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REVIEW

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Theory and method in the quantitative analysis of "impulsive choice" behaviour: implications for psychopharmacology

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Abstract *Impulsive choice* refers to the selection of small immediate gains in preference to larger delayed gains, or the selection of large delayed penalties in preference to smaller immediate penalties. Current theoretical interpretations of impulsive choice are reviewed, and a synthesis of these ideas, the "multiplicative hyperbolic model of choice", is presented. The model assumes that the value of a positive reinforcer increases as a hyperbolic function of its size, and decreases as a hyperbolic function of its delay and the odds against its occurrence. Each hyperbolic function contains a single discounting parameter which quantifies the organism's sensitivity to the variable in question. The hyperbolic discounting functions combine multiplicatively to determine the overall value of the reinforcer. Equivalent functions are postulated to govern the (negative) value of aversive events, the net value of an outcome reflecting the algebraic sum of the positive and negative values. The model gives rise to a quantitative methodology for studying impulsive choice, based on a family of linear indifference (null) equations, which describe performance under conditions of indifference, when the values of the reinforcers are assumed to be equal. This methodology may be used to identify individual differences in sensitivity to the magnitude, delay and probability of reinforcement. The methodology is also suitable for the quantitative evaluation of the effects of some pharmacological interventions on discounting parameters. Recent psychopharmacological studies of impulsive choice are reviewed, and the utility of indifference equations for extending this work, and developing a quantitative psychopharmacology of impulsive choice is discussed.

Key words Impulsive choice · Delay of reinforcement · Probability of reinforcement · Hyperbolic discounting functions · Indifference equations

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Introduction

The purpose of this paper is to review a theory of choice behaviour which may have significant implications for our understanding of "impulsiveness" and "self-control". We will argue that the theory has potential for describing and analyzing choice behaviour in a broad range of experimental settings, which includes, but is not restricted to, the range of situations which has traditionally formed the basis of laboratory models of "impulsive choice". We will attempt to show how the theory gives rise to a quantitative behavioural methodology which may provide a basis for disentangling some of the interacting factors that determine choice behaviour. Finally, we will discuss the implications of the theory for studies of the psychopharmacology of impulsive choice. First, however, it is necessary to consider the conceptual basis of "impulsiveness".

Definitions of impulsiveness

"Impulsiveness" features in current psychiatric taxonomies (e.g. DSM-IV: American Psychiatric Association 1994), both as a problematic behavioural tendency exhibited by patients suffering from various psychiatric conditions, and as the name for a group of disorders in which this tendency is the most prominent clinical feature ("impulse control disorders"). Impulsiveness is defined as "the failure to resist an impulse, drive or temptation to perform an act that is harmful to the person or to others" (American Psychiatric Association 1994, p. 609). This definition, though helpful in clinical settings, lacks the operational quality that would allow its application in the animal behaviour laboratory. Moreover, it fails to distinguish clearly between impulsiveness and aggression, features which often coexist in individual patients, but which are usually regarded as distinct entities by psychologists.

Personality theorists have devised a number of measures of impulsiveness and self-control, mostly, but not

exclusively, based on self-report questionnaires (Barratt 1981, 1983; Barratt and Patton 1983; Eysenck et al. 1985). These measures have helped us to see impulsiveness/self-control as a behavioural feature that can vary between individuals and which may correlate with biological variables (Barratt 1983; Eysenck and Eysenck 1978). However, such measures are primarily descriptive of behavioural tendencies in complex social situations, and as such are unlikely to illuminate more fundamental behavioural processes that may be entailed in impulsiveness in animals.

The term "impulsiveness" has been applied to many different aspects of the operant behaviour of humans and animals, for example the emission of premature responses in schedules in which reinforcement is made contingent upon pausing (Gordon 1979; van den Broek et al. 1987a; Sagvolden and Berger 1996), emitting short-latency incorrect responses in conditional discrimination tasks (Kagan 1966; van den Broek et al. 1987b; Harrison et al. 1997; Evenden 1999), failure of responding to decline in extinction schedules (Berger and Sagvolden 1998; Sagvolden et al. 1998), premature termination of sequences of responses (Evenden 1998), impaired temporal differentiation of responding (Walker 1982; van den Broek et al. 1992), and choice of smaller earlier reinforcers in preference to larger delayed reinforcers (Ainslie 1975; Herrnstein 1981; Mazur 1987; Logue 1988). It seems unlikely that such disparate behaviours reflect a unitary underlying behavioural process; however, deficits in "behavioural inhibition" (Soubrié 1986), "waiting capacity" (Thiébot et al. 1985), timing (Siegman 1961; Barratt 1981), "behavioural switching" (Ho et al. 1998), and tolerance of delay of gratification (Mischel 1966; Logue 1988) have been proposed to encompass many of these behavioural phenomena.

The theoretical model discussed in the present paper is relevant to interpretations of impulsiveness in terms of "intolerance of delay". It is *not* our contention that the theory is able to account for all the diverse behaviours that have traditionally labelled as "impulsive". Rather, we will try to show that the model can help us to understand one such behaviour, "impulsive choice", by enfolding it into a more general theory of operant choice behaviour.

Impulsive choice refers to the selection of small short-term gains in preference to larger delayed gains, or the selection of larger delayed losses in preference to smaller immediate losses. A homely example of the former situation might be a child's impulsive decision to spend his weekly pocket money on chocolate bars, rather than saving it towards a larger delayed goal, such as the purchase of a bicycle. A familiar example of the latter situation might be a student's impulsive decision to accept the potentially severe but delayed consequences of poor examination performance, rather than suffer the inconvenience of an evening spent poring over his lecture notes (see Ainslie 1975; Deluty 1981; Herrnstein 1981).

