EFFECTS OF STIMULANT DRUGS ON SELF-CONTROL CHOICES IN PIGEONS: DETERMINING BEHAVIORAL MECHANISMS OF DRUG ACTION

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ABSTRACT

The present study attempted to identify behavioral mechanisms of stimulant effects on "self-control" choices in pigeons. The experimental procedure required pigeons to respond on a single random interval (RI) 1 min schedule in order to choose between a smaller, more immediate reinforcer (1 s food after 2 s delay) and a larger, more delayed reinforcer (4 s food after 2 to 40 s delay). While the signaled delay to the smaller option remained 2 s throughout the session, the signaled delay associated with the larger option increased across five, 10 min blocks from 2 s to 40 s. In this way delay-discount functions were obtained within each experimental session. Once stable delay-discount functions were obtained, methylphenidate (MPD) (0.0 – 17.0 mg/kg) and methamphetamine (METH) (0.0 – 3.0 mg/kg) were administered via i.m. injections. Using a logarithmic variation of Herrnstein's matching law, an attempt was made to separate changes in the sensitivity to delay (S_D) from changes in the sensitivity to amount (S_A). Overall, MPD and METH increased choices of the larger, more delayed reinforcer. Moreover, MPD's and METH's primary effects were a decrease in S_D, although concomitant decreases in S_A occasionally occurred. It is concluded that quantitative methods such as those used here may prove useful in elucidating behavioral mechanisms of drug action.

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I would like to thank my committee members: Dr. Raymond Pitts, Dr. Mark Galizio, Dr. Christine Hughes, Dr. Carol Pilgrim, and Dr. Kim Sawrey for the professional and academic support they provided throughout this project. I would like to especially thank my mentor, Dr. Raymond Pitts. If it had not been for his vast knowledge in this field, his outstanding academic guidance, and his patience, the completion of this thesis would have been substantially more difficult. I greatly appreciate all of the knowledge Dr. Pitts has shared with me during this experience.

DEDICATION

I would like to dedicate this thesis to my mother, Rose I. Hank, for her continual support, understanding, and patience during the last 3 years. Her endless confidence and frequent words of encouragement have once again helped me to achieve my goals. Thank you mom.

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INTRODUCTION

Behavior Pharmacology and Environmental Modulation of Drug Effects

A number of factors can determine the behavioral effects of drugs. An important class of variables is pharmacological. For example, the dose, the route of administration, and the receptor system with which a drug interacts all influence whether or not it affects behavior and the particular behavioral effects observed (see Cooper, Bloom, & Roth, 1996; Goodman & Gillman, 1996). However, determining drug effects cannot be limited to pharmacological variables. Another class of variables, environmental factors, can play a major role in determining behavioral effects of drugs. Research concerning these variables has flourished over the years and through this, the sub-discipline of Behavior Pharmacology has emerged.

Behavioral Pharmacology is the study of the effects of drugs on behavior using methods of the Experimental Analysis of Behavior. The main concern of Behavioral Pharmacology lies with the behavioral actions of drugs rather than their chemical or neurochemical effects (see Branch, 1991). According to Branch, "A major goal of Behavioral Pharmacology is to describe, characterize, and quantify how drugs modify or otherwise interact with fundamental behavioral processes" (p.42). As Witkin and Katz (1990) point out, behavioral pharmacologists attempt to determine how drugs affect specific behaviors or behavioral processes. Demonstrations of environmental/behavioral influences on drug action are numerous; an exhaustive review is beyond the scope of the present paper. A few examples, however, should provide an indication of the powerful role of these variables.

In an early study, Teitlebaum and Derks (1958) studied drinking in rats wherein licking a tube produced water. They compared licking controlled by shock postponement versus licking controlled by water deprivation. Some doses of amphetamine increased rates of licking maintained by shock postponement, but decreased licking rates induced by water deprivation. Although all rats engaged in licking, this behavior functioned differently depending on whether or not it was controlled by shock postponement or water deprivation. Thus, formally (topographically) similar responses were affected differentially by a drug, depending upon their controlling variables.

Interestingly, drugs can produce similar effects on topographically dissimilar responses when such behaviors are controlled by comparable environmental conditions (e.g., Kelleher, Fry, Deegan, & Cook, 1961). In the Kelleher et al. study, the environmental variable controlling behavior was the same for all rats (i.e., food presentation under a fixed interval, or FI, schedule). However, for some rats, the response required to obtain food was a lever press, whereas for other rats the response was a press on a wall-mounted disk. Although the two responses were topographically dissimilar, effects of amphetamine on the two responses were very similar; the same was true for meprobamate. Following the administration of these drugs, pigeons decreased pause time (s) under the FI schedule and responded in similar temporal patterns regardless of the response topography. Thus, it was demonstrated that when formally different responses fell under similar functional control, behavioral effects of drugs were similar. These data suggest that a functional analysis of variables controlling behavior (as provided through the Experimental Analysis of Behavior) is necessary for understanding drug effects.

In a landmark study showing functional control over drug effects, Dews (1955) showed that the schedule of reinforcement maintaining behavior was an important determinant of the behavioral effects of drugs. Key pecking by pigeons was maintained under either a fixed ratio (FR) 50 (reinforcement delivered after every 50^{th} peck) or an FI 15 min (reinforcement delivered after the first peck following a 15 min time interval) schedule of food presentation. Under control (non-drug) conditions, rates and patterns of responding differed under the two schedules. Specifically, responding under FR 50 produced a steady and high rate of responding, whereas responding under FI 15 min increased across the duration of the timed interval. Thus, overall response rates generally were higher under the FR 50 schedule compared to those under the FI 15 min schedule. Doses of pentobarbital then were administered prior to selected experimental sessions. Figure 1 shows that pentobarbital's effects depended upon the schedule of reinforcement and/or the control response rate maintained by that schedule. For example, the 1.0 mg dose decreased response rates under the FI 15 min schedule and increased response rates under the FR 50 schedule. These data were of particular interest because they demonstrated that "stimulant" or "depressant" drug effects were not invariant properties of a drug, but rather, they can depend on the schedule of reinforcement. In other words, in order to understand and accurately predict drug effects on behavior, environmental factors must be taken into account.

After Dew's (1955) study, many researchers began to report other types of environmental control over behavioral effects of drugs. These controlling variables have been reviewed by numerous researchers (e.g., Barrett, 1987; Branch, 1991; Sanger & Blackman, 1976;). Aside from reinforcement schedules and the selected parameters of

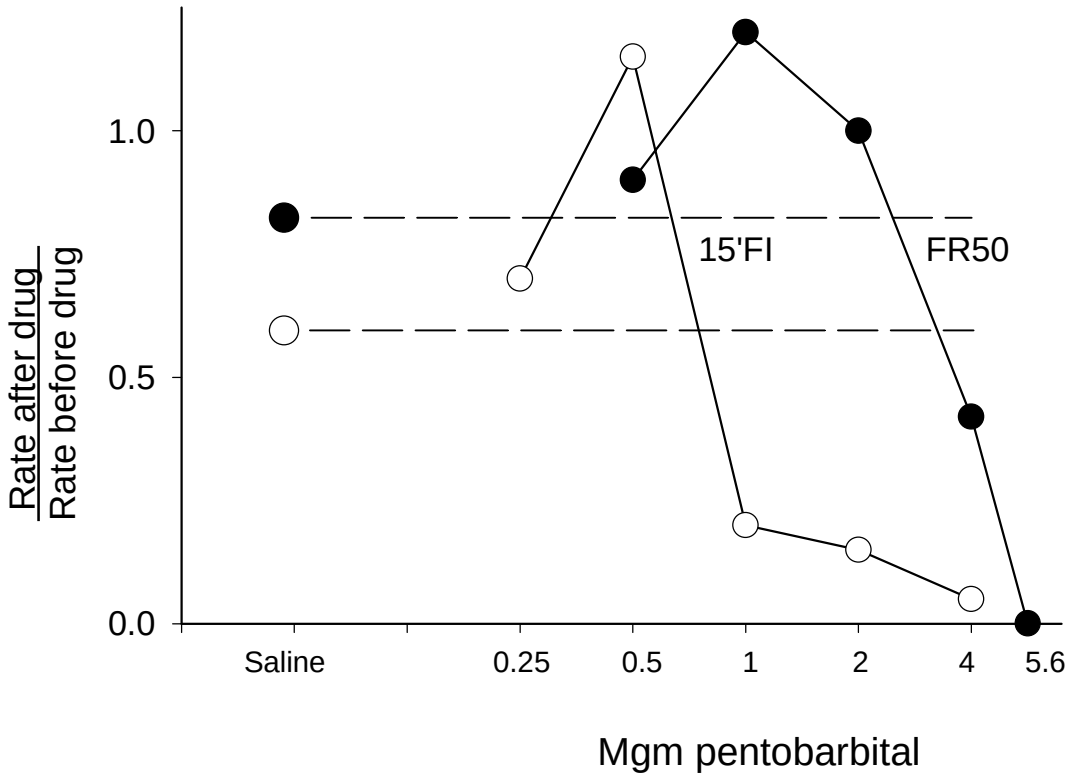

Figure 1. Responding maintained by FI 15' (open circles) and FR 50 (closed circles) plotted as a function of pentobarbital dose. Redrawn from Dews, P.B. (1955). Studies on behavior I: Differential sensitivity to pentobarbital of pecking performance in pigeons depending upon the schedule of reward. *Journal of Pharmacology and Experimental Therapeutics, 113*, 393-401.

the schedules, other important environmental determinants include the type of consequence (e.g., Barrett, 1976), the degree of deprivation of the scheduled reinforcer (e.g., Hughes, Pitts, & Branch, 1996; Schaal, Miller, & Odum, 1995), the nature/degree of stimulus control (e.g., Laties & Weiss, 1966; Thompson & Corr, 1974), whether or not behavior is punished (e.g., Dworkin, Bimle, & Miyauchi, 1989), and the type and intensity of punisher (e.g., Branch, Nicholson, & Dworkin, 1977; McMillan, 1975).

Rate Dependency

With the demonstration that drug effects could be modulated by specific environmental conditions, a means to organize the data was needed. In addition to providing procedures for the systematic study of behavioral effects of drugs and showing that environmental variables could modulate these effects, many investigators suggested that the Experimental Analysis of Behavior could make an additional contribution: a theoretical framework to aid understanding of the relation between the environment and the behavioral effects of drugs.

In a critical early study, Dews (1958) found that effects of methamphetamine (METH) on responding maintained under several different reinforcement schedules depended upon control response rates, regardless of the particular schedule used to produce them. That is, drug effects on performance were similar across different schedules if those schedules produced similar control response rates. Furthermore, the rate at which responding occurs under certain reinforcement schedules was shown to be a controlling factor, even when comparable rates were produced by different consequences (e.g., Kelleher & Morse, 1968).

Subsequent research showed that under a variety of schedule conditions with a variety of consequences, a drug's effect could be predicted on the basis of the baseline, non-drug rate of responding (see Sanger & Blackman, 1976). For many drugs, particularly the so-called stimulants, certain doses tended to raise relatively low response rates and lower relatively high response rates. With these research findings, an emphasis regarding the relation between response rates obtained in the absence of drug and response rates obtained in the presence of drug developed. Thus, it appeared that Behavioral Pharmacology had its first general principle: "rate dependency."

As the notion of rate dependency gained recognition, it seemed as though a theoretical framework to aid understanding about the relation between the environment and the behavioral effects of drugs was underway. Yet, although a drug's behavioral effect often can be predicted on the basis of control rate under some conditions, rate dependency as a general theoretical framework appears to be limited (see Branch, 1984). As the concept of rate dependency suggests, if control response rates are similar, drug effects will be similar, regardless of reinforcement schedules and controlling variables; however, data from a number of important studies suggests that this relation does not hold under all conditions. For example, amphetamine or cocaine increase low rates under schedules of positive reinforcement, but decrease or have no effect on low rates of behavior suppressed by punishment (e.g., Dworkin et al., 1989) or behavior under strong stimulus control (e.g., Laties & Weiss, 1966). Therefore, under some conditions, the baseline rate may not be the best predictor of drug effects (Barrett, 1976). In addition, detailed analyses of drug effects on performance under FI schedules suggest that rate-dependent effects of drugs may be an artifact of more molecular processes (Branch &

Gollub, 1974). Thus, the concept of rate dependency seemed to demonstrate limited generality, leading many behavioral pharmacologists to question the adequacy of this notion. A more adequate theoretical framework to help describe the relation between the environment and behavioral drug effects was once again sought.

Behavioral Mechanisms of Drug Action

Thompson and Schuster (1968) suggested what might be considered an alternative theoretical view to the notion of rate dependency. They suggested that Behavioral Pharmacologists seek to identify "behavioral mechanisms of drug action." Interest concerning this alternative view began to increase among behavioral pharmacologists as limitations surrounding the concept of rate dependency started to surface (see Branch, 1984). Indeed, Branch (1991, p. 21) suggested, "The goal of Behavioral Pharmacology is to identify *behavioral mechanisms* of drug action." Unfortunately, what behavioral mechanisms are and how they are identified have been difficult questions to answer. In some respects, the answers still remain unclear.

Thompson suggested that "by behavioral mechanism of drug action we refer to a description of a drug's effect on a given behavioral system expressed in terms of some more general set of environmental principles regulating behavior" (1984, p. 5). He went on to say that:

> specifying the behavioral mechanism(s) responsible for an observed effect involves identifying the environmental variables which typically regulate the behavior in question and characterizing the manner in which the variables' influence is altered by the drug (1984, p. 5).

In other words, the way a drug affects behavior depends on how the environmental variables that control behavior are changed. It is crucial to understand what basic processes maintain behavior under normal, non-drug conditions, and moreover, how drugs interact with these behavioral processes. Unfortunately, identifying a behavioral mechanism of drug action is a difficult and complex process. Despite the plentiful literature describing this notion, conclusive data illustrating specific behavioral mechanisms are lacking (see Witkin & Katz, 1990).

Consider "self-control" situations, for example. Processes associated with reinforcement (e.g., reinforcement amount and reinforcement delay) have been shown to influence self-control under normal (non-drug) conditions. Self-control, from a behavior analytic view, includes a choice between a smaller, more immediate reinforcer and a larger, more delayed reinforcer. Choosing the smaller, more immediate reinforcer is considered the "impulsive" choice. Choosing the larger, more delayed reinforcer is considered the self-control choice. If a particular drug increases self-control, a determination of the behavioral mechanism of drug action would include an analysis concerning how the drug affected such controlling variables as reinforcement amount and/or delay. An understanding regarding how reinforcement amount and/or delay affect self-control in the absence of a drug must be established before drug administration in order to help determine how a drug interacts with these controlling variables. In short, the present study is designed to help identify potential behavioral mechanisms associated with effects of stimulants on choice, or more specifically, on self-control choice.

Experimental Analysis of Choice

An individual has a choice when two or more behavioral options are simultaneously available. For example, in everyday life people choose which clothes to wear and which television programs to watch. In any case, one behavioral option is chosen over another, and this choice depends upon many variables. An important tool for studying choice is the concurrent schedule of reinforcement. This schedule is described as one with two or more schedules simultaneously and independently occurring, with different responses required for each schedule (Ferster & Skinner, 1957). For example, in animal studies, rats can learn to press two levers and pigeons can learn to peck two response keys in an operant chamber. On a concurrent schedule, reinforcement on one schedule may be contingent upon a peck or lever press to one operandum and reinforcement on another schedule may be contingent upon a peck or lever press to the other operandum. It is not necessary, however, to have reinforcement delivered after every response. In fact, reinforcement is usually delivered according to intermittent reinforcement schedules.

Concurrent variable-interval (VI) schedules are typically used to study choice. On a VI schedule, reinforcement is delivered following a response that occurs after a variable length of time has elapsed since the last reinforcer. In a concurrent VI VI schedule, two independent VI schedules are assigned to two different behavioral options, and both schedules are in effect simultaneously. For example, in a concurrent VI 1 min VI 1 min schedule, reinforcement is set up on average once every minute for each behavioral option. Therefore, responses on each option are required to receive all available reinforcers.

Modifying certain reinforcement contingencies has been shown to affect choice systematically. In other words, through controlled laboratory research, researchers have found that choice can be predicted quite accurately when certain variables are manipulated. Among these variables are rate, amount, and delay of reinforcement.

In a classic experiment, Herrnstein (1961) gave pigeons the opportunity to peck either of two response keys. Herrnstein exposed the pigeons to different combinations of VI schedules (conc VI 3 min VI 3 min, VI 2.25 min VI 4.5 min, VI 8 min VI 9 min, and VI 1.5 min VI∞ [Ext.]). He found that the relative number of responses allocated to each key varied systematically as a function of their relative rate of reinforcement. Specifically, he found that the proportion of responses on a given key equaled or matched the proportion of reinforcers obtained via that key. This relation is described by a simple mathematical equation known as the matching law. The matching equation states that:

$$B_L/(B_L+B_S) = R_L/(R_L+R_S), \qquad (1)$$

where B denotes the rate of behavior, R denotes the rate of reinforcement, and the subscripts denote the two behavioral options (larger reinforcer and smaller reinforcer). Overall, Herrnstein found this equation to be a good descriptor of an organism's behavior under concurrent VI schedules of reinforcement.

Catania (1963) and Neuringer (1967) compared reinforcement magnitude on single-key and two-key procedures and found matching with reinforcement amount. Reinforcement amount was defined as the number of seconds pigeons had access to grain. Both studies reported choice was a function of reinforcement amount. In Neuringer's study, for example, the reinforcement amount available for pecking one response key ("standard") was always 2 s, and the reinforcement amount available for

pecking the other ("variable") key was manipulated across experimental conditions. Rate and delay of reinforcement were held constant. Response rates on the variable key were found to increase as the reinforcement amount associated with that key increased, while response rates on the standard key decreased. These data can be described with a version of the matching equation adopted to reinforcement amount:

$$B_L/(B_L+B_S) = A_L/(A_L+A_S), \qquad (2)$$

where A_L and A_S denote the reinforcement amounts delivered according to each behavioral option.

Chung (1965) and Chung and Herrnstein (1967) studied how choice behavior was affected by reinforcement delay. In Chung and Herrnstein's study, for example, contingencies were initially programmed on a concurrent VI 1 min VI 1 min schedule. After stable response rates were obtained, the delay to reinforcement following a peck on the left ("standard") key became 8 s for one group and 16 s for another group. The delay to reinforcement following a peck on the right ("experimental") key ranged from 1 to 30 s. A darkened chamber signaled reinforcement delay. During this delay responses were ineffective and no reinforcement was delivered. Rate and amount of reinforcement were held constant. As the delay associated with responding on the experimental key increased, response allocation to that key decreased. The overall finding concerned the matching of relative response rate to the reinforcement delay. Thus, with respect to reinforcement delay:

$$B_L/(B_L+B_S) = D_L/(D_L+D_S), \qquad (3)$$

where D_L and D_S denote the delays of reinforcement associated with each behavioral option. Note that "immediacy" can be considered the reciprocal of delay (I = 1/D).

Demonstrations that rate, amount, and delay have comparable effects on choice led Baum and Rachlin (1969) to suggest a general form of the matching law that subsumes these three variables:

$$B_L/(B_L+B_S) = R_LA_LD_S/ (R_LA_LD_S+R_SA_SD_L) \quad (4)$$

This relation has been demonstrated for a variety of species (including humans) with a variety of response types, and with a variety of reinforcers (see Conger & Killeen, 1974; Davison & McCarthy, 1988; McDowell, 1988). Thus, matching appears to be a very general phenomenon.

Choice and Self-Control

Much of the research concerning choice involves laboratory settings in which animals have a choice between two behavioral options. As stated earlier, these two options are presented simultaneously. For instance, a pigeon has a choice between pecking two response keys in an operant chamber. On each option, reinforcement amount and delay may differ. One behavioral option may deliver a smaller reinforcement following a short delay (e.g., 2 s food delivered immediately), while the other option may deliver a larger reinforcement following a longer delay (e.g., 4 s food delivered after a 2 s delay). This particular type of choice procedure has been termed a self-control procedure.

From a behavior analytic standpoint, the notion of self-control does not describe a specific internal locus of control. Instead, behavior analysts focus on a temporal locus of control. One behavior analytic way to view self-control is to describe behavior under conditions in which an organism chooses between a smaller, more immediate reinforcer and a larger, more delayed reinforcer (Rachlin, 1974; Rachlin & Green, 1972).

Therefore, the focus of self-control involves comparing control by immediate versus distant consequences.

This temporal locus of control is demonstrated in the frequent self-control choices organisms engage in every day. Take the millions of dieters, for instance, who must choose between sticking to their diet and "being bad." Do they choose the salad or splurge for the burger? By choosing the burger, the dieter receives an immediate, yet possibly smaller reinforcer (i.e., the taste of the burger and satiation are immediately delivered, yet consumption of the burger will not help facilitate weight loss in the future). In contrast, choosing the salad delivers perhaps a larger, yet delayed reinforcer (i.e., the salad may not taste as good or satiate the dieter for an extended period of time, but will help with future weight loss, which in fact, is the long-term goal of the dieter). When students have upcoming tests, do they study or spend a night out with friends? Going out with friends will deliver immediate reinforcement, however, this reinforcement may be smaller in that it will only last a few hours. On the other hand, studying may deliver a delayed, yet larger reinforcement (i.e., a better grade at the end of the semester). If a pigeon has a choice between 4 s of food delivered immediately and 6 s of food delivered after a 2 s delay, which option would the pigeon choose? Thus, the question is one that involves *when* certain consequences are delivered.

In the pigeon's case, choosing the smaller, more immediate reinforcer (i.e., 4 s food immediately) is considered the impulsive choice. The pigeon receives reinforcement immediately, but receives a smaller amount. Choosing the larger, delayed reinforcement (i.e., 6 s food after a 2 s) is called the self-control choice. In this case, the pigeon receives a larger reinforcement, but only after waiting the passage of a longer

delay. When pigeons and other animals (including children) have a choice between receiving a small reinforcer immediately or receiving a larger reinforcer later, they often engage in impulsive behavior (e.g., Logue, 1988; Mazur & Logue, 1978; Mischel, Shoda, & Rodriguez, 1992; Rachlin & Green, 1972). That is, what seems to be the less optimal choice in the long run is the one preferred more often.

What happens to behavior as a function of delay has attracted much attention. If both the smaller and larger reinforcer options were delivered immediately, the natural choice would be the larger of the two. However, once a delay is implemented for one of the behavioral options, response allocation to the different behavioral options may change. When this delay is increased, response allocation to that particular option becomes less likely. It can be stated then, that as delay duration increases, the probability that an organism will choose the option associated with that reinforcer decreases. Therefore, organisms are less likely to choose a larger reinforcer with increasing delay durations. Thus, the delay "discounts" the effectiveness of the reinforcer, hence the term "delay discounting" (Mazur, 1987, 1988). In Figure 2, reinforcer effectiveness is plotted as a function of delay duration, resulting in a steep concave curve. As the delay duration increases, reinforcer "value" decreases. This is what Mazur referred to as a "delay-discount function."

Mazur (1987, 1988) demonstrated these delay-discounting functions through an adjusting delay procedure. In this type of procedure, an organism chooses between two reinforcement alternatives (smaller vs. larger). The delay for one alternative (the "standard" key) remains constant within sessions, while the delay to the competing

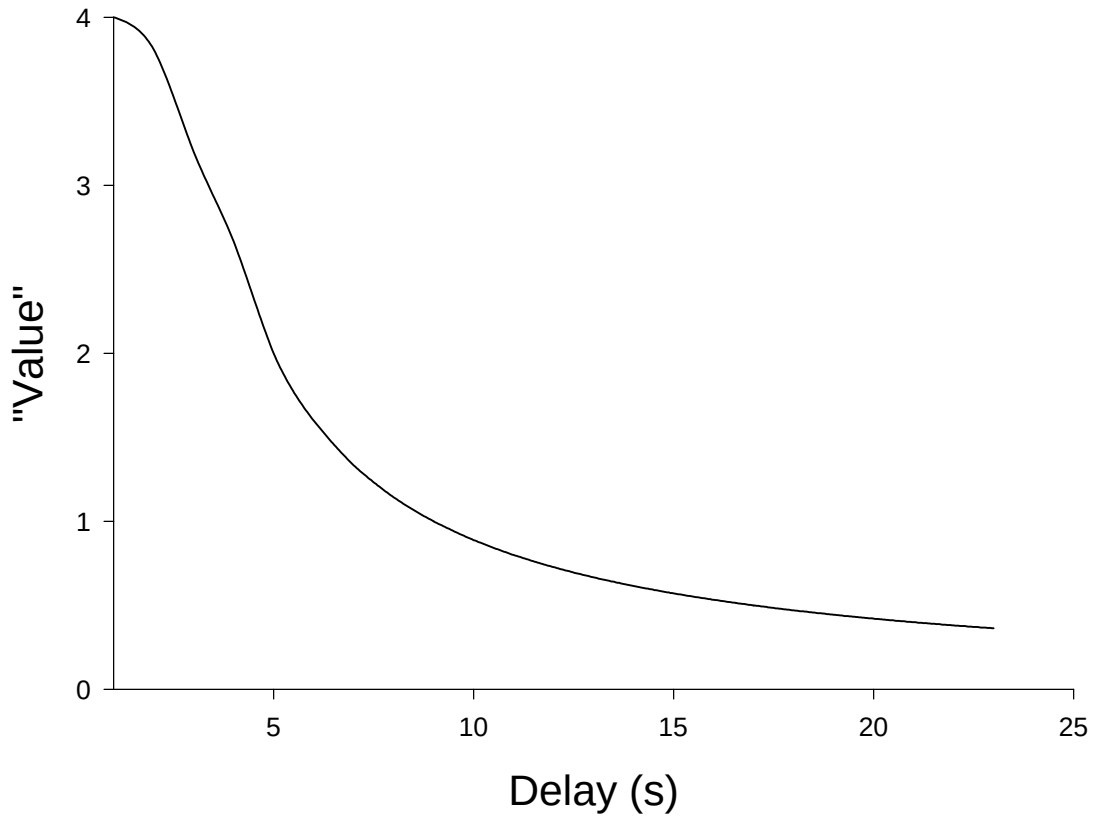

Figure 2. Reinforcer effectiveness (“value”) as a function of delay duration. As the delay duration increases, reinforcer “value” decreases.

alternative (the “adjusting” key) changes within sessions. For example, the delays to both the standard and adjusting alternatives may be 6 s at the start of the session. The adjusting delay is determined by how the organism allocates choices between the two alternatives. If the standard alternative is chosen in the next two trials, the adjusting delay decreases by 1 s. However, if the adjusting alternative is chosen in the first two trials, the adjusting delay increases by 1 s. If each alternative is chosen once in the first two trials, the adjusting delay remains the same. Mazur developed this procedure to measure indifference points, or the delay value at which both alternatives will be chosen equally. By manipulating the delay values associated with the standard choice, indifference points change systematically. That is, the adjusting delay value associated with the large reinforcer changes as a function of delay to the standard key. When reinforcement delay is increased, responding on that option decreases, thus further demonstrating profound effects of delay.

