Response Expectancy as a Determinant of Experience and Behavior

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ABSTRACT. Response expectancies, defined as expectancies of the occurrence of nonvolitional responses, have generally been ignored in theories of learning. Research on placebos, hypnosis, and fear reduction indicates that response expectancies generate corresponding subjective experiences. In many cases, the genuineness of these self-reported effects has been substantiated by corresponding changes in behavior and physiological function. The means by which response expectancies affect experience, physiology, and behavior are hypothesized to vary as a function of response mode. The generation of changes in subjective experience by corresponding response expectancies is hypothesized to be a basic psychological mechanism. Physiological effects are accounted for by the mind-body identity assumption that is common to all non-dualist philosophies of psychology. The effects of response expectancies on volitional behavior are due to the reinforcing properties of many nonvolitional responses. Classical conditioning appears to be one method by which response expectancies are acquired, but response expectancy effects that are inconsistent with a conditioning hypothesis are also documented.

Expectancy is a central construct in a number of influential theories of learning (Bolles, 1972; Rotter, 1954; Tolman, 1932). Bolles (1972) classified expectancy constructs into two types. R-S expectancies are beliefs about the relation between behavior and environmental consequences. S-S expectancies are beliefs that certain stimulus events or cues predict the occurrence of other stimulus events. The purpose of this article is to draw attention to a type of expectancy that has demonstrable effects on experience, behavior, and physiological function but that has not been considered in previous learning theories. Response expectancies are expectancies of the occurrence of nonvolitional responses, either as a function of behavior (R-R expectancies) or as a function of specific stimuli (S-R expectancies). In Rotter’s (1954) social learning theory, the occurrence of a response is hypothesized to be a function of the expectancy that the behavior will be reinforced and of the value of the expected reinforcement. However, Rotter’s theory is limited to the prediction of voluntary behavior. The response expectancy hypothesis described in this article extends social learning theory in two ways. It defines a construct that predicts the occurrence of nonvolitional responses, and it considers the effect of nonvolitional response expectancies on voluntary behavior.

Nonvolitional responses are responses that are experienced as occurring automatically, that is, without volitional effort (cf. Beck, 1976). They include emotional reactions (e.g., fear, sadness, elation), sexual arousal, conversion symptoms, pain, and so forth. Because nonvolitional responses have positive and negative reinforcement value, expectancies of their occurrence affect the probability that a person will engage in particular voluntary behaviors. For example, agoraphobics avoid a wide variety of situations because of their expectancy that entering those situations will result in the occurrence of panic attacks (Goldstein & Chambless, 1978). In addition, a considerable body of data indicates that nonvolitional responses are elicited and/or enhanced by the expectancy of their occurrence. These data are drawn from three areas of investigation: placebo effects, fear reduction, and hypnosis.

Placebo Effects

Placebos are substances that are administered in the guise of active drugs but that do not in fact have the pharmacological properties attributed to them. Placebos can reduce clinical pain, increase arousal to erotic stimuli, reduce generalized anxiety and depression, cause or reduce feelings of nausea, and induce feelings of alertness, tension, relaxation, or drowsiness. These subjective responses are frequently accompanied by corresponding physiological changes, for example, changes in pulse rate, blood pressure, galvanic skin response, gastric function, penile tumescence, skin conditions, and possibly endorphin activity in the brain (Barber, 1978; Marlatt & Rohsenow, 1980; Ross & Olson, 1982; Ross & Buckalew, 1983).

Placebo effects generally correspond to people’s knowledge or beliefs about the kind of drug they believe they are receiving, and for that reason, a causal relation between expectancy and placebo reaction has generally been assumed. However, classical conditioning has been proposed as an alternative to the idea that placebo effects are due to expectancies (Glied-
man, Gantt, & Teitelbaum, 1957; Herrnstein, 1962; Knowles, 1963; Wickramasekera, 1980). According to these formulations, administrations of active treatment constitute conditioning trials, during which treatment effects are associated with a variety of concurrent and antecedent stimuli including the pill, capsule, or syringe in which the medication was administered. Rather than being viewed as an alternative to expectancy, classical conditioning can be understood as one method by which expectancies are formed (Bolles, 1972; Reiss, 1980). However, if the data on placebo effects could be accounted for solely in terms of conditioning, then the concept of response expectancy would be redundant. Therefore, the data reviewed in this section are evaluated with respect to three questions: (a) Do classical conditioning trials contribute to the elicitation of placebo responses? (b) Are there placebo responses that cannot be accounted for in terms of conditioning? (c) What are the effects of expectancies of responses that are the opposite of that which would be predicted on the basis of previous conditioning trials?

**Conditioned Placebo Responses**

Support for the classical conditioning hypothesis has been drawn from early reports on the use of drugs (e.g., morphine) as unconditioned stimuli in conditioning experiments with laboratory animals (Pavlov, 1927/1960). More recently, “placebo effects” have been elicited in rats by means of classical conditioning procedures (Herrnstein, 1962; Ross & Schnitzer, 1963). In these studies, injections of inert solutions, or simply the insertion of a needle, produced responses similar to those produced by a previously administered drug. Pihl and Altman (1971) replicated this phenomenon using amphetamine as the unconditioned stimulus but found a “reverse” placebo effect when saline was substituted for a tranquilizer. Although chlorpromazine produced a decrease in activity from baseline activity, subsequent administration of a saline solution resulted in a significant increase over baseline activity levels. The authors concluded that “the applicability of the conditioning model may be relative to the nature of the active substance utilized” (p. 94).

