RATE DEPENDENCY, BEHAVIORAL MECHANISMS, AND BEHAVIORAL PHARMACOLOGY

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Behavioral pharmacology has become increasingly independent of the experimental analysis of behavior. At its beginning, behavioral pharmacology was closely related to the experimental analysis of behavior, with developments in each field aiding the other. Early attempts to systematize data in behavioral pharmacology culminated with the development of the rate-dependency concept, but as this principle was found to have more limited generality than originally was hoped, a theoretical void developed. This circumstance was followed by increased reliance on pharmacological theory as a basis for experimentation and interpretation, with an attendant decrease in emphasis on environmental variables and behavioral interpretations. Lack of interplay between behavioral pharmacology and the experimental analysis of behavior is detrimental to both disciplines because each could contribute significantly to the other. The current trend might be reversed if more research were directed at elucidating behavioral mechanisms of drug action.

Key words: behavioral mechanisms of action, rate dependency, drugs, drug discrimination, drug self-administration

Behavioral pharmacology as a unique, identifiable entity seems to be disappearing. Most research in modern behavioral pharmacology is aimed at pharmacological rather than at behavioral questions. As a result, it may now be appropriate to classify behavioral pharmacology as a subdiscipline within pharmacology, and as one that is largely divorced from the experimental analysis of behavior. In what follows I hope to clarify this assertion by describing the history that has led to the current state of affairs and to argue that the trend, if it continues, will leave unanswered many important and interesting questions. Finally, I shall suggest briefly how the trend might be changed.

One can debate the origins of the discipline of behavioral pharmacology, but there is little doubt that a report by Dews (1955) represents an important landmark. Dews

trained food-deprived pigeons to peck a response key under either a fixed-ratio (FR) 50 or fixed-interval (FI) 15-min schedule of food presentation. Once consistent performances were established, test sessions occasionally were preceded by intramuscular injections of a range of doses of pentobarbital. The results of the experiments are summarized in Figure 1, which illustrates proportional changes in response rates (compared to nondrug rates) following drug administration. The outcome was, and still is, truly astonishing, particularly at the 1- and 2-mg doses. Responding under the FI schedule was depressed by these doses, whereas responding under the FR schedule was enhanced. Thus, the effect of the drug on the same behavior (key pecking), occurring for the same reason (food reinforcement), depended critically on how events were scheduled. In addition, Dews also made direct observations of the pigeons and reported that neither 1 mg nor 2 mg produced any noticeable behavioral changes. Thus, not only was it demonstrated that scheduling can have profound effects on the way in which a drug affects well controlled, motivated behavior, but also that the

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Fig. 1. Log dose-effect curves, showing effects of pentobarbital on pigeons' pecking. Each point represents the arithmetic mean of the ratios for the same four birds at each dosage level on each schedule. Open circles represent effects for birds on an FI 15-min schedule; solid circles: birds on FR 50. (From Dews, 1955; reprinted by permission.)

behavioral control techniques produced measurements that were much more sensitive to the drug's effects than were observations of gross behavior patterns.

Dews' findings pointed to a new area of inquiry. Before his work, research on psychological effects of drugs had focused mainly on how topographically or functionally similar activities are affected by drugs—for example, how "aggression" is affected. Dews' work, however, indicated that environmental variables can modify the ways in which drugs influence topographically or functionally similar activities. Thus, in order to understand how a drug influences behavior, it is not enough just to know what the pattern of behavior is and what the drug is. Current and previous environments also must be considered. The task for the new area of investigation, behavioral pharmacology, then, was to develop a data base for development of a theory or theories of how current and past environments modify drug effects.

Dews' work was followed by a rapid increase in research using techniques derived from the experimental analysis of behavior. The ability to generate consistent, reproducible patterns of behavior in individual

subjects opened the door to a systematic analysis of behavioral effects of drugs, an analysis that had not been possible before the techniques were developed. Behavioral pharmacology thus was born, and its coming of age was certified, in a sense, with the publication of its first textbook (Thompson & Schuster, 1968). Throughout this early period, researchers frequently reported additional effects like those first described by Dews (1955). That is, the growing literature was filled with demonstrations that subtle features of experimental environments and histories could influence the way in which a drug affects behavior. In addition to demonstrations that reinforcement scheduling can modify a drug's behavioral effects, there were reports implicating other environmental variables such as type of discriminative stimulus (e.g., Laties & Weiss, 1966), training history (e.g., Terrace, 1963), and presence or absence of punishment (e.g., Geller & Seifter, 1960; Morse, 1964). As the data base grew, two approaches to organizing and systematizing it were developed. One emphasized behavioral mechanisms of action, and the other emphasized the relation between drug effects and baseline performance (the concept of rate dependency).