Drug Effects on Self-Control Choices

Psychoactive drugs alter behavior and can be administered to treat neuropsychological illness (Julien, 1995). Encompassed within psychoactive drugs are the psychostimulants. These include drugs such as cocaine, amphetamine, and methylphenidate (MPD), an amphetamine-like stimulant. Psychostimulants have a high potential of abuse (i.e., they function as reinforcers) and some major effects on behavior include sleep reduction and increased general activity (e.g., locomotor activity).

One medical use of psychomotor stimulants such as amphetamine and MPD is with the treatment of attention deficit hyperactivity disorder (ADHD). ADHD affects approximately 6% of school-age children (Julien, 1995). Children diagnosed with

ADHD are often characterized as “inattentive and hyperactive,” and their behavior is often described as impulsive. It is commonly believed that administration of these psychomotor stimulants helps children with ADHD behave less impulsive and alternatively, to behave in a more self-controlling manner. Thus, it could be that the effectiveness of delayed events is increased through administration of these particular drugs.

An increasing amount of research concerning the effects of psychostimulants (i.e., cocaine and *d*-amphetamine) on self-control in non-humans has been conducted. For example, Logue et al. (1992) examined these behavior changes using an adjusting-delay procedure. It was found that indifference points decreased significantly under chronic administration of 15 mg/kg cocaine. That is, the rats chose the smaller, more immediate reinforcer much more frequently, thus illustrating impulsive behavior. Charrier and Theibot (1996) studied the effects of psychotropic drugs on self-control in rats using a discrete-trials procedure. They found decreased self-control choices after moderate doses of *d*-amphetamine (0.25 – 1.0 mg/kg) compared to that during non-drug sessions. Moreover, Evenden and Ryan (1996) used a lever-pressing procedure with rats to determine the effects of *d*-amphetamine. Rats could choose between receiving one food pellet immediately and three or five food pellets at varying delays (in increasing order within a session). Doses of 0.3 and 1.0 mg/kg *d*-amphetamine were administered, and it was demonstrated that 1.0 mg/kg significantly decreased choices of the larger reinforcer, suggesting a decrease in self-control (an increase in impulsivity). It seems then, that the data concerning behavioral effects on self-control suggest psychostimulants increase sensitivity to delay. However, as further research shows, this is not always the case.

More recent data suggest the opposite effect when evaluating drug effects on self-control. Using an adjusting-amount procedure, Richards, Sabol, and de Wit (1999) observed an increase in impulsive behavior only after a chronic post-session dose of 4.0 mg/kg METH. During acute administration of METH, however, doses of 0.5, 1.0, and 2.0 mg/kg decreased impulsive behavior. In an adjusting-amount procedure, the animals choose between a smaller, more immediate reinforcer and a larger, more delayed reinforcer. However, the smaller alternative is an immediate adjusting amount of water, whereas the larger alternative is a delayed fixed amount of water. In this case, choosing the immediate adjusting (smaller) amount of water would be an impulsive choice and choosing the delayed fixed (larger) amount of water would be a self-control choice. The amount of water is adjusted for the immediate choice to determine indifference points at which animals will choose both alternatives equally. If, after drug administration, indifference points decrease, impulsivity is said to have increased. On the other hand, if indifference points increase, impulsivity is said to have decreased. Wade, de Wit, and Richards (2000) found amphetamine (0.5 and 1.0 mg/kg) increased indifference points, indicating increased choices allocated to the larger (delayed fixed amount of water) alternative. Other research using a somewhat different procedure has provided what are considered similar results regarding effects of *d*-amphetamine and impulsive behavior (de Wit, Crean, & Richards, 2000; Feola, Richards, & de Wit, 2000). In these studies, a discrete-trials procedure known as the "stop task" was implemented. The stop task is a procedure designed to measure "behavioral inhibition" (i.e., the ability to inhibit, or stop, an initiated response). In this procedure, the inability to inhibit the initiated response is considered an example of impulsive behavior. It was reported in these studies that *d*-

amphetamine increases the ability to stop an initiated task. Thus, contrary to previous results, these recent data suggest that stimulants such as amphetamines decrease impulsive behavior (i.e., increases preference for a larger, more delayed reinforcer or increases the capacity to cease an initiated response). A number of procedural differences may have contributed to the variation in the results of the more recent studies and those of the earlier studies (e.g., the absence of steady-state procedures and forced choice trials in both the Charrier and Theibot, (1996) and Evenden and Ryan, (1996) experiments). In any event, the prevailing view seems to be that stimulants increase self-control.

Consistent with that view, research concerning methylphenidate (MPD) also seems to suggest an increase in self-control choices. Schroeder, Mann-Koepke, Gualtier, Eckerman, and Breese (1987) demonstrated increased self-control choices in humans with doses of 0.3 and 0.15 mg/kg MPD. Similarly, using an adjusting-delay procedure to measure indifference points, Bullock (1999) reported similar data with pigeons. Self-control choices were shown to increase under 5.6 and 10.0 mg/kg MPD.

In a study recently conducted in our laboratory (Pitts & McKinney, unpublished observations), rats were given a choice between a smaller, more immediate and larger, more delayed reinforcement. The delay for the smaller reinforcer was always 0 s. Delays to the larger reinforcer ranged from 0 – 50 s in an increasing order within sessions. Thus, the delays to both alternatives began at 0 s, but the delay to the larger reinforcer increased within sessions (ending at 50 s). For each delay value, rats were given 5 choice trials. When the delay for both alternatives was 0 s, the larger reinforcer was chosen exclusively. However, as seen in Figure 3, once the delay reached 20 s the

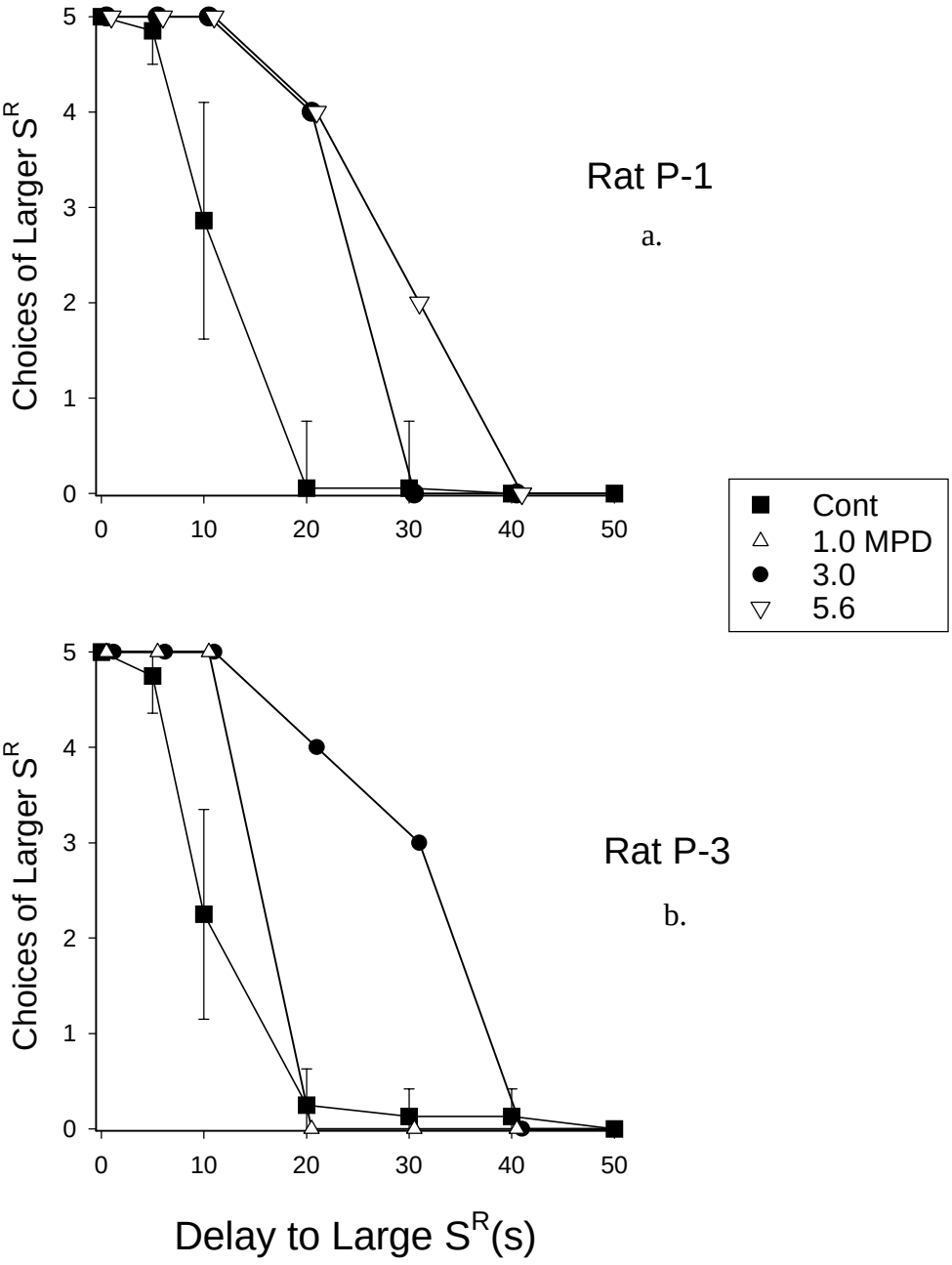

Figure 3. The number of choices maintained by the larger reinforcer as a function of delay to this option. Closed squares represent control performance. Open upward, closed circles, and open downward triangles represent choices maintained by 1.0, 3.0, and 5.6 mg/kg MPD, respectively. Data are presented for subjects P-1 (a) and P-3 (b). Vertical lines represent 95% confidence intervals for control performance.

choice of the larger reinforcer was abandoned and the smaller, more immediate reinforcer was chosen. The interesting result here is when doses of 1.0, 3.0, and 5.6 mg/kg MPD were administered, the rats chose the larger reinforcer at considerably longer delays than under control conditions. MPD administration shifted the functions rightward (Figure 3), indicating these doses increased self-control.

In summary, although the data for amphetamine are somewhat mixed, the results of most of the studies suggest that stimulants increase self-control. The present study will address the question concerning how MPD and METH increase self-control choices with respect to amount and delay contingencies.

The Present Study

Several of the results reviewed above suggest that MPD and METH increase choices to the larger more delayed reinforcer. But what aspect of reinforcement is affected? In other words, what are the behavioral mechanisms of this action? Are these drugs changing effects of delay, such that longer delays are more readily "tolerated"? On the other hand, are they altering effects of amount, such that the larger reinforcer becomes relatively more effective? Most investigators have suggested that stimulant effects on self-control are due to changes in the effects of delay (e.g., Wade et al., 2000). However, other literature suggests the possibility of an alternative account. For example, Heyman (1992) used a version of the matching law applied to single VI reinforcement schedules to characterize MPD's effects. This version is as follows:

$$B=kR/(R + R_e) \qquad (5)$$

where B is response rate, R is reinforcement rate and k and R_e are two free parameters. In this case, changes in *k* (i.e., asymptote) were interpreted as changes in motor

performance, whereas changes in R_e (i.e., rate at which asymptote is reached) were interpreted as changes in reinforcement efficacy. Rats were exposed to various VI schedules (ranging from 3 s to 108 s). This in turn produced varying reinforcement rates (ranging from 30 to 1,100/hr). Overall, it was demonstrated that MPD, particularly 1.0 and 2.0 mg/kg, decreased R_e without affecting *k*. This effect is similar to that produced by increasing reinforcement magnitude. Therefore, reinforcement variables altered by MPD may not be limited to effects on delay of reinforcement, but may also include effects on amount of reinforcement. It is known from the matching law that increasing the amount for an option shifts choice to that option. It is also known that decreasing the delay for an option increases choice for that option. Again, the primary question concerns identifying these drugs' behavioral mechanisms on self-control. None of the previous results demonstrate conclusively which aspects of reinforcement are modulated. The present study attempts to identify whether the sensitivity to reinforcement delay and/or amount is being changed by MPD and METH.

In the present study, pigeons performed key-pecking responses in an operant chamber to assess their distribution of self-control and impulsive choices. All pigeons chose between larger and smaller reinforcers. The delay associated with the smaller reinforcer remained constant throughout sessions, while the delay associated with the larger reinforcer increased across the session to obtain within-session delay-discount functions. A single random interval (RI) 1 min schedule was used. A variation of the logarithmic version of the matching law was used to quantify MPD and METH's effects on the distribution of responses at each delay (see data analysis, equation 7). The present study employed two free parameters, both mathematically derived from the data. One

parameter was used to estimate changes in the sensitivity to effects of delay (S_D) and the other parameter estimated changes in the sensitivity to effects of amount (S_A). Drug induced changes in these parameters were subjected to analysis to determine if they might possibly serve as behavioral mechanisms of drug action.

METHOD

Subjects

Four experimentally naïve male White Carneau pigeons (*Columba livia*), designated 1985, 1863, 1809, and 1845, served as pigeons. All pigeons were housed individually in a colony room (70 – 75 degrees F) operating under a 12-hr light/dark cycle. Free access to water and health grit was provided. Initially, free access to mixed grain was provided to all pigeons. After obtaining stable weights for 5 consecutive days, mean weights for each pigeon were calculated. From those mean weights, 80% body weights were obtained. Afterward, pigeons were maintained at these 80% body weights through experimenter-regulated access to the mixed grain for the entire study.

Apparatus

The two experimental operant chambers (BRS/LVE, Inc. model SEC-002) used were 35.0 cm deep by 30.5 cm wide by 36.0 cm high. On one wall of each chamber were three response keys, horizontally arranged, spaced 8.5 cm apart (center to center), 2.5 cm in diameter, and 26 cm from the floor. Each side key measured 9.0 cm from its adjacent wall. The keys could be trans-illuminated red, yellow, or green; approximately 0.25 N of force was needed to activate each key. A 1.2-watt white houselight was located 6.5 cm directly above the center key. Located 5 cm to the left of the white houselight was a green houselight and located 5 cm to the right of the white houselight was a red

houselight. A 5.0 by 6.0 cm aperture, through which mixed grain could be obtained, was located on the same wall 11.0 cm directly below the center key. A solenoid-operated food hopper provided timed access to mixed grain. When grain was presented, all key lights and houselights were off, and a white light illuminated the opening. Each chamber was equipped with a ventilation fan and white noise was present in the room to mask extraneous sounds during operating hours in the experimental chamber. A computer using MED-PC® 2.0 software and MED® Associates interfacing (Georgia, VT) collected data and controlled experimental programs. This computer was located in an adjacent room.

Behavioral Procedure

Preliminary Training

Following adaptation to the chamber, all pigeons were magazine trained. Pecking the center key then was shaped through differential reinforcement of successive approximations. During this training, the center key was illuminated yellow. Daily sessions were conducted for two days under an FR 1 schedule, in which each peck to the center key resulted in 3.5 s access to grain. Sessions terminated after the 30^{th} food presentation.

After pecking the center key was established, the side keys were illuminated and operative. Sessions were conducted for two consecutive days, each consisting of 30 trials. One side key was illuminated per session. The color of the illuminated key was randomly determined from trial to trial. A peck to the illuminated side key delivered 3.5 s access to food under FR 1. A 10 s blackout period, during which the chamber was dark, followed each reinforcer delivery.

In the next two daily sessions, both side keys were operative and each side key could be illuminated red or green. Key color was randomly determined on a trial-by-trial basis. Sessions operated under a multiple FR1 FR1 schedule, where each side key was associated with an independent FR1 schedule. Thus, there were two components: a left-side key component (red or green) and a right-side key component (red or green). Components were presented sequentially and each component was activated for three reinforcers. Each condition (red left, red right, green left, green right) was presented 10 times in random order.

Pigeons then were randomly assigned to key color/position conditions. As a result, two pigeons experienced red on the left side key and green on the right side key. The other two pigeons experienced green on the left side key and red on the right side key. Once randomly assigned, these conditions remained unchanged for the entire study.

During the remaining training sessions, a red key and a green key were illuminated simultaneously. A single RI 1 min schedule was introduced. Over successive sessions, the RI increased from 2 s to 1 min. Therefore, on average, reinforcement was available once every minute. Under this schedule, reinforcement availability was determined by a probability gate pulsed at every second of each session (except during reinforcement). For each second, the probability that reinforcement would be available for the next response was .0167 (1/60). Once reinforcement was set up, a .5 probability determined whether reinforcement was delivered after a peck to the red key or the green key (Figure 4). A 5 s changeover delay (COD) was in effect to prevent a peck on either key from being reinforced within 5 s of a changeover from a peck on the opposite key. A post-reinforcement timeout (60 s – [delay + amount]) following reinforcement delivery

Behavioral Procedure

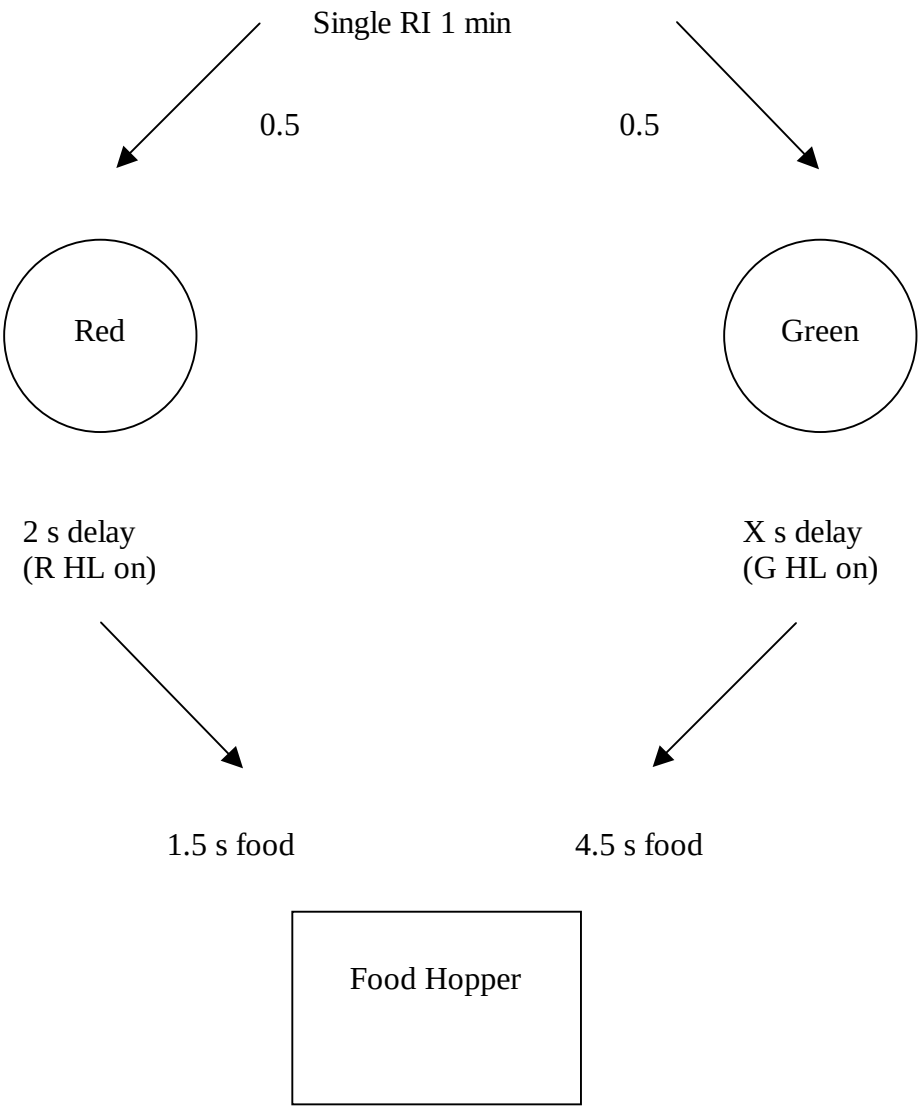

Figure 4. Diagram of the experimental procedure. Key light and houselight colors were counterbalanced across pigeons.

was also in effect. During this timeout the chamber darkened. A 2 s delay was associated with both the smaller reinforcer (1.5 s access to mixed grain) and larger reinforcer (4.5 s access to mixed grain). For each reinforcement amount, the .5 s was included to allow the pigeon time to bring its head to the food hopper (see Epstein, 1981). For the remainder of this paper, the values 1 s and 4 s will be used to represent the reinforcement amounts used in this study. During the delays, all lights were turned off except for the colored houselight that corresponded with the illuminated key color. Key-color position and the key corresponding to the larger reinforcer were counterbalanced across pigeons and held constant throughout the experiment. Therefore, each pigeon was randomly assigned to one of the following four conditions: (a) left key red and right key green with the larger reinforcer corresponding to the left key (1985), (b) left key red and right key green with the larger reinforcer corresponding to the right key (1809), (c) left key green and right key red with the larger reinforcer corresponding to the left key (1863), and (d) left key green and right key red with the larger reinforcer corresponding to the right key (1845).

Experimental Procedure

After stable responding on the RI 1 min schedule with 2 s delay for both alternatives was obtained (and preference was shown for the larger reinforcer), a within-sessions delay manipulation was added. Sessions were blocked into five, 10 min time segments (excluding delay time, reinforcement time, and post-reinforcement blackout time). The signaled delay to the smaller reinforcer remained 2 s throughout the study, however, the delay to the larger reinforcer increased within sessions, in which delays of 2 s, 10 s, 20 s, 30 s, and 40 s were programmed across these five, 10 min blocks. Thus,

the delay ratio (D_L/D_S) across these five, 10 min blocks was 1, 5, 10, 15, and 20. This way, an entire delay-discount function was obtained within each session. A blackout period of 75 s was also programmed between each block. During this period all lights inside the chamber darkened. Sessions were conducted 5 days per week (Monday through Friday). Stability criterion included visual inspection of the data. Behavior was considered stable when data showed minimal variability and no trends for 10 consecutive sessions.

Pharmacological Procedure

Once stability was obtained on the behavioral procedure, MPD and METH were administered acutely prior to selected sessions through intra-muscular injections to the breast region in a volume of 1.0 ml/kg. Doses of MPD ranged from 1.0 to 17.0 mg/kg (expressed in terms of the total salt) and doses of METH ranged from 0.3 to 3.0 mg/kg; saline was also administered. At least two determinations of each drug dose and saline were conducted. Effects of doses were determined in a mixed order, with the constraint that no dose was given a second time until all doses were given once. Each determination of a dose effect curve was preceded by an assessment of the effects of saline.

The injection area alternated between the left and right breast muscle. Injections were administered approximately every Tuesday and Friday, provided data obtained the preceding day were within the range of the preceding 10 non-injection sessions. Injections took place 15 min prior to the sessions. Control sessions were defined as the sessions immediately prior to an injection session. MPD was administered first, followed by METH. A 30-day “washout” period was included, during which sessions were

conducted as usual, with the exception that pigeons were not exposed to any drug administrations.