Two studies have purported to demonstrate the acquisition of a placebo response in human beings by means of conditioning trials. Lang and Rand (1969) obtained increases in heart rate of at least 15 beats per minute as an unconditioned response to glyceryl trinitrate in three women. Substitution of a placebo after numerous additional conditioning trials elicited pronounced tachycardia in two of these subjects but a much lesser heart-rate increase in the third, who “stated that she knew she had been given a placebo” (p. 913). Although generalization from the behavior of only three subjects is difficult at best, the failure to obtain a clear conditioned response from the subject who suspected that a placebo had been substituted for the active drug fits a conditioned expectancy model better than it does a conditioned response model.

In the other study claiming to demonstrate a conditioned placebo response in human subjects (Knowles, 1963), the reaction time of six habitual coffee drinkers was assessed following the ingestion of regular or decaffeinated placebo coffee and also in a no-liquid control condition. Although the ingestion of both caffeinated and decaffeinated coffee produced a decrease in reaction time, the design of the study was such that conditioning could not have occurred during the course of the experiment for four of the six subjects. Subjects were assigned in pairs to three experimental groups. Each group experienced one of the following orders of treatment administration: (a) placebo, caffeine, control; (b) control, placebo, caffeine; or (c) caffeine, control, placebo. Because the placebo was administered prior to the caffeine in orders (a) and (b), these four subjects the effects of decaffeinated coffee could not have been due to a conditioning trial within the experimental period. The claim that conditioning had taken place rests on the fact that all six subjects were habitual coffee drinkers. However, the assumption that similar effects would not have occurred in subjects who were not coffee drinkers was not tested.

Consistent with the results of Pihl and Altman’s (1971) study on laboratory rats, the effects of conditioning trials on human response to a placebo appear to vary as a function of the drug used as an unconditioned stimulus. Prior experience with the active drug enhances the effects of placebo marijuana (Carlin, Post, Cornelis, Bakker, & Halpern, 1974; Jones, 1971) and placebo analgesia (Batterman & Lower, 1968). Also, pain relief with placebo analgesia is greater when it is administered after a more potent drug than it is following a less potent drug (Kantor, Sunshine, Laska, Meisner, & Hopper, 1966). In contrast, prior experience inhibits the effects of placebo tranquilizers (Meath, Feldberg, Rosenthal, & Frank, 1956; Rickels, Lipman, & Raab, 1966; Segal & Shapiro, 1959; Zukin, Arnold, & Kessler, 1959). Furthermore, with placebo tranquilizers, there is an inverse relation between the strength of the presumed unconditioned stimulus (UCS) and the magnitude of the presumed conditioned response (CR, Rickels et al., 1966). These data support the hypothesis that conditioning trials enhance the effects of placebo marijuana and analgesia, but they disconfirm con-
conditioning hypotheses as explanations of the effects of placebo tranquilizers.

Unconditioned Placebo Effects

In the previous section, it was assumed that due to stimulus generalization from experience with other medications, some placebo effects might be found in subjects who had not had prior exposure to the specific medication under study. In this section, studies are considered in which the placebo response is either unrelated or in a direction opposite to that of the presumed UCS. As noted by Wickramasekera (1980), "there can be no CR if there were no UCS (active ingredients)" (p. 14). Therefore, placebo responses that are unrelated to the pharmacological effects of a drug cannot be accounted for by conditioning models.

Although the design of Knowles's (1963) study precluded the possibility of demonstrating that the obtained effect was due to conditioning, it did provide evidence that the effect was not due to conditioning. Mean reaction times in the caffeine and placebo conditions were shortest immediately after ingestion and increased with each subsequent assessment. At 60 minutes after ingestion, reaction times for all three conditions were virtually identical. At 90 minutes, reaction times under caffeine and placebo conditions were slower than under control conditions. However, Knowles cited earlier data indicating that effective blood levels of caffeine are not likely to be reached for at least one hour following oral ingestion. The fact that caffeine did not result in shorter reaction times one hour or more after administration suggests that it is not an unconditioned stimulus for decreased reaction time. If this is true, the decreased reaction times observed immediately following ingestion of the caffeinated and decaffeinated coffee could not have been due to conditioning. Thus, Knowles demonstrated a placebo effect in humans in the absence of the conditions that are necessary for classical conditioning to occur.

A similar conclusion can be drawn from a case report of a placebo-induced conversion reaction (Levy & Jankovic, 1983). Phenytoin (Dilantin) produced a variety of idiosyncratic reactions in a patient, including speech distortions; visual, auditory, and tactile hallucinations; and muscle spasms. Neuropsychological testing following the administration of saline in the guise of phenytoin revealed a number of dose-dependent effects, including loss of consciousness, unresponsiveness to intense pain stimuli, disappearance of corneal reflexes, and convulsive seizures. However, administration of phenytoin in the guise of a neutral substance failed to elicit any abnormalities. Because phenytoin did not elicit the placebo-induced responses when it was given in disguised form, it could not have served as an unconditioned stimulus for those responses.

Research on expectancy effects has resulted in the development of the balanced placebo design (Marlatt & Rohsenow, 1980). The balanced placebo design yields a $2 \times 2$ matrix comprising the following four conditions in which subjects are (a) told they will get a drug/receive drug, (b) told they will get a drug/receive placebo, (c) told there will be no drug/receive drug, (d) told there will be no drug/receive no drug. This design allows independent evaluation of pharmacological and expectancy effects and has led to the discovery of placebo alcohol effects that are unrelated to or inconsistent with its pharmacological effects. For example, the pharmacological effect of alcohol is to decrease sexual arousal (Farkas & Rosen, 1976; Rubin & Henson, 1976). However, consistent with common expectations (Brown, Goldman, Inn, & Anderson, 1980), the belief that one has consumed alcohol results in increased sexual arousal to erotic stimuli (Bridell et al., 1978; Wilson & Lawson, 1976a, 1976b). Placebo alcohol also produces increased aggressive behavior (Lang, Goecckner, Adesso, & Marlatt, 1975) and increased craving for and consumption of alcohol (Engle & Williams, 1972; Marlatt, Demming, & Reid, 1973). Because the administration of alcohol in disguised form failed to produce these effects, they cannot be accounted for by a conditioning hypothesis.

