<td>Placebo</td>
<td>Threshold I (Experimenter I) with pill</td>
</tr>
<tr>
<td>Drug</td>
<td>Disabuse: &quot;It was a placebo.&quot;</td>
</tr>
<tr>
<td></td>
<td>No disabuse: &quot;Now the drug hat worn&quot;</td>
</tr>
<tr>
<td></td>
<td>Threshold M (Experimenter I) same.</td>
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performance. The shock intensities were halved such that, for example, when the subject received Shock Number 5 on this second threshold test, he was actually receiving half of what he had received on Threshold I for "Shock Number 5."

Up to this point the experimenter was "blind" as to experimental condition. He now flipped a card from a bidden pile of group-assignment cards, telling him whether the subject was to be in the "placebo" or "drug" group. At this point instructions varied as follows:

**Placebo**: Would you be very surprised if I told you that you didn't get a real drug? We really just gave you a placebo pill; that is, the capsule just contained inactive sugar. We had to do this to control for what is called the placebo effect. When you do drug research, sometimes people are affected just because of the attention that you have given them, and regardless of what you have given them. To control for this effect we have divided our subjects into two groups; one group gets the real drug and another group gets the fake pill. To determine whether the drug has had an effect, we have to compare drug people with placebo people. Do you understand?

The subject's questions as to why be had taken more shock were deflected by statements like, "Well, that could happen, I guess." The subject was then sworn to secrecy and thanked for his participation.

**Drug**: Let me check you eyes to see to what extent your pupils can dilate. This is a measure of the amount of effect the drug has had on you. I am just going to shine a small light in your eye, and there is nothing to be afraid of, just hold your head steady, please. Your pupils are still not constricting completely. The parataxin is beginning to wear off though, and you should be back to normal in another minute or so.

After a second pupillary check, the experimenter said that the parataxin was bad. In fact, worn off. The subject was then thanked for his participation.

**Threshold I**: The second experimenter entered shortly after the first had left, but before he could launch into the cover story for "his experiment" the first experimenter returned and asked the subject to fill out a questionnaire which "the Department requires in experiments which use thugs." The second experimenter suggested that the subject do this during a natural pause in the experiment. (This ploy resulted in subject's construing the questionnaire as part of the "first experiment" without, however, his actually seeing it until after Threshold I!) The second experimenter then proceeded to explain that he was studying the effects of intersensory stimulation upon pain sensitivity. He was running his study in tandem with the first experiment simply because of the availability of the apparatus and for convenience of scheduling. For purposes of experimental design, the subject would have to undergo another threshold test "which you probably are already familiar with from the first experiment." After this initial threshold test, the subject would be given another, during which auditory stimulation would be interspersed. The same threshold test as in Threshold I was then administered. Naturally, this experimenter was "blind" as to the subject's condition and performance. Following this, the subject completed the questionnaire. Prior to debriefing, the last nine subjects (five placebo and four drug) were asked for a rating of a sample shock. The shock was that which the subject had indicated as "painful or unpleasant" on Threshold I, this bit of information having been surreptitiously left behind by the first experimenter. Several further questions were asked of all subjects before complete debriefing, namely (to placebo subjects): "I see that you took more shocks on Mr.______'s second threshold test. Since the capsule was actually a placebo, how do you account for this?" To all subjects: "Did you see my experiment related in any way to Mr. ______'s?" "How many shocks did you expect to take on the threshold test I just gave you?" All subjects were then thoroughly debriefed.

**Results**

*Success of the manipulations*. The questionnaire which the subjects had filled out "for the Department" provided supporting data that the desired effects had been achieved by the various manipulations. All! subjects in the placebo group did, in fact, regard the pill they had received as a placebo and, except for two subjects, the drug subjects believed that their capsule was a true drug and that it had produced a change in skin sensitivity. The data of two drug subjects, who suspected that they had gotten a placebo and that the shock intensities had been halved, were excluded. None of the subjects considered the "two experiments" as related.

**Threshold performance. It should be noted** first that all subjects took more shocks on Threshold II than on Threshold I before announcing pain and tolerance. There were also no differences between placebo and drug groups when Threshold II was compared to Threshold I ($5 < 1$). Thus, the two groups of subjects behaved similarly through the second threshold test, as one would expect from the random assignment of subjects to the groups.

The data of primary interest are the differences between Threshold III and Threshold I for those points at which subjects reported pain and inability to continue. As can be seen in Table 2, subjects who were denied the cognition that they had received a drug (placebo) took an average of 1.77 more shocks at the third threshold test before reporting pain, while subjects who
entered the third test still believing that the drug had effected the changes during Threshold II did not, on the average, improve at Threshold III as compared to Threshold I. The difference between the improvement of placebo and drug subjects, however, is marginally significant. More striking are the changes for tolerance. Here placebo subjects endured 1.15 more shocks, while drug subjects took —.73, this difference being statistically reliable.

