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INSOMNIA AND THE ATTRIBUTION PROCESS[1]

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Insomniac subjects were given placebo pills to take a few minutes before going to bed. Some subjects were told that the pills would cause arousal (arousal condition), and others were told that the pills would reduce arousal (relaxation condition). As predicted, arousal subjects got to sleep more quickly than they had on nights without the pills, presumably because they attributed their arousal to the pills rather than to their emotions, and as a consequence were less emotional. Also as predicted, relaxation subjects got to sleep less quickly than usual, presumably because they assumed that their emotions were unusually intense since their arousal level was high even after taking an arousal-reducing agent. The results have relevance for Schachter's theory of emotions and Kelley's attribution theory. Pragmatically, the findings suggest the feasibility of a therapy based on reattribution of symptoms, and indicate that traditional suggestion effect practices should be modified.

In an important experiment on emotion published in 1962, Schachter and Singer exposed subjects to situations designed to elicit either anger or euphoria. Prior to their exposure to these situations, subjects were injected with adrenalin, a drug which produces autonomic arousal. Some of the subjects were told that they were being injected with a drug which would cause autonomic arousal, while other subjects were given no information about the arousal effects which the injection would produce. The uninformed subjects were far more emotional—either euphoric or angry, depending on the experimental condition—than were informed subjects. The experiment has been taken as evidence of the emotional plasticity of the state of autonomic arousal. Individuals in a state of arousal may experience very disparate emotional states or no emotional state at all, depending on the cognitions which attend the arousal. A perhaps equally important implication of the experiment has received little attention. Not only were informed subjects less emotional than uninformed subjects, they were also less emotional than control subjects who received a placebo. This trend, though statistically not significant, suggests that informed subjects overcompensated for the injection. They perhaps attributed not only adrenalin-produced arousal to the injection, but naturally occurring arousal as well. As a consequence, informed subjects were less emotional than they "should" have been, given the emotion-eliciting situations in which they were placed.

In order to determine whether people can be induced to believe that part of their naturally occurring arousal is due to an artificial, external source, Nisbett and Schachter (1966) gave sugar pill placebos to subjects who were about to undergo a series of electric shocks. Some of their subjects were told that the pill would produce palpitation, breathing rate increase, and "butterflies in the stomach." Other subjects were told that the pill would produce a variety of symptoms which were not autonomic in nature. Those subjects who believed themselves to be in a state of drug-produced arousal found the shocks less painful than did other subjects, and were willing to tolerate higher shock intensities. Furthermore, an internal analysis revealed that toleration of shock was a direct function of the extent to which subjects ascribed their arousal to the pill. The experiment indicates that it is indeed possible to persuade subjects that their naturally occurring arousal has an external origin. As a consequence, such subjects lower

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their estimation of the intensity of the stimulus which actually produced the arousal.

It is useful to discuss this research in the context of the attribution theory of Kelley (1967) stemming from Heider's (1958) work. Briefly, Kelley proposes that many cognitive and motivational phenomena are the result of the individual's perception of causes for the psychological effects which he observes in himself. In this process of causal attribution, the individual can make mistakes, that is, attribute an effect to the wrong cause. Such errors may have pronounced effects on his subsequent motives and beliefs. Thus, the subjects in Schachter and Singer's (1962) experiment may be viewed as victims of an experimentally produced attribution error. Uninformed subjects in that experiment who were injected with adrenalin mistakenly attributed their arousal to the situation in which they found themselves, rather than to the injection. As a consequence, they became emotional. Similarly, subjects in Nisbett and Schachter's (1966) experiment, who were told that their placebo pills would produce arousal, mistakenly attributed shock-produced arousal to the pills, and as a consequence found the shock to be less aversive than it "really" was.

Ross, Rodin, and Zimbardo (1969) have proposed that the reattribution of arousal symptoms may be of use in alleviating maladaptive emotional states. Ross et al. conducted an experiment similar to that of Nisbett and Schachter. Their subjects were encouraged to attribute the arousal symptoms accompanying fear of electric shock to a loud noise piped in over a headset. Such subjects were shown to be less fearful than subjects who could only attribute their arousal symptoms to fear of electric shock. Following Valins' (Valins, 1966; Valins & Ray, 1967) suggestion that cognitive manipulations of perceived *level* of arousal may have therapeutic applications, Ross et al. proposed that manipulations of the perceived *source* of autonomic arousal may also have therapeutic uses. The present experiment was an attempt to produce such a therapeutic lessening of a maladaptive emotional state by means of a reattribution of arousal symptoms.