The multiplicative hyperbolic model of choice

The model attempts to combine a number of quantitative principles that are believed to contribute to the determination of reinforcer value. (The *value* of a positive reinforcer has the status of an intervening variable which may be inferred from, and measured in terms of, relative preference in concurrent [choice] schedules of reinforcement: see Rachlin et al. 1991). The model attempts to bring together certain well-established principles of choice behaviour in a novel way; it does not invoke any novel, unproven principles. The model is founded on the proposition that reinforcer value is determined by the product of a series of hyperbolic functions, each of which governs the influence of a particular feature of the reinforcing stimulus. In principle, any number of features could be co-opted into the model. However, for the present purposes, only the magnitude, delay and probability of reinforcement will be considered. Some aspects of the model have been discussed previously (see Bradshaw and Szabadi 1992; Ho et al. 1997, 1998); here, we outline the logic of the model and illustrate how it may be used to examine the effects of interventions that influence the sensitivity of organisms to delayed and probabilistic reinforcers. We will only consider the application of the model to steady-state behaviour, as most of the evidence cited in support of the model derives from the analysis of choice behaviour in situations in which the subjects have received extensive experience of the reinforcement contingencies.

Postulate 1

The value of a positive reinforcer presented immediately following an operant response (*instantaneous* value, V_i^+) is assumed to be an increasing hyperbolic function of its physical magnitude or quantity, q:

$$V_i^+ = \frac{1}{1+Q^+/q}. \qquad (1)$$

Q^+ is the discounting parameter for the reciprocal of reinforcer magnitude. This relation is implied by Herrnstein's (1970) hyperbolic response-strength equation, and has been assumed by a number of other models of positive reinforcement (e.g. Vaughan 1985)[1]. Herrnstein's equation has received extensive experimental confirmation both with animals (see de Villiers 1977; Heyman and Monaghan 1987; Bradshaw and Szabadi 1989) and with humans (see Bradshaw and Szabadi 1988; Kollins

[1] The dependent variable in Herrnstein's (1970) equation is "response strength" rather than reinforcer value, whereas Vaughan (1985) uses the hyperbolic function to define value. The nature of the function governing the translation of value into overt behaviour is an important issue which is not addressed in this paper. One of the advantages of the indifference-point approach advocated in this paper is that it circumvents the problem of specifying this function by focusing on situations in which the values of the choice alternatives are equal (see below)

et al. 1997). [Strictly speaking V_i^+ is the reinforcer value relative to a hypothetical maximum value, V_{max} (Vaughan 1985). However, in choice procedures in which relative response measures are used, V_{max} cancels out of the relevant equations; therefore it will be omitted from the present exposition (see also Bradshaw and Szabadi 1992).]

Postulate 2

The value of a positive reinforcer whose delivery is delayed for some time, d, after an operant response (V_d^+) is assumed to be a decreasing hyperbolic function of d:

$$V_d^+ = \frac{1}{1 + K^+ \cdot d}. \quad (2)$$

K^+ is the discounting parameter for delay of reinforcement. This equation was proposed by Mazur (1987), following earlier work by Ainslie (1975; Ainslie and Herrnstein 1981), and has received extensive confirmation in experimental studies of animal and human choice behaviour (see King et al. 1992; Kirby 1997; Mazur 1997). The notion of hyperbolic "delay discounting" is at variance with the normative exponential discount function advocated by classical microeconomic theory (Samuelson 1937; Fishburn and Rubinstein 1982), but is compatible with a normative model based on maximization of the rate of gain in repetitive choices (Kacelnik 1997). The superiority of the hyperbolic equation as a descriptor of delay discounting by individual animals and humans has been firmly established by demonstrations of preference reversal. It has repeatedly been shown that preference for a smaller earlier reinforcer reverses (i.e. the larger more delayed reinforcer comes to be preferred) as the delays to both reinforcers are progressively increased by equivalent amounts (Ainslie and Herrnstein 1980; Green et al. 1981, 1994; Christensen-Szalanski 1984; Bradshaw and Szabadi 1992; Kirby and Herrnstein 1995). Preference reversal is incompatible with exponential discounting, and as such, it has been regarded as "irrational" by some economists (Olson and Bailey 1981; Becker and Murphy 1988). It is, however, a robust empirical phenomenon, which is entirely to be expected on the basis of hyperbolic delay discounting (see Rachlin 1974; Herrnstein 1981; Ainslie and Haslam 1992; Rachlin and Raineri 1992).

Postulate 3

The value of a positive reinforcer that occurs with a probability p following an operant response (V_p^+) is assumed to be a decreasing hyperbolic function of the "odds-against" ratio, θ (where θ=[1/p]–1):

$$V_p^+ = \frac{1}{1 + H^+ \cdot \theta}. \quad (3)$$

H^+ is the discounting parameter for the odds against occurrence of a reinforcer. This equation has been shown by Rachlin and colleagues (Rachlin et al. 1986, 1991) to provide a good description of choice between hypothetical probabilistic reinforcers by human subjects. Subsequent studies have confirmed the applicability of the equation to choice between monetary and other material reinforcers in "gambling" experiments (Rachlin and Siegel 1994; Green et al. 1997). The identity of the forms of Equations 2 and 3 has led some authors to argue that delay and "odds against occurrence" are functionally equivalent in the determination of choice behaviour (Rachlin et al. 1986; Rachlin and Raineri 1992; Myerson and Green 1995; Green and Myerson 1996). However, Ostaszewski et al. (1998), using human subjects, have shown that time and probability discounting are differentially affected by monetary inflation, suggesting that the two discounting parameters are independently manipulable. To date, the evidence supporting Equation 3 has derived mainly from experiments in which human subjects have been required to choose between hypothetical (usually monetary) rewards. It will be important in future experiments to extend this work to animals making choices between tangible reinforcers such as food (see Kacelnik 1997).