Data Analyses

Overall response rates maintained by the larger and smaller reinforcers were calculated for the last 5 min of each session block (number of responses/ 5 min). Data obtained during the last 5 min of each 10 min block were used for data analysis. This particular sample of data was used for analysis to help insure stable behavior in each session component. The following equation, based on the matching law, served as the starting point for the analysis of preference:

$$B_L/B_S = (A_L/A_S)^{SA} / (D_L/D_S)^{SD} \quad (6)$$

where B_L/B_S denote the ratio of responses, A_L/A_S denote the ratio of amount (4), and D_L/D_S denote the ratio of delays (as the delay to the large reinforcer increased, the ratio increased). The terms S_A and S_D represent the sensitivity of the response ratio (B_L/B_S) to effects of amount and delay, respectively.

If the sensitivity to amount is relatively large, a given change in the ratio of amount (A_L/A_S) would provide a relatively large change in the ratio of responses (B_L/B_S). If the sensitivity to delay was relatively large, a given change in the ratio of delay (D_L/D_S) would provide a relatively large change in the ratio of responses (B_L/B_S). Therefore, the larger parameter would indicate which variable had a greater effect.

According to Equation 6, an increase/decrease in the amount ratio (A_L/A_S) would increase/decrease the response ratio (B_L/B_S). Furthermore, an increase/decrease in the delay ratio (D_L/D_S) would decrease/increase the ratio of responses (B_L/B_S). As the delay (D_L) to the larger reinforcer (D_L) increases, responding to the option delivering the larger

reinforcer would decrease. On the other hand, if the delay to the larger reinforcer (D_L) decreases, responding to the option delivering the larger reinforcer would increase. Here, amount has a positive effect, whereas delay has a negative effect. For the present study, equation 6 was logarithmically transformed:

$$\log(B_L/B_S) = S_A\log(A_L/A_S) - S_D\log(D_L/D_S) \qquad (7)$$

When data are logarithmically transformed, typical delay-discount functions appear as straight lines. In this way it becomes easier to separate and further determine how changes in sensitivity to amount and/or delay modulated behavior.

Figure 5 shows a hypothetical delay-discount function based upon equation 7. In accordance with the present study, the amount and delay values were represented by the following: A_L=4, A_S=1, D_L=2-40 s, and D_S=2 s. In Figure 6 (the top graph), the sensitivity to amount is hypothetically increased from 1 to 2, shown by the dotted line. This dotted line is characterized by an upward move and a change in the y-intercept. In other words, when the sensitivity to amount is changed (Figure 6, top graph), there appears to be a constant proportional change in the ratio of responses (B_L/B_S) at all values of the delay ratio (D_L/D_S). Thus, there is a change in the y-intercept, but not the slope. In contrast (Figure 6 the bottom graph), the sensitivity to delay is hypothetically decreased from 1 to .75, also shown by a dotted line. When the sensitivity to delay is altered (Figure 6, bottom graph), a change in the slope but not the y-intercept occurs. Thus, proportional change in the response ratio (B_L/B_S) is greater as the delay ratio (D_L/D_S) increases.

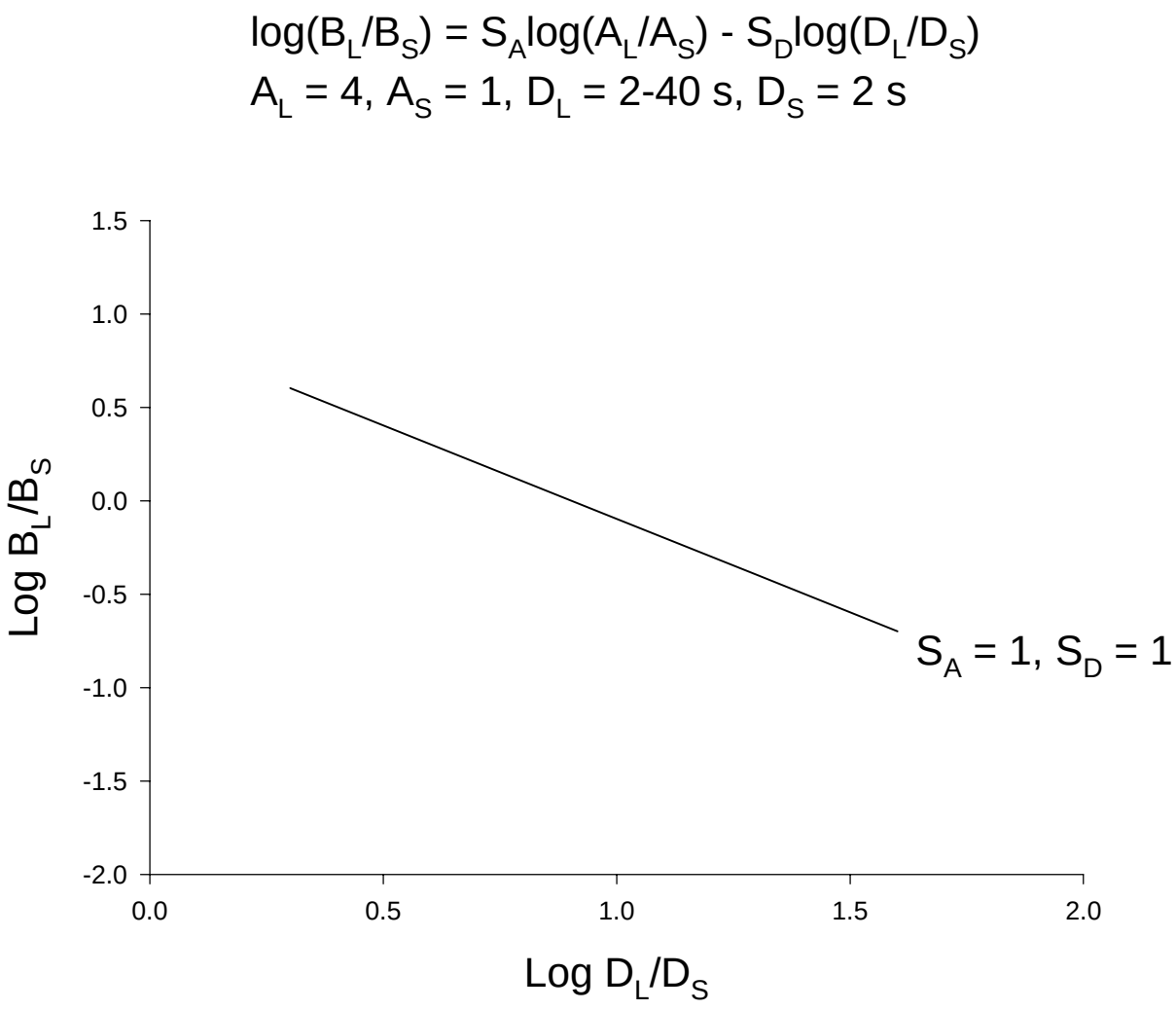

Figure 5. Logged response ratios plotted as a function of logged delay ratios when the sensitivity to amount and delay are both 1. In this example, $A_L = 4$, $A_S = 1$, $D_S = 2$ s, and $D_L = 2$ to 40 s.

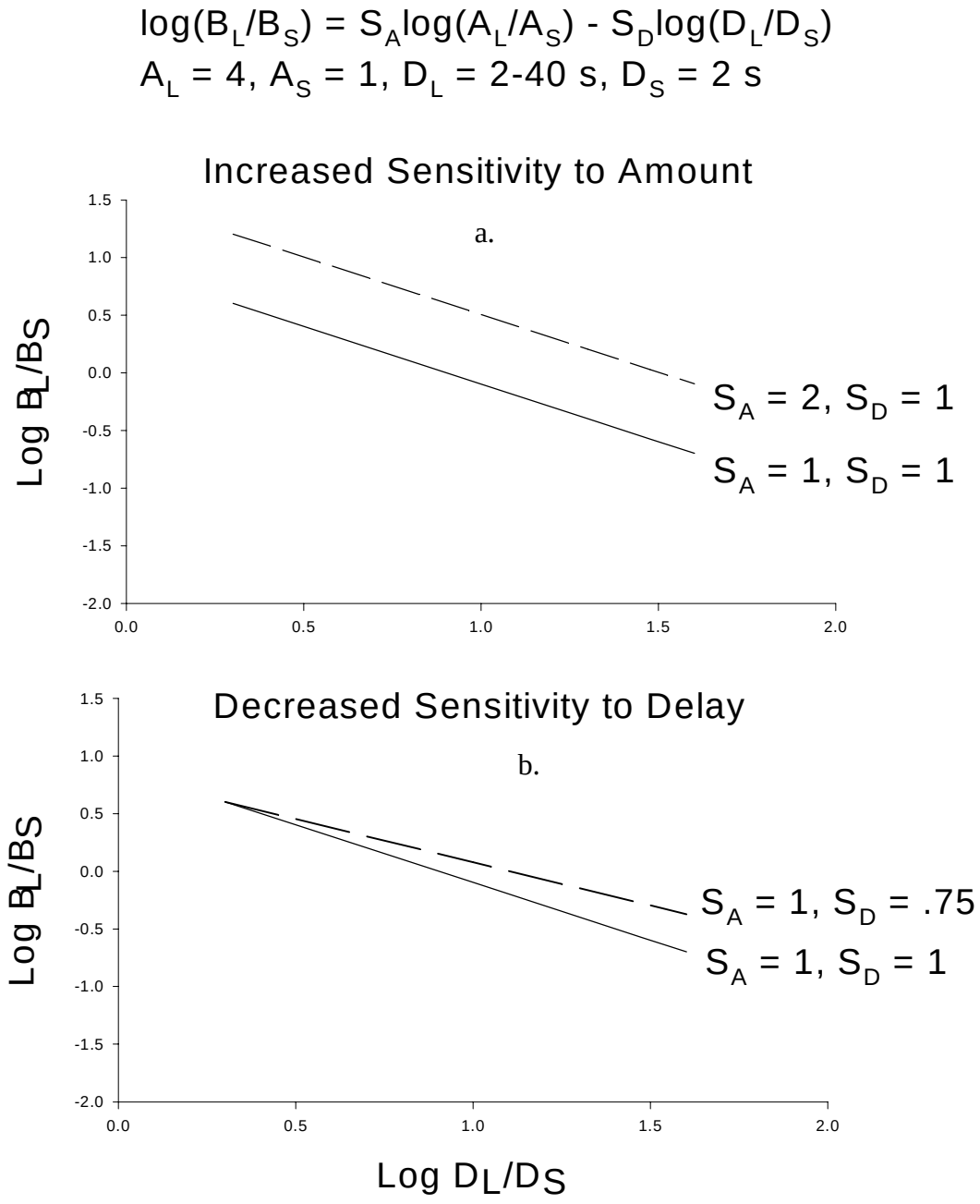

Figure 6. Logged response ratios as a function of logged delay ratios. The solid line represents the function when S_A and S_D are both equal to 1. Top panel (a): The dotted line represents this same function when S_A is increased to 2 while S_D remains 1. Bottom panel (b): The dotted line represents the function when S_D is decreased to .75 and S_A remains 1.

In the present study, a value of B_L/B_S was obtained at each value of D_L/D_S for each pigeon. Using the logged equation (equation 7), a quantitative analysis was then carried out by fitting a regression line to the data points. Values of S_A and S_D were obtained under non-drug conditions and under the doses of MPD and METH. Slope and y-intercept values for each session were calculated and compared in the non-drug and drug conditions to observe changes in either measure. According to this analysis, a drug-induced change in the y-intercept would be interpreted as a change in the sensitivity to amount. In contrast, a drug-induced change in the slope would be interpreted as a change in the sensitivity to delay.

For the present study, data were summarized in two ways. First, response rates that were calculated for both options over the last 5 min of each component were averaged for control, saline, and each dose. Using those averaged response rates, response ratios (large/small) were calculated for each component for control, saline, and each drug dose. These ratios were then subjected to analysis by the logged equation; slope and y-intercept values were determined for each condition, along with corresponding r^2 values. Second, in order to characterize the day-to-day variation in the discount functions within pigeons, and the changes produced by each drug dose, individual discount functions for each session were calculated. In other words, for each pigeon, a response ratio (L/S) was calculated for each component for each control, saline and drug session. These ratios (from each session) were then analyzed using the logged formula (equation 7). Average slope and y-intercept values were then obtained for control, saline and each dose. These were used to construct dose-effect functions for MPD and METH.

Finally, rate-dependent effects were assessed using the response rates for the larger and smaller options in each component (last 5 min) under control and after each drug dose. These response rates were averaged for each component in control and drug conditions. The average values for each component under control conditions were used as the "control rate". In order to determine how drug administration affected responding on each option, the average response rate obtained after each drug dose was divided by the average control rate. The resulting value was then multiplied by 100 (percent control rate). Once these values were calculated, the percent control rate was plotted as a function of control rate on log/log coordinates.

RESULTS

Control Performance

Figure 7 presents delay-discount functions obtained during control sessions prior to MPD injections for each pigeon. Data averaged for all four pigeons are also presented. These graphs show the ratio of responses (L/S) as a function of the ratio of delays (L/S). A ratio greater than 1.0 indicates more responses on the option associated with the larger reinforcer; a ratio of less than 1.0 indicates more responses on the option associated with the smaller reinforcer.

In general, a decreasing, negatively decelerating function was obtained for all pigeons. All pigeons except 1809 behaved in a way to produce a ratio greater than 1.0 in the beginning of the session (when delays were equal). Pigeons 1863, 1845, and 1985 chose the larger option between 1.8 and 3.0 times more often at the start of the session. As the session progressed (the delays to the larger reinforcer increased), choices on the larger option decreased to produce a ratio that approached 1.0. For 1809, the response

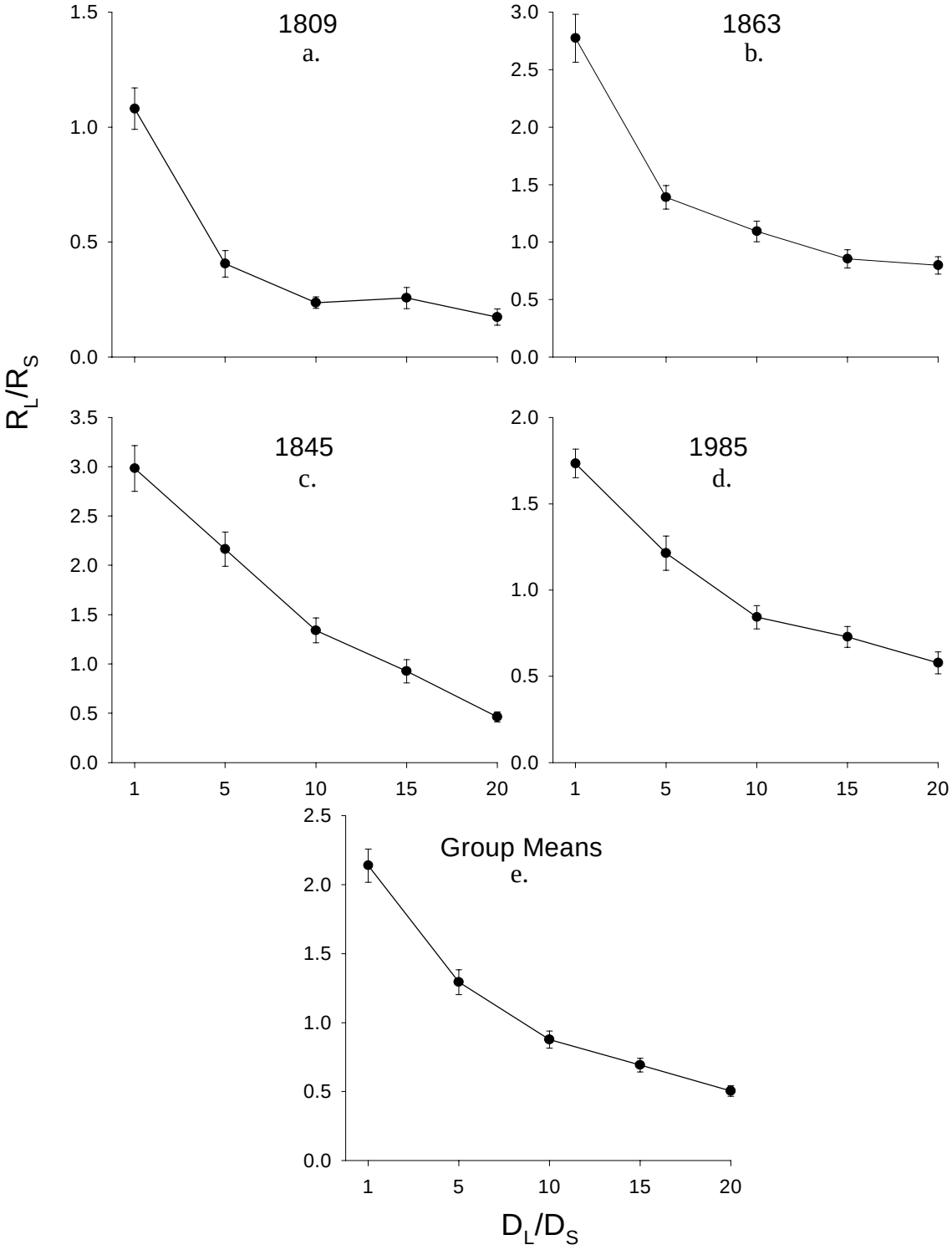

Figure 7. Response ratios (L/S) plotted as a function of delay ratios (L/S) for sessions prior to a MPD injection (16-20 sessions). Each data point represents an averaged response ratio with each increasing delay for the larger reinforcer. Data are presented for 1809 (a), 1863 (b), 1845 (c), 1985 (d), followed by group means (e). Vertical lines represent standard error of the mean. Note different y-axis scales for individual plots.

ratios obtained across all blocks were the lowest of all four pigeons. In 1809's case, a large number of responses were still allocated to the smaller reinforcer, even when delays to both options were equal, indicating a bias for the smaller reinforcer.

Figure 8 shows logged response ratios as a function of logged delay ratios for each pigeon. In each graph, regression lines were fit to the data using the method of least squares; r^2 values are also presented. A value of 0.0 on the y-axis represents indifference between the two behavioral options. With this logarithmic transformation, the response ratio becomes a linear, decreasing function of the delay ratio for all pigeons. Individual r^2 values ranged between 0.81 and 0.99. The r^2 value for the group function was 0.96.

An analysis of the absolute response rates that composed the delay-discount functions are shown in Figure 9. Figure 9 characterizes response allocation to each behavioral option under control conditions. These graphs show the average responses per minute obtained for both behavioral options as a function of the delay to the larger reinforcer for each pigeon. Response rates averaged for all pigeons are shown following the individual plots. Closed circles represent response rates maintained by the larger reinforcer while open circles represent response rates maintained by the smaller reinforcer.

As indicated in Figures 7 and 8, three of four pigeons (1863, 1845, and 1985) allocated more responses on the option associated with the larger reinforcer during the first block, when the delays to both options were equal (2 s). As the session continued, and the delay to the larger reinforcer increased, response rates maintained by the larger reinforcer decreased for all pigeons. Pigeons 1863, 1845, and 1985 showed a

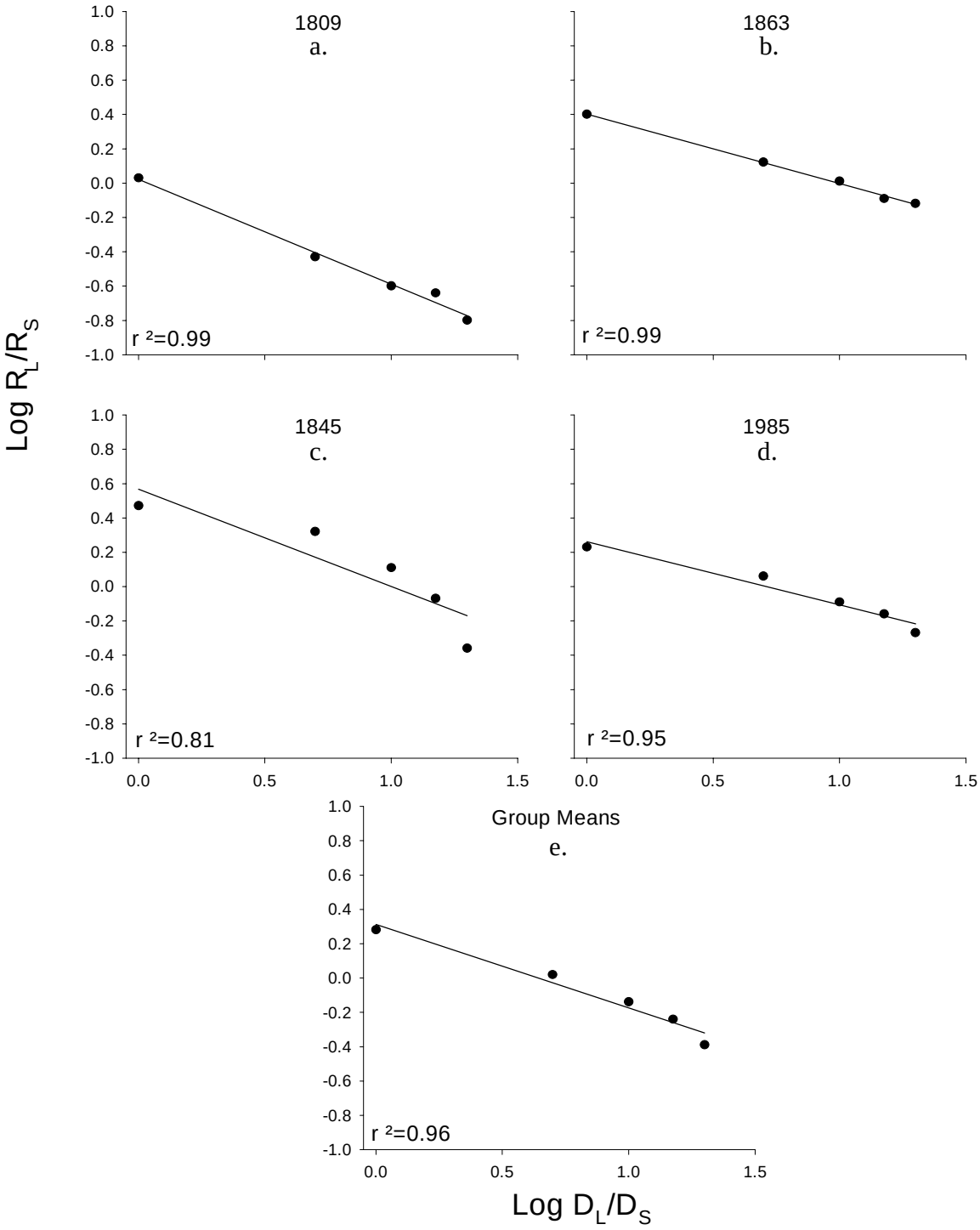

Figure 8. Logged response ratios plotted as a function of logged delay ratios. The logged average response ratios for sessions prior to a MPD injection (16-20 sessions) are presented for each pigeon (a through d), followed by group means (e). Regression lines fit to the data using the method of least squares are presented along with corresponding r^2 values.

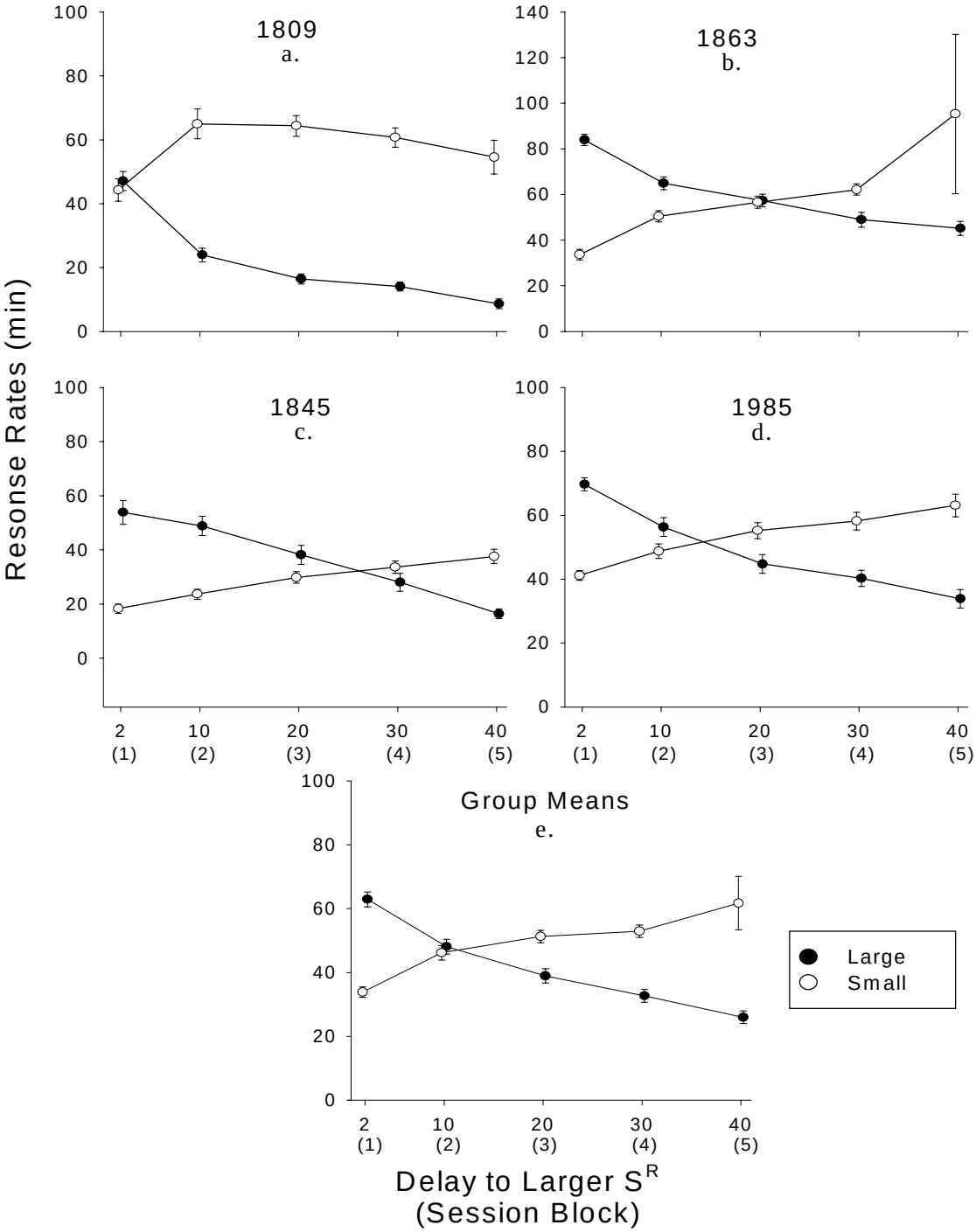

Figure 9. Response rates (min) plotted as a function of the delay to the larger reinforcer for each of the five session blocks. Data points are averaged response rates maintained by both reinforcers for those sessions prior to a MPD injection (16-20 sessions). Data are presented for 1809 (a), 1863 (b), 1845 (c), 1985(d), and group means (e). Vertical lines represent standard error of the mean. Closed circles represent choices maintained by the larger reinforcer, while open circles represent choices maintained by the smaller reinforcer. Note different scales for individual plots.

corresponding increase in response rates maintained by the smaller reinforcer across blocks. Pigeon 1809 chose the smaller reinforcer more often throughout most of the session. Although response rates maintained by both options were comparable in block 1, the response rates maintained by the smaller reinforcer increased in block 2, when the delay to the larger reinforcer increased to 10 s. For the remainder of the session, rates maintained by the smaller option stayed relatively constant for this pigeon.