The constituents of behavioral mechanisms of action were elaborated by Thompson and Schuster (1968). The notion of mechanism of action is as integral a part of pharmacology as is the concept of structure-activity relationship, and it can be illuminated by an analogy from cardiovascular pharmacology. Suppose, for example, that a given dose of a particular drug increases blood pressure. An analysis of the mechanism(s) of action would involve determination of how the drug interacted with factors that normally play a part in the control of blood pressure—that is, the way in which the drug affected heart rate, stroke volume, peripheral vascular resistance, baroreceptor function, etc. would be studied. If the drug increased peripheral vascular resistance and did not modify other parameters known to influence blood pressure, then the mechanism by which the

drug produces hypertension would be identified. (Of course, this is a simplified example.) By analogy, elucidation of a behavioral mechanism of drug action depends critically on knowing the factors that normally influence the behavior under study. For example, an operantly conditioned performance may be controlled by such things as deprivation, discriminative stimuli, conditioned reinforcers, unconditioned reinforcers, or a delay between response and reinforcer. Elucidation of the behavioral mechanisms of action of a drug that affects such performance would involve examining how the drug interacts with these factors.

Thus, the point of view that drug effects can be characterized through behavioral mechanisms of action necessarily depends on our knowing how behavior is controlled in the absence of the drug (more on this later). This dependence helps explain why behavior-analytic procedures and theory are useful to those who are interested in studying behavioral effects of drugs. Through the experimental analysis of behavior, procedures have been developed that directly identify variables that control behavior; thus one can readily identify factors involved in the normal (i.e., nondrug) maintenance of the behavior of interest. In short, the assumption that a drug's behavioral effects can be characterized through behavioral mechanisms of action implies that the experimental analysis of behavior is an integral part of behavioral pharmacology.

The other approach to organizing data emphasized the relations between rates of responding under drug and nondrug conditions. This rate-dependency concept implies that the variables responsible for the behavior are important only insofar as they determine the rate of responding. Thus, if two different sets of conditions (e.g., different reinforcement schedules) generate equivalent response rates, then a drug will produce similar effects in the two conditions.

That a drug's effect on responding might depend on the baseline rate was first suggested by Dews (1958) with respect to methamphetamine: "When a control perfor-

Fig. 2. Relationship between mean rates of responding in individual 25-s segments of a 500-s repeating cycle, under normal conditions and the increment in rate following amobarbital. Abscissa: log of rate as responses per session (i.e., in a total of 500 s because each 25-s period occurred once in each of the 20 cycles). Ordinate: change in log rate following amobarbital. For recording purposes, the 500-s interval was divided into 20 periods of 25 s; therefore, there are 20 points on each graph—10 representing periods when the houselight was present (shown as circles) and 10 representing periods when the houselight was not present (shown as $\blacktriangle$). The line through them was calculated by least squares from all points. On the $X = 0$ ordinate are plotted all points at which the mean rate of responding was 1 or fewer responses per 500 s; this arbitrary assignment makes possible the logarithmic plot. Exclusion of these indeterminate points would not appreciably affect the regression line. (From Dews, 1964; reprinted by permission.)

mance contains long inter-response times, administration of low effective doses . . . tends to reduce their number and length. Higher doses . . . also tend to increase the length of short inter-response times" (p. 145). Later, when studying effects of amobarbital

on responding under an FI 500-s schedule with interpolated extinction periods, Dews (1964) plotted, on double logarithmic coordinates, proportional changes in response rate in successive segments of the FI as a function of baseline rate in those segments. The results, displayed in Figure 2, showed that change in rate was a power function of corresponding control rate. Thus, the way in which the temporal pattern of responding was disrupted by the drug was consistent with the view that the drug's effect could be predicted from the rate under nondrug conditions.