Postulate 4

It is proposed that the overall value of a positive reinforcer is jointly determined by the above three hyperbolic functions:

$$V^+ = V_i^+ \cdot V_d^+ \cdot V_p^+ = \frac{1}{1 + Q^+/q} \cdot \frac{1}{1 + K^+ \cdot d} \cdot \frac{1}{1 + H^+ \cdot \theta}. \quad (4)$$

The three parameters of this equation, Q^+, K^+ and H^+, reflect the extent to which the effects of the corresponding independent variables, q, d and ϑ, are modulated. For example, if H^+=0, value is impervious to reinforcer probability, whereas if H^+=1, value increases as a linear function of reinforcer probability. The multiplicative combination of discount functions has previously been advocated, in the case of delay and probability, by Rachlin and Raineri (1992). Analogous multiplicative combination of reinforcement "sensitivity" parameters has also been advocated in the context of the Generalized Matching Law (Logue 1988; Leon and Gallistel 1998).

Postulate 5

It is postulated that an equivalent set of equations describe the (negative) values of aversive events (for evidence, see deVilliers 1977; Deluty 1981; Lowenstein and Prelec 1992); thus the negative value of an aversive event is assumed to be influenced by the independent variables q^-, d^- and θ^-, modulated by the parameters Q^-, K^- and H^-. A more complex situation arises when outcomes of choices entail both rewards and penalties. It is postulated that the net value of such an outcome is deter-

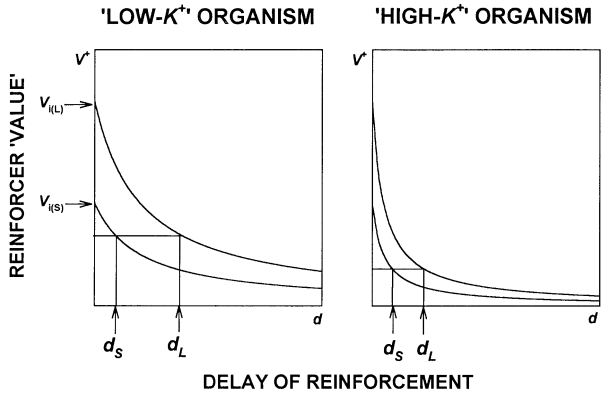

Fig. 1 Hyperbolic time-discounting functions for a large (*L*) and a small (*S*) reinforcer in organisms with low and high discount rates (K^+, cf. Equation 2). $V_{i(L)}$ and $V_{i(S)}$ are the instantaneous values of the reinforcers (i.e. the values when no delay is imposed). A delay (d_S) is imposed on the small reinforcer. The *horizontal line* identifies the delay to the larger reinforcer (d_L) which equates its value to that of the small reinforcer. Note that this "indifference delay" is shorter for the "high-K^+"organism than for the "low-K^+"organism

mined by the algebraic sum of the positive and negative values: $V=V^+-V^-$. For example, the value of a large probabilistic positive reinforcer combined with a small but certain aversive consequence is

$$V=\left[\frac{1}{1+H^+\cdot\theta}\cdot\frac{1}{1+Q^+/q}\right]-\left[\frac{1}{1+Q^-/q^-}\right]. \quad (5)$$

Potential gains and losses may exert quantitatively different effects upon choice, depending on the relative values of the corresponding discounting parameters. For example, the well known "loss-aversion" principle, exemplified by the greater sensitivity of most humans to delayed or probabilistic losses than to delayed or probabilistic gains of equivalent magnitude, may be accounted for by the inequalities $K^+\neq K^-$ and $H^+\neq H^-$ (see Lowenstein and Prelec 1992).

Postulate 6

It is assumed that discounting parameters are relatively stable properties of individual organisms, which reflect their sensitivity to particular features of reinforcing stimuli. To the extent to which they may vary between individuals of the same species, they may be regarded as "personality dimensions" (Herrnstein 1981; Herrnstein and Prelec 1992). However, unlike most other personality dimensions, they are amenable to study in animals, and are susceptible, or so we assume, to experimental manipulation using biological interventions.

Figure 1 illustrates how individual differences in discounting parameters may influence preference. The two graphs show time-discounting functions for a large and a small reinforcer for organisms with low and high values of K^+. Consider first a "low-K^+"organism (left hand graph) faced with a choice between a large reinforcer of instantaneous value $V_{i(L)}$, and a smaller reinforcer of instantaneous value $V_{i(S)}$. If delays are introduced between the subject's choice response and the delivery of the reinforcers, both reinforcers lose value at a rate determined by K^+. Let us impose a brief delay on the delivery of the smaller reinforcer, d_S. The horizontal line shows the value of the small delayed reinforcer. The point of intersection of this horizontal line with the time-discounting function for the larger reinforcer defines the delay that would have to be imposed on the larger reinforcer in order to devalue it to such an extent that it becomes equivalent to the smaller reinforcer (d_L). The right-hand graph shows the corresponding functions for an organism with a high value of K^+. In this case, the values of both reinforcers decline more steeply, and at the standard delay, d_S, the value of the smaller reinforcer is less than it is for the "low-K^+"organism. However, the steep decline in value of the large reinforcer means that in order to match the value of the smaller reinforcer, d_L needs to be considerably shorter for the "high-K^+"organism than for the "low-K^+"organism. Thus, even a relatively short pre-reinforcer delay may lead a "high-K^+"organism to spurn a large reinforcer in favour of a smaller, more immediate reward.