Effects of Methylphenidate (MPD)

Figure 10 shows effects of the intermediate doses of MPD (3.0, 5.6, and 10.0 mg/kg) using the log-ratio plots. Closed circles represent data from control sessions and open symbols show data obtained from drug sessions. Squares, upward triangles, and inverted triangles show effects of 3.0, 5.6, and 10.0 MPD, respectively. In general, 1.0 mg/kg produced negligible effects and 17.0 mg/kg produced substantial decreases in overall responding, and thus, data for these doses are not shown here. The logged response ratios for all MPD doses and saline are presented in Table 1.

The upper panels of Figure 10 show that 1809 and 1863 chose the larger reinforcer more often at longer delays following drug administration compared to control sessions. For 1809, MPD decreased the slope to a large extent following all doses, although this effect did not appear systematically related to dose. The decrease in slope for this pigeon was accompanied by a substantial decrease in y-intercept following 3.0 mg/kg. For 1863, MPD also decreased the slope. This effect occurred without a change in y-intercept. For 1845 and 1985, the primary effect of MPD was to produce a decrease in the y-intercept. That is, the L/S ratios decreased at the smaller delay ratios, but were relatively unchanged at larger delay ratios. For 1845, the decrease in the y-intercept

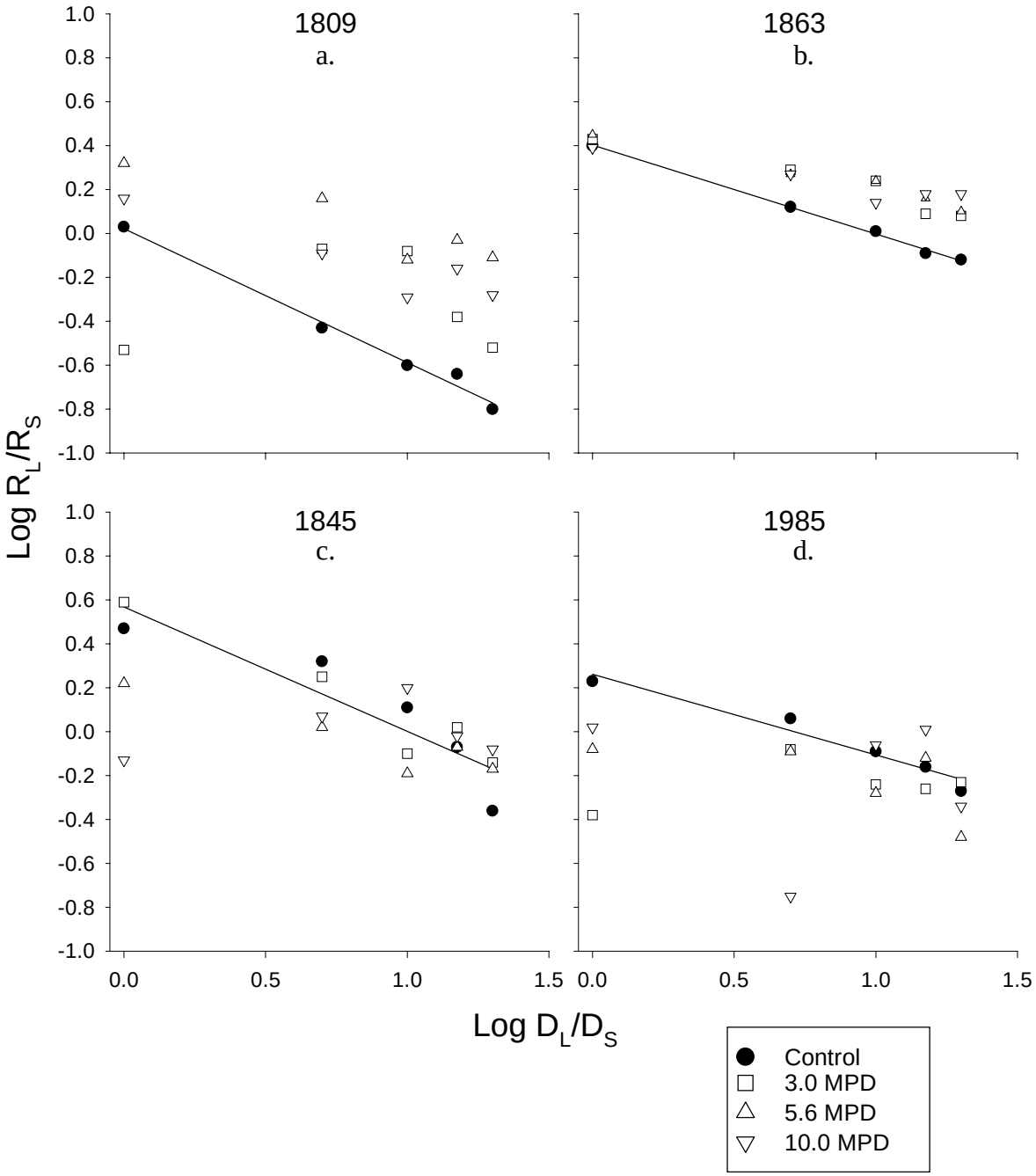

Figure 10. Logged response ratios plotted as a function of logged delay ratios. Data points represent mean values for 1809 (a), 1863 (b), 1845 (c), and 1985 (d). Each data point for control is a mean of 16-20 sessions. Each data point representing a MPD dose is a mean of 2-4 determinations. Closed circles represent control performance and open symbols represent MPD performance. Squares, upward triangles, and inverted triangles represent 3.0, 5.6, and 10.0 mg/kg MPD respectively. Regression lines were fit to the data using the method of least squares. The corresponding r^2 values for 1809, 1863, 1845, and 1985 were .99, .99, .81, and .95 respectively.

		MPD Dose (mg/kg)						
Subject/Block		Control	Saline	1.0 mg/kg	3.0 mg/kg	5.6 mg/kg	10.0 mg/kg	17.0 mg/kg
1809								
	1	.03	-.07	-.07	-.53	.32	.16	-.32
	2	-.43	-.46	-.18	-.07	.16	-.09	-1.04
	3	-.60	-.62	-.46	-.08	-.12	-.29	-1.09
	4	-.64	-.69	-.65	-.38	-.03	-.16	-.72
	5	-.80	-.71	-.64	-.52	-.11	-.28	-.83
1863								
	1	.40	.32	.31	.43	.45	.39	1.55
	2	.12	.22	.10	.29	.27	.27	.47
	3	.01	.07	.08	.24	.24	.14	.15
	4	-.09	-.07	-.21	.09	.16	.18	.51
	5	-.12	-.19	-.34	.08	.10	.18	.15
1845								
	1	.47	.44	.30	.59	.22	-.13	-1.08
	2	.32	.16	.04	.25	.02	.07	.67
	3	.11	.01	-.12	-.10	-.19	.20	1.32
	4	-.07	-.05	-.04	.02	-.07	-.02	-.18
	5	-.36	-.16	-.08	-.14	-.17	-.08	-.48
1985								
	1	.23	.21	-.05	-.38	-.08	.02	-.49
	2	.06	.02	-.19	-.08	-.09	-.75	-.04
	3	-.09	.00	-.36	-.24	-.28	-.06	-.90
	4	-.16	-.11	-.41	-.26	-.12	.01	.20
	5	-.27	-.29	-.38	-.23	-.48	-.34	-.23

Table 1. Mean logged response ratios for each subject for control, saline, and MPD sessions.

appeared to be an increasing function of dose. Thus, for all pigeons, MPD decreased the steepness of the slope. For 1809 and 1863 this occurred mainly as an effect of increasing choices of the option maintained by the larger reinforcer at longer delays. For 1845 and 1985, this effect was mainly due to a decrease in choices of the larger option during the first block (i.e., a decrease in y-intercept).

Figure 11 shows dose-effect curves for slopes (left) and y-intercepts (right) for each pigeon at control, saline and MPD doses (note that slopes are absolute values). This figure shows that the primary effect of MPD for 1809 and 1863 was to decrease the slope. For both 1809 and 1863, at least one MPD dose decreased the slope without changing the y-intercept. Although a saline effect was observed with 1863 for slope, the slope values obtained following drug administration were compared to values maintained under control conditions. For 1809, 17.0 mg/kg substantially decreased the y-intercept relative to other doses. For 1863, none of the doses significantly affected the y-intercept. In contrast, the primary effect of MPD for 1845 and 1985 was to decrease the y-intercept. For 1845, the decrease in y-intercept following 1.0, 5.6, and 10.0 mg/kg was accompanied by a slight decrease in slope. For 1985, the y-intercept decreased to a large extent following all doses. A slight decrease in slope accompanied the decrease in y-intercept following 3.0 mg/kg for 1985.

An analysis of the response rates that composed the discount functions are presented in Figures 12 through 16. Figures 12 through 16 characterize response allocation to each behavioral option for control sessions (all sessions prior to a MPD injection) and sessions following administration of 3.0, 5.6, and 10.0 mg/kg MPD.

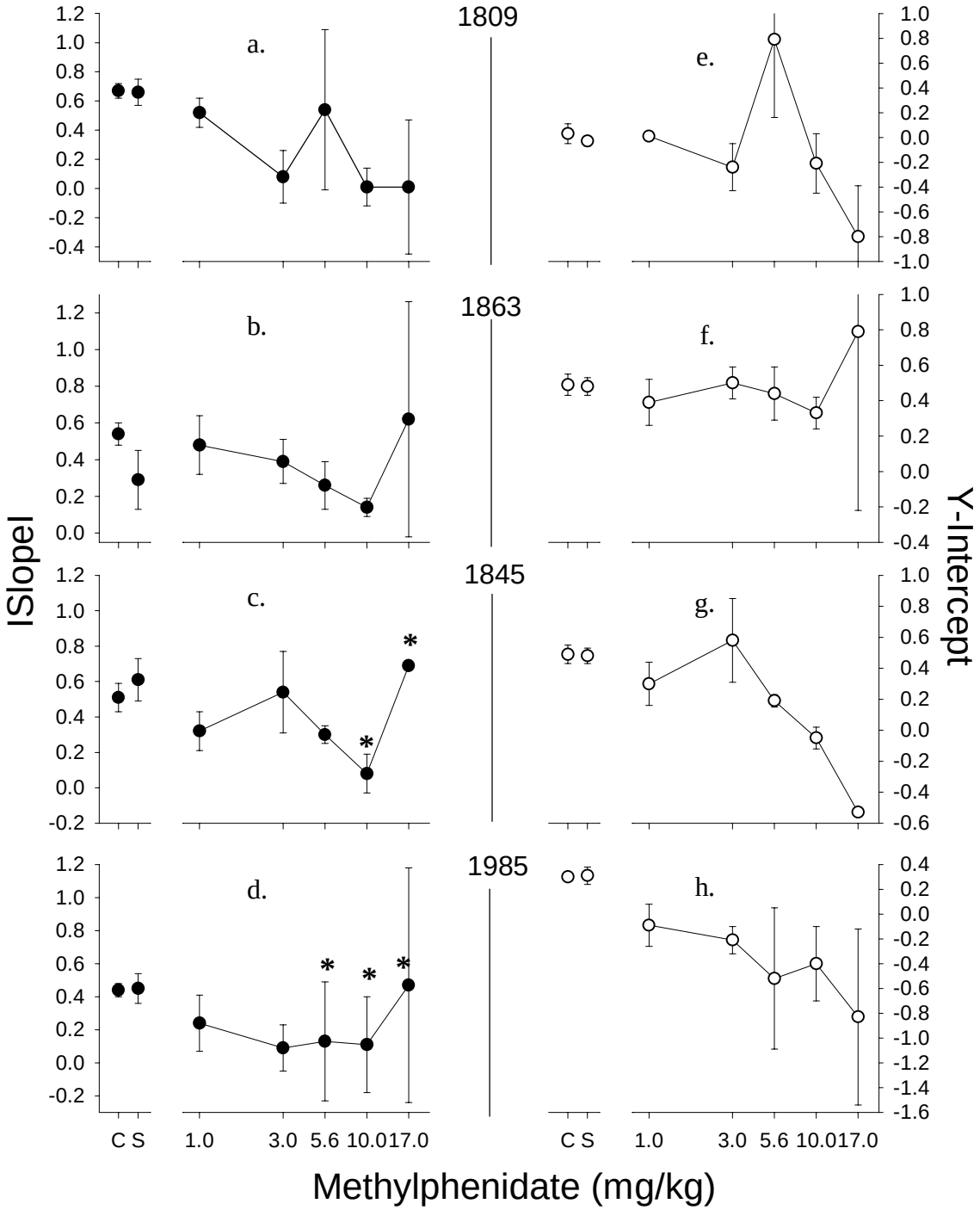

Figure 11. Dose effect functions of slopes (a through d) and y-intercepts (e through h) for control, saline, and all MPD doses. The slope values presented (a through d) are absolute values. Mean values for saline and MPD doses were derived from 2 – 4 determinations (except for 1845 at 17.0 mg/kg which was only administered one time). Mean values for control were derived from 16-20 determinations. Vertical lines represent standard error of the mean. Note that all slopes were negative except for those marked by a *. Also note different y-axes.

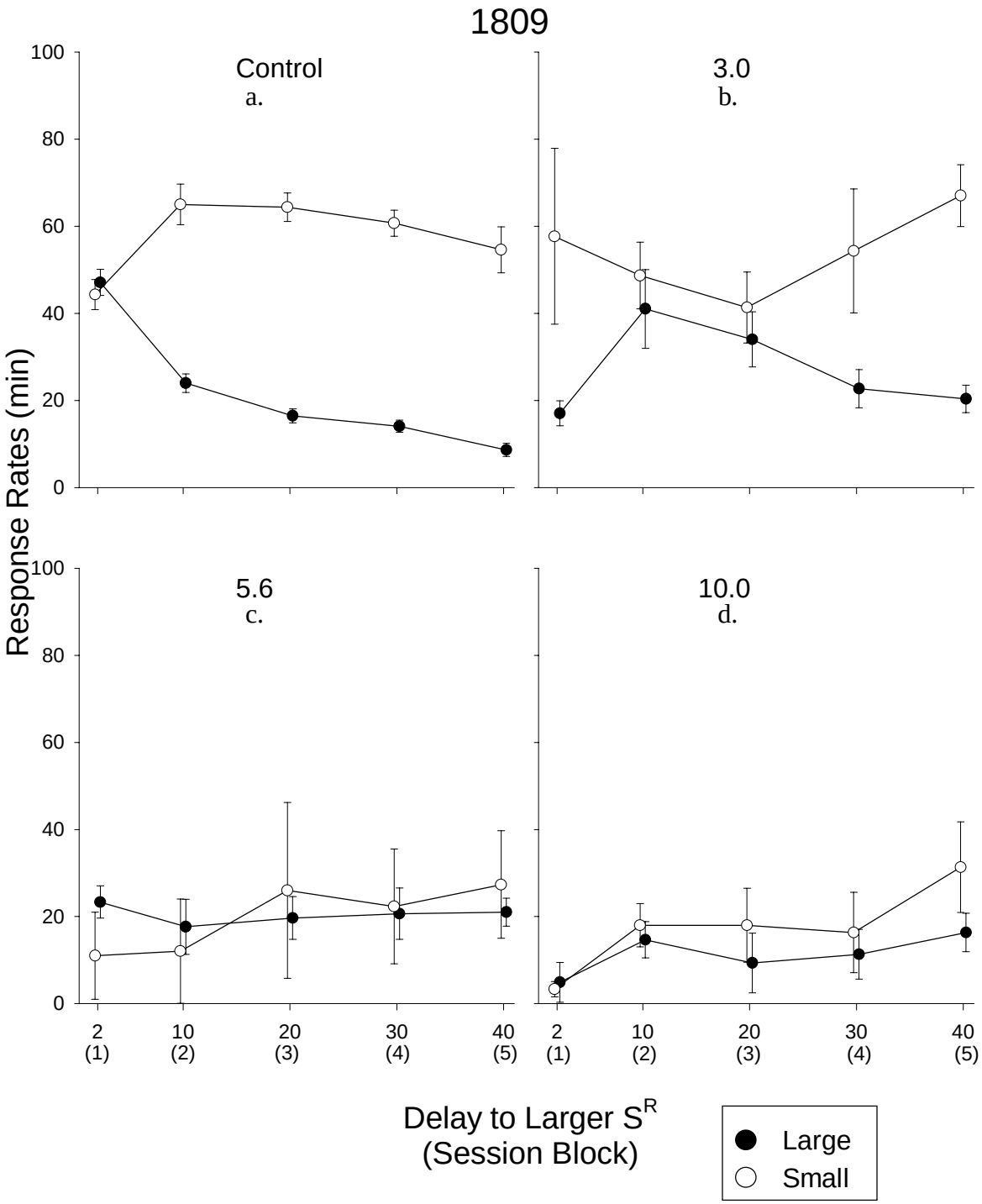

Figure 12. Response rates (min) plotted as a function of the delay to the larger reinforcer for each session block. Data points represent mean values and vertical lines represent standard error of the mean for 1809. Mean values for control were derived from 16-20 sessions. Mean values for MPD doses were derived from 2-4 determinations. Closed circles represent choices maintained by the larger reinforcer and open circles represent choices maintained by the smaller reinforcer. Separate plots are shown for control (a), 3.0 (b), 5.6 (c), and 10.0 (d) mg/kg MPD.

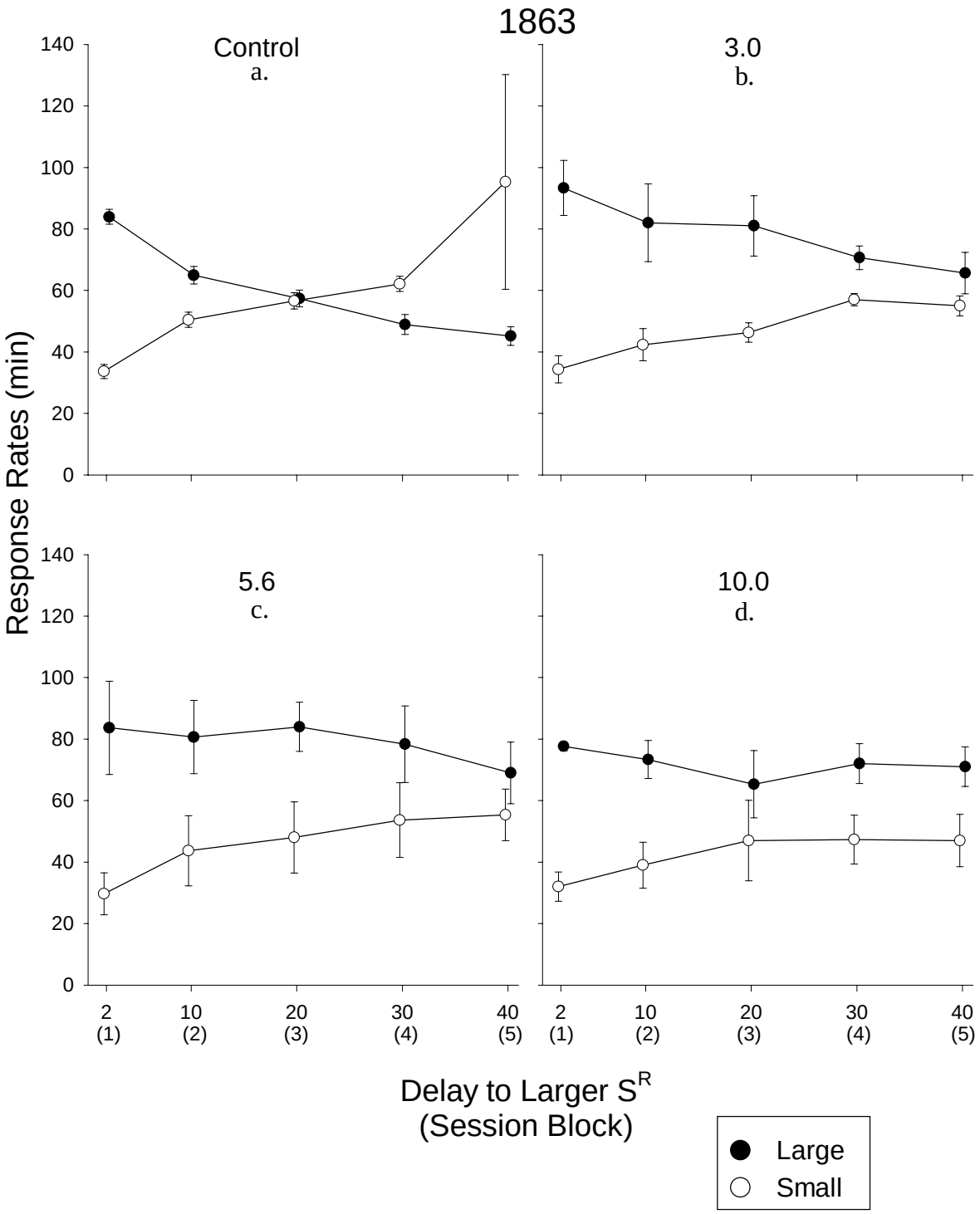

Figure 13. Response rates (min) plotted as a function of the delay to the larger reinforcer for each session block. Data points represent mean values and vertical lines represent standard error of the mean for 1863. Mean values for control were derived from 16-20 sessions. Mean values for MPD doses were derived from 2-4 determinations. Closed circles represent choices maintained by the larger reinforcer and open circles represent choices maintained by the smaller reinforcer. Separate plots are shown for control (a), 3.0 (b), 5.6 (c), and 10.0 (d) mg/kg MPD.