In anxiety-eliciting heterosexual situations, the belief that one has consumed alcohol is associated with decreased heart rate in males (Wilson & Abrams, 1977) and increased heart rate in females (Abrams & Wilson, 1979). It seems highly unlikely that alcohol as an unconditioned stimulus would have opposite pharmacological effects on male and female heart rates.

Marlatt and Rohsenow (1980) maintain that these placebo phenomena are consistent with a conditioning model, suggesting that the "conditioned response is a compensatory reaction, one that is in the opposite direction to the unconditioned response." (p. 192). Although this is consistent with the data on sexual arousal, it is inconsistent with the data on alcohol craving, aggression, and social anxiety, in which cases no unconditioned responses were observed. It is also inconsistent with most placebo effects, which correspond to the pharmacological effects of the drug under investigation. Thus, if conditioning accounts for placebo alcohol effects on sexual arousal, it cannot account for other placebo effects. Conversely, if most placebo effects are due to conditioning, then the effect of placebo alcohol on sexual arousal is an anomaly.¹

¹ Since writing this article, Siegel's (1983) work on classically conditioned drug tolerance has come to my attention. Siegel and his colleagues have convincingly demonstrated that classical conditioning produces compensatory conditioned responses (i.e., a CR that is opposite in direction to the UCR) for some responses (those for which tolerance develops) to some drugs. This phenomenon
Placebo and drug effects are widely assumed to be additive. For example, Lasagna, Mosteller, von Felseringer, and Beecher (1954) found that morphine elicited a mean pain relief rating of 95% among patients who consistently reported relief from placebos (placebo reactors) as compared to 54% among consistent nonreactors to placebos. The 41% difference between placebo reactors and placebo nonreactors in pain relief following morphine administration is presumably due to the placebo effect of having been given medication. In other words, “the response to any UCS will include two components. The first component will be a CR (placebo response) and the second component a UCR (e.g., specific effect of a drug)” (Wickramasekera, 1980, p. 8). On the basis of his classical conditioning model, Wickramasekera predicted that, as a fractional component of the UCR, the placebo component ought to be weaker than the pharmacological effect. I have located three studies in which the placebo and pharmacological components of stimulant or depressant medications have been experimentally isolated (Frankenhaeuser, Post, Haggdahl, & Wrangsjoe, 1964; Lyerly, Ross, Krugman, & Clyde, 1964; Ross, Krugman, Lyerly, & Clyde, 1962). The results of these three studies reveal expectancy effects that are of a magnitude equal to or greater than the pure pharmacological effects of stimulant and tranquilizing drugs. Because these placebo effects are more than fractional components of the corresponding pharmacological effects, they constitute disconfirmatory evidence for Wickramasekera’s conditioning model.

**Summary**

Classical conditioning procedures can produce a placebo response in animals with some drugs but produce a reverse placebo response with tranquilizers. Similarly, studies with human subjects have reported results that were consistent with conditioning theory when analgesia or marijuana was the unconditioned stimulus but have resulted in disconfirmatory results with tranquilizers. Although placebo responses generally mimic the effects of the active drug, when people have response expectancies that are contrary to the pharmacological effects of the active drug, their response to placebo is consistent with their expectations rather than with the drug’s pharmacological effects. Finally, the placebo component of drug administration can be as powerful as or more powerful than the pharmacological component of drug effects. These data suggest that classical conditioning may be one method by which response expectancies are formed but that the effects of conditioning trials are mediated by expectancy.

**Expectancy and Fear**

People fear and avoid situations in which they expect aversive consequences (Beck, 1976; Rotter, 1954). However, expectations of harm cannot account for the degree of fear that is experienced by phobic individuals, because fear is defined as phobic only if it is “recognized by the individual as excessive or unreasonable in proportion to the actual dangerousness of the object, activity or situation” (American Psychiatric Association, 1980, p. 225). A number of investigators have proposed that the expectation of intense fear is a cause of this excessive fear and avoidance (Goldstein & Chambless, 1978; Kirsch, Tenen, Wickless, Saccone, & Cody, 1983; Reiss, 1980, Reiss & McNally, in press). Because the experience of intense fear is extremely aversive, the expectancy of its occurrence provides strong motivation for avoidance. For example, people with severe snake phobias avoid looking at pictures of snakes because of the unpleasant feelings that the pictures provoke. Similarly, agoraphobics are less afraid of supermarkets, shopping malls, and all the other situations that they avoid than they are of the panic attacks they fear might occur.

Evidence of the role of fear expectancies in phobias is provided by therapy outcome studies in which “placebo” control procedures have been employed. These procedures are designed to induce an expectancy of improvement, and it is generally assumed that their effectiveness is due to the same underlying mechanisms governing the effectiveness of placebo drugs. However, attempts at defining the term placebo in a way that is appropriate to both pharmacological and psychological contexts have not been successful (Crittelli & Neumann, 1984; Kirsch, 1978; Kirsch, 1985a). Therefore, “placebo” psychotherapy procedures will be termed expectancy modification procedures. Expectancy modification is a specific active psychological mechanism that can be an effective therapeutic component of psychological treatment.

**Expectancy Modification and Fear Reduction**

The seminal study in which an expectancy modification procedure was used to control for “placebo” effects in the evaluation of a psychological treatment was Paul’s (1966) research on systematic desensi-
zation. Paul's expectancy modification procedure was more effective than a no-treatment control, indicating that expectancy modification can produce a significant reduction in anxiety. Although most studies comparing desensitization to expectancy modification procedures have reported greater fear reduction for desensitization (Kazdin & Wilcoxon, 1976), Borkovec and Nau (1972) demonstrated that many expectancy modification procedures are perceived as less credible than systematic desensitization. Independently, McReynolds, Barnes, Brooks, & Rehagen (1973) demonstrated that a more compelling expectancy modification procedure was as effective as desensitization and more effective than Paul's (1966) "attention placebo" in reducing fear of snakes.