The state of insomnia seems a promising candidate for a first attempt at a therapeutic intervention using the reattribution technique. Emotionality at bedtime can be a chief proximal cause of insomnia. The high level of mental activity and the alertness produced by an emotion are incompatible with sleep and could delay sleep onset. The present line of theorizing would suggest that to the extent that an insomniac goes to bed in a state of autonomic arousal and associates that arousal with cognitions which are emotionally toned, he should become more emotional and have greater difficulty getting to sleep. However, if the insomniac were to take a "drug" which he believed to be capable of producing arousal symptoms, he might attribute part of his arousal to the drug, and might perceive the emotionally toned cognitions to be less intense. As a consequence, such a subject might become less emotional. Insomniac subjects given a placebo which they believe to be an arousal agent might therefore paradoxically get to sleep more quickly than usual.

Such an experiment also provides a framework within which to test a second hypothesis of theoretical and practical interest. If the belief that arousal has been produced by a drug leads to lowered emotionality, then the belief that arousal has been reduced by a drug should lead to increased emotionality. The subjects who mistakenly believe themselves to be under the influence of an arousal-decreasing agent should become highly emotional. Such subjects should say to themselves, in effect, "If I feel as aroused as I do now, when a drug is operating to lower my arousal, then I must be very aroused indeed." More formally, such subjects should perceive any arousal which they experience to be an underrepresentation of the intensity of their emotionally toned cognitions. If such a subject experiences normal arousal, but thinks it has been "drug reduced," he will infer that his cognitions are unusually powerful. Emotionality should thus be increased, and the state of insomnia should become worse for such subjects.

Thus, it was hypothesized that (*a*) insomniacs given placebo pills, which they believe capable of arousing them, will attribute their naturally occurring arousal to the pill, will therefore experience less intense emotions, and will fall asleep more quickly than usual;

and (*b*) insomniacs given placebo pills, which they believe capable of calming them, will attribute more than their naturally occurring arousal to emotionally toned cognitions, will therefore experience more intense emotions, and will fall asleep less quickly than usual.

METHOD

The experimental test of the hypotheses required (*a*) recruiting subjects who suffer from insomnia; (*b*) leading some subjects to believe that a pill would increase their arousal at bedtime, and leading others to believe that a pill would decrease it; and (*c*) measuring changes in the delay of sleep onset.

Subjects

Forty-two subjects were recruited by signs posted on the campus of Yale University. The signs were headed "Insomniacs wanted for psychological research on dreams." Attached to the posters were cards which volunteers completed and mailed in. The experiment was described to volunteers over the telephone as one on dream-content analysis which would take two ½-hour sessions and pay $3.00. Subjects were told that "light sleepers" were being recruited because "they tend to have more dreams and to remember them better than deep sleepers."

Ages of the subjects ranged from 19 to 26 years, with a mean age of 22.1 years. Thirty-three of the 42 subjects were male, and all but 2 were undergraduates or graduate students. As a group, the subjects appeared to be people who had considerable difficulty in getting to sleep. They reported taking 42.56 minutes, on the average, to get to sleep on the 2 nights preceding the first experimental session. This is comparable to the 59.06 minutes characteristic of the "poor" sleepers in Monroe's (1967) study of insomnia and very much more than the 7.18 minutes reported by his "good" sleepers.

Procedure

Subjects were seen individually in two 30-minute sessions, the first session on a Wednesday and the second on Friday of the same week. On Wednesday, subjects answered questions about their sleep on the 2 previous nights, Monday and Tuesday. The experimenter then explained the alleged purpose of the study. "I am interested in the possible effects of level of bodily activity on dream content. I think there might be some relationship between how active your body is internally, during sleep, and what you dream about."

Subjects were then told that they would be given a drug in the experiment:

> In order to find out the effects of bodily activity, I'm going to give you a drug to take tonight and tomorrow night. Of course, the drug is harmless; it's a nonprescription drug. It will have no effect on your ability to work or study.

