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Comparative Study of the Causal Reasoning of Response-reinforcer Contingency

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I. Introduction

In a variable-ratio (VR) schedule, the number of responses required to deliver a reinforcer varies between successive reinforcers. In a variable-interval (VI) schedule, a reinforcer is delivered for the first response following completion of an varied interreinforcement interval (IRI). When these two schedules provide the same rate of reinforcement, the response rate to the VR is typically higher than to the VI (e.g., Baum, 1993; Peele, Casey, & Silberberg, 1984). For example, Peele et al. recorded the IRIs generated by pigeons on a VR schedule, and then used those IRIs to create VI intervals. When response rates in their first experiment are averaged across subjects, they found rates were 27% higher on the VR than on the VI even though each schedule provided approximately the same rate of reinforcement.

While it is apparent that VR and VI schedules that provide the same reinforcement rate shape different response rates, why do they do so? Two kinds of accounts have been proposed. One is a molecular account which attributes this rate difference to between-schedule differences in the relation between the time between two successive responses (interresponse time or IRT) and the probability of reinforcement (Peele et al., 1984). In particular, on VIs the probability of reinforcement is an increasing and bounded function of IRT duration, while on VRs the probability does not change with the

duration of the IRT. If response emission is controlled by this relation, the differential reinforcement of long IRTs on VI, but not VR, should result in longer IRTs on VI than VR, and, in consequence, lower rates to the former schedule because IRT duration and response rate are inversely related.

The second account is often considered to be molar because it, unlike the IRT reinforcement account offered above, is based on aggregations of IRTs (response rate) and reinforcements over time (reinforcement rate). This model attributes the VR-VI rate difference to differences in the response-rate, reinforcement-rate feedback functions for these two schedules (Baum, 1981). Specifically, marginal increases in response rates produce higher rates of reinforcement on VR than they do on VI. If animals are sensitive to this feedback-function difference, the higher rates seen on VR schedules can be rationalized.

Is organism sensitive to the molecular causality between IRT duration and reinforcement probability, or to the molar causality between response-rate and reinforcement-rate? Tanno and Sakagami (2008; also see Tanno, Silberberg, & Sakagami, 2009, 2010) have suggested that the response-rate difference between VR and VI schedules may be largely attributable to a molecular causality. They made their case by comparing response rates on a VR schedule with those maintained by other schedule types that were purpose-built to alter the molar relationship between response rate and reinforcement rate. Although their between-schedule comparisons permitted the emergence of molar control over response rate (i.e., based on the feedback relation between response rate and reinforcement rate), it did not appear. Instead, the sole factor that seemed responsible for rate differences between VR and all comparison schedules was differences in the reinforced IRT distributions. They interpreted this result as establishing the primacy of a molecular account of the VR-VI rate difference based on between-schedule differences in IRT reinforcement, thereby endorsing molecular modeling of single-schedule effects.

II. Molecular theory successfully predicts response rates under VR and VI schedules: Simulation study (Tanno & Silberberg, submitted)

To further confirmation of the molecular account for the response-rate dif-

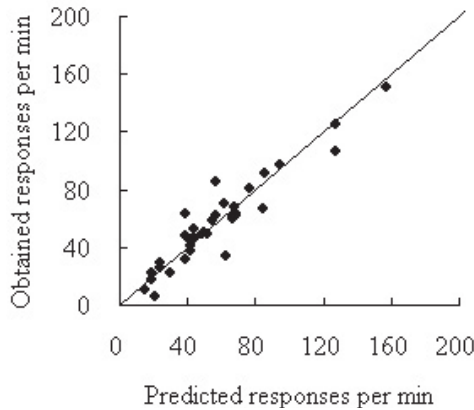


Figure 1. Obtained vs. predicted response rates in responses/min from the molecular model. Diagonal line indicates perfect matching between these two rates.

ference between VR and VI schedules, we conducted the following simulation study. The model assumes that response rate is determined by all IRTs that occur between successive reinforcers. The contribution of these IRTs to rate determination decays exponentially as a function of their distance from reinforcement, with exponential parameter λ . For each response, the model selects an IRT at random from the last 300 exponentially weighted IRTs. The likelihood that an IRT would be selected equals the reciprocal of its duration divided by the sum of the reciprocals of all 300 IRTs. This IRT defines the mean value of an exponential distribution of IRTs. An IRT is chosen at random from this distribution for emission in computer-simulated performances on a variety of aperiodic schedules.