To date, at least 11 studies have shown that credible expectancy modification procedures are capable of producing as great a reduction in fear as that produced by systematic desensitization (Gatchel, Hatch, Maynard, Turns, & Taunton-Blackwood, 1979; Kirsch & Henry, 1977; Kirsch et al., 1983; Lick, 1975; Marcia, Rubin, & Efran, 1969; McGlynn, 1971; McGlynn, Gaynor, & Puhr, 1972; McGlynn, Reynolds, & Linder, 1971; McReynolds et al., 1973; Slutsky & Allen, 1978; Tori & Worell, 1973). In addition, Gelder et al. (1973) found an expectancy modification procedure to be as effective as desensitization in treating animal and tissue damage phobias. Although desensitization was more effective than expectancy modification in the treatment of agoraphobia and social fear, Gelder et al.'s expectancy modification treatment failed to elicit outcome expectancies comparable to those elicited by desensitization. Expectancy modification treatments also have been reported to be as effective as cue-controlled relaxation, relaxation as an active coping skill, and heart-rate biofeedback (Gatchel et al., 1979; Gatchel, Hatch, Watson, Smith, & Gaas, 1977; Russel & Lent, 1982).

Many of the causal mechanisms that have been hypothesized as explanations of the effects of desensitization have been based on classical conditioning models. These hypothesized mechanisms include reciprocal inhibition (Wolpe, 1958), counterconditioning (Davison, 1968) and extinction (O'Leary & Wilson, 1975). In a test of conditioning hypotheses, Kirsch and Henry (1977) compared the effects of systematic desensitization and two credible expectancy modification procedures. One of these procedures was specifically designed to rule out conditioning hypotheses. In an "operant desensitization" condition, visualizations of anxiety-related scenes were paired with painful electric shocks, which subjects were told would "punish the anxiety." Both expectancy modification procedures were as effective as standard systematic desensitization in reducing public-speaking fear. Because aversive stimuli are assumed to be the unconditioned stimuli leading to the acquisition of fear as a conditioned response, the substantial degree of fear reduction produced by "operant desensitization" cannot be accounted for by extinction or counterconditioning. Furthermore, because the addition of the electric shock was the only procedural difference between "operant" and traditional desensitization, it is reasonable to suspect that the effects of the two procedures were due to a common causal mechanism. Substantial correlations between pretreatment ratings of treatment credibility and treatment outcome measures suggest that expectancy modification was the common causal agent.

In a subsequent study (Kirsch et al., 1983), 46 snake-phobic adults were assigned to either systematic desensitization, a credible expectancy modification treatment, or a delayed-treatment control condition. Anxiety expectancies were assessed by having subjects rate the degree of fear they thought they would experience at each step of a behavioral approach test. These ratings accounted for more than 50% of the variance in pre- and posttreatment approach behavior. In addition, immediately after the treatment was described, but before it was initiated, subjects were asked to predict their posttreatment fear. Within-cell correlations indicated that these predictions were especially accurate for desensitization subjects, accounting for about 60% of the variance in posttreatment approach behavior. These data can be interpreted most parsimoniously as indicating that systematic desensitization is an effective procedure for modifying fear expectancies, which in turn alter experienced fear.

**Response Expectancy and Self-Efficacy**

As defined by Bandura (1977), self-efficacy is the belief that one is able to execute a behavior upon which reinforcement is contingent. Defined in this manner, self-efficacy is conceptually distinct from response expectancy, which refers to the occurrence of nonvolitional responses. Bandura has hypothesized that alterations in efficacy judgments are central to the effects of all psychotherapies and has supported this contention with reports of high correlations between responses to snake-approach self-efficacy questionnaires and posttreatment approach to a previously feared snake. However, recent data raise serious questions about the construct validity of snake-approach self-efficacy questionnaires.

Kirsch (1982) asked snake-fearful students to complete two self-efficacy questionnaires, one pertaining to approaching a live snake, the other to tossing a wad of paper into a basket from various distances. Subjects were then asked if increasing levels of hypothetical incentives would enable them to perform snake-approach and paper-toss tasks that they had previously rated as beyond their ability. Whereas hypothetical incentives as low as $20 were sufficient to alter most subjects' efficacy judgments for approach-
ing a snake, such incentives as one million dollars and saving one's own life or the life of another failed to alter most subjects' efficacy judgments for the paper-toss task. When asked to explain this discrepancy, subjects expressed certainty of their ability to approach a snake if it were necessary to do so. Kirsch (1982) concluded that efficacy questionnaires measured subjects' willingness to approach a snake given their expected fear and the incentives for approach.

In another study (Kirsch et al., 1983), efficacy ratings and fear response expectancies had virtually identical power in predicting behavioral approach and ratings of experienced fear. In addition, the two expectancy measures were highly correlated during pretreatment \( r = -.75 \) and posttreatment \( r = -.90 \) assessment periods. When two measures are that highly correlated and have virtually identical predictive power, it is reasonable to suspect that they are measuring the same construct. Examination of the items on the two scales suggests that both measure expected fear. The anxiety expectancy scale asks people to predict their level of fear at each step of the approach test. Efficacy scales ask them to predict the number of steps they will accomplish. If behavior on an approach test is a measure of fear, then predicted approach (i.e., efficacy ratings) is a measure of expected fear.

Although self-efficacy and response expectancy are conceptually distinct, when measured in fear-provoking situations there is considerable overlap between them. This is because fear-related efficacy questionnaires do not measure people's self-efficacy. Instead, they measure people's willingness to approach a feared stimulus. Social learning theory predicts volitional behavior as a function of expected reinforcement. In the absence of sufficient incentive, people are unwilling to engage in behavior that they believe will lead to intense fear. Thus, when external consequences are held constant, high correlations are obtained between fear response expectancies and supposed measures of self-efficacy (cf. Kirsch, 1985b).