Possible side effects of the drug, which constituted the experimental manipulation, were then described. While the experimenter excused himself to get the pills, subjects answered a bogus questionnaire about the frequency and type of dreams they usually experienced. When this was completed, subjects were given two sugar pill placebos, with instructions to take one that night, Wednesday, and the other on the next night, Thursday, about 15 minutes before going to bed. Subjects were instructed to continue taking any other medications as usual.[3] The drug side effects were then reiterated. To complete the cover story, subjects were given short dream-report forms which they were told to take home and complete whenever they awoke on the next 2 mornings. Finally, subjects answered a questionnaire designed to check whether they knew what symptoms the experimenter had told them to expect from the pill. The experimenter was prepared to correct any mistakes, but all subjects were aware of the appropriate side effects, and correction was never necessary.

Subjects returned on Friday of the same week for a session scheduled at the same time as the Wednesday session, and answered questions about their sleep on Wednesday and Thursday nights. Additional questions were asked about the experimental manipulation and the effects of the pills. Subjects were then interviewed, debriefed, and paid $3.

Manipulating Attribution

Arousal condition. The subjects' attribution of arousal was manipulated by varying the described side effects of the placebo pills. The pill was described to one group of subjects, those in the arousal condition, as a drug which would increase their level of arousal.

> This drug will increase your bodily activity. It works on the sympathetic nervous system, which is the system that arouses you and sends adrenalin through your system. The pill will increase your heart rate and it will increase your body temperature. You may feel a little like your mind is racing. In general it may arouse you.

These side effects were selected from arousal symptoms which pretest subjects reported as being typical of a night with insomnia. When subjects who have received this side-effect description go to bed, they should believe themselves to be under the influence of an arousal-producing drug. To the extent that they experience arousal symptoms, they should attribute them to the pill, rather than to emotional cues. This attribution should result in lowered emotionality, with a consequent decrease in the time needed to fall asleep.

Relaxation condition. For subjects in the other experimental group, those in the relaxation condition,

[3] Only three subjects, all in the control condition, were taking sleeping pills, and they all took equal doses of their drug on each of the 4 nights of the experiment.

the pill was described as one which would decrease arousal.

> This drug will lower your bodily activity. It works on the parasympathetic nervous system, which is the system that relaxes you. The pill will lower your heart rate. It will decrease your body temperature so that you will feel a little cooler. And it will calm down your mind. In general, it will relax you.

When subjects who have received this side-effect description go to bed, they should believe themselves to be under the influence of an arousal-reducing drug. Any arousal which they experience should be perceived as an underrepresentation of their true level of arousal. Such subjects should therefore attribute greater intensity to emotional cues than would otherwise be the case. This should result in heightened emotional states and a consequent increase in the time needed to fall asleep.

Control condition. As a control on any possible variations in sleep behavior from the earlier to the later part of the week, and as a check on any possible effects of simply being in an experiment, a control group was included. These subjects were not given pills, and were asked just to report on their dreams. They were given the same cover story about the experimenter's interest in the relation between bodily activity and dream content, but were told: "You have been placed in a control group. We want to see what kind of dreams you report on your own, without me giving you a drug." The control group and each of the experimental groups contained 14 subjects.[4]

Measurement

Sleep onset. In order to avoid possible suspicion as to the true nature of the experiment, the subjects were never directly asked how long it took them to fall asleep. Instead, subjects were asked to estimate for each of the 4 nights of the experiment, (*a*) when they went to bed and (*b*) when they fell asleep. The time it took each subject to fall asleep was computed by subtracting the time he reported going to bed from the time he reported falling asleep. This measure of sleep onset constituted the chief dependent variable.

Arousal symptoms. In order to determine the extent to which arousal symptoms were experienced at bedtime and were attributed to the pills, subjects were asked to report on arousal symptoms for the 2 preexperimental nights and the 2 experimental nights. All questions were answered on either 5- or 7-point scales. In order to measure the level of experienced arousal, the following were asked: "How warm or cold did you feel?" and "How much did your mind race?" In order to determine the extent to which arousal was attributed to the pills, subjects were asked how much the pills affected their body temperature, mental activity, and alertness.