Methodological implications: the application of "indifference-point" ("null-equation") methods to the experimental analysis of choice behaviour

The model outlined in the preceding section provides a conceptual framework for interpreting naturally occurring individual, strain and species differences in operant choice behaviour, as well as the effects of neurobiological interventions on choice behaviour. As discussed above, an organism with high value of K^+ will be predisposed to select small immediate reinforcers in preference to larger delayed reinforcers. Similarly, a biological intervention that results in an increase in the value of K^+ will promote such impulsive choice behaviour. It is important to note, however, that while an increase in the value of K^+ should, ex hypothesi, promote preference for small immediate rewards over larger delayed rewards, an increased preference for small immediate rewards does not necessarily imply an increase in the value of K^+. This can be seen from inspection of Equation 4, which shows that a change in the instantaneous value of a reinforcer (via a change in Q^+) may influence choice behaviour in a qualitatively similar manner to that induced by a change in K^+ (see also Herrnstein 1981). This is an important consideration in the interpretation of empirical preference data. For instance, a number of pharmacological interventions have been found to bias choice in favour of a small immediate reinforcer over a larger delayed reinforcer (see below for references). These effects have generally been interpreted in terms of increases in sensitivity to (or "reduced tolerance" of) delay of reinforcement (in the terminology of the present model, an increase in the value of K^+). However, Equation 4 shows

that it may be very difficult to exclude an alternative interpretation, namely that the treatments altered the subjects' sensitivity to *magnitude* of reinforcement (i.e. reduced the value of Q^+). Discrimination between these two interpretations is not feasible using simple preference data, but may be accomplished using "indifference-point" or "null-equation" methodology.

Null-equation methodology is recognized as an important tool in quantitative pharmacology, which enables conventional dose-response curves to yield up such elusive properties of pharmacological receptors as agonist and antagonist dissociation constants (MacKay 1981; Black and Leff 1983; Hughes and MacKay 1985; Kenakin 1993). Analogous methods are routinely employed in the quantitative analysis of operant choice behaviour (see Mazur 1987, 1997). However, their potential for quantitative psychopharmacology has never been systematically explored. The following example illustrates how methods based on null equations may help us to disentangle some of the interacting factors that determine choice between reinforcers, and may thereby help to clarify the behavioural processes that are involved in the effects of drugs on choice behaviour.

The crux of these methods is that the subject is provided with a choice between two reinforcers, *A* and *B*, and the size, delay or probability of one of them is varied until the subject comes to choose the two reinforcers with equal frequency (i.e. until the subject becomes "indifferent" between the two reinforcers). Under these conditions, it is assumed that the values of the two reinforcers are equal:

$$V_A = V_B \tag{6}$$

(Mazur 1987). One of the advantages of focusing on indifference points is that no assumptions need be made about the relation between reinforcer value and behavioural output; one need only assume that indifference implies equality of value (i.e. Equation 6). Null-equation methods have proved their worth in the analysis of drug-receptor interaction for the analogous reason that they circumvent the problem of non-linear relations between receptor occupancy and biological response which for so many years confounded attempts to estimate dissociation constants from conventional dose-response curves (see Kenakin 1993).

Equation 6 may be expanded by substitution of the appropriate terms from Equation 4. For instance, consider a situation in which an organism chooses between two reinforcers of equal magnitude: *A*, delivered after a brief delay (d_A) with a probability $p_A<1$, (i.e. $\theta_A>0$), and *B*, delivered after a longer delay (d_B) with unit probability. Indifference should be obtained when

$$\frac{1}{1+K^+\cdot d_B} = \frac{1}{1+K^+\cdot d_A}\cdot\frac{1}{1+H^+\cdot\theta_A}. \tag{7}$$

Rearrangement of this equation yields the following linear relation:

$$d_B = d_A(1+H^+\cdot\theta_A) + \frac{H^+\cdot\theta_A}{K^+}. \tag{8}$$

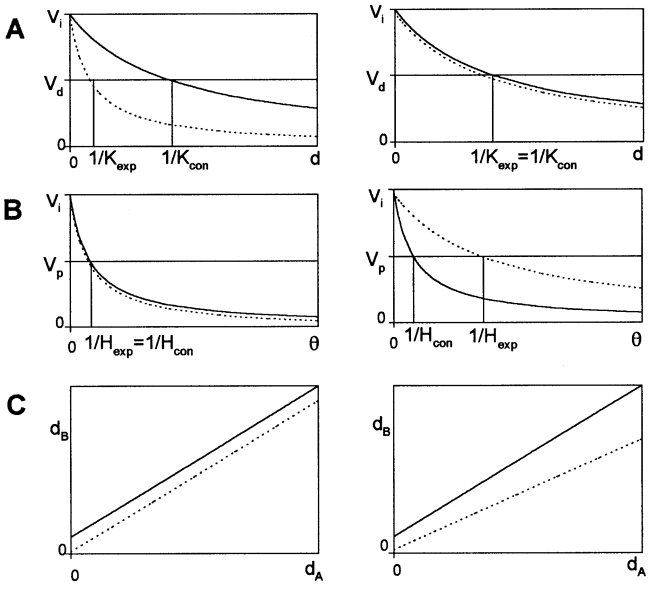

Fig. 2A–C Derivation of the linear indifference function for two reinforcers, *A* and *B*, differing in delay and probability (Equation 8). *Continuous lines* show the "baseline" functions; *dotted lines* show how these functions are altered by a variable that increases K^+ (*left-hand graphs*) and a variable that reduces H^+ (*right-hand graphs*). **A** Relation between value (*V*) and delay (*d*) (Equation 2). **B** Relation between value (*V*) and "odds against" ratio (θ) (Equation 3). **C** Relation between the indifference delay to the certain reinforcer and the delay to the probabilistic reinforcer (Equation 8). Note that a change in H^+ alters the slope of the linear function (*right-hand graph*), whereas a change in K^+ alters the intercept without affecting the slope (*left-hand graph*)

Experimental determination of the indifference values of d_B corresponding to a range of values of d_A should allow a linear function to be obtained (d_B versus d_A). The slope and intercept of this function may be used to derive numerical estimates of H^+ and K^+ (H^+=[slope–1]/θ_A; K^+=[slope–1]/intercept). These estimates may then be compared between subjects that have undergone a relevant neuropharmacological intervention and an appropriate control group.

Figure 2 illustrates functions derived for a hypothetical variable that increases K^+ (i.e. a variable that increases the rate at which reinforcers lose their value as a function of delay; left-hand column), and a hypothetical variable that reduces H^+ (i.e. a variable reduces the rate at which reinforcers lose their value as a function of declining probability; right-hand column); row A illustrates Equation 2, row B Equation 3, and row C Equation 8. Note the change in slope of Equation 8 that results from a change in H^+, but not from a change in K^+.

Ho et al. (1997) derived a similar analysis of choice between reinforcers differing in delay and *quantity*, rather than delay and probability. The relevant linear indifference equation is:

$$d_B = \frac{1}{K^+}\cdot\left[\frac{V^+_{i(B)} - V^+_{i(A)}}{V^+_{i(A)}}\right] + d_A\cdot\left[\frac{V^+_{i(B)}}{V^+_{i(A)}}\right] \tag{9}$$

which expands to:

$$d_B = \frac{1}{K^+} \cdot \left[\frac{\frac{1}{1+Q^+/q_B} - \frac{1}{1+Q^+/q_A}}{\frac{1}{1+Q^+/q_A}} \right] + d_A \cdot \left[\frac{1+Q^+/q_A}{1+Q^+/q_B} \right] \quad (9a)$$

(see Bradshaw and Szabadi 1992; Ho et al. 1997, for derivation). The unwieldy slope and intercept terms of Equation 9a do not lend themselves well to a simple solution for Q^+. However, group differences in Q^+, or effects of interventions on this parameter, can readily be inferred from the slope of the linear relation, which is sensitive to changes in Q^+, but not to changes in the only other free parameter in the equation, K^+. K^+, on the other hand, may be estimated quantitatively from the plot of d_B versus d_A, using the formula K^+=[slope–1]/intercept (see Ho et al. 1997)[2].

The linear relation defined by Equation 9 is well supported by empirical data. Figure 3 shows linear indifference functions for pigeons obtained by Mazur (1987), and Fig. 4 shows data obtained by Ho et al. (1997) using rats. The latter study compared two groups of rats maintained under different food deprivation conditions (80% and 90% of free-feeding body weight). The slope of the function was less steep for the animals maintained under the more severe deprivation condition, indicating a lower value of Q^+ under this condition. This is consistent with evidence for motivation-enhancing effects of more severe deprivation (Bradshaw et al. 1983; Heyman and Monaghan 1987). The values of K^+ obtained from the linear regressions ([slope–1]/intercept) were also lower under the more severe condition, indicating an inverse relation between deprivation and the rate of time-discounting (see Bradshaw and Szabadi 1992; Wogar et al. 1992; Ho et al. 1997). It should be noted, however, that this effect of deprivation on time discounting has not been consistently observed; for example Richards et al. (1997) found no effect of satiation on the value of K^+ in water-deprived rats.

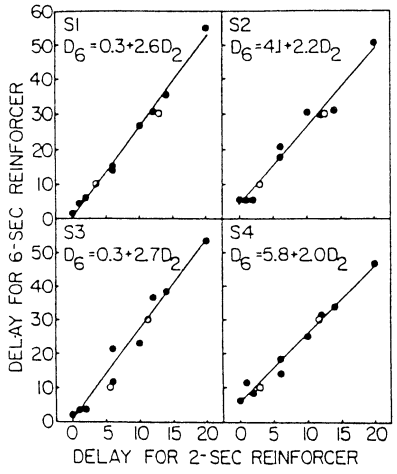

Fig. 3 Linear indifference functions for four pigeons (data from Mazur 1987; reproduced by permission). Indifference delay for a large reinforcer is plotted against the delay to a smaller reinforcer in linear co-ordinates

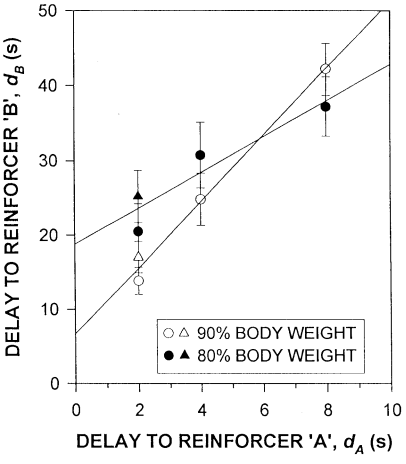

Fig. 4 Linear indifference functions for rats maintained at 80% (*filled symbols*, mean±SEM, n=10) and 90% (*open symbols*, mean±SEM, n=10) of their free-feeding body weights; *triangles* indicate redeterminations (data from Ho et al. 1997). Indifference delay for a larger reinforcer is plotted against delay to a smaller reinforcer in linear co-ordinates (Equation 9). Note the steeper slope and lower intercept for the animals maintained under the milder deprivation condition