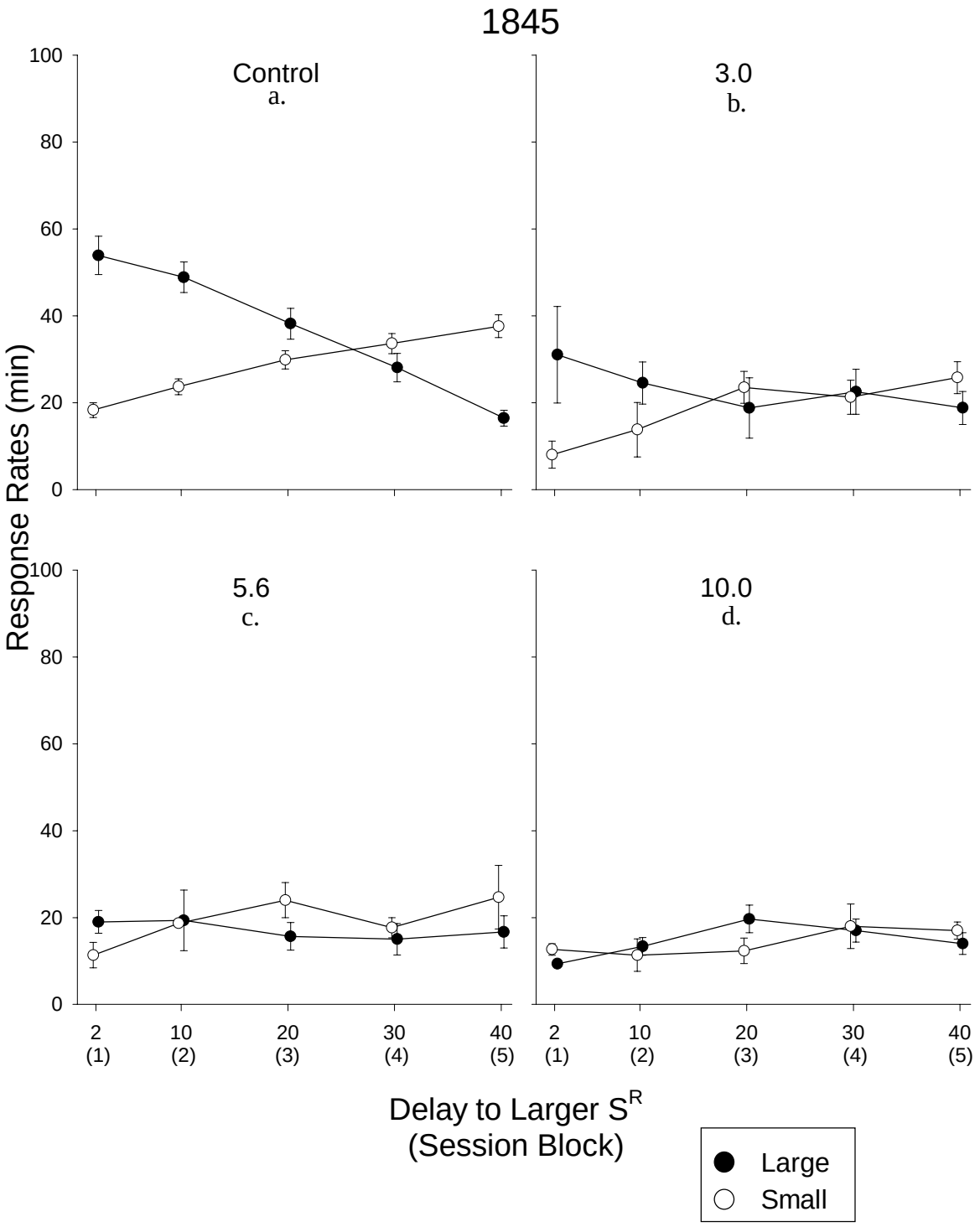

Figure 14. Response rates (min) plotted as a function of the larger reinforcer for each session block. Data points represent mean values and vertical lines represent standard error of the mean for 1845. Mean values for control were derived from 16-20 sessions. Mean values for MPD doses were derived from 2-4 determinations. Closed circles represent choices maintained by the larger reinforcer and open circles represent choices maintained by the smaller reinforcer. Separate plots are shown for control (a), 3.0 (b), 5.6 (c), and 10.0 (d) mg/kg MPD.

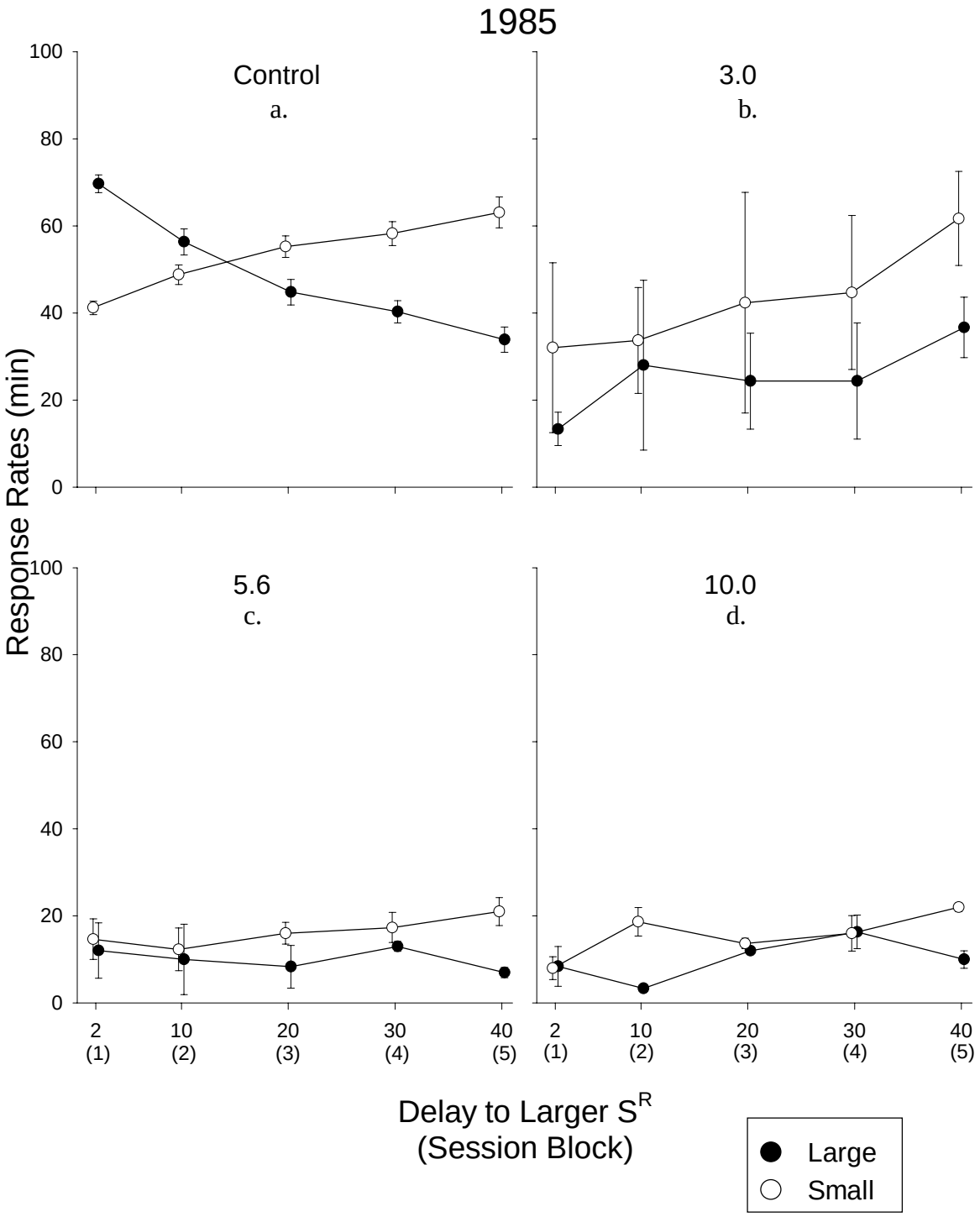

Figure 15. Response rates (min) plotted as a function of the delay to the larger reinforcer for each session block. Data points represent mean values and vertical lines represent standard error of the mean for 1985. Mean values for control were derived from 16-20 sessions. Mean values for MPD doses were derived from 2-4 determinations. Closed circles represent choices maintained by the larger reinforcer and open circles represent choices maintained by the smaller reinforcer. Separate plots are shown for control (a), 3.0 (b), 5.6 (c), and 10.0 (d) mg/kg MPD.

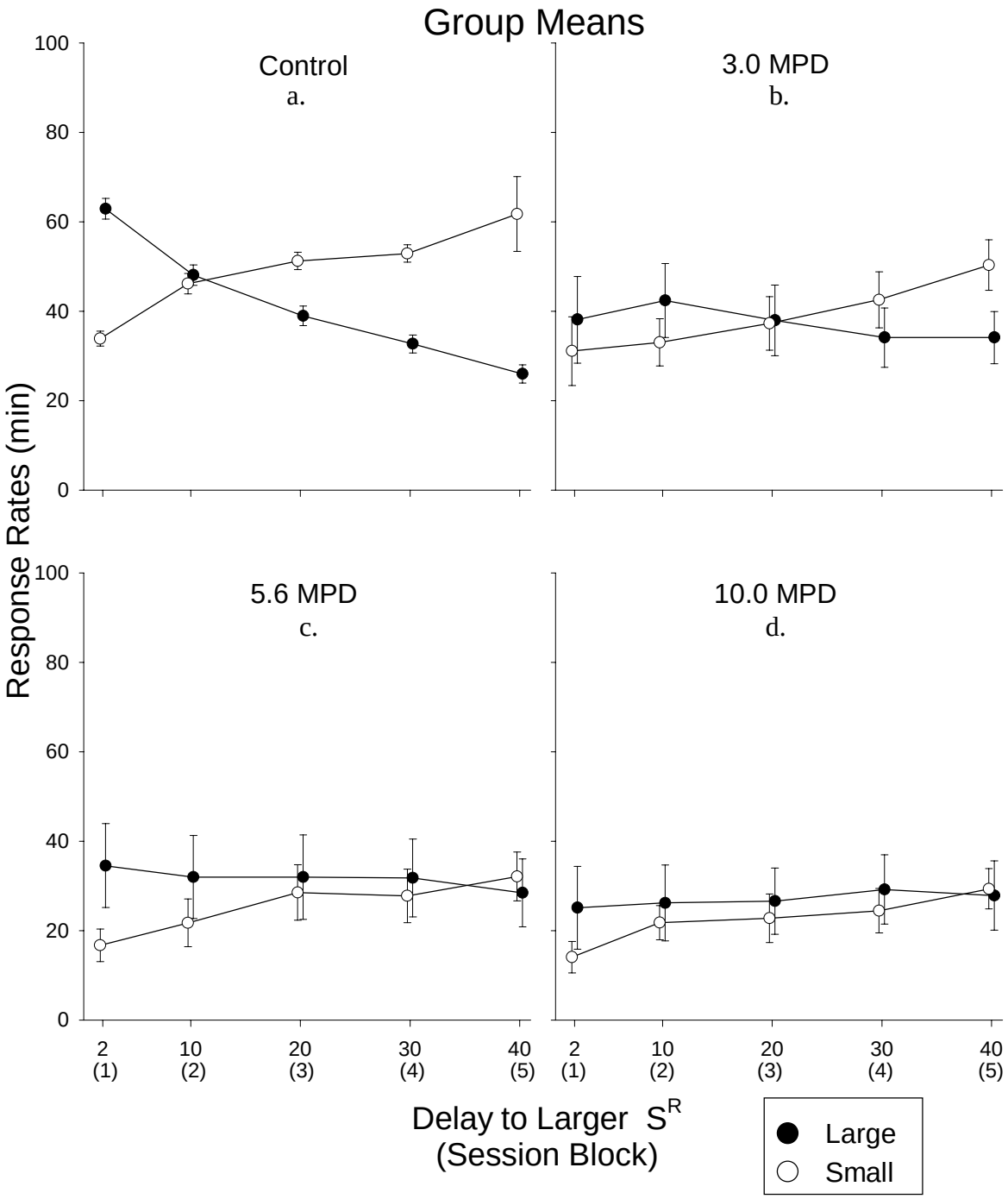

Figure 16. Response rates (min) plotted as a function of the delay to the larger reinforcer for each session block. Data points represent group averages and vertical lines represent standard error of the mean. Closed circles represent choices maintained by the larger reinforcer and open circles represent choices maintained by the smaller reinforcer. Separate plots are shown for control (a), 3.0 (b), 5.6 (c), and 10.0 (d) mg/kg MPD.

Figures 12, 13, 14, and 15 show data for 1809, 1863, 1845, and 1985 respectively. Figure 16 shows data averaged for all four pigeons.

For all birds, these doses tended to flatten the functions for both the larger and smaller reinforcers. This is illustrated to a large extent at 5.6 and 10.0 mg/kg for all pigeons (see Figure 16) in that response rates maintained by both reinforcers were relatively unchanged as a function of increasing delay. In addition, intermediate doses of MPD, particularly 5.6 and 10.0 mg/kg MPD, generally brought the response rates maintained by the larger and smaller reinforcers together.

Although 1809 (Figure 12) continued to exhibit a response pattern that favored the smaller reinforcer following administration of MPD, the overall difference in the response rates for both options decreased dramatically. In general, for 1809 (Figure 12) and 1863 (Figure 13), lower rates maintained by the larger reinforcer increased and higher rates maintained by the smaller reinforcer decreased at the longer delays (20-40 s). For 1809, but not 1863, rates maintained by the larger reinforcer decreased at the shortest delay. Flattened functions for 1845 (Figure 14) and 1985 (Figure 15) occurred as a result of reduced rates maintained by the larger reinforcer at the shorter delays (resulting in the decreased y-intercepts shown in Figure 11) and reduced rates maintained by the smaller reinforcer at all delays.

Further inspection of Figures 12 to 16 reveals the points at which the rates maintained by the smaller and larger options come together and crossover. For the group (Figure 16), the point at which rates maintained by both options crossover appear to consistently move in a rightward direction as doses increased. It seems that as MPD doses increase, pigeons generally begin to tolerate longer delays and choose the option

associated with the larger reinforcer more often across all session blocks. The group curve, however, is not fully representative of individual pigeons (except 1863). Crossover effects varied across pigeons. In some cases the crossover point shifted right (e.g., 1809 following 5.6 mg/kg and 1863 following all doses), but in other cases the crossover point shifted left (e.g., 1845 following 3.0 and 5.6 mg/kg).

Figures 17 through 20 show rate dependency plots for behavior maintained by the smaller and larger reinforcers following 3.0, 5.6, and 10.0 mg/kg MPD. Figures 17, 18, 19, and 20 show data for 1809, 1863, 1845, and 1985, respectively. In these graphs percent control (rate under drug/rate under control x 100) is plotted as a function of control rate for each block. That is, each data point represents a specific block of the session. Data representing the larger and smaller options are combined in each plot for each dose; filled circles represent data for the larger option and open circles represent data for the smaller option. Regression lines fit to all data points (large and small) are presented for each MPD dose, as are the corresponding r^2 values.

There was evidence of rate-dependent effects for all pigeons. For 1809 (Figure 17), rate-dependent effects were seen at the intermediate doses; lower rates tended to increase and higher rates tended to decrease following MPD administration. For 1863 (Figure 18), rate-dependent effects, although less pronounced than the effects observed with 1809, were obtained at the intermediate doses as well. Interestingly, similar effects were not always achieved at comparable control rates maintained by the larger and smaller options. Rates maintained by the larger reinforcer tended to be elevated to a greater extent relative to comparable rates maintained by the smaller reinforcer (Figure 18 for all doses). For 1845 (Figure 19), rate-dependent effects were observed at 5.6 and

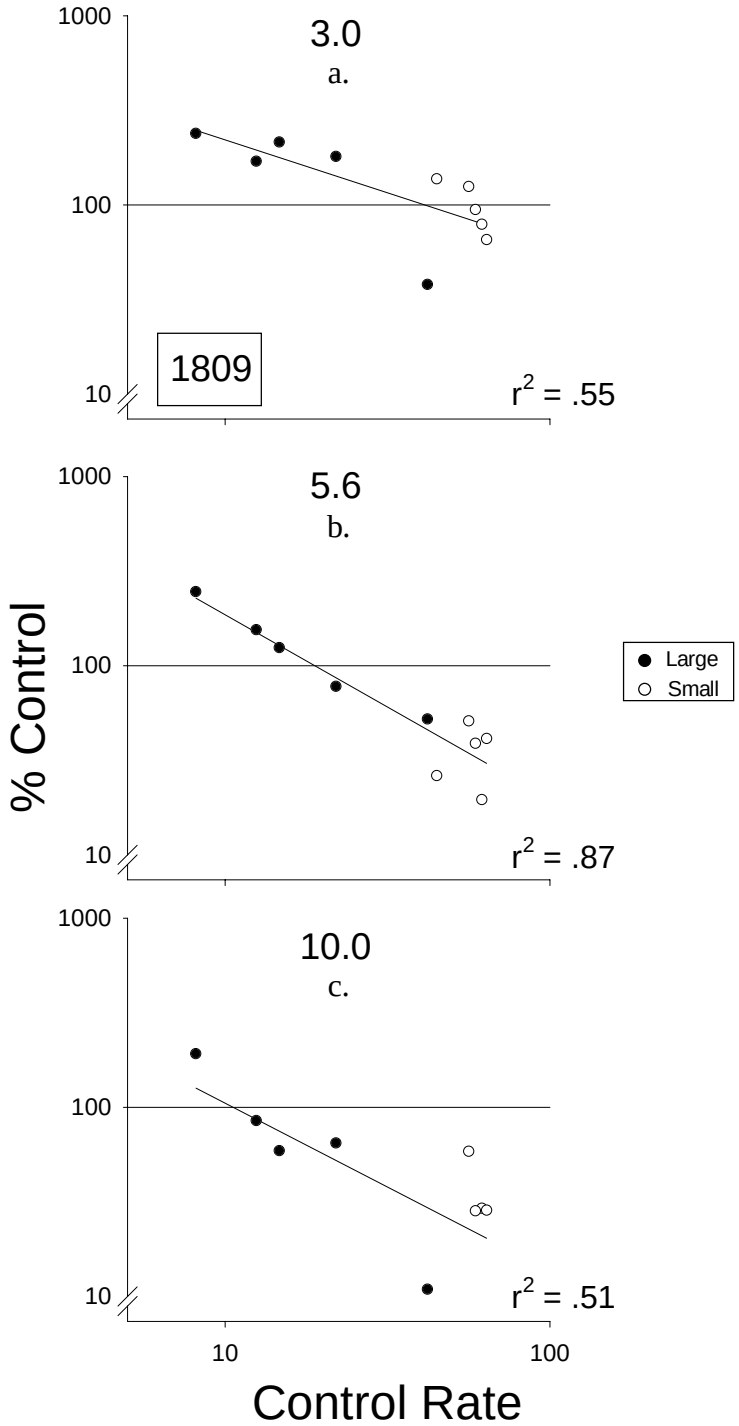

Figure 17. Percent control plotted as a function of control rate for 3.0 (a), 5.6 (b), and 10.0 (c) mg/kg MPD. Filled circles represent behavior maintained by the larger reinforcer and open circles represent behavior maintained by the smaller reinforcer. Data points are mean values and corresponding r^2 values are presented for 1809. Each data point is a mean of 2-4 determinations.

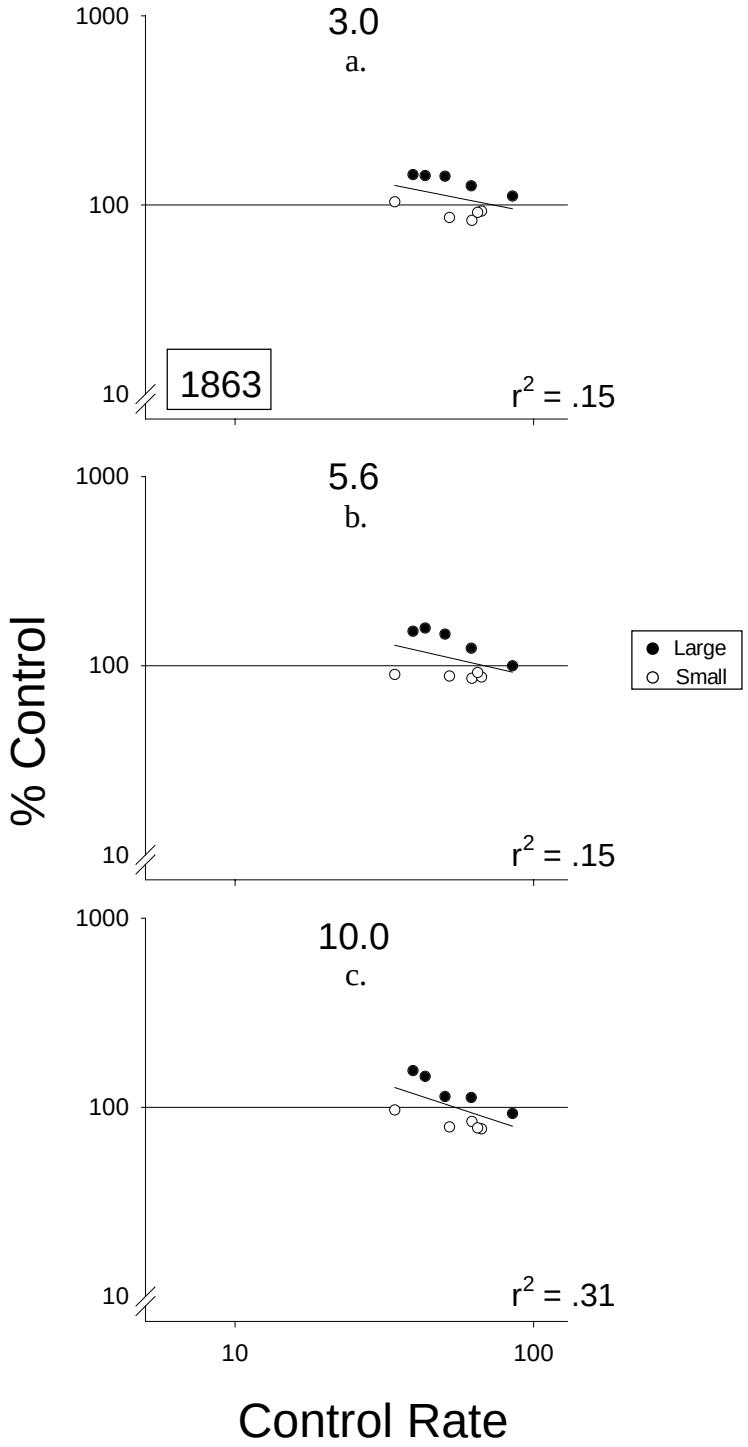

Figure 18. Percent control plotted as a function of control rate for 3.0 (a), 5.6 (b), and 10.0 (c) mg/kg MPD. Filled circles represent behavior maintained by the larger reinforcer and open circles represent behavior maintained by the smaller reinforcer. Data points are mean values and corresponding r^2 values are presented for 1863. Each data point is a mean of 2-4 determinations.

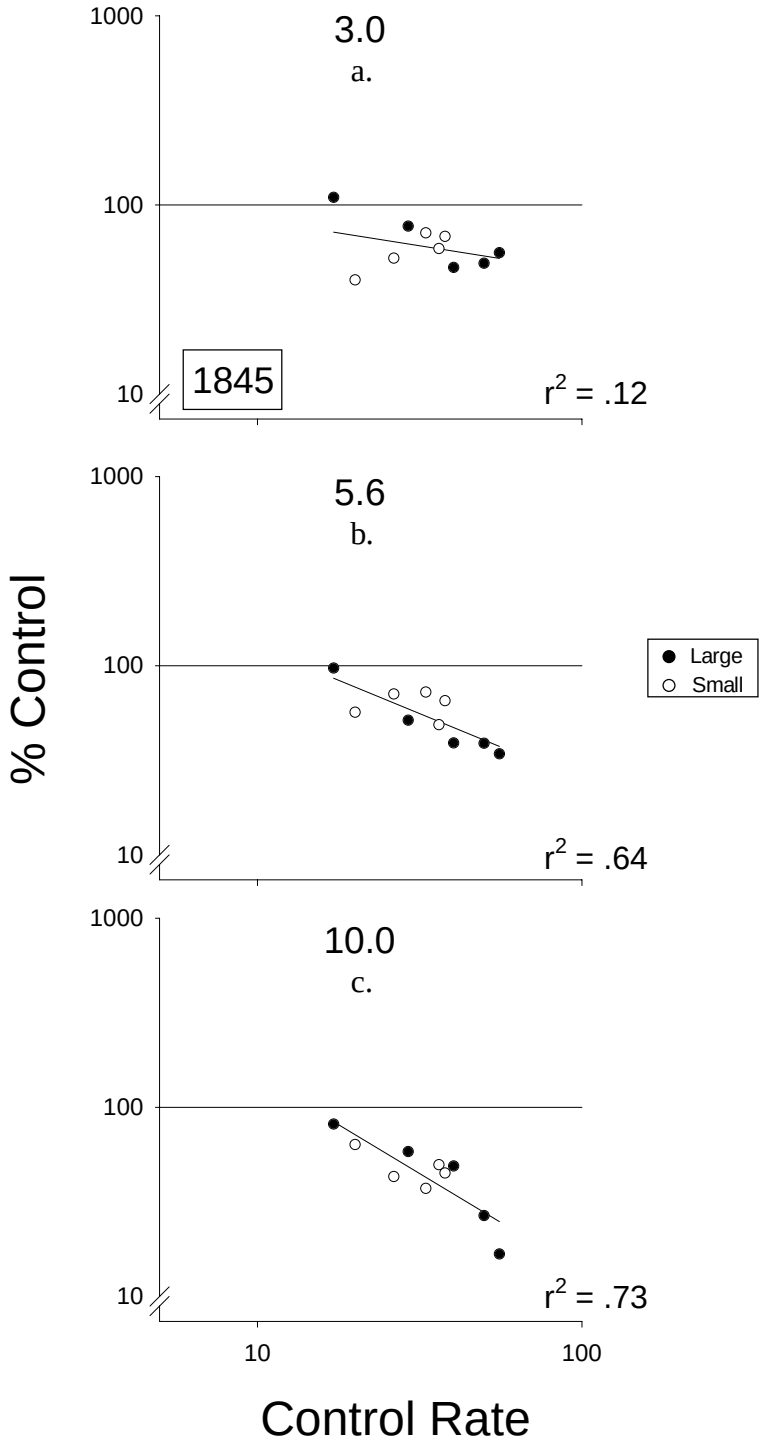

Figure 19. Percent control plotted as a function of control rate for 3.0 (a), 5.6 (b), and 10.0 (c) mg/kg MPD. Filled circles represent behavior maintained by the larger reinforcer and open circles represent behavior maintained by the smaller reinforcer. Data points are mean values and corresponding r^2 values are presented for 1845. Each data point is a mean of 2-4 determinations.