Perhaps a better way of looking at the data is to consider the improvement from Threshold I to III as a percentage of the improvement from I to II. Analyzing the data in this manner allows us to evaluate the percentage of "drug-improved" behavior which is subsequently maintained. Such an analysis also has the virtue of correcting for different degrees of behavior change from Threshold I to II. It can be seen in Table 2 that placebo subjects maintained a greater percentage of their "drug-improved" behavior than did drug subjects. The principal hypothesis was therefore confirmed: disabusing subjects of the cognition that a drug had effected improvement in their shock-taking behavior leads to greater generalization of this "drug effect" to the "undrugged" state.

Some additional findings are also of interest. It will be recalled that following Threshold III the subject was asked how many shocks he had thought he would take on that threshold. Of the 13 placebo subjects 10 (77%) indicated that they had expected to take more shock on the third threshold than on the first, while of the 11 drug subjects none expressed the expectation of taking more. This difference is highly significant ($p < .001$), suggesting that being disabused of a drug-attribution cognition increases subjects' expectations about the number of shocks they will be able to take on a subsequent test.

It will also be recalled that nine subjects were asked, following Threshold III, to rate a single shock on a scale ranging from 0 (not at all painful or unpleasant) to 100 0 (extremely painful or unpleasant); the shock given each subject was that rated as painful on Threshold I. Analysis of these data reveals that placebo subjects considered this shock to be somewhat less painful than did drug subjects ($t = 2.15, p < .10$, two-tailed). All subjects also rated the perceived pain of the last shock that they had taken on the final threshold, In spite of the fact that the placebo subjects had taken stronger shocks, they rated their final shocks as being no stronger than did the drug subjects. These two sets of ratings suggest that the placebo subjects reevaluated the painfulness of the shocks.

**Study II**

**Method**

A replication was conducted with an apparatus which permitted the delivery of shocks in smaller incremental steps. Thresholds I and III now consisted of a series of shocks increasing by 100-microampere steps while Threshold II increased in 50-microampere steps. It was thought that by using smaller increments the dependent measure would more readily reflect the subjects' ability to withstand shock. The electrodes and their placement were the same as in Study I. An added feature was a remote control unit that enabled the experimenter to deliver the shocks at some distance from the subjects, as well as to reduce by one-half the intensities of all shocks for Threshold II. As in the first study, all shocks were 500 milliseconds in duration.

This replication was identical to Study I except for a procedural change designed to improve the
deception. The subject was told that he would have to set the shock intensities himself to make the experimenter have to monitor various physiological changes at a console at the other end of the room. It was felt that the deception would thereby be even more compelling since the subject would now be less likely to think that the experimenter was reducing the intensity of the shocks. Dummy electrodes were taped to the forearm of the subject's nonpreferred hand for this purpose, and the shock box was positioned so that the subject could read and manipulate the decade switches. The instructions were to increase each successive shock by 100 microamperes. In all other respects the procedure followed by the first experimenter was the same as that of the first experiment. With the exception that the subject was setting the shock intensities, the second experimenter also followed the same procedure as in the first experiment.

Results

As in Study I, the manipulations were successful. Placebo subjects regarded their pill as a placebo while drug subjects believed it was a drug and attributed their improvement at Threshold II to it. Furthermore, all subjects noted significant decreases in their pain sensitivity at Threshold II and all believed that the two experiments were unrelated. The data of one drug subject were excluded, however, because he thought that the drug was still active during the third threshold test.

Threshold performance. As in Study I, the first and second thresholds of the two groups did not differ. The placebo and drug groups did differ, however, in their change-scores between threshold III and Threshold I for reports of pain and of tolerance. It can be seen in Table 3 that once again there is marginal significance for pain, the placebo subjects showing a mean increase of 4.54 shocks as compared to the increase of 1.10 shocks of the drug group. If we combine the probability levels for Studies I and II (cf. Stauffer et al., 1949), the musical differences for pain are significant at the .03 level. Considering the point at which subjects stopped taking shock, placebo subjects also raised their tolerances significantly more than drug subjects. Analyzing the improvement on Threshold III as a function of the improvement on Threshold II leads to similar data: placebo subjects maintained a greater percentage of their "drug-improved" behavior than did drug subjects.*

As in Study I, subjects were asked how many shocks they had expected to take on Threshold III. Of the 11 placebo subjects 9 (82%) expected to take more shock than they had on Threshold I, while of the 10 drug subjects none had this expectation. This difference is highly significant (p < .001), replicating the corresponding finding from Study I.