Equations 8 and 9 predict that the absolute, as well as the relative, sizes of the reinforcers will affect choice behaviour. This is because the non-linear relation between the instantaneous value of a reinforcer (V_i^+) and its physical size (q), specified by Equation 1, entails a diminution of the ratio of the instantaneous values of the two reinforcers (A and B) if the sizes of both are multiplied by

[2] Numerical determination of Q^+ may be possible using two pairs of reinforcers, as in the following example, which was kindly suggested to us by an anonymous reviewer. Consider an experiment consisting of two phases, in which subjects choose between 1 and 2 food pellets (phase I), and between three and six food pellets (phase II). In each case, the smaller reinforcer (A) is delivered immediately, and the indifference delay to the larger reinforcer (B) is determined experimentally ($d_{B(I)}$ and $d_{B(II)}$). For each phase, the null equation is

$q_A/(q_A+Q^+)=q_B/[(q_B+Q^+)(1+K^+)]$.

Substituting the numbers of food pellets for q_A and q_B, we may derive the following:

$1/(1+Q^+)=2/[(2+Q^+)(1+K^+d_{B(I)})]$ (phase I)

$3/(1+Q^+)=6/[(2+Q^+)(1+K^+d_{B(II)})]$. (phase II)

Rearranging and combining these two equations, and solving for Q^+, yields:

$Q^+=(6d_{B(II)}-2d_{B(I)})/(d_{B(I)}-d_{B(II)})$.

The practical utility of this interesting approach remains to be determined. One potential problem may arise from the reliance on two indifference points, rather than a linear regression based on a range of points, which may result in unacceptably broad confidence intervals

the same factor, x (where $x>1$)[3]. Consistent with this prediction are Mazur's (1988) finding that rats' preference for a larger probabilistic reinforcer over a smaller certain reinforcer was diminished when the sizes of both reinforcers were quadrupled, and Wogar et al.'s (1992, 1993) findings of a reduction of the indifference delay for the larger of two reinforcers when the sizes of both reinforcers were tripled. Similar findings have also been obtained with humans (Kirby and Maraković 1996). Such results have sometimes been said to indicate an effect of reinforcer size on the rate of time- and probability-discounting (Green and Myerson 1996; Kirby and Maraković 1996). However, it should be noted that from the perspective of the present model, the discounting parameters, K^+ and H^+, are not sensitive to reinforcer size; the ability of reinforcer size (q) to alter choice behaviour arises from the modulation of reinforcer value by Q^+.

It will be apparent from the foregoing discussion that linear indifference equations may be derived for many choice situations other than those described in detail above. It seems otiose to enumerate these, as they are all derivatives of Equations 4 and 5. However, it may be worth emphasizing that different linear equations, based on different experimental designs, may be used to estimate the same parameter. For example, H^+ may be determined from an experiment involving choice between reinforcers of equal magnitude but different delays and probabilities (cf. Equation 8), or from an experiment involving choice between non-probabilistic reinforcers differing in magnitude and delay (cf. Equation 9). The availability of multiple avenues to the estimation of the same discounting parameter provides an obvious opportunity for checking on the internal coherence of the model and the consistency of the effects of pharmacological interventions on the discounting parameters.

Techniques for measuring indifference points

The approach advocated in this paper entails lengthy parametric experiments, in which indifference points are measured under steady-state conditions in a series of conditions spanning a broad range of values of the independent variable under investigation. The rate-limiting step in such studies is the determination of individual indifference points. Several concurrent schedule (choice) techniques are available for obtaining indifference points; some more time consuming than others. Two main classes of concurrent schedule are recognized, free-operant and discrete-trials.

Free-operant concurrent schedules

In these schedules, the subject has continuous access to two operanda which are associated with different outcomes. A powerful variant of concurrent schedules, which lends itself well to studies of delayed or probabilistic reinforcement, is the *concurrent chain* schedule, in which equal "initial link" schedules (for example, a pair of concurrent variable-interval schedules) provide access to mutually exclusive "terminal link" schedules (for example, response-independent reinforcer delivery following different delays, i.e. fixed-time schedules: see Zeiler 1977). Relative response rate in the initial links provides the measure of preference, and equality of relative response rates is taken to indicate equality of the reinforcing values of the terminal links (Autor 1960; Herrnstein 1964). Of course, precise indifference may not be obtained in any one pair of schedules; however, the indifference point may be estimated from a series of concurrent schedules by application of the Generalized Matching Law (Baum 1974; Logue 1988).

Gibbon and Church (1981) described a concurrent chain schedule, the "time-left" procedure, that takes advantage of interval timing behaviour to derive a continuous, graded measure of preference within a single session, thus enabling an indifference point to be obtained in one training condition (see also Gibbon and Fairhurst 1994). In this procedure, equal initial-link schedules provide access, at unpredictable times after the start of a trial, to two mutually exclusive terminal links which deliver reinforcement either after a fixed delay or, after a variable delay, at the end of the trial. Early in the trial, the fixed delay is shorter than the variable delay, whereas towards the end of the trial, the variable delay is shorter. Preference for the variable delay accordingly increases from approximately zero at the start of the trial, to near 100% at the end of the trial. The locus of the indifference point (50% choice of the variable delay) depends on the length of the fixed delay and the sizes of the reinforcers offered in the two terminal links. Gibbon and Fairhurst (1994), in a parametric study using this technique, have shown that the indifference delay is a linear function of the length of the fixed delay, in accord with Equation 9.