Other types of data that strongly supported the rate-dependency concept were those from experiments that examined the relations between variables related to type or potency of reinforcers and a drug's behavioral effects. A well known example is the

work of Kelleher and Morse (1964), who studied squirrel monkeys under FI and FR schedules. For monkeys in one group, in the presence of a white stimulus the first response after 10 min terminated a train (1/s) of brief electric shocks (FI 10-min). In the presence of a red light 30 responses terminated the light and brief shocks that were scheduled every 30 s. For monkeys in the other group, the same multiple schedule was in effect except that completion of the same schedule requirements resulted in presentations of food. Thus, the study involved a comparison of similar performances maintained either by positive or negative reinforcement. The effects of *d*-amphetamine and chlorpromazine are shown in Figure 3. The schedules according to which consequences were arranged were considerably more important determinants of the drug

Fig. 3. Effects of *d*-amphetamine sulfate and chlorpromazine hydrochloride on rates of responding under multiple fixed-interval, fixed-ratio schedules of positive and negative reinforcement. Three monkeys were studied on each multiple schedule. Each drug was given intramuscularly immediately before the beginning of a 2½-hr session. At least duplicate observations were made for each monkey at each dose level. Summary dose-effect curves for the four component schedules were obtained by computing the means of the percentage changes in average response rates from control to drug sessions. The dashed line at 100% indicates the mean control level for each component. The vertical lines in the middle of the figure indicate the ranges of control observations, including saline injections, expressed as percentages of the mean control value. Note the general similarity of the pairs of dose-effect curves for fixed-interval and for fixed-ratio components. (From Kelleher & Morse, 1964; reprinted by permission.)

effects than were the events scheduled. (See Cook & Catania, 1964, and Waller & Waller, 1962, for other examples.)

Because the two kinds of schedules, FR and FI, engendered different response rates, it is a short conceptual leap to suggest that the differences in drug effects depend on the baseline rate. This leap was made in an influential review by Kelleher and Morse (1968), who successfully applied the rate-dependency concept to data from a range of experimental procedures and thus provided behavioral pharmacology with its first unifying principle—rate dependency. Most of the extant data concerning effects of drugs on schedule-controlled behavior were consistent with rate dependency. In addition, Dews' discovery of the orderly relationships between control and drug-influenced rates of responding under FI schedules (see Figure 2) had been replicated widely and systematically. Thus, not only was the rate-dependency concept consistent with a broad array of data, it also yielded quantitative predictions in some circumstances. It is not surprising, then, that the view assumed a predominant interpretive role in behavioral pharmacology.

Interestingly, little attention was paid to the fact that rate dependency relegated the independent variables that control behavior to uninfluential positions. Rate dependency describes a correlation between two dependent variables—response rate under baseline conditions and rate under drug. Thus, taken to its extreme, rate dependency implies that the bases for differences in response rate are largely irrelevant. For example, if equivalent response rates were produced by a variable-ratio (VR) 100-response schedule and a variable-interval (VI) 15-s schedule with conditioned reinforcement added, rate dependency implies that drug effects would be the same under the two schedules in spite of the fact that the controlling variables may be different. This in turn implies that an analysis of drug effects in terms of behavioral mechanisms such as stimulus control, type of reinforcement, or conditioned reinforcement is unnecessary, for

drugs do not interact independently with such mechanisms. Consequently, as researchers analyzed further the dependence of drugs effects on baseline rate, the analysis of drug effects in terms of behavioral processes was de-emphasized. Aiding the ascension of the rate-dependency view was the fact that the first thorough explication of the concept of behavioral mechanisms of action (Thompson & Schuster, 1968) was published in the same year as Kelleher and Morse's review. Thus, just as a framework emphasizing behavioral mechanisms appeared, from which research to identify and establish organizing principles could be generated, an impressive case was made for adopting a ready-made and well substantiated single organizing principle that could supersede them.