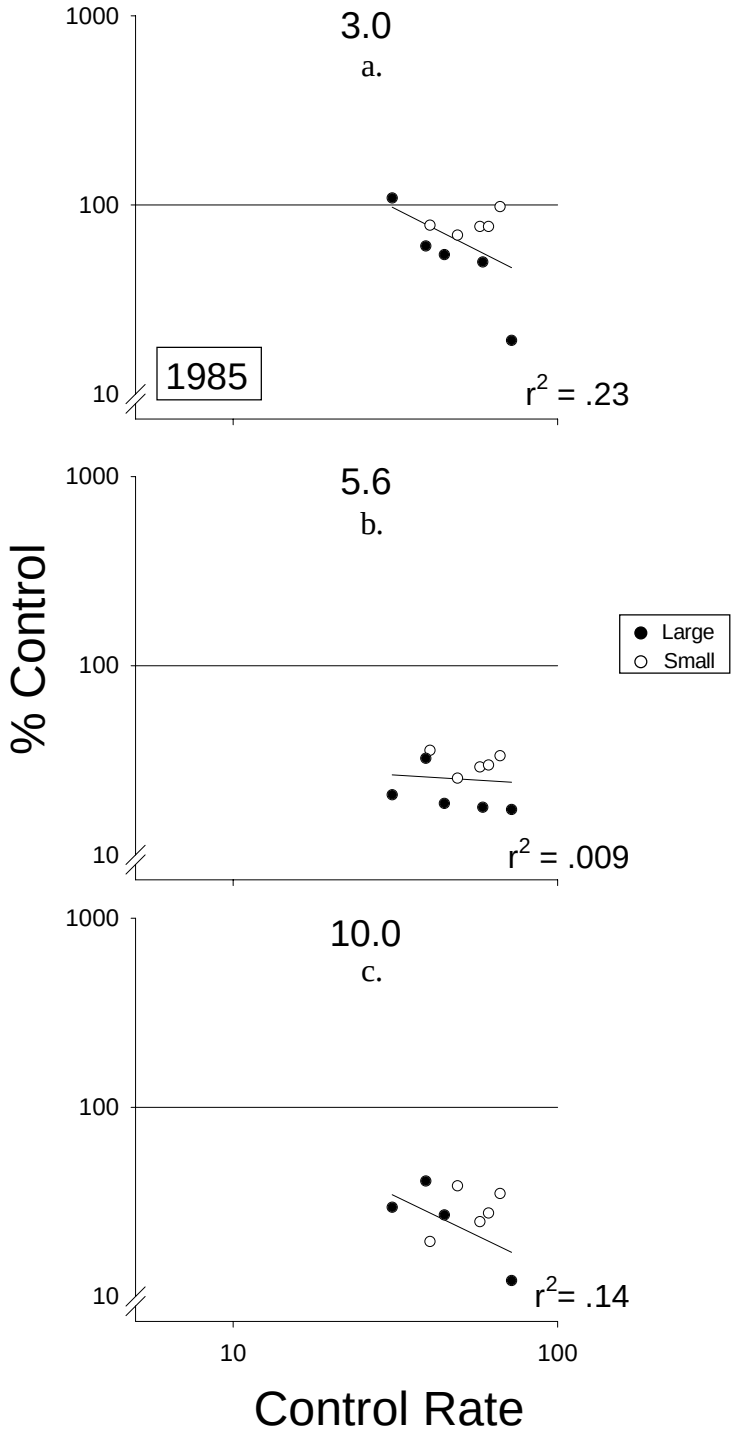

Figure 20. Percent control plotted as a function of control rate for 3.0 (a), 5.6 (b), and 10.0 (c) mg/kg MPD. Filled circles represent behavior maintained by the larger reinforcer and open circles represent behavior maintained by the smaller reinforcer. Data points are mean values and corresponding r^2 values are presented for 1985. Each data point is mean of 2-4 determinations.

10.0 mg/kg and for 1985 (Figure 20) rate-dependent effects were observed mainly at 3.0 mg/kg and slightly at 10.0 mg/kg. For both 1845 and 1985, MPD tended to decrease all response rates; when rate-dependent effects occurred, higher rates were decreased more than lower rates.

Effects of Methamphetamine (METH)

Figure 21 shows effects of the intermediate doses of METH (1.0, 1.7, and 3.0 mg/kg) on the logged discount functions. Regression lines fit to data from control data and corresponding r^2 values are also presented. Closed circles represent data obtained from control sessions and open symbols represent data obtained from drug sessions. Squares, upward triangles, and inverted triangles represent effects of 1.0, 1.7, and 3.0 mg/kg, respectively. Effects of saline and 0.3 mg/kg were generally negligible and are therefore not presented in these graphs; the logged response ratios for saline and all METH doses are presented in Table 2.

All three METH doses decreased the slopes of the functions for all four pigeons. For 3 pigeons (1809, 1863, 1845), this resulted primarily from an increase in relative responding maintained by the larger option at longer delays. Note that on occasion, doses also decreased the y-intercept (e.g., 1.7 and 3.0 mg/kg for 1863). For 1985, the slope change was accompanied by large, dose related decreases in the y-intercept. Note, however, that 3.0 mg/kg METH increased relative responding maintained by the larger reinforcer at longer delays (e.g., inverted, unfilled triangles).

Figure 22 shows individual dose-effect curves for slopes (left) and y-intercept (right) under control, saline, and at all METH doses (note that slopes are absolute values). In accordance with Figure 21, Figure 22 illustrates further that METH flattened the

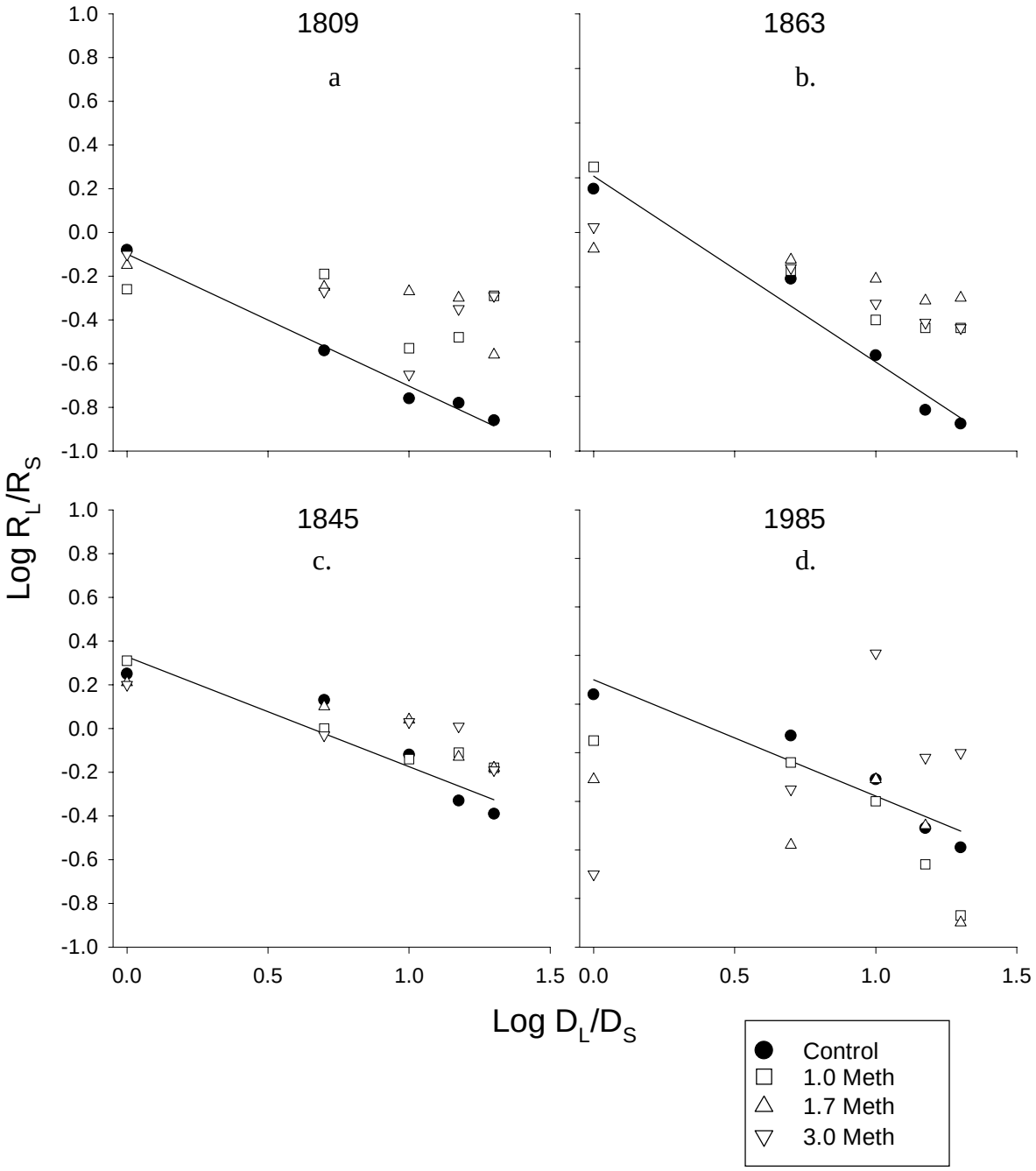

Figure 21. Logged response ratios plotted as a function of logged delay ratios. Data points represent mean values. Each data point for control is a mean of 15-17 sessions. Each data point representing a METH dose is a mean of 2-4 determinations. Closed circles represent control performance and open symbols represent METH performance. Square, upward triangles, and inverted triangles represent 1.0, 1.7, and 3.0 mg/kg METH, respectively. Regression lines were fit to the control ratios using the method of least squares. The corresponding r^2 values for 1809 (a), 1863 (b), 1845 (c), and 1985 (d) were .99, .97, .87, and .90, respectively.

		Meth Dose (mg/kg)					
Subject/Block		Control	Saline	.3 mg/kg	1.0 mg/kg	1.7 mg/kg	3.0 mg/kg
1809							
	1	-.08	-.21	-.17	-.26	-.15	-.10
	2	-.54	-.50	-.50	-.19	-.25	-.27
	3	-.76	-.65	-.67	-.53	-.27	-.65
	4	-.78	-.85	-.69	-.48	-.30	-.35
	5	-.86	-.94	-.75	-.29	-.56	-.29
1863							
	1	.36	.33	.36	.44	.14	.22
	2	.03	-.13	.10	.06	.10	.07
	3	-.25	-.46	-.25	-.12	.03	-.06
	4	-.45	-.47	-.25	-.15	-.05	-.13
	5	-.50	-.36	-.36	-.15	-.04	-.15
1845							
	1	.25	.26	.44	.31	.21	.20
	2	.13	.09	.04	0.00	.10	-.03
	3	-.12	-.19	-.28	-.14	.04	.03
	4	-.33	-.38	-.30	-.11	-.13	.01
	5	-.39	-.72	-.48	-.18	-.18	-.19
1985							
	1	.24	.35	.20	.05	-.11	-.50
	2	.07	.10	-.08	-.04	-.38	-.15
	3	-.11	-.13	.24	-.20	-.11	.41
	4	-.31	-.32	-.27	-.46	-.30	-.02
	5	-.39	-.57	-.64	-.67	-.70	0.00

Table 2. Mean logged response ratios for each subject for control, saline, and METH sessions.

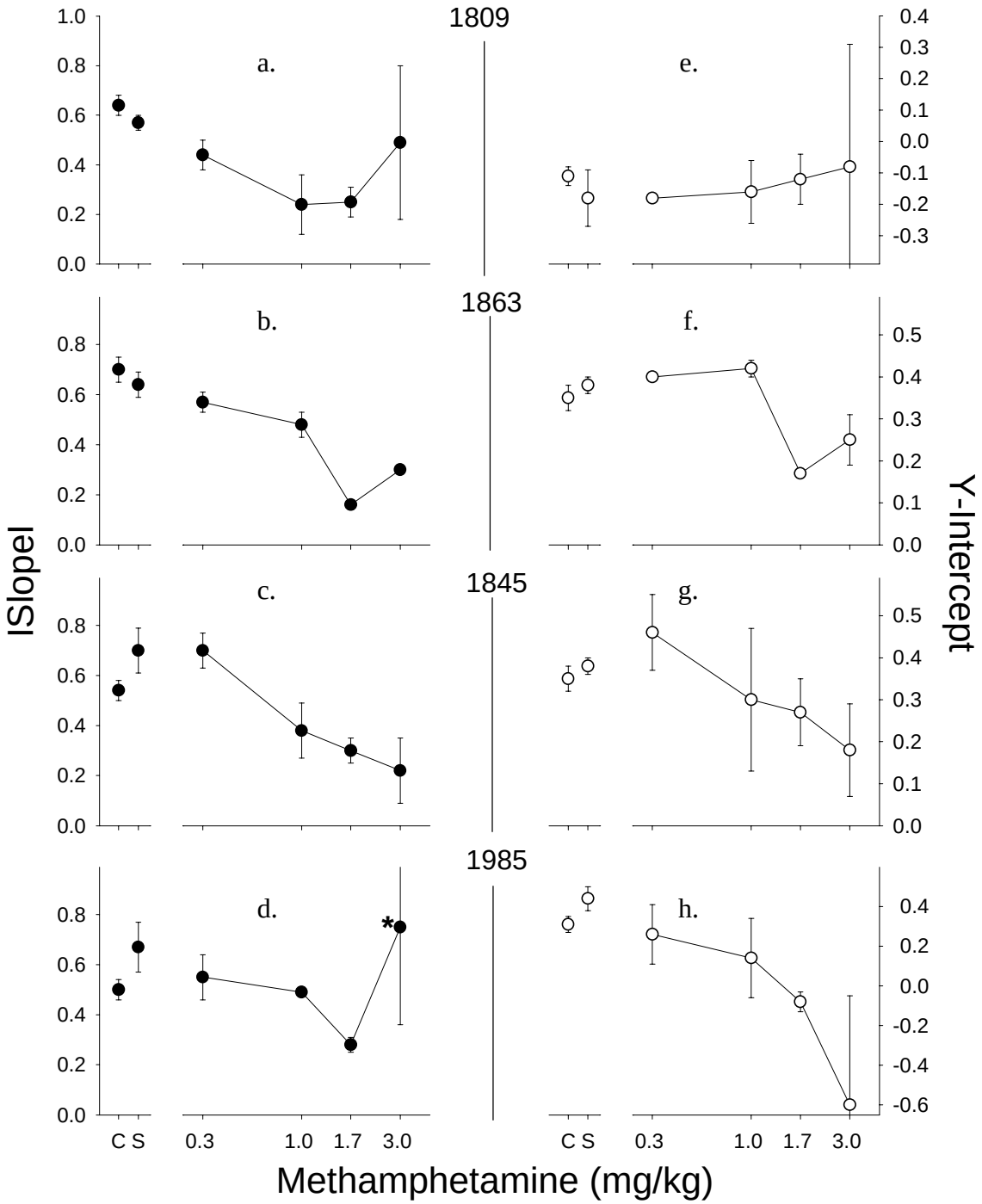

Figure 22. Dose effect functions for slope (a through d) and y-intercepts (e through h) for control, saline and all METH doses. The slope values (a through d) presented are absolute values. Mean values for control were derived from 15-17 sessions. Mean values for saline and METH doses were derived from 2 – 4 determinations. Vertical lines represent standard error of the mean. Note that all slopes were negative except for those marked by a *. Also note different y-axes.

discount functions for all pigeons. For 1809, 1863, and 1845, METH clearly decreased the slope at several doses. For 1809, decreases in slope were not accompanied by substantial changes in y-intercept. Although some y-intercept decreases occurred along with slope decreases in some instances for 1863 and 1845 (particularly at 1.7 and 3.0 mg/kg), all had at least one METH dose that affected the slope but not the y-intercept (e.g., 1863 at .3 and 1.0 mg/kg, 1845 at 1.0 mg/kg). For 1985, the primary effect of METH was to decrease the y-intercept in a dose-dependent fashion, although both 1.7 and 3.0 mg/kg flattened the function (3.0 mg/kg actually made the function positive).

The response rates that composed the discount functions shown in Figure 21 are presented in Figures 23 through 27. Figures 23 through 27 show response rates maintained by both behavioral options for control sessions (all sessions prior to METH injection) and sessions following administration of 1.0, 1.7, and 3.0 mg/kg. Figures 23 through 26 characterize data for individual pigeons; figure 27 shows data averaged for all four pigeons.

All doses of METH tended to flatten both functions. For pigeon 1809 (Figure 23), rates maintained by the smaller reinforcer were decreased at all delays and rates maintained by the larger reinforcer either did not change or were slightly increased at 1.0 and 1.7 mg/kg. With 1863 (Figure 24), response rates maintained by the smaller reinforcer were decreased in all blocks (except block 1) and rates maintained by the larger reinforcer increased at the longer delays (20-40 s). Pigeon 1845 (Figure 25) showed a decrease in rates maintained by the smaller reinforcer at all delays. For this pigeon a decrease in rates maintained by the larger reinforcer was observed only at the shorter delays; at the longer delays rates remained relatively unchanged. For pigeon 1985

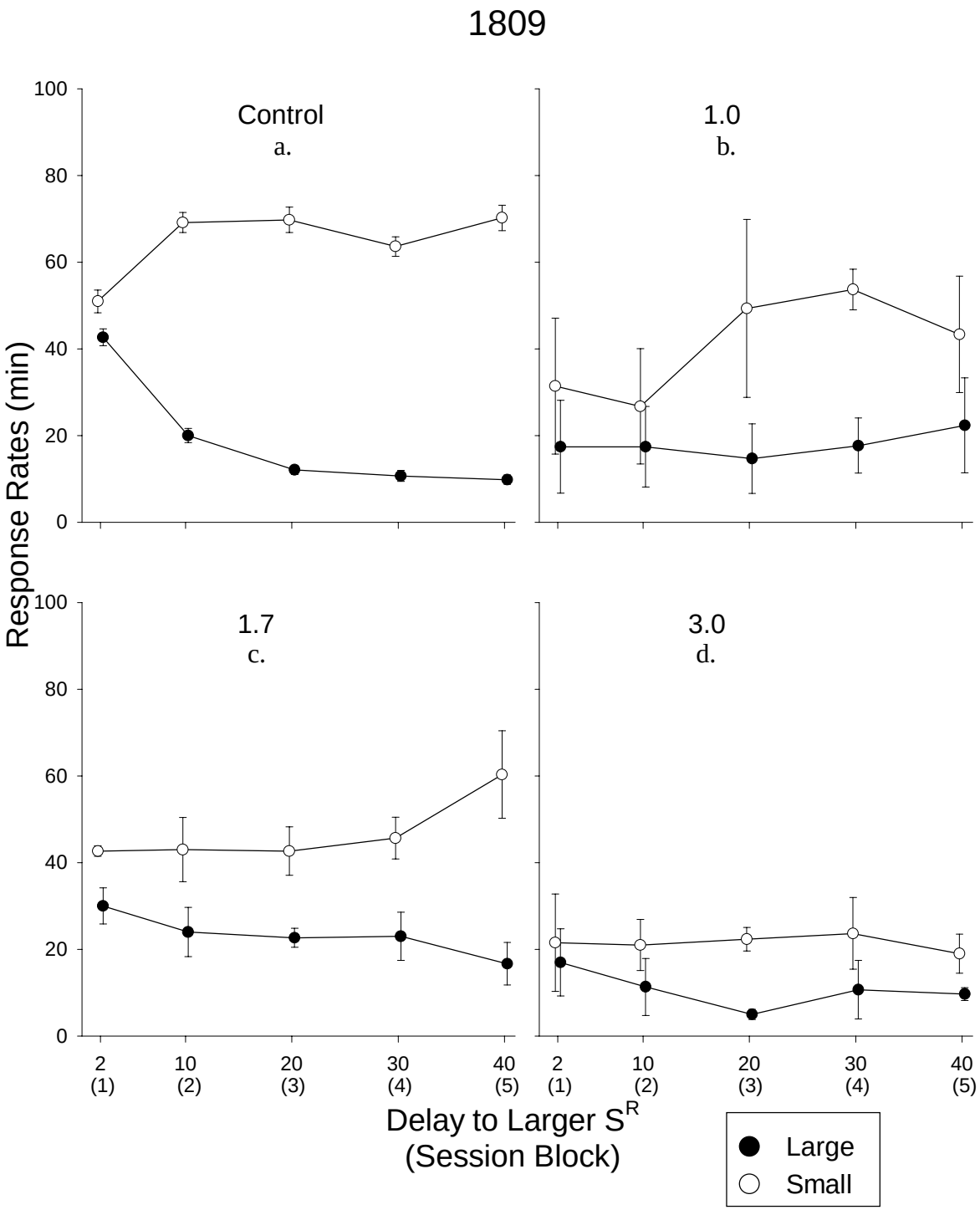

Figure 23. Response rates (min) plotted as a function of the delay to the larger reinforcer for each session block. Data points represent mean values and vertical lines represent standard error of the mean for 1809. Mean values for control were derived from 15-17 sessions. Mean values for METH doses were derived from 2-4 determinations. Closed circles represent choices maintained by the larger reinforcer and open circles represent choices maintained by the smaller reinforcer. Separate plots are shown for control (a), 1.0 (b), 1.7 (c), and 3.0 (d) mg/kg METH.

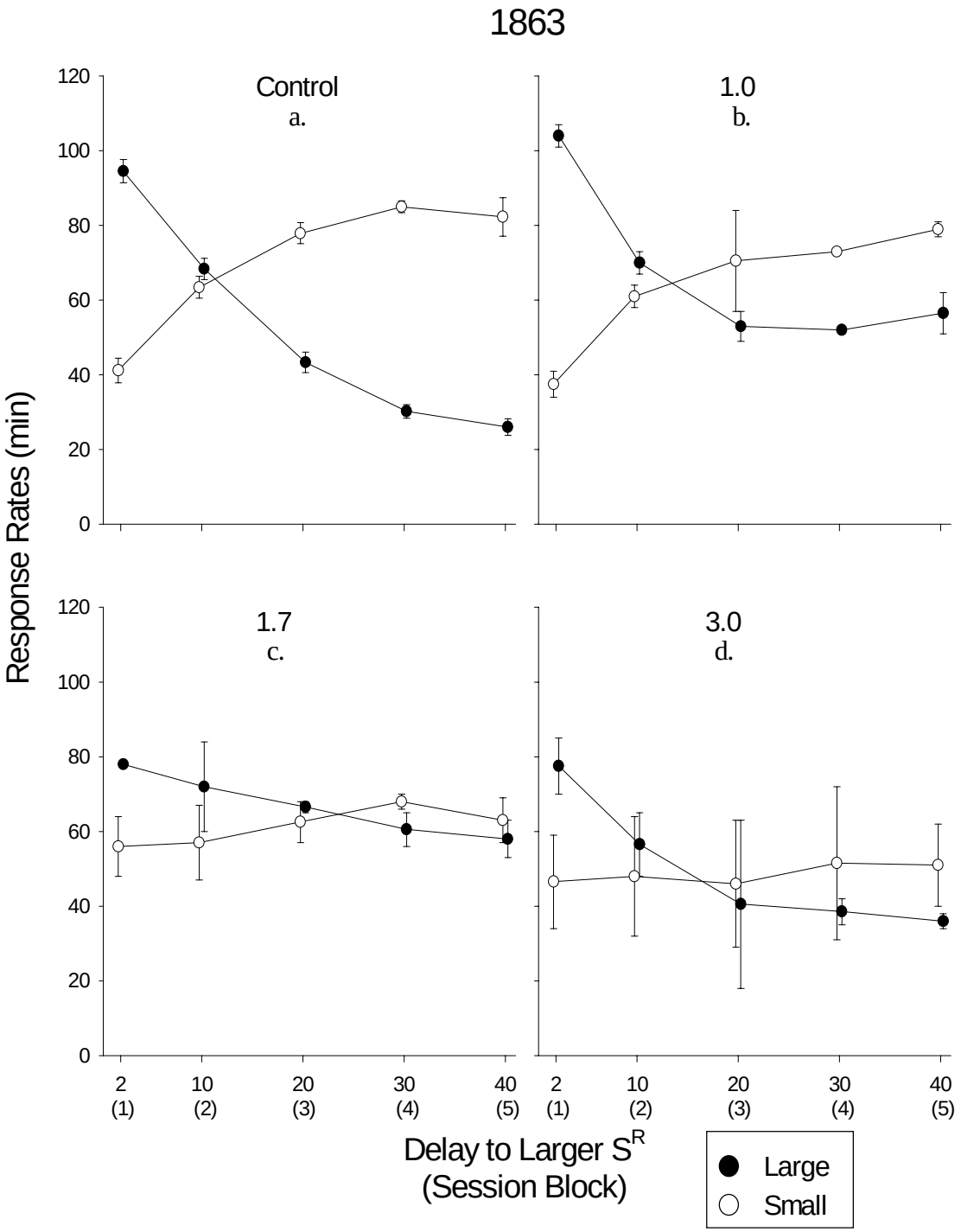

Figure 24. Response rates (min) plotted as a function of the delay to the larger reinforcer for each session block. Data points represent mean values and vertical lines represent standard error of the mean for 1863. Mean values for control were derived from 15-17 sessions. Mean values for METH doses were derived from 2-4 determinations. Closed circles represent choices maintained by the larger reinforcer and open circles represent choices maintained by the smaller reinforcer. Separate plots are shown for control (a), 1.0 (b), 1.7 (c), and 3.0 (d) mg/kg METH.