A perennial problem with the use of free-operant schedules in psychopharmacology is the difficulty of separating motivational effects from "motor debilitating" effects of treatments (see Robbins and Evenden 1985). Although, in the case of concurrent free-operant sched-

[3] This situation differs from the "matching" situation that pertains in conventional concurrent free-operant schedules, where absolute reinforcer size is not expected to influence preference. Herrnstein (1970) showed that the response strengths (measured as response rate, R) in two components, A and B, of a concurrent schedule may be defined as $R_A=k.r_A/(r_A+r_B+r_o)$ and $R_B=k.r_B/(r_A+r_B+r_o)$, where r is reinforcement rate, and k and ro are parameters expressing the maximum response rate and the rate of unscheduled reinforcement (see Bradshaw and Szabadi 1988, 1989 for discussion of the interpretation of these parameters). These formulae yield the familiar matching relation $R_A/R_B=r_A/r_B$ (Herrnstein 1970). Provision of reinforcers of different sizes in A and B biases choice in favour of the larger reinforcer (Miller 1976; Bradshaw et al. 1981); however, multiplication of the sizes of both reinforcers by the same factor should not influence the extent of the bias. The difference between the two situations arises because in the above example, the denominators of the response strength functions are identical, and therefore cancel out to yield the matching relation, whereas this is not the case when exclusive choices are made between delayed or probabilistic reinforcers (see Herrnstein 1981 for discussion)

ules, the use of relative response rate measures may circumvent this problem, these schedules introduce another problem in that they usually allow the subject to switch back and forth between the component schedules without restriction. This switching is highly sensitive to certain pharmacological interventions, for example central 5-hydroxytryptamine (5-HT) depletion (Al-Zahrani et al. 1996; Al-Ruwaitea et al. 1997, 1999b), and changes in switching rate are known to affect some timing and preference indices derived from concurrent schedule performance, including the indifference delay in the time-left procedure (Al-Ruwaitea et al. 1997, 1999a).

Discrete-trials concurrent schedules

These schedules usually consist of a series of trials in each of which the subject has the opportunity to make one choice between two mutually exclusive alternatives. Preference is measured as proportional choice of one alternative across a number of trials. Discrete-trials concurrent schedules tend to engender exclusive preference; this is the expected outcome of repeated exposure to choice between mutually exclusive alternatives, *A* and *B*, since alternative *A* will always be chosen whenever $V_A > V_B$, and vice versa (see Herrnstein 1981). However, indifference points may be determined by systematic manipulation of a relevant independent variable (e.g. delay to one reinforcer), and estimation of the value of the variable corresponding to 50% choice of each alternative by linear interpolation (e.g. Bradshaw and Szabadi 1992).

Al-Ruwaitea et al. (1999a) described a discrete-trials version of the time-left procedure (see above), in which opportunities to choose between a small reinforcer delivered after a short fixed delay and a larger reinforcer delivered after a variable delay (at the end of the trial) were provided at different times during an 84-s trial. The resulting plots of percent choice of the larger reinforcer against time from trial onset were used to derive indifference points, which were shown to be altered by central 5-HT depletion. This schedule offered some advantages over the conventional free-operant time-left procedure, in that it precluded repetitive switching whose occurrence had previously been shown to obscure the effect of 5-HT depletion on indifference delays in the conventional procedure (Al-Ruwaitea et al. 1997).

An important technical development in the estimation of indifference points was Mazur's introduction of the *adjusting delay* schedule (Mazur 1987, 1997). In this schedule, the subject undergoes a series of trials in which the sizes of the two reinforcers and the delay to the smaller reinforcer are fixed, while the delay to the larger reinforcer is allowed to vary in accordance with the subject's choices. When the subject shows a preference for the larger reinforcer, the delay to that reinforcer is increased; when preference shifts to the smaller reinforcer, the delay to the larger reinforcer is reduced. Extended training on this schedule results in the establishment of a quasi-stable delay to the larger reinforcer, which is taken as the indifference delay.

Richards et al. (1997) recently described an ingenious adaptation of Mazur's method to the measurement of indifference points for reinforcer magnitude. Richards et al.'s (1997) *adjusting amount* schedule provides rats with repeated opportunities to choose between a delayed water reinforcer of fixed volume and an immediate water reinforcer the volume of which is adjusted in accordance with the animals' choices. An interesting feature of Richards et al.'s results is the very rapid adjustment of choice behaviour to changes in the delay to the fixed-volume reinforcer, which enabled a family of five indifference volumes to be determined within a 15-week period. This represents a considerable saving of training time compared to other indifference point protocols (e.g. Mazur 1987; Bradshaw and Szabadi 1992; Ho et al. 1997; Al-Ruwaitea et al. 1999a), which could benefit future neurobehavioural applications of indifference point methodology.

Implications for the psychopharmacology of impulsive choice

There is a small but growing literature on the effects of pharmacological interventions on choice behaviour. A major stimulus for this work was Soubrié's (1986) proposal that the ascending 5-HTergic pathways may play an important role in enabling organisms to tolerate delay of reinforcement, and that dysfunction of these pathways may therefore promote selection of small immediate reinforcers in preference to larger delayed reinforcers.