Through the middle to late 1970s, however, data accumulated revealing limits to the rate-dependency concept. For example, McKearney (1974) and Barrett (1976) showed that the type of consequent event responsible for maintaining behavior can modify a drug's effect on operant behavior. In contrast to the type of findings reported by previous researchers (e.g., Cook & Catania, 1964; Kelleher & Morse, 1964; Waller & Waller, 1962), these investigators found that reinforcement-related variables modulated drug effects. For example, McKearney (1974) showed that morphine produced increases in responding engendered by an FI 5-min schedule of electric-shock presentation, whereas it decreased comparable responding produced by an FI 5-min schedule of food presentation. In this case, the key variable was not the baseline rate of responding but rather the scheduled consequence. In addition to these and other demonstrations that baseline response rate is not the exclusive predictor of drug effects (e.g., McKearney, 1970; McKearney & Barrett, 1975), alternative views of data originally viewed as supporting rate dependency appeared (e.g., Branch & Gollub, 1974; Gonzalez & Byrd, 1977). Specifically, it was suggested that drug-induced disruption of the temporal distribution of responding under

FI schedules (see Figure 2) could be viewed more as a systematic diminution of temporal differentiation of responding than as an indication of rate dependency. Because of these experiments delineating limits to the applicability of the rate-dependency principle, behavioral pharmacology was bereft of a theoretical organizing principle.

Thus, the promise of broad generality for rate dependency was not realized and, in hindsight, it may have been unfortunate for the field that the concept was so compelling. The demise of the rate-dependency view left most drug-behavior relationships unanalyzed with respect to behavioral mechanisms of action. For example, the effect reported by Dews (1955) described above (see Figure 1) still remains unanalyzed. The roles played by rate of reinforcement, response topography, differentiation of interresponse times (IRTs), type of reinforcer, and many other variables have yet to be determined. This is, of course, an unsatisfactory state of affairs. Dews' results and the many like it are remarkable. Thus, it would seem eminently worthwhile that such effects be analyzed to determine if and how behavioral mechanisms are involved. That is, if we can understand how drugs interact with "basic" processes such as reinforcement, IRT differentiation, and the like, we will be able to predict drug effects across a wide range of procedures without being limited to "botanizing."

It is important to note here that rate dependency and behavioral mechanisms of action are not necessarily incompatible concepts. In many cases predictions about drug effects can be made on the basis of baseline rates (see Dews & Wenger, 1977). These relations, however, should not be taken as substitutes for analyses of their origins. Instead, rate dependence might better be viewed as a clue to the behavioral mechanisms involved. Many variables that control response rate have been analyzed (e.g., see Catania & Reynolds, 1968; Morse, 1966; Zeiler, 1977), yet the likely contributions of these to the determination of drug effects have received comparatively little study.

As the inadequacy of interpretations based

upon rate dependency became evident, a corresponding shift towards research focused upon behavioral mechanisms of action did not develop. Instead, the field was led in a new interpretive direction by two initially independent lines of inquiry: the study of drugs as discriminative stimuli and the development of receptor binding techniques in neuropharmacology.

That the interoceptive effects of drugs can serve as discriminative stimuli has been known since the 1930s (see Girden & Culler, 1937). Modern behavioral control techniques have allowed examination of drugs as discriminative stimuli with individual subjects (see Colpaert, 1978, and Winter, 1978, for reviews), and the establishment of stimulus control of behavior with a particular dose of a drug leads to two questions. First, will similar control be exerted by other doses of the same drug? Second, will similar control be exerted by other drugs that are known to have similar or dissimilar molecular structures or physiological actions? A great deal of research has been directed to these two questions, and it consistently has produced orderly data. Doses lower than the training dose typically exert less discriminative control than the training dose or higher doses (Kuhn, Appel, & Greenberg, 1974). Drugs that are known to be pharmacologically similar to the training drug usually tend to control responding in a manner similar to that seen with the training drug, whereas drugs known to be pharmacologically dissimilar usually do not (Overton, 1977). Because of these regularities, drug-discrimination procedures are reliable preparations that are widely used in determining if different drugs are pharmacologically similar. That is, these procedures have achieved status much like that of classic, isolated-organ preparations.