In addition, subjects were asked how much they suffered from insomnia on each night, what drug they thought the pills actually contained, and what medications they had taken during the week. Finally, subjects were asked in an open-ended interview how the manipulation had affected them and whether they had suspected the true purpose of the experiment.

[4] The first four subjects in the control condition were given pills "to change bodily activity level," but were told that they would not perceive any side effects. These subjects behaved like the other control subjects, and the two groups are combined for purposes of analysis.

RESULTS

Subjects who were encouraged to attribute their arousal to the placebo (arousal condition) should have attributed less arousal to emotional cues, should consequently have experienced less intense emotional states, and should have gotten to sleep more quickly than usual. Subjects who were encouraged to believe that the placebos had calming properties (relaxation condition) should have assumed that the arousal they experienced was an underrepresentation of that produced by emotional cues, should consequently have experienced more intense emotional states, and should have gotten to sleep less quickly than usual. If arousal subjects are to attribute less arousal to their cognitions than relaxation subjects, it is essential that they attribute more of their arousal to the pill. For preexperimental and experimental nights, subjects were asked how much arousal they experienced (how much their minds raced and how warm or cold they felt); and for experimental nights, subjects were asked how much arousal the pill produced (how drowsy or alert the pills made them feel, how much the pills made their minds race, and how warm or cold the pill made them feel). Differences in reported arousal for preexperimental and experimental nights were slight and nonsignificant. Arousal subjects reported trivially more arousal on experimental than on preexperimental nights (+.25, on a 12-point scale consisting of the sum of the two arousal items), and relaxation subjects reported trivially less arousal (−.57, on the scale). Differences in attribution of arousal to the pill were quite marked, however. On each of the items which assessed beliefs about pill effects, arousal subjects reported more arousal as a consequence of having taken the pill than did

relaxation subjects. The difference between the sum of the three items was highly significant ($t = 4.36, p < .001$).[5]

The differential attribution of arousal was associated with substantial differences in the time it took for subjects to fall asleep. Table 1 presents subjects' reports of the amount of time it took to fall asleep on preexperimental nights (Monday and Tuesday), and on experimental nights (Wednesday and Thursday), reported as mean number of minutes per night. It may be seen that changes were in the predicted direction. The analysis of variance of the changes in sleep-onset time was significant at the .02 level ($F = 5.03$, $df = 2/39$). Moreover, the individual treatment effects were significant. The change of nearly 12 minutes in time to get to sleep reported by subjects in the arousal condition was a significant improvement. The 15-minute change in time to get to sleep reported by subjects in the relaxation condition was a significant worsening. Subjects in the control condition reported only a trivial improvement of less than 2 minutes. Both of the hypotheses were therefore confirmed.

Examination of the sleep-onset means for the preexperimental nights showed that the arousal group took longer to get to sleep on those nights than the other groups. It therefore is important to demonstrate that the improvement shown by the arousal group was not simply due to regression. That the improvement was not due to regression is indicated by the following facts: (*a*) The difference in the highly variable sleep-onset times for preexperimental nights was not significant ($F = 1.45$, $df = 2/39$). (*b*) The elevated mean for the arousal group was entirely due to the presence in this group of two individuals who took an extremely long time to get to sleep on preexperimental nights. When these subjects were excluded from consideration, the experimental effect was still present. Eight of the remaining 12 subjects fell asleep more quickly on experimental nights, and only 2 fell asleep less quickly ($p = .05$). (*c*) Most importantly, an analysis of covariance of the change scores with preexperimental sleep onset as the covariate quite clearly indicated that regression did not account for the experimental effects. With all of the subjects included in this analysis, the F ratio was 5.38 ($df = 2/38$, $p < .01$). The fact that the covariance F was slightly higher than the F for the simple analysis of variance indicates that the experimental effects actually counteracted, to a degree, the effects of regression.

[5] All probability values are based on two-tailed tests.

TABLE 1

MEAN TIME TO GET TO SLEEP PER NIGHT, IN MINUTES, AS A FUNCTION OF EXPERIMENTAL CONDITION

Statistic	Arousal	Control	Sedation
Average of preexperimental nights	53.22	38.40	36.09
Average of experimental nights	41.52	36.96	51.24
Mean change	11.70	1.44	−15.15
t	2.25	.27	2.16
p	<.05	*ns*	<.05

Note.—$n = 14$ in each condition.