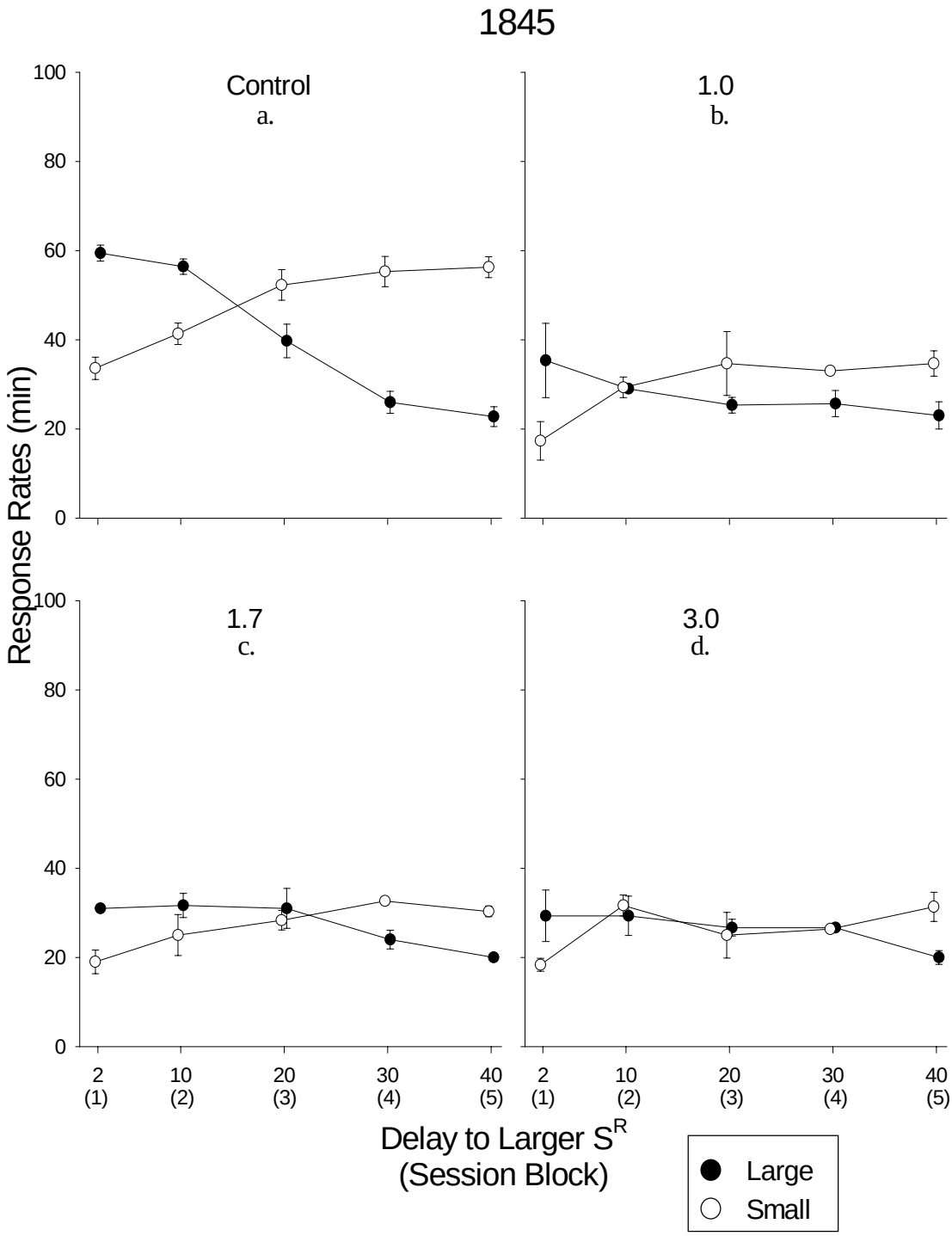

Figure 25. Response rates (min) plotted as a function of the delay to the larger reinforcer for each session block. Data points represent mean values and vertical lines represent standard error of the mean for 1845. Mean values for control were derived from 15-17 sessions. Mean values for METH doses were derived from 2-4 determinations. Closed circles represent choices maintained by the larger reinforcer and open circles represent choices maintained by the smaller reinforcer. Separate plots are shown for control (a), 1.0 (b), 1.7 (c), and 3.0 (d) mg/kg METH.

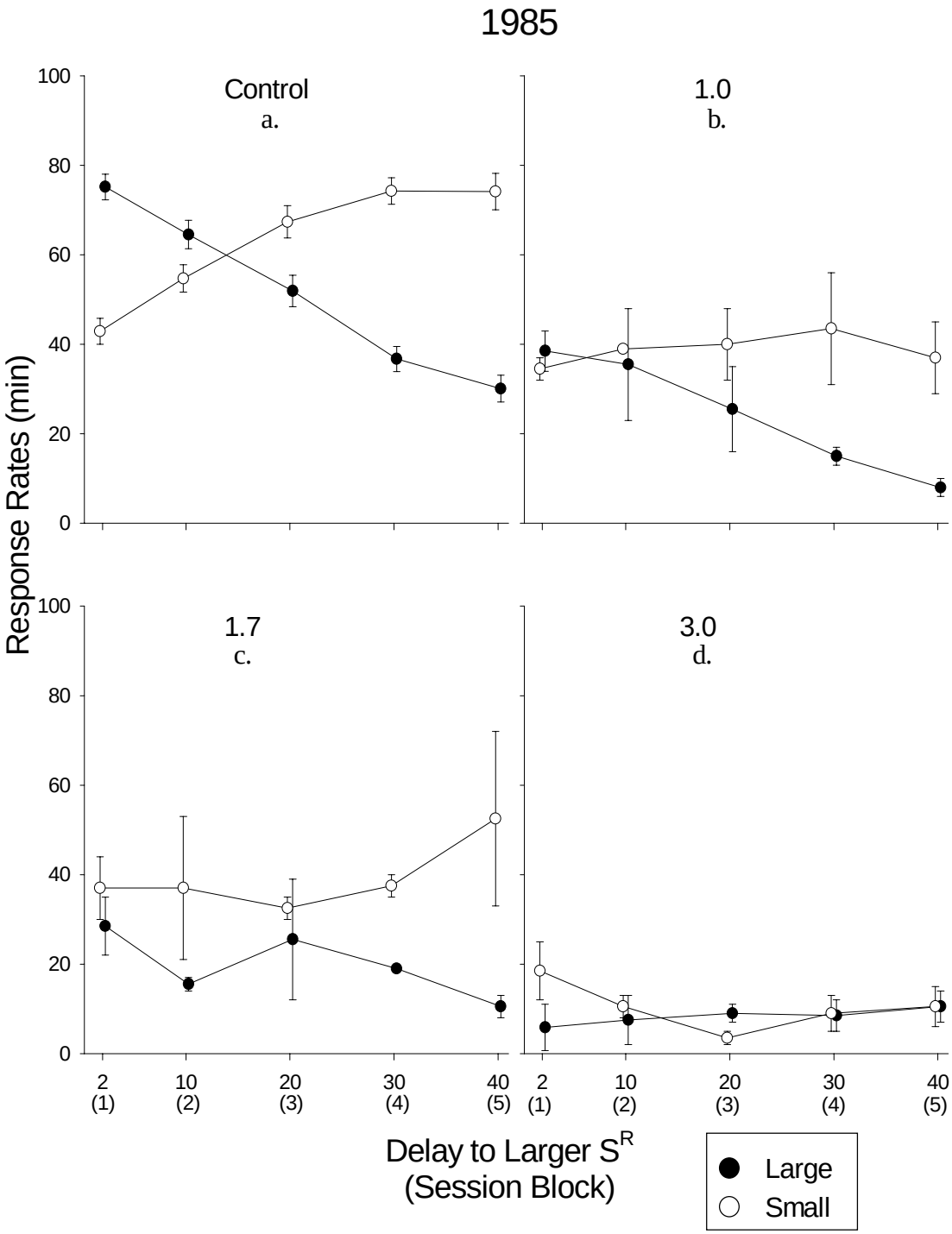

Figure 26. Response rates (min) plotted as a function of the delay to the larger reinforcer for each session block. Data points represent mean values and vertical lines represent standard error of the mean for 1985. Mean values for control were derived from 15-17 determinations. Mean values for METH doses were derived from 2-4 determinations. Closed circles represent behavior maintained by the larger reinforcer and open circles represent behavior maintained by the smaller reinforcer. Separate plots are shown for control (a), 1.0 (b), 1.7 (c), and 3.0 (d) mg/kg METH.

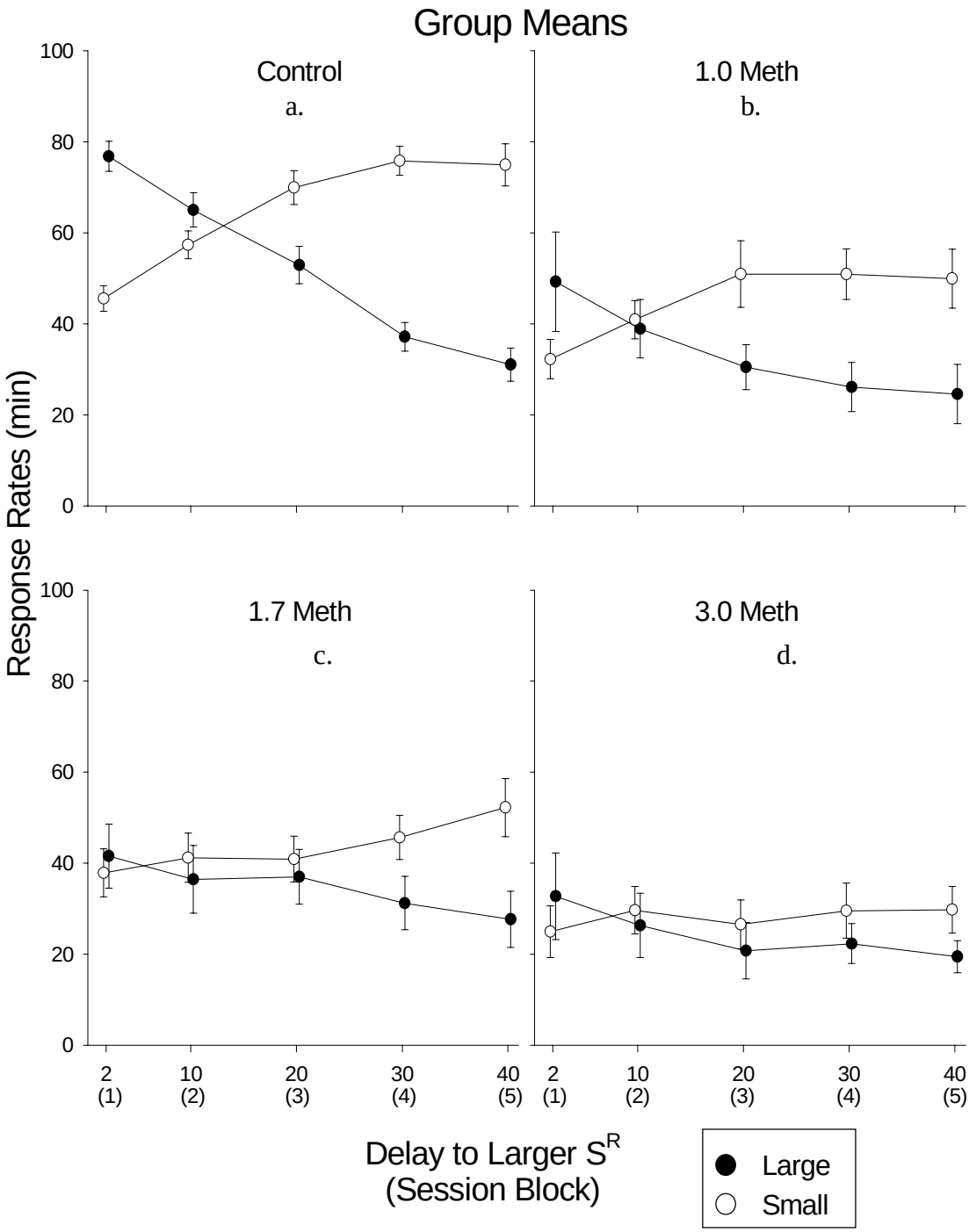

Figure 27. Response rates (min) plotted as a function of the delay to the larger reinforcer for each session block. Data points represent group averages and vertical lines represent standard error of the mean. Closed circles represent choices maintained by the larger reinforcer and open circles represent behavior maintained by the smaller reinforcer. Separate plots are shown for control (a), 1.0 (b), 1.7 (c), and 3.0 (d) mg/kg METH.

(Figure 26), METH doses tended to decrease rates maintained by the larger reinforcer at all delays, although at 1.7 mg/kg, a larger decrease occurred at the shorter delays compared to the longer delays (thus the y-intercept change shown in Figure 21). For this pigeon, rates maintained by the smaller reinforcer were decreased most readily at the longer delays.

With respect to crossover points, Figure 23 shows that, taken as a group, rates began to crossover earlier in the session, resulting in a leftward shift (note this averaged shift is qualitatively different compared to the shift following MPD doses). However, this was not representative of the data for individual pigeons. For example, 1809 (Figure 23) consistently chose the smaller option more often following all doses at all delays, therefore no crossover point was observed. For 1863 (Figure 24), the crossover point following 1.0 and 1.7 shifted rightward compared to control. For 1845 (Figure 25) and 1985 (Figure 26), crossover points did not appear to shift in any systematic fashion.

Figures 28 through 31 show rate dependency plots for behavior maintained by the smaller and larger reinforcers following 1.0, 1.7, and 3.0 mg/kg METH; r^2 values are also presented. Figures 28, 29, 30, and 31 show data for 1809, 1863, 1845, and 1985, respectively. Rate-dependent effects were evident for all pigeons except 1985 following at least one dose. Rate-dependent effects were most clearly apparent with 1809 (Figure 28) particularly at 1.0 and 1.7 mg/kg. In this case, several lower control rates were increased and all higher control rates were decreased. Rate dependency was also obvious for 1863 (Figure 29), although to a lesser degree. With 1863, rate-dependent effects were obtained following all doses, but lower rates were increased more than higher rates were decreased, especially at 1.0 and 1.7 mg/kg. For 1845 (Figure 30) all rates were decreased

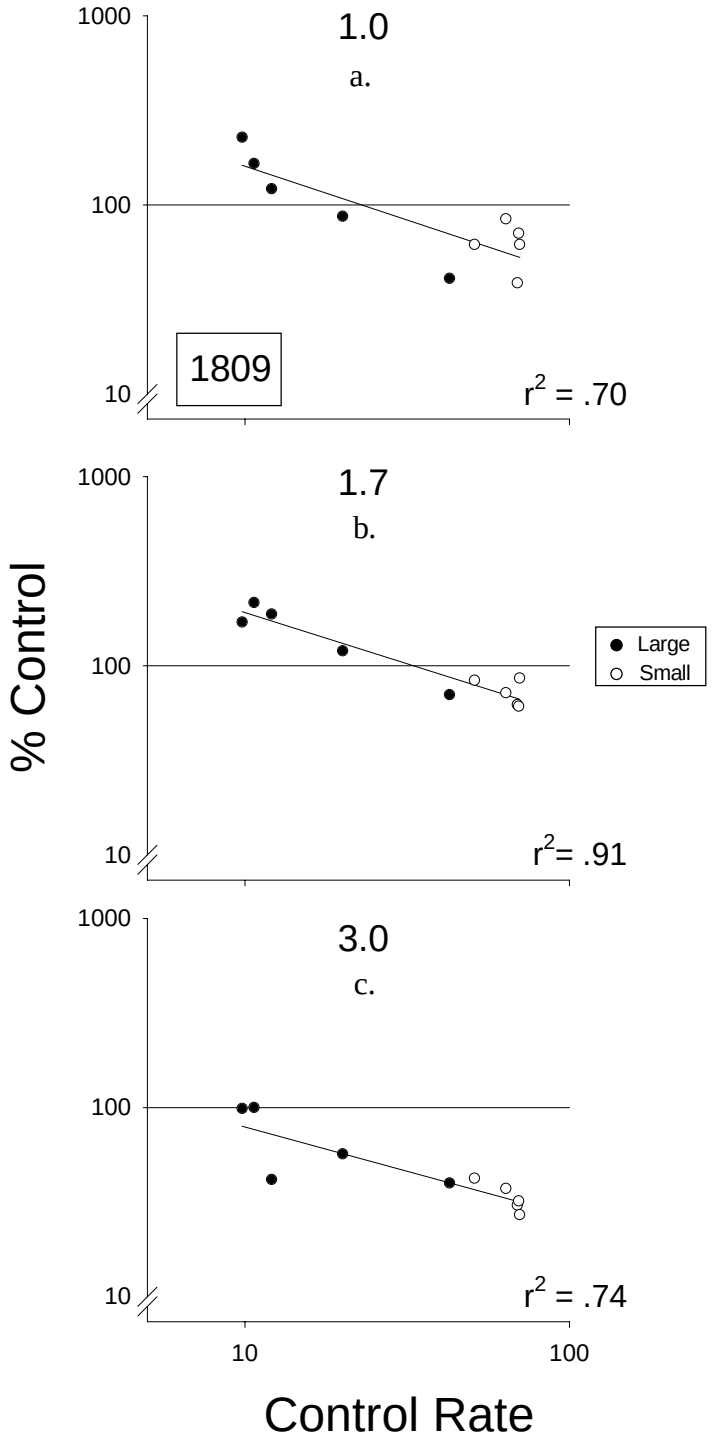

Figure 28. Percent control plotted as a function of control rate for 1.0 (a), 1.7 (b), and 3.0 (c) mg/kg METH. Filled circles represent behavior maintained by the larger reinforcer and open circles represent behavior maintained by the smaller reinforcer. Data points are mean values and corresponding r^2 values are presented for 1809. Each data point is a mean of 2-4 determinations.

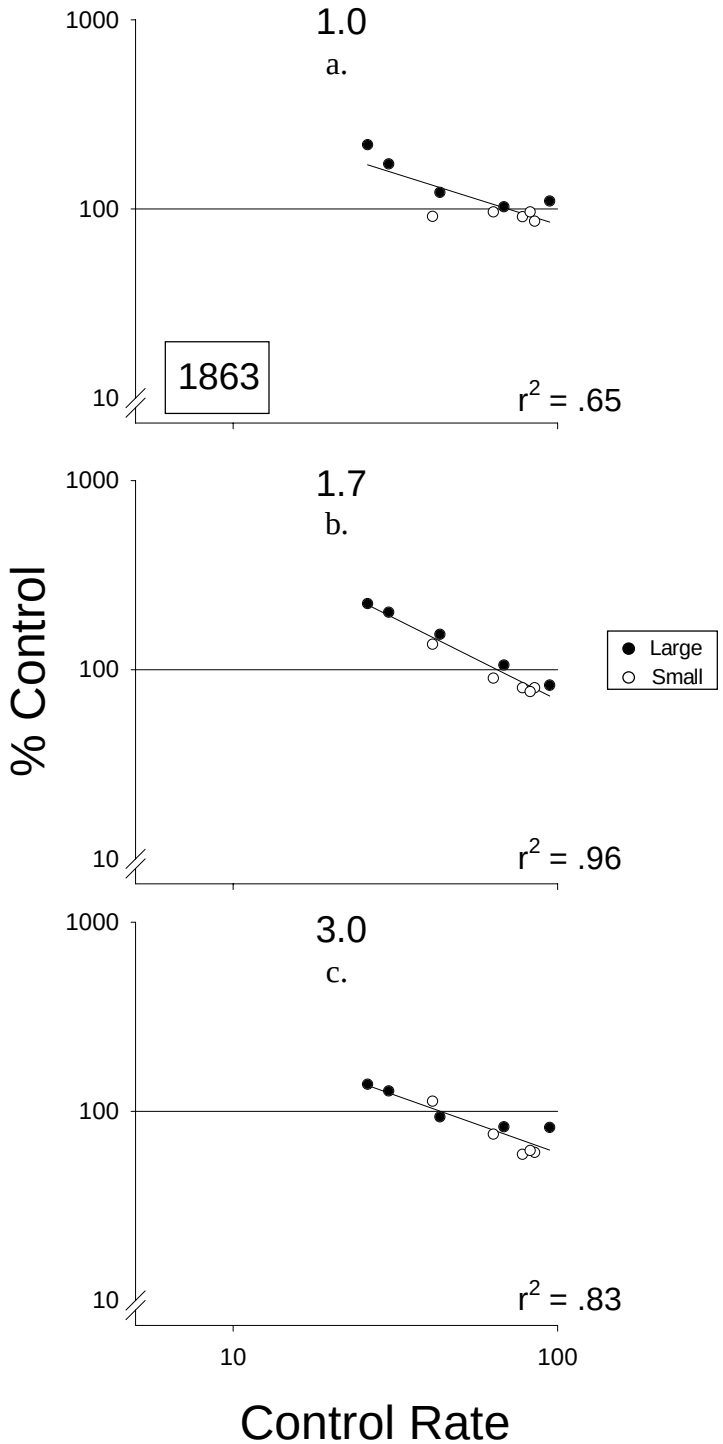

Figure 29. Percent control plotted as a function of control rate for 1.0 (a), 1.7 (b), and 3.0 (c) mg/kg METH. Filled circles represent behavior maintained by the larger reinforcer and open circles represent behavior maintained by the smaller reinforcer. Data points are mean values and corresponding r^2 values are presented for 1863. Each data point is a mean of 2-4 determinations.

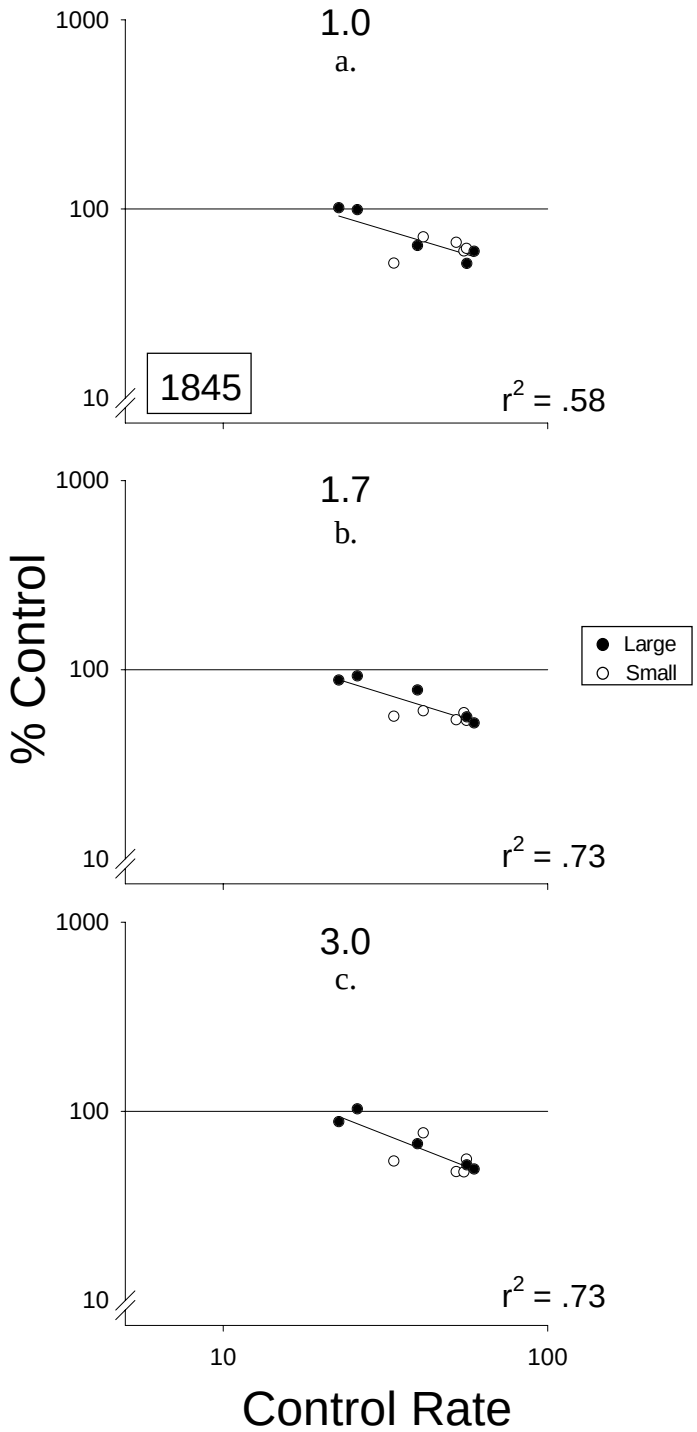

Figure 30. Percent control plotted as a function of control rate for 1.0 (a), 1.7 (b), and 3.0 (c) mg/kg METH. Filled circles represent behavior maintained by the larger reinforcer and open circles represent behavior maintained by the smaller reinforcer. Data points are mean values and corresponding r^2 values are presented for 1845. Each data point is a mean of 2-4 determinations.