**Summary**

Subjective and behavioral manifestations of specific fears can be predicted by procedures designed to modify response expectancies. More credible expectancy modification procedures produce greater alterations in expectancy and fear, and procedures that are as credible as systematic desensitization are generally as effective in reducing fear. The effects of these procedures cannot be accounted for by classical conditioning but are highly correlated with changes in response expectancies. These data support Reiss's (1980) hypothesis that the experience of fear is in part a function of the expectancy of its occurrence and the hypothesis that fear reduction can be brought about by altering the expectancy of its occurrence. In addition, Emmelkamp (1982) has hypothesized that the effects of in vivo exposure treatments are mediated by expectancy. According to Emmelkamp, the self-observed habituation that occurs during in vivo exposure is interpreted as evidence of improvement, thereby altering subjects' expectancies.

**Hypnosis**

There are three kinds of cognitions that ought to affect response expectancies in hypnotic situations: (a) perceptions of the situation as more or less appropriate for the occurrence of hypnotic responses; (b) perceptions of the response as being appropriate to the role of a hypnotized subject (Sarbin, 1950; Sarbin & Coe, 1972), and (c) judgments of one's hypnotizability. If response expectancies affect hypnotic responses, then hypnotic responses ought to be affected by each of these variables. More specifically, the probability of occurrence of a particular response ought to vary as a function of the degree to which subjects perceive the situation as hypnotic, perceive the response as characteristic of the experience and behavior of good hypnotic subjects, and judge themselves to be good subjects (cf. Barber, Spanos, & Chaves, 1974).

**Situational Perceptions**

In the 18th century, a variety of procedures were used by mesmerists to induce "crises." These included stroking or making "passes" over the patient bare-handed or with a magnet, having patients sit around a bucket of water containing iron filings, having the subject stand by a "magnetized" tree or drink "magnetized" water, and having the person sit with his or (more frequently) her knees pressed between the thighs of the mesmerist who applied pressure to the hypochondria (the area between the rib cage and the navel) or the ovarium. In modern placebo terminology, 18th-century induction procedures were nontpecific in the sense that no particular components were necessary for successful induction of a crisis. Experiments conducted by the French Royal Commission established to investigate mesmerism concluded that belief in the appropriateness of the situation was sufficient to elicit crises in susceptible subjects. For example, crises could be elicited by falsely informing people that they were being magnetized through a closed door (Franklin et al., 1784/1965; Franklin et al., 1785/1970).

Although there is still variety in induction procedures, most hypnotic inductions involve suggestions for deep relaxation. However, equivalent degrees of response enhancement have been produced by task motivational instructions (Barber, 1969), brief training in the use of imaginative strategies (Council, Kirsch, Vickery, & Carlson, 1983; Katz, 1978, 1979; Vickery, Kirsch, Council, & Sirkin, 1985), and a variety of expectancy modification procedures (Council et al., 1983; Glass & Barber, 1961; Wilson, 1967). The only
component that is common to these methods of enhancing suggestibility is the credibility of the enhancement procedure. As had the French Royal Commission two centuries earlier, Sheehan & Perry (1976) have concluded that “it is not the procedural conditions per se that are important but whether or not the subject perceives them as part of a context that is ‘appropriate’ for displaying hypnotic behavior” (p. 72).

**Role Perceptions**

During the 18th and early 19th centuries, the behavior of magnetized subjects was as variable as the induction rituals that were employed to bring that behavior about. Some people coughed; others laughed. Some markedly increased their breath rates; others became very relaxed. Feelings of warmth and of cold were reported, as were sensations of pain or numbness. Sensitivity to stimulation might be heightened or lowered, sometimes heightened and then lowered. Many, but by no means all, magnetized subjects exhibited convulsive seizures (Deleuze, 1825/1965; d’Eslon, 1784/1965; Franklin et al., 1785/1970).

Just as hypnotic procedures have become standardized over the course of time, so too has the role of hypnotic subject. The hypnotized subject typically sits passively with eyes closed, shows little or no spontaneous speech or movement, and speaks slowly and softly in response to questions. Sarbin (1950) hypothesized that the behavior of hypnotized subjects is in part a function of their expectations of how a good subject ought to behave. Subsequent data support this hypothesis. For example, people who are informed that hypnotized subjects exhibit dominant arm catalepsy are likely to do so when hypnotized (Orne, 1959; Sheehan, 1971); being informed that spontaneous amnesia is a characteristic of hypnosis substantially increases the likelihood of its occurrence (Young & Cooper, 1972); and information about the difficulty of responding to suggestions affects subsequent responses (Barber & Calverley, 1964; Botto, Fisher, & Soucy, 1977; Klinger, 1970).

Role perceptions also affect self-reports of the experience of “trance.” Henry (1985) constructed a questionnaire consisting of opposing subjective experiences that might be associated with hypnosis. The criterion for item inclusion was an endorsement rate of not more than 60% in either direction by subjects who had not previously experienced hypnosis. In general, subjects’ experience of trance matched their preconceptions. Depending on their preconceived notions, good hypnotic subjects experienced “trance” as a state in which time passed either more slowly or more quickly than usual, logical thought was either more or less difficult than normal, the hypnotist’s voice sounded closer or farther away than before, sounds were experienced as muffled or more clear than usual, the subject felt more or less involved than usual, and so on.