It is noteworthy that the experimental effects occurred only for subjects who believed the pill descriptions. In this first attempt at a therapeutic intervention, the experimenters were not uniformly successful in persuading the subjects that they were being given a real drug. At the final session, subjects were asked if they had believed that the pills they took were arousers, relaxers, or something else, such as a placebo. Two of the subjects in the arousal condition and six of the subjects in the relaxation condition indicated that they had not believed that either of their pills contained a drug with the properties described by the experimenter. Table 2 presents sleep-onset times for believing and disbelieving subjects in the arousal and relaxation conditions. It may be seen that disbelieving arousal subjects took slightly longer to get to sleep on experimental nights than on preexperimental nights, and disbelieving relaxation subjects fell asleep somewhat more quickly on experimental nights. This suggests that the experimental effects occurred only when subjects reinterpreted the meaning of their symptoms in lights of the "knowledge" that they had taken a drug with effects on arousal state.

TABLE 2

CHANGE IN SLEEP-ONSET TIME, IN MINUTES, AS A FUNCTION OF EXPERIMENTAL CONDITION AND BELIEF OR DISBELIEF IN PILL DESCRIPTIONS

Group	Arousal	Relaxation
Believers	14.28 (12)	−29.40 (8)
Disbelievers	−3.72 (2)	3.78 (6)

Note.—Numbers in parentheses indicate number of subjects.

It will be recalled that subjects were asked how much they suffered from insomnia on each of the preexperimental and experimental nights. The data for reported suffering do not at all resemble the data for sleep onset. Arousal subjects actually reported a trivial increase in suffering on experimental nights (.214 points on a 7-point scale), and relaxation subjects reported a trivial decrease (.071 points). Subjects apparently did not base their reports of suffering on the relative amount of time it took them to get to sleep. The correlation between the change in reported time to get to sleep and change in reported suffering was only .07.

Why is it that reports of suffering do not reflect the same patterns as reports of sleep onset? It may be that this result is merely another instance of a general, rather paradoxical finding common to studies employing cognitive manipulations of feeling states. Differences in verbal reports of feelings in these studies are usually much weaker than differences in physiological, behavioral, or behavioroid measures (Davison & Valins, 1969; Nisbett & Schachter, 1966; Valins & Ray, 1967; Zimbardo, 1966). This does not explain the present pattern of results, since the general pattern in studies of this type is itself unexplained. However, it is important to note that this is not the first study to obtain such a discrepancy.

Whatever the reason for the discrepancy, the data on reported suffering are comforting in one respect. They serve to reduce the likelihood that the data on sleep onset were produced by possible demand characteristics inherent in the design. Demand characteristics are at work if subjects, sensing what results the experimenter expects or would like to obtain, behave in such a way as to yield those results. Demand characteristics could have produced the data in the present experiment if subjects in the arousal condition sensed that the experimenter expected their insomnia to improve, and if subjects in the relaxation condition sensed that the experimenter expected their insomnia would become worse. If such biases were at work, it seems likely that they would have been reflected in subjects' answers to the straightforward question, "How much did you suffer from insomnia?" The fact that subjects did not respond in the predicted ways to such a direct question makes it appear unlikely that their reports of sleep onset were produced by a desire to please the experimenter.[6]

DISCUSSION

Therapeutic Applications

Insomniac subjects were led to believe that a pill produced their arousal symptoms at bedtime and were consequently able to fall asleep more quickly than usual. The goal of demonstrating the potential usefulness of reattribution therapy has therefore been realized. It would be premature, however, to propose that the reattribution technique has widespread therapeutic implications. The present study represents only a single therapeutic attempt, using only one technique, to achieve moderate improvement of unknown duration in a rather mild pathological condition. Moreover, the failure to obtain improve-