Research on receptor binding techniques blossomed at the same time behavioral pharmacologists were devoting more study to drug discrimination, and led to an explosive growth of knowledge about drug receptors in the central nervous system (see Snyder, 1984, for a recent review). Identification and

characterization of specific receptors for numerous behaviorally active drugs not only have advanced significantly the field of neuropharmacology but also have provided a theoretical structure that can be used easily to organize the data from drug-discrimination research (and other research in behavioral pharmacology as well). For example, several types of opiate receptors have been identified (see Martin, Eades, Thompson, Huppler, & Gilbert, 1976) that are differentially sensitive to different groups of drugs. When drugs in these groups show similar discriminative properties, but drugs in other groups do not, there is a ready-made organizational scheme into which the data may neatly be fit. That is, the drugs that show similar discriminative properties can be assumed to bind to the same receptor. Thus, neuropharmacology has provided a reductionistic theoretical base for interpreting a drug's behavioral effects.

Such explanations, then, are nonbehavioral (cf. Skinner, 1950) and may be contrasted with the types of accounts that might be developed were an analysis of behavioral mechanisms of action to be carried out successfully. In any event, the successful application of receptor theory in the interpretation of some behavioral effects of drugs diverted research away from examination of environmental variables. However, one branch of behavioral pharmacology, the study of drug self-administration, seems to have survived this shift in emphasis.

A drug is said to be self-administered if behavior can be established and maintained by drug administration as a consequence of the behavior. Many techniques have been developed for studying drug self-administration (see Griffiths, Brady, & Bradford, 1979, and Johanson, 1978, for reviews of some of these). Research on drug self-administration has been directed at characterizing precisely the reinforcing effects and at developing procedures that will predict whether humans will abuse certain drugs. This research frequently emphasizes behavioral factors in the establishment and maintenance of drug self-administration,

and pharmacological principles usually do not provide a theoretical base for interpreting results. Instead, the field is characterized by a steady accumulation of data demonstrating environmental modification of drug self-administration, although to date, no coherent behavioral framework has emerged that systematizes the findings. Thus, in the field of drug self-administration, in contrast to other areas in behavioral pharmacology, behavioral analyses of drug effects continue to be attempted because it is recognized that inferences about receptor binding do not provide a complete account of why a drug is self-administered (see Goldberg, Spealman, Risner, & Henningfield, 1983).

The current state of behavioral pharmacology, then, is one in which research is directed mainly (but not exclusively) at drug discrimination and at drug self-administration. Drug-discrimination procedures have assumed the status of basic pharmacological preparations and are used mainly to investigate strictly pharmacological questions. Even work examining drug effects on operant behavior maintained by conventional consequences usually is directed at pharmacological rather than behavioral questions. Except for the study of drug self-administration, then, behavioral pharmacology has evolved largely into a subdiscipline within pharmacology wherein drug similarity is examined with intact organisms. Obviously, this is an important enterprise, but by being absorbed into pharmacology the field has lost the emphasis that made it unique. Behavioral pharmacology no longer seems to be a field directed primarily at analyses of how past and current environments modify drug effects.

It is not simply that rate dependency has been shown to be less ubiquitous than it once was seen to be and that receptor theory has provided an interpretive base that have led behavioral pharmacology to its current state. To some extent, the challenges of behavioral pharmacology are based on those faced generally by the experimental analysis of behavior. Whereas pharmacological prin-

ciples provide an organized, logical framework from which experimental ideas can be derived and results can be interpreted, behavioral principles, by contrast, currently are not as widely accepted (see Seligman, 1970) and thus tend to provide a weaker interpretive base. This should come as no surprise, however, and also should not be viewed with excessive alarm. Pharmacology is a more well developed science than is psychology. What I hope, however, is that the attractiveness and orderliness of pharmacological theory will not keep the field from pursuing a behavioral analysis of drug action. Receptor theory tells us little about the important facts involved in producing reinforcement-schedule-dependent drug effects. Presumably, whether a subject is responding under an FI or FR schedule will not influence to what receptor a drug will bind, but it is clear that whether a subject is responding under an FI or FR schedule can modify profoundly the type of effect seen. And this modification is in need of analysis—a behavioral analysis.