**Expectancy Ratings and Responsiveness**

Self-reported expectancies of naive subjects typically account for about 10% of the variance in hypnotizability (Melei & Hilgard, 1964; Saavedra & Miller, 1983; Shor, 1971; Wadden, 1983). However, correlations between expectancy and response account for approximately 25% of the variance when trance induction is replaced with brief training in goal-directed imagining (Kirsch, Council, & Vickery, 1984). Similarly, Barber and Calverley (1969) reported that expectancy was more highly correlated with responsiveness when subjects were instructed to place themselves in hypnosis than when a formal trance induction was used.

Council and Kirsch (1983) assessed response expectancies prior to administering a traditional trance induction and again after the induction but prior to administration of dependent variable suggestions. They also obtained subjective reports of trance depth immediately after the trance induction. Responsiveness to suggestions was significantly correlated with both pre- and postinduction expectancies, but the correlation between responsiveness and postinduction expectancies was significantly greater than that between responsiveness and preinduction expectancies. Although trance depth reports were also correlated with responsiveness, regression analyses revealed that only postinduction response expectancies contributed unique variance to the prediction of responsivity. A path analysis supported the hypothesis that hypnotic inductions enhance responsiveness by altering response expectancies.

**Modification of Hypnotic Response Expectancies**

Procedures designed to modify subjects’ expectancies of hypnotic responding have been shown to affect responses to suggestions. For example, Vickery and Kirsch (1985) told subjects that repeated testing resulted in increased, decreased, or unchanged levels of hypnotic response, and found that responsiveness varied as a function of that information. Similarly, manipulating expectancies by providing bogus feedback from “personality scales” affects responsiveness (Gregory & Diamond, 1973; Saavedra & Miller, 1983). Wilson (1967) increased subjects’ response expectancies by providing them with experiences designed to convince them that they were experiencing a variety of imagined perceptual phenomena. For example, while suggesting that subjects imagine that the room was red, Wilson imparted a red tinge to the room by means of a small hidden light bulb. These subjects were later tested for responsiveness to standard hypnotic suggestions, without mention of “hypnosis” or the use of a hypnotic induction procedure. Not only
did the experimental subjects achieve significantly higher scores than control subjects, but their scores were such that none would be classified as “low hypnotizables” as that term is typically defined (e.g., Weitzenhoffer & Hilgard, 1962).

**Imaginative Involvement and Response Expectancy**

The idea that hypnotic experiences are a function of imagination dates back to the 18th century (Franklin et al., 1785/1970). The modern counterpart to this notion, the concept of absorption in imaginative activities (Tellegen & Atkinson, 1974), has provided a point of convergence for state and nonstate theorists (Barber et al., 1974; E. Hilgard, 1965; J. Hilgard, 1979; Sarbin & Coe, 1972). However, recent data suggest that the relation between absorption and hypnotic responsiveness is mediated by expectancy. For example, Council et al. (1983) reported that although Tellegen and Atkinson’s (1974) absorption scale predicted subjects’ responses to hypnotic suggestions, it was more highly correlated with response expectancies, which in turn were even more highly correlated with responsiveness. Furthermore, when variance associated with expectancy was statistically controlled, the relation between absorption and hypnotizability was nonsignificant.

As noted above, bogus feedback from personality tests can alter subjects’ responsiveness to hypnotic suggestions. Because items on the absorption scale are face obvious, Council and Kirsch (1983) hypothesized that administering it in the context of an experiment on hypnosis might be a way of indirectly providing subjects with bogus feedback. Thus, the association between absorption and responsiveness might be an artifact of the scale’s effect on subjects’ response expectancies. In a test of that hypothesis, they administered the scale to 64 subjects in the context of a hypnosis experiment and to an additional 64 subjects in a context that was unrelated to hypnosis. Regression analyses revealed a significant interaction between scores on the absorption scale and the context in which the scale was administered. Absorption was significantly correlated with responsibility and expectancy only when the scale was administered in a context that was clearly associated with a subsequent hypnotic experience.

Barber et al. (1974) hypothesized that positive attitudes, motivations, and expectancies lead subjects to engage in goal-directed fantasies, which in turn generate suggested behaviors and experiences. However, when instructed to do so, good hypnotic subjects are able to respond to suggestions while imagining conflicting events (Spanos, Weekes, & de Groh, 1984; Zamansky, 1977). In addition, Council and Kirsch (1984) demonstrated that the effects of intentionally imagining along with hypnotic suggestions (parallel imagery) and the effects of imagining events that were incompatible with suggestions (counter imagery) varied as a function of the instructional set that was provided to subjects. Parallel imagery enhanced responding among subjects who were provided with a convincing rationale indicating that this would be its effect, but it inhibited responsiveness among subjects given a negative instructional set. Similarly, counter imagery inhibited responsiveness only among subjects who were given a response-inhibition rationale.

High test–retest correlations across a wide variety of hypnotic inductions, training procedures, and measures of responsiveness have led to the conclusion that hypnotizability is a relatively stable trait (Perry, 1977). However, having given subjects information designed to elicit conflicting expectancies, Council and Kirsch (1984) obtained test–retest correlations accounting for only 10% of the variance in hypnotic responsiveness. In contrast, their expectancy manipulation accounted for approximately 46% of the variance, suggesting that previously reported correlations reflected stable response expectancies rather than the presence of an underlying personality trait (cf. Spanos, 1982).

**Summary**

The data reviewed above reveal that “hypnosis” refers to a wide variety of procedures by means of which many people’s response expectancies can be temporarily altered. Both the efficacy of the procedure and the nature of the response depend primarily on subjects’ beliefs and expectancies. Similarly, the degree of responsiveness varies as a function of expectancy. Although hypnotizability has been viewed as a relatively stable trait, in at least some situations conflicting expectancy instructions can account for more variance than “trait hypnotizability,” suggesting that high test–retest correlations on measures of hypnotic response may be due to the presence of relatively stable response expectancies. It is possible that, with sufficiently strong response expectancies, all individuals would show high levels of hypnotic response.