[6] One further artifactual possibility deserves some mention. It may have occurred to the reader that relaxation subjects might have gone to bed earlier than their usual bedtime, in the expectation that the pill would help to put them to sleep. If so, relaxation subjects might have been less tired than other subjects and might have gotten to sleep less quickly for this reason. Actually, there was a slightly greater tendency for relaxation subjects to go to bed earlier on experimental nights than there was for arousal subjects ($t = 1.57$, $p < .15$). However, the two groups reported almost identical degrees of tiredness at bedtime on experimental nights (5.32 for relaxation subjects; 5.31 for arousal subjects). Moreover, there was no correlation between the tendency to go to bed earlier on experimental nights and the tendency to take longer to get to sleep on experimental nights ($r = .12$ for relaxation subjects; $r = .04$ for arousal subjects). Thus, it seems unlikely that the tendency of relaxation subjects to go to bed earlier was responsible for the worsening in sleep-onset time.

ment in subjects' self-report of suffering must temper the authors' enthusiasm. One should demand of a therapeutic technique that it produce improvement in subjective state, not merely improvement of behavior. Nevertheless, the present findings are encouraging for a first attempt. It is hoped that the present investigation will prompt the study of other applications of the reattribution technique. To that end, the authors speculate briefly on extensions and improvements of the present method.

One undesirable aspect of the technique used in the present study, from the standpoint of producing lasting improvement, was the reliance on pills and deception. More permanent and less "gimmicky" techniques for achieving reattribution may be possible, however. For example, insomnia sufferers could be told that their arousal at bedtime is due to a general condition of high base-line autonomic arousal. (There is evidence that insomniacs do in fact have higher base-line arousal; Monroe, 1967). Just as some people have high metabolic rates, insomniacs might be told, others have a high rate of autonomic functioning. This technique, like the pills, might offer a nonemotional attribution for naturally occurring bedtime arousal, yet at the same time would eliminate the need for the patient's continuing belief in placebos.

A second extrapolation of the present method might allow for temporary use of the pill technique. Let us consider what would happen to a patient whose condition improved through the use of placebos, and who then discovered that he had been hoaxed. An experimental model of such a situation has been examined by Davison and Valins (1969). Their subjects were given a series of electric shocks and were then given placebos which, they were told, might affect their sensitivity to shock. This was followed by a series of shocks surreptitiously decreased in intensity. At this point half of the subjects were told that their pills were really placebos, and the other half were told that the drug was wearing off. In a third set of shocks, "dehoaxed" subjects were able to tolerate shocks of greater intensity. This experiment suggests that dehoaxed subjects benefited from the belief that they themselves, instead of a drug, were responsible for their behavioral improvement. Similarly, insomniacs might also benefit from learning that a mere reattribution of arousal had caused improvement. This might make it apparent to such individuals that their suffering is not inevitable and that their attitudes toward their symptoms exert an influence on the symptoms.

The Attribution Process

In addition to demonstrating that reattribution techniques are of potential therapeutic value, the present study was concerned with shedding more light on the attribution process itself. It should be admitted at the outset of a discussion of process that we have no definitive means of showing that the sleep-onset changes were produced by the differential attribution of arousal symptoms to the pills. Sleep-onset changes may have been produced by a variety of processes which are theoretically less interesting. It is conceivable, for example, that before going to bed, the arousal subjects were worried about the possibility that the pill would make them uncomfortably aroused, and were relieved to find that it did not have this effect. This feeling of relief might have made it easier for arousal subjects to get to sleep. Similarly, relaxation subjects might have been happily anticipating a state of relaxation at bedtime. Their disappointment (and/or resentment) upon realizing that they were not in such a state might have prevented them from going to sleep. It is also possible that there were attention shifts which made it easier for arousal subjects to fall asleep and harder for relaxation subjects to fall asleep. For example, arousal subjects may have concentrated on their symptoms rather than their worries. Or it is possible that there were differences in behavior before bedtime which made it easier for arousal subjects to fall asleep and harder for relaxation subjects to fall asleep.

The scope of the present experiment was such as to make it impractical to control for all possible alternative explanations. Thus, there is little which can be said to counter these alternatives, except to point out that they detract little from the practical interest of the present research, and to note that there was nothing in the formal or informal com-

ments of the subjects to lend plausibility to any of these alternatives. There is, however, a remaining alternative mechanism which is wholly consistent with the attribution-theory framework and highly plausible in view of some of the comments made by subjects.