Several studies have investigated the effects of lesions of the 5-HT pathways, and acute treatment with drugs whose mode of action is believed to involve these pathways, on choice between small immediate and large delayed rewards. Wogar et al. (1993) examined the effect of destruction of the ascending 5-HTergic pathways by intra-raphe injections of the selective neurotoxin, 5,7-dihydroxytryptamine, on performance on Mazur's adjusting-delay schedule. The 5-HT-depleted rats showed lower indifference delays to the larger reinforcer than sham-lesioned control rats. Although this result is consistent with an effect of the lesion on the time-discounting parameter K^+, other interpretations cannot be ruled out, because only a single indifference point was determined for each group (see below). Al-Ruwaitea et al. (1999a) compared 5-HT depleted and sham-lesioned rats' tolerance of delay to reinforcement using a discrete-trials time-left schedule. Indifference delays to a larger reinforcer were obtained for four values of the delay to a smaller reinforcer. The lesioned group's indifference delays were significantly shorter than those of the sham-lesioned group, suggesting an increase in the value of K^+. However, to date, there have been no studies in which quantitative estimates of K^+ have been compared between 5-HT-depleted and intact animals.

Performance in impulsive choice paradigms is also affected by acute treatment with drugs that interact with 5-HTergic mechanisms. Using a T-maze procedure in which rats chose between a small immediate reward and a large delayed reward, Bizo et al. (1988) found that 5-HT reuptake inhibitors (clomipramine, zimeldine) increased the proportion of choices directed towards the larger delayed reinforcer; benzodiazepines had the opposite effect (Thiébot et al. 1985; Thiébot 1986), as did the 5-HT depleting agent *p*-chlorophenylalanine (PCPA) (Thiébot 1992). Using the same paradigm, Poulos et al. (1996) found that the 5-HT releasing agent *d*-fenfluramine reduced choice of the smaller immediate reinforcer, while the 5-HT_{1A} receptor agonist 8-hydroxy-2(di-*n*-propylamino)tetralin (8-OH-DPAT) had a biphasic effect, promoting choice of the smaller immediate reinforcer at lower doses, and reducing it at higher doses.

However, these findings have not been found to generalize to other delayed reinforcement paradigms. Charrier and Thiébot (1996) found that PCPA, benzodiazepines, selective 5-HT uptake inhibitors, and the 5-HT_{1A} receptor agonists buspirone, MDL-7305-EF and 8-OH-DPAT, failed to affect transitional choice between reinforcers differing in magnitude and delay in a discrete-trials lever-pressing task.

Evenden and Ryan (1996) found no effect of the tricyclic antidepressant imipramine and the selective 5-HT uptake inhibitor citalopram on the indifference delay for a five-food-pellet reinforcer compared to an immediate one-pellet reinforcer. *d*-Amphetamine promoted choice of the immediate reinforcer, while diazepam and the non-selective 5-HT receptor antagonist metergoline had the opposite effect. The effect of *d*-amphetamine resembled the effect of another psychostimulant, cocaine, which in sub-chronic treatment, has also been found to promote impulsive choices (Logue et al. 1992).

Tomie et al. (1998) examined the effect of ethanol on impulsive choice using a discrete-trials lever-pressing task. Ethanol differentially affected the choice behaviour of rats that tended to make impulsive choices under baseline conditions, moderate doses promoting preference for the smaller immediate reinforcer. Rats that were less liable to make impulsive choices under baseline conditions were less sensitive to the effects of ethanol.

The results reviewed in this section show that impulsive choice is sensitive to a variety of pharmacological interventions. However, the data show considerable variability across studies, and in some cases frankly contradictory results have been obtained with ostensibly similar experimental protocols. There are, no doubt, several reasons for such discrepancies. However, one potential cause of variability is clearly suggested by the model presented in this paper. As discussed above, the multiplicative hyperbolic model proposes that when subjects make choices between reinforcers that differ in size and delay, indifference delays are determined both by K^+ and by Q^+. Separation of the effects of an intervention on these two parameters cannot be accomplished using a single indifference point; quantitative analysis using a range of indifference points is required (e.g. by fitting an indifference function such as that specified by Equation 9). The effect of an intervention on the value of a single indifference point may, in some cases, be quite misleading, as can be seen from Fig. 4, which illustrates how an intervention that affects both K^+ and Q^+ may alter both the slope and the intercept of the indifference function in such a way that tolerance of delay may appear to be increased at some values of delay and reduced at others. It is to be hoped that future studies employing parametric experimental designs may help to clarify some of the ambiguities contained in the extant data on the psychopharmacology of impulsive choice.

Conclusions

Our understanding of choice behaviour has advanced considerably during the past decade, thanks to parallel theoretical and methodological developments in the experimental analysis of behaviour. The aim of this paper has been to provide a synthesis of some of these developments, and to show how they may be used to address questions of interest to psychopharmacologists investigating the neural basis of impulsiveness. From the standpoint of the model discussed in this paper, impulsive choice, that is the selection of small immediate gains in preference to larger delayed gains, or the selection of larger delayed penalties in preference to smaller immediate penalties, is not intrinsically pathological. It arises from the particular combination of the sizes, delays, and probabilities of the positive and negative outcomes facing organism at the moment of choice, and from organismic characteristics which may vary between individuals, and which are summarized by discounting parameters for these outcome features. The model gives rise to a family of quantitative methods for disentangling and measuring these parameters. We hope that these methods will be of value in future studies of the psychopharmacology of impulsive choice.

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