**Causal Mechanisms**

A number of intervening variables have been hypothesized to mediate expectancy effects on particular responses. For example, Frank (1973) proposed that placebos engender feelings of hopefulness associated with faith in improvement, feelings that in turn reduce generalized depression and anxiety in psychiatric outpatients. Hopefulness has also been hypothesized to promote physical healing (Frank, 1973), possibly by enhancing immunologic function (Simonton, Matthews-Simonton, & Creighton, 1978). Similarly, placebo-induced pain reduction may be mediated by decreased anxiety (Evans, 1974) or the release of endorphins in the brain (Fields & Levine, 1981).

Each of these hypothesized mechanisms is spe-
specific to particular types of response. Although some of them may be related to other response expectancy effects (e.g., anxiety reduction may be an important factor in hypnotic pain reduction), the wide range of nonvolitional responses that are affected by expectancy and the similarly wide range of situations in which these effects occur suggest the heuristic value of a more general response expectancy hypothesis. For example, endorphin activity does not appear to be related to hypnotic pain reduction (Spiegel & Albert, 1983). Furthermore, because one cannot feel hopeful and hopeless at the same time, it is difficult to imagine how the hopefulness hypothesis might account for placebo-induced side effects in patients who also experience positive effects from the same placebo. Nor can it easily explain the opposing effects of stimulant and tranquilizer placebos on healthy, nonpatient volunteers for a drug experiment (e.g., Frankenhaeuser, Jarpe, Svan, & Wrangsjö, 1963). Because these subjects are not being treated for a disorder, the concept of faith in improvement is not applicable. Similarly, the fact that simple instructions about the effects of repeated testing produce opposing alterations in the probability of responding to standard hypnotic suggestions (e.g., hand lowering, taste hallucination, posthypnotic amnesia) cannot be readily explained by any of these hypothesized mediating mechanisms.

The response expectancy hypothesis is not intended to replace the mechanisms described above. There is ample evidence that hopefulness produces symptomatic relief in psychiatric outpatients (e.g., Frank, Nash, Stone, & Imber, 1963; Shapiro, Struening, & Shapiro, 1980), and although the evidence is scant (Hall, 1984; Jemmott & Locke, 1984), the hypothesis that positive emotional states enhance immunologic function is intriguing and merits further investigation. Similarly, data linking placebo pain relief to endorphin release are extremely important, although they pose the question of how placebos produce an increase in endorphin activity. Endorphin release may be more profitably viewed as one of the effects of placebos, rather than as the cause of placebo effects. The purpose of the response-expectancy hypothesis is to supplement previous hypotheses in order to account for expectancy effects on a wider range of responses.

Response expectancies affect subjective experience, overt behavior, and physiological function. The mechanisms by which these effects are produced can best be understood by separate consideration of each of these response systems. In brief, I propose that (a) there is an unmediated causal relation between response expectancy and subsequent experience, (b) some of the physiological changes that are associated with response expectancies can be accounted for by the *identity assumption* that is logically required of all nondualist philosophies of mind, and (c) volitional behavior is affected by response expectancies because of the reinforcement value of nonvolitional responses.

**Response Expectancy and Subjective Experience**

In critiquing the introspective methods of prebehaviorist experimental psychology, Dodge (1912) introduced the idea that an expectancy can produce a corresponding conscious experience. Since that time, response expectancy effects have been viewed as artifacts to be controlled for in psychological research. However, given the data reviewed above, it seems reasonable to view those effects as evidence of a basic psychological mechanism that is capable of affecting a wide range of responses. As a basic psychological mechanism, response expectancies generate corresponding subjective experiences without additional intervening mechanisms. In this respect, the response expectancy hypothesis is not different from other hypothesized causal relations between cognitions and subsequent experience. Beck (1976), for example, proposed that sadness is caused by thoughts of loss, anger by the belief that one has been wronged, fear by expected harm or loss, and anticipatory excitement by expectations of positive reinforcement. The response expectancy hypothesis asserts that the expectancy of a subjective experience has a direct (i.e., unmediated) corresponding effect on experience.

**Response Expectancy and Physiological Function**

Part of the difficulty in understanding the apparent causal relation between response expectancies and physiological function results from the assumption of mind–body dualism that is implicit in the formulation of the question. All monist solutions to the mind–body issue involve some form of identity thesis. For example, double-aspect or neutral monist positions view mental and physical descriptions as different aspects of a single underlying reality. Similarly, materialist philosophies hold that mental events do not cause physical events; rather, they are identical to physical events in the same way that water is identical to H₂O. Water does not cause nor is it caused by H₂O; it is H₂O. The common factor in these views is the implication of physical counterparts to any subjectively described event.

The only logical alternative to the identity assumption is to allow the existence of mental events without physical correlates, which implies a dualist mind–body position. However, if cognitions do not correspond to physical processes, either they do not affect behavior or physiological function, which is inconsistent with considerable data, or the law of conservation of energy is invalid. In contrast, the identity thesis implies an isomorphic relation between mental and physical causality. If mental events correspond to physical events, then a statement that one mental...
event (e.g., expectancy) causes another mental state (e.g., fear) is equivalent to a statement that the physiological state corresponding to the first mental state causes the physiological state corresponding to the second mental state. In other words, physiological changes accompany changes in mental states because any particular mental state corresponds to some particular physical state. Physiological changes are not generated by the expectancy of their occurrence but by the expectancy of corresponding subjective experiences. Changes in pulse rate, for example, are not brought about by the expectancy of their occurrence but by expectancies of alterations in subjectively experienced arousal levels.

The identity thesis does not imply totally dissimilar patterns of physiological response for different psychological states. For example, increased arousal is associated with a variety of affective states (Reisenzein, 1983; Schacter & Singer, 1962). Conversely, because a single subjective term (e.g., anxiety) can be used to describe a variety of experiences, different instances in which the same label is used may correspond to different states of the nervous system (e.g., Tyer & Lader, 1974). The identity thesis merely asserts that any particular instance of a subjective experience is associated with some specific state of the organism. The principle of causal isomorphism adds that any pattern of causal relations between psychological variables implies a similar causal relation among corresponding physiological variables.