Sleep-onset changes may have been produced not by an alteration in the perceived intensity of emotionally toned cognitions, as was proposed in the introduction, but by another consequence of the initial attribution error. Informal conversations with subjects revealed that many of them appeared to worry about the fact that they were insomniacs—about their inability to control such a basic function as sleep and about the state of insomnia as evidence of more general pathology. There is good reason to believe that the experimental manipulations would have had an effect on worries such as these. Arousal subjects were told, in effect, that on experimental nights their insomnia would be caused by a drug. On experimental nights, therefore, arousal subjects did not have to view their symptoms as evidence of inadequacy or pathology. They may have worried less about their condition and may have gotten to sleep more quickly for this reason. Similarly, relaxation subjects were told, in effect, that on experimental nights they should experience fewer insomnia symptoms than usual. On experimental nights, therefore, relaxation subjects would have had to view anything less than a noticeable reduction in their symptoms as evidence of a particularly bad bout with insomnia. Upon failing to experience such a reduction, they might have worried more than usual about their condition and consequently have gotten to sleep less quickly. The attribution error may not have resulted in a change in emotionality across the board, then, but only in a change in degree of worry about the condition of insomnia. To the extent that worry about insomnia further interferes with sleep, such changes could have produced the experimental results.

Whether or not such processes occurred in the present experiment, it seems likely that there are pathologies involving a vicious cycle of the following type: (*a*) occurrence of symptoms, (*b*) worry about symptoms, and (*c*) consequent exacerbation of symptoms. For example, males with problems of impotence probably respond with alarm to signs of detumescence in the sexual situation. Alarm, of course, would increase the likelihood of continued loss of erection. If it were possible to change the meaning which detumescence has for the individual, alarm and consequent impotence might be prevented. Such an individual might be given a drug, for example, and told that it might occasionally produce momentary detumescence; or he might be assured that occasional detumescence in the sexual situation was characteristic of most normal males, or even that it was characteristic of particularly virile males. A cycle of symptoms, worry about symptoms, and intensified symptoms might be expected to occur with a number of other behaviors as well, including perhaps stuttering, extreme shyness, and excessive awkwardness in athletic situations. With each such condition, an externalization of the symptoms or a reinterpretation of the symptoms in nonpathological terms might help to break the cycle.[7]

Suggestion and Attribution

A striking aspect of the present findings is their apparent contradiction of the body of thought and research dealing with the concept of suggestion effect. On the surface, the present experiment resembles a conventional study of suggestion or placebo effects, for example, an experiment showing that administration of a "pain killer" placebo produces a reduction of pain symptoms. Yet the predictions and the obtained results of the present study were exactly opposite to those which would be indicated by suggestion theory. Subjects given a "stimulant" actually got to sleep more quickly, and subjects given a "relaxant" got to sleep less quickly. How can the present results be reconciled with the characteristic findings in the area of suggestion effects? The answer probably lies in the fact that subjects in the present experiment were quite familiar with the symptoms of insomnia. Thus, subjects had two items of information, the first supplied by the experimenter's sug

[7] The authors are indebted to Stanley Milgram for pointing out that this exacerbation cycle is probably characteristic of a number of pathologies.

gestion, and the second supplied by the subject's own past experience with insomnia symptoms: (*a*) Subjects knew that they had taken a drug which was supposed to affect insomnia symptoms, and (*b*) subjects knew that their actual experience of insomnia symptoms was about the same as it usually was. Subjects should have inferred from these facts that the arousal produced by their emotions was of a different magnitude than usual. Arousal subjects should have assumed that the magnitude of emotion-produced arousal was less than usual, and relaxation subjects should have assumed the magnitude was greater. Such an additional implication, stemming from an awareness of typical symptom level, is not characteristic of most suggestion experiments, with the following notable and very instructive exception.

Experiments designed to test the effectiveness of tranquilizers must have a control condition in which subjects are given placebos which they believe to be tranquilizers. Such a placebo control condition closely resembles the relaxation condition in the present experiment. The present line of reasoning leads to the expectation that such subjects would become more anxious upon realizing that their arousal level is still rather high, despite the "fact" that they are taking tranquilizers. Work done by Rickels and his colleagues (Rickels, Baumm, Raab, Taylor, & Moore, 1965; Rickels & Downing, 1963; Rickels, Lipman, & Raab, 1966) shows that this is often the case and indicates that the subjects who get worse are precisely those with the greatest awareness of typical symptom level.