Where is the field likely to go from here? Drug-discrimination procedures will continue to be used because they can validate, in intact organisms, pharmacological classifications of drugs that are developed using *in vitro* preparations. Such procedures also can be used to identify previously unsuspected pharmacological similarities among drugs. There are, however, potential dangers in making inferences about drug receptors on the basis of behavioral data. Even the simplest activity on the part of an organism involves a multitude of neurotransmitters and receptor types. Extreme care must be taken to ensure that similarities in drug effects do not depend on some common (or opposing) “downstream” effect if one is to make inferences about receptors. For example, Drug A may bind to Receptor X and Drug B to Receptor Y, yet the neuronal systems activated by stimulation of Receptors X and Y may converge on another system (perhaps activated by stimulation of Receptor Z) that is the basis for the interoceptive stimulus on which the discrimination (or other effect) is

based. Thus, the fact that Drugs A and B produce similar discriminative effects (or that their discriminative effects can be blocked by the same drug) does not mean necessarily that they bind to the same receptor type.

It also seems certain that advances will continue in the study of drug self-administration. Drug abuse is an important social problem, so the continued accumulation of data related to factors that influence drug taking is of significance.

Less certain is the future of research on other drug-behavior interactions. If such research is to continue toward any useful end, patience and continued progress in the experimental analysis of behavior will be necessary. Patience is dictated by two considerations. First, available data illustrate the enormous complexity of the subject matter of behavioral pharmacology. Seemingly minor changes in the variables controlling behavior can have major influence on the way a drug affects behavior. Consider a study by Byrd (1974), who trained chimpanzees to press a key under a multiple FR 30 FI 10-min schedule of food presentation. Effects of a range of doses of chlorpromazine were determined under two conditions. In one, each key press was followed immediately by a 20-ms audible click. In the other, there were no programmed auditory consequences of a key press. This seemingly minor difference (“feedback” vs. no “feedback”) significantly altered chlorpromazine’s effects on responding under the FI schedule but not under the FR schedule. Specifically, addition of “feedback” potentiated the rate-increasing effect of the drug during the FI schedule, but did not change the dose-effect curve for responding under the FR schedule. Obviously, the drug-environment interactions here are complex, and complex phenomena usually cannot be analyzed quickly.

The second consideration dictating patience is that experiments in behavioral pharmacology are time-consuming. Once a behavioral baseline has been established, drug testing cannot be carried out more frequently than once every 3 to 7 days, depend-

ing on how long it takes for the drug to be cleared from the subject's system and on how quickly the baseline is recovered. Because several doses must be tested, usually more than once each, determination of a dose-effect curve takes anywhere from 4 to 10 weeks to complete. Furthermore, a single dose-effect curve is just the beginning of an analysis. Elucidation of behavioral mechanisms of action requires that dose-effect curves be assessed across a range of behavioral parameters, and, of course, a new baseline must be established under each of these sets.

Recently, a method that may help reduce the time necessary to conduct behavioral analyses of drug action has been employed in some studies. The technique, cumulative dosing, allows determination of a dose-effect curve in a single session (see Wenger, 1980). Test sessions are divided into segments separated by timeout periods. Drug injections are then made during sessions (i.e., during the timeout periods) as well as before them. The effective dose during any segment is the sum of all amounts injected over the entire session. Thus, over the course of the session, a subject is exposed to increasing amounts of the drug. Although this procedure holds much promise, its use is restricted generally to drugs that have a long duration of action, and there are circumstances under which dose-effect curves determined by the method differ significantly from curves generated via the more traditional technique (Thompson, Moerschbaecher, & Winsauer, 1983).

Even with the advent of new procedures like cumulative dosing, it is unlikely that progress in behavioral pharmacology will be rapid unless the number of practitioners were to grow suddenly. There is every likelihood, however, that steady, cumulative progress can be made, but we shall have to be both persistent and patient.

The future of behavioral pharmacology depends not only on patience but also on the vigor of the experimental analysis of behavior. If we are to characterize drug effects in terms of their interactions with fundamental