At the psychological level, the problem is to establish the causal relations between specific cognitive states (e.g., irrational thoughts, images, response expectancies) and subsequent subjective experiences (e.g., alterations in mood or pain perception). At the physiological level, the problem is to identify the central and peripheral concomitants of each of these states. Although the physiological concomitants of expectancies are not known, some of the physiological concomitants of resulting experiences have been identified. Expectancy-induced changes in fear and subjective arousal levels are frequently accompanied by corresponding changes in pulse or heart rate, skin conductance, and blood pressure (Beiman, 1976; Blackwell, Bloomfield, & Buncher, 1972; Brodeur, 1965; Frankenhaeuser, Jarpe, Svan, & Wrangsjö, 1963; Frankenhaeuser, Post, Hagdahl, & Wrangsjöe, 1964; Grayson & Borkovec, 1978; Kirsch & Henry, 1979; Lick, 1975). Expectancy-induced changes in male sexual arousal are accompanied by increases in penile tumescence (Briddell et al., 1978; Wilson & Lawson, 1976a). Expectancy-induced pain reduction may be accompanied by increased endorphin activity in the brain (Fields & Levine, 1981) and by reduced respiratory and electromyographic responses (Barber & Hahn, 1962). However, the most difficult problem in identifying complete causal sequences involves the discovery of central state correlates of cognitions and other subjective experiences.

Response Expectancy and Volitional Behavior

According to Rotter (1954), the probability of a behavioral response is a function of the expectancy that the response will lead to reinforcement and of the subjective value of the reinforcement. Because many nonvolitional experiences (e.g., pain, fear, sexual arousal) have obvious positive or negative reinforcement value, they are among the reinforcements determining the probability of behavior upon which they are believed to be contingent. Thus, alcohol consumption is partially determined by expectancies that it will lead to enhanced social and physical pleasure, enhanced sexual performance, increased confidence, shorter sleep-onset latencies, pain reduction, and reduced tension (Brown et al., 1980).

Reiss and McNally's (in press) expectancy model of phobias is generally consistent with Rotter's social learning theory and with the response expectancy hypothesis proposed in this article. According to their model, phobic avoidance can be predicted as a function of expected harm (a reinforcement expectancy), expected fear (a response expectancy), and the degree to which fear is experienced as aversive (i.e., the reinforcement value of the anticipated fear). Similarly, response expectancies and their reinforcement values affect the likelihood of initiating sexual contact, ingesting medication or other drugs, volunteering to be an hypnotic subject, and so forth.

Response Expectancy and Social Learning

By way of summary, the following propositions integrate the response expectancy hypothesis presented in this article with some of the hypotheses proposed by other cognitive theorists.

1. The probability of a volitional response is a function of the expectancy that the response will lead to reinforcement and of the value of the expected reinforcement (Rotter, 1954). These expectancies have been termed R–S expectancies by Bolles (1972). Operant conditioning procedures are one means by which R–S expectancies are acquired.

2. Certain stimuli have primary or unlearned reinforcement value (Rotter, 1982). In addition, stimuli can acquire reinforcement value through their association with other reinforcing stimuli. The secondary reinforcement value of a stimulus is determined by the expectancy that it will lead to other reinforcements (Rotter, 1954). Classical conditioning is one means by which these S–S expectancies are formed (Bolles, 1972).

3. From propositions 1 and 2, it can be deduced that the probability of a volitional response is determined by the sum total of expected reinforcements.
and of their primary reinforcement values (Kirsch, 1985b).

4. Nonvolitional responses can be elicited by unconditioned stimuli. In addition, some cognitions (e.g., perceptions of loss and of having been wronged, and expectancies of harm and of positive reinforce-
ment) function as unconditioned stimuli for specific emotional states (sadness, anger, fear, and anticipatory excitement; Beck, 1976).

5. Nonvolitional responses can also be elicited by the expectancy of their occurrence, either as a function of specific stimuli (S–R expectancies) or as a function of voluntary responses (R–R expectancies). Classical conditioning is one means by which response expectancies are acquired (Reiss, 1980). In addition, verbal persuasion, attributional processes, modeling, and self-observation are involved in the acquisition and modification of response expectancies.

6. The effects of unconditioned stimuli and re-
response expectancies are additive. Therefore, response expectancies can enhance, inhibit, or reverse the ef-
fects of unconditioned stimuli (Frankenhaeuser, Post, 
Hagdahl, & Wrangsjoe, 1964; Lyerly et al., 1964; Ross et al., 1962; Wolf, 1950).

7. The probability of occurrence of a nonvol-
itional response varies directly with the strength of the expectancy of its occurrence and inversely with the magnitude or difficulty of the expected response. Therefore, a weak expectancy for a large change in subjective response is likely to be disconfirmed, whereas a strong expectancy for a small change is likely to be confirmed, thereby strengthening the expectancy and initiating a cycle for continued change.

8. Nonvolitional responses have reinforcement value. The reinforcement value of some nonvolitional responses may be due to expectancies of further sub-
sequent reinforcement. Other nonvolitional responses (e.g., the experience of pain, sadness, or fear) function as primary reinforcers, in that they have reinforcement value that is independent of expectancies of subsequent additional reinforcement. It should be noted, however, that even these may acquire secondary reinforcement value if followed by social reinforce-
ment (e.g., sympathy).

9. Because nonvolitional responses have rein-
firment value, they are among the reinforcers that determine volitional behavior. Specifically, the prob-
ability of a volitional response is a function of the sum total of expected stimuli and nonvolitional re-
sponses and of the primary reinforcement value of those stimuli and responses.

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