Subjects were also asked to rate a single shock on a scale from 0 to 100, the shock given being that which had been rated as painful by each respective subject on Threshold I. Whereas marginal significance had been found in Study I (placebo subjects tending to rate this as less painful than drug subjects), the difference in this replication was not significant. This finding is also supported by the ratings of the last shock of the third threshold. Placebo subjects rated this shock as significantly more painful than did drug subjects.

Moreover, we find within the placebo group a "genuine" effect; that is, relative to their own Threshold I, these subjects took significantly more shocks before announcing pain (p < .01) and before stopping (p <.01). This "true effect" was not found in Study I, the actual increase for those placebo subjects not being significant, although it was significantly greater than the decrease observed for the drug group.

<table>
<thead>
<tr>
<th>Condition</th>
<th>N</th>
<th>I(^0) shocks</th>
<th>II</th>
<th>III</th>
<th>III-I(^\prime)</th>
<th>III-I/II-I(^\prime)</th>
<th>% improvement</th>
<th>DO-Tolerance</th>
<th>% improvement</th>
</tr>
</thead>
<tbody>
<tr>
<td>Placebo</td>
<td>11</td>
<td>10.40</td>
<td>26.40</td>
<td>14.98</td>
<td>4.54</td>
<td></td>
<td>37%</td>
<td>21.27</td>
<td>48.00</td>
</tr>
<tr>
<td>Drug</td>
<td>10</td>
<td>10.20</td>
<td>24.90</td>
<td>11.30</td>
<td>1.10</td>
<td></td>
<td>13%</td>
<td>21.60</td>
<td>43.60</td>
</tr>
</tbody>
</table>

subjects (p < .02). These data suggest that of the cognition that they had received a drug may be seen as using a placebo caged-vice superior behavior change, and not as placebos are generally used, namely, as a control for the administration of a drug and the attendant expectations of gain over and above the actual drug effect.

The mechanism by which attribution effected maintenance of the behavior change has not adequately been isolated in this experiment. It was expected that one or more of three processes would be initiated when a subject observed that his changed behavior (on Threshold II) was due to himself: reevaluation (the subject would infer that the shocks were not as painful as he originally thought); changes in expectations and aspirations (the subject would infer that his subsequent behavior would resemble his behavior on Threshold II and he would be motivated to take more shock); generation of positive affect (the subject would be pleased and proud of his improved performance on Threshold II, and these positive feelings would raise his threshold for pain, perhaps by reducing his anxiety). Of these processes, we have data relevant to reevaluation and changes in expectations.

If reevaluation occurred we would expect that prior to the third threshold the placebo subjects would have inferred or convinced themselves that the shocks were really not as painful as they had originally believed. Their performance on the third threshold would then reflect this reevaluation. In accord with this reasoning, placebo subjects did take more shocks on the third threshold before announcing pain and before stopping than they did on the first threshold. Drug subjects did not. Although the changes in pain thresholds were not as strong as the changes in tolerance, both may be considered a reflection of reevaluation (the pain threshold data are undoubtedly weaker when one considers that subjects seemed to find this a difficult judgment, one which in fact results in variances more than twice as large as that of the tolerance threshold). However, if reevaluation did occur, we might also expect that the placebo subjects would have rated the shocks lower in painfulness than the drug subjects when given the opportunity to do so.

DISCUSSION

The results of Study I and the replication support the hypothesized importance of attribution in generalizing drug-produced behavior changes to the nondrug state. In our analogue to psychoactive drug therapy, subjects were deceived successfully into believing that a drug had significantly raised their pain thresholds and increased their tolerances for electric shock. The crucial attributions were then differentially manipulated by disabusing some subjects (placebo) of the that their behavior change could have been caused by the capsule. Deprived of this drug attribution, subjects evidently attributed their improvement to themselves, subsequently maintaining these changes to a greater degree than manifested by control subjects, who continued to attribute their improvement at the second threshold to the drug.

It is important to distinguish our findings from the well-known "placebo effect" (cf. Frank, 1961; Honigfeld, 1964). The placebo effect refers to the phenomenon whereby individuals change with an inactive substance in a direction in which they think they should change. In our own studies above, this would refer to the improvement that might have been observed at Threshold II over and above the surreptitious halving of all shock intensities. This is for our purposes uninteresting; furthermore, conclusions cannot be drawn from our data because improvement from Threshold I to II could be accounted for by adaptation, for which we did not have a control group. Our main results deal with the differences between Thresholds I and III, that is, with the persistence of our analogue drug-produced behavioral change from Threshold II. Our disabusing the placebo subjects

Our distinction between self-attributed and behavior change may seem similar in some ways to Rotter's (1966) distinction between internal and external control. We hesitate to stress this apparent similarity, however, inasmuch at Rotter restricts himself to locus of reinforcement, whereas it is difficult to find reinforcement operating meaningfully in our paradigm,
after the third threshold. There was a tendency for this to occur in the first experiment but not in the second.