behavioral processes, it obviously is necessary that we understand those processes. On this front, too, there is reason to be hopeful. The recent emphasis in the experimental analysis of behavior on dynamic behavior-environment interactions (e.g., extinction, forgetting, acquisition) bodes well for behavioral pharmacology in at least three ways. First, new techniques are being developed for studying dynamic changes in behavior in individual subjects. That is, the tacit principle that legitimate individual-subject research necessitates a steady-state behavioral baseline seems finally to be waning. Behavioral pharmacologists thus are being presented with a wider range of procedures that will allow them to determine experimentally how drugs interact with dynamic behavioral change. Second, and more important, research on the dynamics of behavior should lead to the development of a theoretical base that is most applicable to the goals of behavioral pharmacology. Almost invariably, administration of a drug alters the relations that exist between environment and behavior—that is, the contingencies of reinforcement. For example, if a drug lowers response rate under a ratio schedule, inter-response-time related contingencies and the usual relation between response rate and reinforcement rate will be altered. We need to know the contribution of these temporary changes in contingencies if we are to understand the drug effects. Finally, the recent extension of behavior-analytic techniques to new phenomena also opens the door to new areas of inquiry for behavioral pharmacology. For example, although it has hardly begun, the experimental analysis of naturally occurring behavior already has set the occasion for drug research (e.g., Peffer-Smith, Smith, & Byrd, 1983).

It appears, then, that the future of behavioral pharmacology holds the promise of using developments, both methodological and theoretical, in the experimental analysis of behavior. There exist already conceptual views from experimental analysis of behavior that have not yet been utilized by behavioral pharmacologists. Two examples

are Nevin's (1974) notions concerning response strength and McDowell and his colleagues' (McDowell, Bass, & Kessel, 1983; McDowell & Kessel, 1979) rate equation from which one can extract a measure of reinforcer value. (Interestingly, Nevin's view may have much in common with the rate-dependency concept. Specifically, instead of response rate [origin unimportant], response strength [origin unimportant] could be an important predictor of drug effects.) Employment of either of these approaches, however, requires observation of behavior under a variety of circumstances to yield estimates of either response strength or of reinforcer value. Thus, drug research based on these new conceptualizations will progress slowly, but to the extent that the views offer a chance to clarify the behavioral nature of drug action, the price of effort will be modest.

As a final point it should be noted that the interaction between behavioral pharmacology and the experimental analysis of behavior is not unidirectional. Drug-oriented experimentation can speak effectively to behavioral issues. For example, Branch, Nicholson, and Dworkin (1977) found that pentobarbital increased responding suppressed by electric-shock punishment, but that it did not increase responding suppressed to a similar degree by timeout punishment. This finding suggests that positive and negative punishment are functionally distinct, something that could not be concluded from simple observation of the rate-suppressing effects of the two procedures. Similarly, if Nevin's or McDowell and Kessel's formulation is used to direct research in behavioral pharmacology and is found to provide a useful theoretical base, then in some sense the view will be validated. Thus, behavioral pharmacology can be viewed as an integral part of the experimental analysis of behavior in which drugs can be used to validate or invalidate hypotheses, as well as to test the generality of concepts.

To summarize, the future of behavioral pharmacology seems predictable on two

fronts but less so on the third. Pharmacologically oriented research using drug-discrimination paradigms will continue because the preparation has become a standard part of the pharmacologist's arsenal. Research on drug self-administration also will advance because of the obvious social relevance of such work. The picture for the rest of behavioral pharmacology is less clear. We know that environmental variables can modify a drug's behavioral effects, but after nearly 30 years, we have yet to devise a conceptual scheme into which to fit our findings. Thus, if traditional behavioral pharmacology is to continue, it must go on in somewhat a theoretical vacuum. My suggestion is that we focus more closely upon behavioral mechanisms of action. Even though the approach was first outlined carefully over 15 years ago (Thompson & Schuster, 1968), it has never been a dominant one in the field. First, it was relegated to the background by rate dependency, and subsequently by receptor-based theorizing. Now is the time to give it its due. Behavioral pharmacologists have shown repeatedly that it is inappropriate to speak of a drug's effect on "behavior" or even on a particular functional unit of behavior (e.g., a key peck). Rather, the effects must be viewed, minimally, as an interaction among the current environment, the conditioning history (see Barrett & Witkin, in press), and the drug. Obviously, such an interaction cannot be characterized or summarized in purely pharmacological terms. The work involved in assessing behavioral mechanisms of action may be tedious and time-consuming, and it may not be successful until our understanding of behavioral processes improves. Yet if behavioral pharmacology is to exist as a unique discipline, and not simply as an arm of pharmacology, such work must be done.

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