Figure 1 shows results from this simulation. With one or two free parameters (exponential decay and minimum length of emitted IRT; average of these estimations are 0.5 and 0.21, respectively), this model accommodates approximately 90% of the variance seen in response rates obtained in previous studies.

III. Comparative study of the causal reasoning (in progress)

Studies of humans, however, have produced conflicting outcomes. Tanno (2009) examined human performances under regulated probability interval (RPI) schedule. Under the RPI schedule, the n th response was reinforced according to the following equation:

$$P(n) = IRI * t / m, \quad (1)$$

where $P(n)$ is the probability of reinforcement of the n th response, t is the total duration from the $(n - 1 - m)$ IRT to the $(n - 1)$ IRT, and m is the IRT memory size. IRT memory size was set at 30. Therefore, t / m was equal to the average duration of the last 30 IRTs with a lag of one. The definition of t meant that $P(n)$ was independent of the IRT of the n th response, and should therefore result in RPI reinforcer rates that were the same as those for *IRI*. If humans are sensitive to the schedule's molecular property, the rate under an RPI schedule should approximate those observed under a VR schedule, as has observed in rats (Tanno & Sakagami, 2008). However, Tano (2009) showed that humans' response rates under RPI schedule approximated those under the VI schedule, which is consistent with the molar prediction.

The purpose of the present experiment is to test the generality of the inconsistency in causal reasoning between animals and humans. While we showed that molecular theory successfully predicts response rates under several types of schedules (see Figure 1), species of animals used in that studies were limited to rats and pigeons. In the present experiment we replicated the experiments of Tanno and Sakagami (2008) and Tanno (2009) in commonmarmosets. If commonmarmosets show high response rates under RPI schedule, as well as rats and pigeons, it suggests that sensitivity to molar variable is a unique characteristic of humans. In contrast, if commonmarmosets shows low or intermediate response rates under RPI schedule, it demonstrates the sensitivity to molar variables in primates. This experiment is still in progress, so here we describe the plan of the experiment.

Method

Subjects

Three commonmarmoset (2 male and 1 female; *Callithrix jacchus*) served as the study subjects; they were both provided by CLEA Japan, Inc., and were approximately 18 months old at the start of the experiment. They were individually housed in a temperature-controlled room on a 12-h light/dark cycle where they had continuous access to water, and were fed to maintain them at free-feeding weights after each session.

Device

The experiment was conducted in one operant chamber with internal dimensions of 31.5 cm (length) by 33.5 cm (width) by 31.5 cm (height), produced by SHIMAELE, Co., Ltd.. The front wall was made of aluminum and the other three walls, the ceiling, and the floor were made of stainless steel grids. A water bottle was fixed behind the front wall, and the nozzle of which could be inserted into the chamber through a hole located at the center of the front wall, located 11.5cm above the floor and projected 1.2cm into the chamber. Two straight arrays of white LEDs were attached to the chamber's ceiling to provide general illumination. White noise (83-dB) masked extraneous sounds. All experimental events were computer controlled.

Procedure

The commonmarmosets were trained to lever press by auto-shaping and then exposed to a pretraining phase consisting of two or three sessions of exposure to each of the following schedules: continuous reinforcement, VR 5, VR 10, VR 15, and VR 20 in that order. Reinforcers consisted of a 4 s presentation of sucrose solution (5 % of concentration).

In training phase, the commonmarmosets were initially exposed to a VR 20 schedule, and then to inter-reinforcement interval (IRI) yoked VI and RPI schedules. Each condition lasted for 30 sessions, and each session ended after 40 min.

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