A study by Rickels et al. (1966) shows that both prolonged experience with the anxiety state and extensive experience with tranquilizing drugs increase the likelihood that treatment with placebos will produce a worsening of anxiety state. Both experience with anxiety and experience with drugs would of course serve to give the patient a more accurate base line against which to judge the effectiveness of the placebo. Patients with a chronic, long-standing illness or patients who have previously experienced anxiety relief from drugs would readily perceive that the placebo is having little effect. If such patients infer from this fact that their symptoms are unusually severe, they should get worse. This is apparently the case. The results reported by Rickels et al. (1966) are particularly striking for patients whose experience both with drugs and with their illness is extensive. Whereas almost 80% of such patients improved when treated with tranquilizers, fewer than 30% improved on placebos. Although it is not completely clear from the presentation of the data, it seems likely that the majority of the placebo-treated patients got worse. In contrast, over 70% of the acutely ill patients with no previous experience with tranquilizers actually improved on placebo. Other work, by Rickels and Downing (1967) and Rickels et al. (1965), indicates that the higher the anxiety level of the patient, the more likely it is that his condition will worsen when placed on placebo. Patients with the highest anxiety levels would of course be expected to have the greatest awareness of their predrug symptom level. It should be particularly clear to these patients that the drug is having little effect, and they should therefore be particularly likely to infer that they are getting worse.

The findings of Rickels and his colleagues lend considerable support to the present theoretical framework. Their evidence indicates that some patients do indeed get worse when given placebos which they believe to be tranquilizers. The patients who get worse are precisely the ones who would be expected to do so in terms of the present framework. Rickels et al. (1966) professed themselves to be surprised at their findings, as well they might, since theory in the area of suggestion effects is not equipped to deal with the kinds of reversal effects which attribution theory leads us to expect. A clear implication of the present findings and framework is that clinical workers should beware the use of placebos and suggestion. Before resorting to placebos or suggestion, clinicians should probably ask themselves: "Is there a further implication of the suggestion I am making to the patient? If he fails to experience the effects I suggest, can he infer something damaging about himself?"

REFERENCES

DAVISON, G., & VALINS, S. Maintenance of self-attributed and drug-attributed behavior change. *Journal of Personality and Social Psychology,* 1969, **11**, 25–33.

HEIDER, F. *The psychology of interpersonal relations.* New York: Wiley, 1958.

KELLEY, H. Attribution theory in social psychology. *Nebraska Symposium on Motivation,* 1967, **15**, 192–240.

MONROE, L. J. Psychological and physiological differences between good and poor sleepers. *Journal of Abnormal Psychology,* 1967, **72**, 255–264.

NISBETT, R. E., & SCHACHTER, S. Cognitive manipulation of pain. *Journal of Experimental Social Psychology,* 1966, **2**, 227–236.

RICKELS, K., BAUMM, C., RAAB, E., TAYLOR, W., & MOORE, E. A psychopharmacological evaluation of chlordiazepoxide, LA-1 and placebo, carried out with anxious, neurotic medical clinic patients. *Medical Times,* 1965, **93**, 238–242.

RICKELS, K., & DOWNING, R. Drug- and placebo-treated neurotic outpatients. *Archives of General Psychiatry,* 1967, **16**, 369–372.

RICKELS, K., LIPMAN, R., & RAAB, E. Previous medication, duration of illness and placebo response. *Journal of Nervous and Mental Disease,* 1966, **142**, 548–554.

ROSS, L., RODIN, J., & ZIMBARDO, P. G. Toward an attribution therapy: The reduction of fear through induced cognitive-emotional misattribution. *Journal of Personality and Social Psychology,* 1969, **12**, 279–288.

SCHACHTER, S., & SINGER, J. E. Cognitive, social and physiological determinants of emotional state. *Psychological Review,* 1962, **69**, 379–399.

VALINS, S. Cognitive effects of false heart-rate feedback. *Journal of Personality and Social Psychology,* 1966, **4**, 400–408.

VALINS, S., & RAY, A. Effects of cognitive desensitization on avoidance behavior. *Journal of Personality and Social Psychology,* 1967, **7**, 345–350.

ZIMBARDO, P. G. The cognitive control of motivation. *Transactions of the New York Academy of Sciences,* 1966, **28**, 902–922.

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