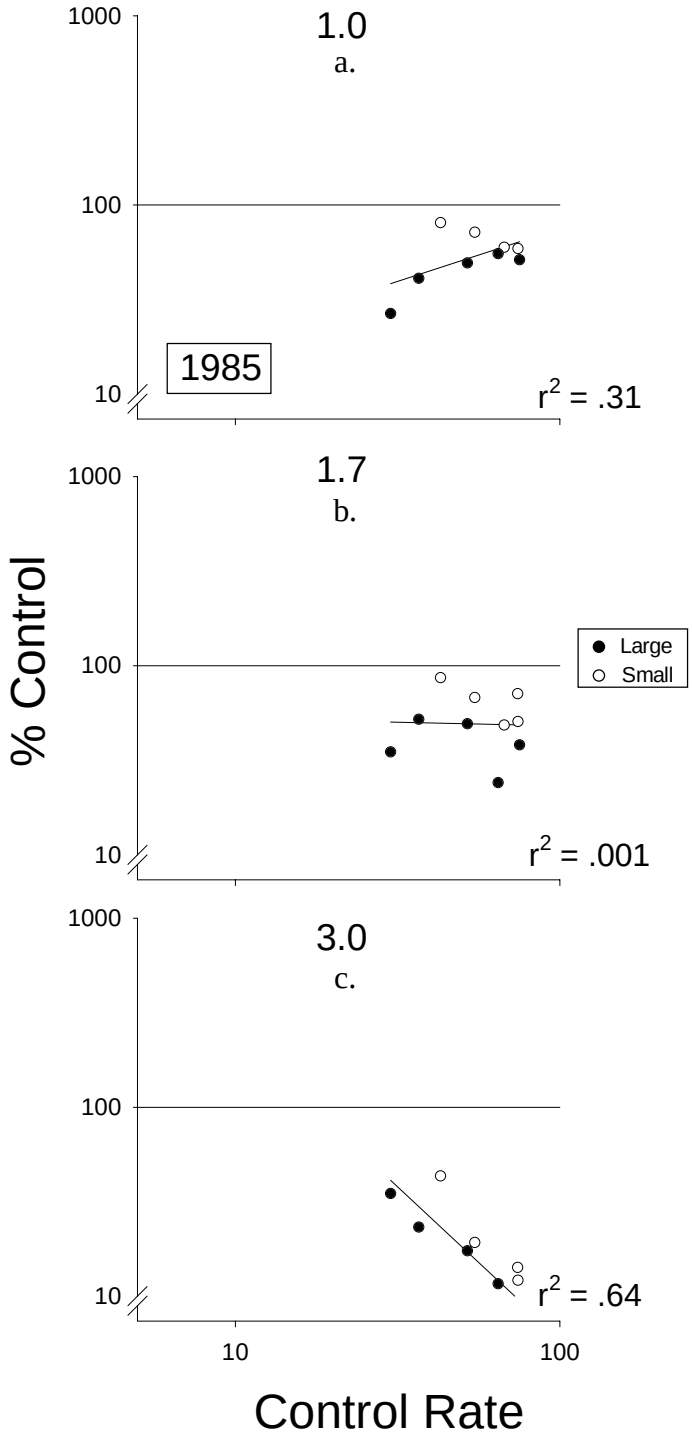

Figure 31. Percent control plotted as a function of control rate for 1.0 (a), 1.7 (b), and 3.0 (c) mg/kg METH. Filled circles represent behavior maintained by the larger reinforcer and open circles represent behavior maintained by the smaller reinforcer. Data points are mean values and corresponding r^2 values are presented for 1985. Each data point is a mean of 2-4 determinations.

following intermediate doses, regardless of control rate. On average, higher rates were decreased to a greater extent compared to lower rates. For pigeon 1985 (Figure 31), higher control rates were decreased more than lower control rates following 3.0 mg/kg.

DISCUSSION

Control Performance

Based on previous studies that have investigated delayed reinforcement (Chung, 1965; Chung & Herrnstein, 1967), it was expected that behavior would show sensitivity to delay. In the present study this was true for all four pigeons under control conditions. That is, as the delay to the larger reinforcer increased across blocks within each session, choices maintained by that option decreased. At the same time, choices maintained by the smaller option increased. Early in the session when delays to both options were equal (2 s), three of the four pigeons (1863, 1845, and 1985) responded on the larger reinforcer up to 3 times more often than on the smaller reinforcer. Choices maintained by the larger reinforcer remained consistently higher relative to the smaller reinforcer for the first 2 blocks, when the delay to the larger reinforcer was 2 and 10 s. Typically, once the delay to the larger reinforcer reached 20 s, the number of responses allocated to the larger reinforcer decreased substantially and choices maintained by the smaller reinforcer became consistently higher. Pigeon 1809's behavior was qualitatively similar to the behavior of the other pigeons in that choices maintained by the larger reinforcer decreased as the choices maintained by the smaller reinforcer increased. However, a bias for the option associated with the smaller reinforcer was observed throughout the experiment. In any case, all four pigeons demonstrated a consistent sensitivity to delayed reinforcement, and reliable within session delay-discount functions were obtained.

Under control conditions, the discount functions of all four pigeons were negatively decelerated. That is, the probability of choosing the larger reinforcer decreased systematically with each succeeding increase in delay, producing a smaller and smaller decrease. These delay-discount functions were quite similar to those typically obtained with other procedures (e.g., Mazur, 1987; Richards, Mitchell, & de Wit, 1997).

One notable advantage to the within-session procedure was that it provided an efficient method to study drug effects. Manipulating the delay within each session allowed for drug effects to be studied on the entire function within sessions and therefore a thorough investigation of how drugs affected delay-discounting was gained. In contrast, when a delay is manipulated either across trials or experimental conditions (e.g., Mazur, 1987; Richards et al., 1997), each drug dose must be administered separately at each delay value in order to study drug-induced changes in the discount function.

Effects of MPD and METH

When compared to the discount functions obtained under control performance, the functions relating absolute response rates maintained by both the larger and smaller reinforcers were flattened following MPD and METH. This effect generally occurred as a result of decreased choices maintained by the smaller reinforcer and increased choices maintained by the larger reinforcer at longer delays (see Figures 16 and 27). On occasion this effect was achieved as a result of decreased choices maintained by the larger reinforcer and increased or unchanged choices maintained by the smaller reinforcer at shorter delays (see Figure 24).

Accordingly, drug-induced changes in the slopes and y-intercepts of the discount functions also were obtained. In cases where the preference for the larger reinforcer

increased at the longer delays, a slope decrease typically was obtained. This drug-induced change in slope appeared as the primary effect following both MPD and METH. This effect was most pronounced with 1809 and 1863 following both drugs, but was also seen to a large extent with 1845, particularly following METH. Y-intercept decreases were observed in cases where preference for the larger reinforcer decreased at the shorter delays, however, these changes occurred only on few occasions. In fact, 1985 was the only pigeon to show a consistent change in the y-intercept following both MPD and METH.

Present Results versus Past Results

On several occasions, moderate doses of MPD and METH increased choices maintained by the larger reinforcer compared to control performance. This increase usually occurred at longer delays later in the session. Although this effect was seen to some degree for all pigeons, it was most substantial for pigeons 1809 and 1863. These results suggested the possibility that MPD and METH attenuated the discounting effect of delay to a large extent for those pigeons. For 1845 and 1985, some evidence of increased choices maintained by the larger, delayed reinforcer was obtained, although the increase for these two pigeons occurred to a much lesser extent.

As stated in the introduction, past studies investigating stimulant effects on self-control have produced mixed findings. It has been reported that stimulants decreased choices maintained by a larger, delayed reinforcer (Charrier & Theibot, 1996; Evenden & Ryan, 1996; Logue et al., 1992). However, more recently it has been reported that stimulants increased choices maintained by a larger, delayed reinforcer and therefore increased self-control (Bullock, 1999; Pitts & McKinney, unpublished observations;

Richards et al., 1999; Wade et al., 2000). The present results provide added support for the view that stimulants increase self-control. Conclusive explanations regarding different outcomes with stimulants and self-control choices have yet to emerge. It is possible that some of the discrepancies across studies are related to procedural differences. For example, the present study and in the studies by Bullock, Pitts and McKinney, Richards et al., and Wade et al. employed some form of forced choice of each alternative. In these past studies, forced-choice trials were arranged in which only one behavioral option was operative per trial and subjects chose each behavioral option once before choosing either option freely. These forced choice trials were implemented to ensure that subjects gain exposure to the different consequences associated with each behavioral option. In the present study, sampling of both options was ensured through a single RI schedule. Thus, subjects were "forced" to sample each option several times throughout the session. In contrast, earlier studies (Charrier & Theibot; Evenden & Ryan) did not implement forced choice trials and, therefore, it is not certain whether subjects in these studies experienced the consequences of the different behavioral options with sufficient regularity.

The use of a signaled delay was another common characteristic found in those studies in which a stimulant-induced increase in self-control was found (Bullock, 1999; Pitts & McKinney, unpublished observations; Richards et al., 1999; Wade et al., 2000). In these studies an explicit and unique stimulus condition was present during the delay periods. In Richards et al. and Wade et al., a tone was sounded during the delay period before the larger reinforcer was delivered. This stimulus change was present each time the larger reinforcer was chosen and was present until reinforcement was delivered. In

the present study, separate lights signaled the delays to the larger and smaller reinforcers. In the studies conducted by Charrier and Theibot (1996) and Evenden and Ryan (1996) where stimulants decreased self-control, no unique stimulus was presented throughout the delays (although the response levers retracted at the onset of each delay and upon delivery of the immediate reinforcer). Based upon studies that have investigated the effects of a signaled delay (Cardinal, Robbins, & Everitt, 2000) the absence of such an explicit stimulus change during the delay period prior to the larger reinforcer may explain why earlier studies (Charrier & Theibot; Evenden & Ryan) found a decrease in self-control. For example, Cardinal et al. reported that amphetamine increased choices maintained by a larger, delayed reinforcer if the delay period was signaled by an explicit stimulus. When the delay period was not signaled by an explicit stimulus, choices maintained by the larger reinforcer decreased; an effect similar to the data reported by Charrier and Theibot and Evenden and Ryan.

The Present Hypothesis

It has been repeatedly shown that under typical, non-drug conditions, increasing the delay to a reinforcer will decrease responses on the corresponding option (Chung, 1965; Chung & Herrnstein, 1967) and increasing the amount of a reinforcer will increase responses on that option (Catania, 1963; Neuringer, 1967). In the present study, it was found that MPD and METH increased responding maintained by a larger, delayed reinforcer. The purpose of the present analysis was to address the following question: Is the delay to reinforcement more readily affected in that longer delays become more "tolerated", or is the amount of reinforcement more readily affected in that a larger reinforcer becomes more effective?

Overall, slope decreases were obtained for all pigeons following at least one dose of MPD and/or METH; for two pigeons (1809 and 1863) substantial decreases were observed following more than one MPD dose (see Figures 10 and 11) and for three pigeons (1809, 1863, and 1845) this was true following several METH doses (see Figures 21 and 22). These slope decreases suggest that MPD and METH affected behavior by decreasing the sensitivity to the effects of delay (i.e., by attenuating delay-discounting). Occasional y-intercept decreases were concomitantly observed (see Figure 10 for 1809 and 1985; see Figure 21 for 1863 and 1985), but these y-intercept decreases were observed to a much lesser extent than the decreases in slope.

Although the overall effects of MPD and METH were characteristically similar, a few small differential effects were observed. In general, both drugs produced similar effects, although the changes produced by METH were more consistent across birds than the changes produced by MPD. METH had a greater tendency to change the slope without affecting the y-intercept. For three of the four pigeons following METH administration, there was at least one dose that changed the slope exclusively. In comparison, MPD primarily changed the slope for two pigeons (1809 and 1863) and the y-intercept for the other two pigeons (1845 and 1985). With 1845, there were far less y-intercept decreases following METH when compared to the changes obtained with MPD. Speculation as to why differential effects were obtained (mainly with 1845) with two very similar drugs may include the fact that MPD was administered first. Thus, the possibility of order effects cannot be completely excluded.

As Thompson's definition states, determining a behavioral mechanism involves two steps: (1)"identifying the environmental variables which typically regulate the

behavior in question" and (2)"characterizing the manner in which those variables' influence is altered by the drug" (1984, p. 2). In summary, it appears as though the present study has met these requirements. That is, it is known that the delay to, and amount of, a reinforcer both systematically affect response allocation between two different behavioral options. The logarithmically transformed version of Herrnstein's matching equation (equation 7) enabled the use of an analysis that allowed for the identification of how these variables were altered following drug administration. A decrease in slope suggested a decreased sensitivity to delay and an increase in y-intercept suggested an increased sensitivity to amount.

The results of the present study, however, were fairly complex. In addition, the identification of a possible behavioral mechanism of drug action is an extremely complex process. Although the results of the present study provide evidence for a decreased sensitivity to delay, conclusive interpretations regarding this as a behavioral mechanism of drug action with respect to self-control should be made cautiously. Several alternative accounts are possible. For example, stimulants produce rate-dependent effects (see Sanger & Blackman, 1976) and changes in timing mechanisms (e.g., Eckerman, Segbefia, Manning & Breese, 1987; Maricq, Roberts, & Church, 1981; Meck, 1981). Stimulants have also been known to increase the effectiveness of conditioned reinforcers (e.g., Cardinal et al., 2000; Files, Branch, & Clody, 1989; Hill, 1970) as well as increase stereotypical, or perseverative, behavior (see Julien, 1995). For this reason, the present results should be considered in the context of these alternative interpretations.

Alternative Interpretations

Stimulants and Rate Dependency

One way in which the stimulant effects have been characterized is through the notion of rate dependency (see Sanger and Blackman, 1976). According to this principle, the effects of stimulants can be predicted based upon response rates maintained under control conditions. Under many circumstances, stimulants tend to increase lower response rates and decrease higher response rates maintained under control conditions. In the present study, rate-dependent effects were observed for all pigeons following at least one dose of MPD and METH.

In most cases where evidence of rate dependency was present, lower response rates were increased and higher response rates were decreased. In a few instances, all rates were decreased, but higher rates tended to be decreased to a greater extent than lower rates (e.g., see Figure 19, 20, and 31). For both drugs, the most pronounced effects were obtained with 1809 and 1863. These two pigeons also demonstrated the greatest decrease in slope. Indeed, for these two pigeons, every intermediate dose of MPD and METH that decreased slope also produced rate-dependent effects. Thus, these data beg the question: Were the decreases in slope merely a by-product of rate dependency or were the rate-dependent effects a by-product of the slope decreases?

Unfortunately, the above question cannot be answered conclusively on the basis of the present data. However, it is important to note that any interpretations made based on rate dependency should be done so with caution. As Branch (1984) pointed out, rate dependency has served as a useful empirical generalization within Behavioral Pharmacology. The notion of rate dependency has provided efficient descriptions of data

under numerous conditions (e.g., see Sanger and Blackman, 1976). Rate dependency does not, however, provide an explanatory analysis of data. To state that a particular stimulant had rate-dependent effects is to say that lower baseline response rates were increased and higher baseline response rates were decreased. In no way does this observation, or description, reveal what possible behavioral processes caused such a change in response rates. In any case, describing data as rate-dependent should not lead behavioral pharmacologists to attribute less importance to other modulating variables (e.g., type of consequence, reinforcement rate, conditioned reinforcement). There is no doubt that rate dependency has provided a concise description of response rate data under a wide variety of conditions, but to utilize the notion of rate dependency for anything more than a description of data could be misleading.

Aside from its explanatory limitations, certain aspects of the present data weaken an interpretation attributing the present results exclusively to rate dependency. First and foremost, if rate dependency was the primary account of response rates under drug conditions, it would be expected that similar control response rates would be changed similarly regardless of other modulating factors (i.e., amount and delay). This was not always the case. In fact, there were several occasions in which comparable control response rates maintained by the larger and smaller reinforcer were affected differentially (see Figures 17, 18, 20, and 31 for specific examples). Second, on occasion the rate-dependent plots appeared somewhat unsystematic. Pigeon 1809 (see Figure 17) provided one of the best overall examples of rate dependency. The response rate plots obtained for this pigeon, however, were a bit disorganized. Specifically, the most disorganized rate-dependency plots for 1809 were at 3.0 and 10.0 mg/kg MPD. Examination of the r^2

values associated with the plots for 3.0 and 10.0 mg/kg MPD revealed values of .55 and .51, respectively. As a different example, 1863's behavior (see Figure 18) was also a noteworthy example of rate dependency and yet the response rates obtained, when plotted as rate-dependent functions, appeared moderately clustered. For 1863, the range of control response rates maintained by the larger and smaller reinforcer was generally limited to higher values. When compared to the rate-dependent functions of the other pigeons, the rate-dependent functions for 1863 did not show substantial decreases with any control rates, regardless of whether they were maintained by the larger or smaller reinforcer. Thus, with this pigeon, the restricted range of the control response rates limits an interpretation based upon rate dependency.

In any case, the purpose here was not to undermine the notion of rate dependency as an empirical generalization. For the present purpose however, it should be stated that although evidence of rate dependency was obtained, changes in the effects of sensitivity to delay and amount also were obtained. To focus on such an empirical generalization may hinder elucidation of relevant behavioral processes at work. In fact, such a focus may actually divert attention from other important modulating variables (see Branch, 1984).

Stimulants and Timing

It has been suggested that stimulants affect temporal discrimination (Eckerman et al., 1987; Maricq et al., 1981; Meck, 1981). Specifically, it has been asserted that stimulants speed up a subject's "internal clock", thus causing an overestimation of the passage of time. Stated in more behavioral terms, a drug-induced overestimation of time can be observed as a subjects' tendency to respond earlier under procedures requiring

temporal discrimination. Thus, it is important to consider the possibility that the present data reflect such a drug effect.

Meck (1983) used a temporal discrimination procedure that varied signal durations. In this study, subjects discriminated between short signal durations and long signal durations (2 vs. 8 s of white noise). Reinforcement was delivered for a response that correctly discriminated the duration as short or long. Intermediate durations (e.g., 2.6, 3.2, 4.0, 6.4 s) were randomly intermixed with an equal probability; responses made on either key were not reinforced following these durations. Following intermediate doses of METH, responses associated with the longer, signaled durations increased. This result suggested that the subjects began to overestimate the duration of the signals and therefore responded as if the shorter durations were in fact, longer.

Maricq et al. (1981) used a peak procedure to study timing disruptions and stimulants. In this procedure, FI 40 s trials and 80 s extinction (EXT) trials were randomly intermixed. With the FI 40s trials, the first response following 40 s was reinforced by food delivery. On the 80 s EXT trials, responses were not reinforced. Response rates maintained by the FI 40 s schedule and on the 80 s extinction trials were plotted as a function of the passage of time. It was found that for FI 40 s, a scalloped pattern emerged with the maximum response rate occurring close to the time of reinforcement. For those trials in which reinforcement was omitted, a typical scalloped pattern of responding was obtained, with the maximum response rate occurring in close approximation of when food was typically delivered on the FI 40 s schedule, followed by a decreasing rate over the remainder of the interval. Maricq et al. reported that METH produced a leftward shift in the time point at which maximum response rates occurred (a

result indicative of overestimating the passage of time) for both reinforcement schedules. In addition, it was reported that constant proportional changes were observed with the response rate functions. Thus, if a subject responded to a 10 s interval as if it were a 20 s interval, then it would respond to a 20 s interval as if it were a 40 s interval. Indeed, METH exaggerated the time intervals and in turn made them "seem" longer.

Despite the results stated above, certain characteristics of the present results do not support an interpretation based on a disruption of timing. To begin with, if timing were to account for the delay of reinforcement effects obtained in the present study, an overestimation of delay duration would have occurred. In adding a constant proportional amount to each delay, the "perception" of the delay would change from, for example, 2 s, 10 s, 20 s, 30 s, and 40 s to 4 s, 20 s, 40 s, 60 s, and 80 s. In the present study, a constant proportional change would be expected with both the larger and smaller reinforcement options since both options were associated with a delay (2 s for the smaller reinforcer and 2 to 40 s for the larger reinforcer). Such a proportional change in the functional effects of both delays would not be expected to produce a change in preference.

Stimulants and Conditioned Reinforcement

It has been reported that stimulants increase choices maintained by a larger, delayed reinforcer when the delay period is associated with an explicit stimulus (Cardinal et al., 2000). Furthermore, it has been suggested that when a stimulus change, or signal, is associated with a delay period, it may begin to function as a conditioned reinforcer (Files et al., 1989; Hill, 1970). If this is true, increased choices maintained by a larger, delayed reinforcer might be due to an increased effectiveness of the conditioned reinforcer during the delay period. As with other procedures used to study self-control

(Richards et al., 1999; Wade et al., 2000), the present study included an explicit stimulus change during the delay period for the larger reinforcer prior to reinforcement delivery. During the delay period, all lights were extinguished except for the colored houselight that corresponded with the illuminated key color just chosen. Unlike other studies (Richards et al.; Wade et al.), however, the smaller reinforcer included in the present study also was associated with a delay. Although this delay was considerably shorter (2 s throughout the session) than that associated with the larger reinforcer, it also was signaled. Presumably, the signal associated with the delay to the smaller reinforcer also served as a conditioned reinforcer. In other procedures used to study self-control, the larger, more delayed reinforcer was the only option associated with a stimulus change (i.e., the smaller reinforcer was presented immediately). Because the delay periods for both behavioral options (2 s associated with the smaller reinforcer and 2 to 40 s associated with the larger reinforcer) were signaled in the present study, it would be expected that a conditioned reinforcement effect would have occurred with both behavioral options. If a conditioned reinforcement effect were to occur for both options, an increase in responding maintained by both reinforcers would have resulted. In the present study, however, it was more often that only choices maintained by the larger reinforcer were increased. Therefore, it seems unlikely that increased choices maintained by the larger, more delayed reinforcer were the result of a conditioned-reinforcement effect.

Stimulants and Perseveration

Another suggested effect of stimulants is that they increase "stereotyped" or "perseverative" behavior (see Julien, 1995). "Perseveration" is a term often used to

describe the tendency of an organism to persist in ongoing behavior. During baseline, pigeons tended to choose the larger reinforcer more often at the beginning of the session, when its delay was relatively short. Increased choices maintained by the larger reinforcer later in the session did follow the administration of MPD and METH, and therefore it is worthwhile to consider the possibility that perseveration may account for certain characteristics of the present data.

Recall that the delay to the larger reinforcer was presented in a fixed, increasing sequence throughout the experiment. Regardless of the pigeons' behavior, the delay to the larger reinforcer was sure to increase across each session block during each session. When this delay was shortest, a preference for the larger reinforcer was observed in most cases. If perseveration was responsible for the results obtained from the present study, response rates maintained by the larger reinforcer would have certainly been expected to increase at longer delays following MPD and METH, since that was the behavior the pigeons were engaged in early in the session. Therefore, it is possible that increased response rates maintained by the larger reinforcer resulted as a drug-induced increase in perseverative behavior rather than an increased preference for the larger reinforcer later in the session.

Although this interpretation cannot be completely ruled out, it is weakened by data from studies by Richards et al. (1999) and Wade et al. (2000). These investigators have reported drug-induced increases in preference for the larger reinforcer under conditions in which perseveration was unlikely (under an adjusting-amount procedure). However, because the possibility of perseverative responding does complicate the interpretation of the present results, future research might include randomly intermixing

the delay values across the session blocks in order to ensure that results were not merely due to an increase in perseveration.

Summary

The present study used a within-session delay manipulation to obtain delay-discount functions. The consistency and reliably of the within-sessions delay-discount functions suggests that the present study offered an efficient method to study drug effects. The results obtained in the present study resembled those reported by Richards et al. (1999) and others, showing that stimulants increase self-control. Although evidence of a decreased sensitivity to delay was obtained with several pigeons, interpretation of the drug effects was complicated by several issues (i.e., rate dependency, perseveration). As stated earlier, the identification of a behavioral mechanism of drug action is a complex and intricate process. Surely one experiment does not provide sufficient evidence to conclude unequivocally that such a mechanism has been identified. In any case, the results gathered from the present study are promising in several respects. Although more research regarding behavioral mechanisms of drug action is necessary, it is certainly hoped that future research will utilize this approach to continue investigating behavioral mechanisms of drug action.

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