It is the absence of strong effects on this measure that leads us to question whether reevaluation of the shocks accounted for the placebo subjects' third threshold performance. Nevertheless, we believe that reevaluation is a viable explanation. The fact that the subjective pain ratings do not reflect reevaluation might very well be due to their having been obtained after the third threshold. At this point in time, placebo subjects might have considered that, although they took more shock on the second threshold than on the first, their third threshold performance was relatively poor. Their inability to match their Threshold II performance might have then led them to infer that the shocks were more-painful than they had thought prior to the third threshold. In other words, subjects might have evaluated the shocks as less painful after the second threshold and consequently took more shock on the third threshold, but, when considering that this last performance was not as good as on the second threshold, they might have inferred that the shocks really were painful. We have here two reevaluations, the second of which may have cancelled the first.

Considering now the subjects' expectations after the second threshold, it will be recalled that in both experiments placebo subjects expected to tolerate more shocks on the third threshold than on the first, whereas drug subjects did not. Although these differences in expectation were predicted, can they alone account for the differences in third threshold performance? If subjects were motivated to match their expectations to take more shock on Threshold III than on I, then behavior on the third threshold could be explained without reference to reevaluation. This is possible and not at all uninteresting. Since we were apparently successful in dissociating the two experiments, subjects would be matching their own expectations and not those of the second experimenter. We believe, however, that this explanation seems to account better for the tolerance data than for the pain data. Tolerance for shock is something that a subject could raise without deceiving the experimenter, whereas, without reevaluation, the recognition of pain could be changed only by deceiving the experimenter. Experimental subjects do not act in this manner! It seems more likely that they would reevaluate the shocks than harm an innocent experimenter.

Although this study does not permit us to accurately specify the mechanism by which the evaluation of one's behavior affects subsequent behavior, it should be clear that attitude-change research has produced data consistent with ours. Both dissonance research and the data that Bern (1967) has gathered supporting his interpretation of that research lead to the conclusion that attitude change is very much influenced by an individual's explanation for his behavior. If an individual believes that he is responsible for engaging in a behavior which is inconsistent with his own attitude, his attitude will more readily change and become consistent with this behavior than if he believes that external forces are responsible for his behavior. Thus, if there is little external justification for engaging in the behavior (e.g., it is effortful), associated with little reward, not very important to the experimenter or science), the individual is likely to interpret the behavior as self-induced. If it is self-induced the individual can best explain his behavior by forming an attitude that can account for it. Thus, in these situations we find more attitude change than in those situations where there are adequate external justifications for engaging in the behavior.

These data and speculations seem also to have interesting implications for the general area of behavior modification. As an extension of general experimental psychology, behavior therapy is essentially environmental, looking to external variables for the alteration of "abnormal" behavior. The literature indicates to us little concern shown to how the individual so manipulated perceives the reasons for his changing. If we make what seems to be the reasonable assumption that the behavior therapy client construes agents of change to be outside himself, we have a situation analogous to that dealt with above, namely, a change brought about...
operant approaches would pose problems for the maintenance of behavior. Inge once the artificially imposed contingencies are withdrawn (cf. Davison, 1969); and the failure thus far to demonstrate enduring behavior change via operant procedures might be accounted for at least in part by the notions of attribution proposed here. If a person realizes that his behavior change is totally dependent upon an external reward or punishment, there is no reason for his new behavior to persist once the environmental contingencies change.

Likewise, consider a recent comment (Davison & Valins, 1968) made on the conflicting results obtained with the use of the barbiturate Brevital as a means to induce deep muscle relaxation during systematic desensitization (cf. Brady, 1966, 1967; Friedman, 1965; Reed, 1966). It was suggested that the use of this drug to impose relaxation upon an individual might create problems for the generalization of desensitization in imagination to the real life situation. It has been observed from experimental studies in desensitization that the transfer of desensitization effects from the imaginal anxiety hierarchy to the life situation is not at all a simple matter (Davison, 1968). The Brevital desensitization work seems to be a situation identical to the core of this paper: the relaxation produced by Brevital is likely to be perceived by the person as not of his own doing. The important notion, "I have been able to control my inactions in this situation," seems more probable when persons relax their own muscles than when they are relaxed by a drug (cf. Vahan & Ray, 1967). This concern is quite separate from the efficacy of Brevital to enable the client to climb his imaginal anxiety hierarchy, just as the experiments ported above do not concern the changes at Threshold II; rather, we would expect that clients who attribute their relaxation during desensitization to a drug rather than to their own efforts will maintain these gains significantly less when confronted with the real situation.


Miller, R. E., Murphy, J. V., & Mirsky, I. A. Persistent effect of chlorpromazine on extinction of an avoidance response. Archives of Neurology and Psychiatry, 1957, 73, 526-530.


(Received May 8, 1968)