Table 2. Identification of bat 111 isolate as VEE virus, type IB by kinetic hemagglutination-inhibition test (*3, 22*). The optimum *p*H for hemagglutination was 6.0. Results are expressed as units inhibited.

Serum dilution	Antigen					
	Bat 111	283424 IB	ICA IB	3880 ID	Mena II IE	Mex64A99 IE
Costa Rica, IB						
1 : 320	> 32	> 16	> 16	> 16	≧ 32	> 32
1 : 640	> 32	> 16	> 16	≧ 16	16	> 32
1 : 1280	≧ 32	≧ 16	≧ 16	4	2	≧ 32
1 : 2560	16	8	8	2	2	2
Mena II, IE						
1 : 80	16	8	≧ 16	4	≧ 32	> 32
1 : 160	< 2	2	8	2	16	≧ 32
1 : 320	< 2	< 2	1	< 2	4	4
1 : 640	< 2	< 2	< 1	< 2	2	1

against the endemic Florida (FE3-7C) strain (*11*) of VEE virus (Table 1). Kinetic hemagglutination-inhibition studies (Table 2) demonstrated the close relation between the isolate and an epidemic strain of VEE virus, type IB. The titer of serum from bat 111 was 1 : 20 by a 90 percent plaque-reduction neutralization test in duck embryo cells (*12*); thus, circulating antibody and virus in the viscera were present simultaneously. Neither antibody nor virus was detected in the other 17 bats, nor was virus recovered from other tissues of bat 111.

Vampires subsist on a strict blood diet and frequently feed on horses; in fact, rabid vampire bats cause more than 10,000 equine deaths every year in Mexico (*13*). In horses infected with the epidemic strain of VEE, viremia titers may approach $10^{7.2}$ suckling mouse intracerebral LD_{50}/ml (*14*). This may be enough to cause infection of a susceptible vampire bat by ingestion of infected blood, since this species commonly consumes 20 to 25 ml of blood a day (*15*). On the other hand, this bat may have been bitten by mosquitoes that had taken a blood meal from an infected horse. Cases of fatal equine encephalitis had occurred within 500 yards of the location of capture of bat 111.

Many arboviruses, including VEE virus (*16*), have been isolated from bats collected in areas where these viruses are endemic (*17*). Moreover, Sulkin and his co-workers have accumulated data which indicate that bats could serve as ideal reservoir hosts for arboviruses (*18*). Vampires cohabit with many other species of bats (*19*), including the Mexican freetail (*Tadarida brasiliensis mexicana*) (*19, 20*), which migrates in massive numbers to the southwestern United States every spring (*21*), and there appears to be ample opportunity for transfer of the virus to areas far from an infected focus.

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Δ^9-Tetrahydrocannabinol: Dose-Related Effects on Timing Behavior in Chimpanzee

Abstract. *Δ^9-Tetrahydrocannabinol, at doses within the effective range for humans, was administered orally to chimpanzees with stable, efficient timing performances maintained by multilink chained schedules of food reinforcement. Reinforcements decreased with increasing dose, because of decreased frequencies of total operant timing responses and decreased accuracy of the timing performances which did occur. Higher doses exerted an effect for up to 3 days.*

One difficulty encountered in interpreting results from animal studies of Δ^9-tetrahydrocannabinol (Δ^9-THC), the presumed principal psychoactive constituent of marihuana, has been the high doses generally needed to produce behavioral effects. This drug, in oral doses of about 0.200 mg/kg, has been reported to produce marihuana-like effects in humans (*1*). As part of an investigation of its behavioral and toxicological effects, oral doses ranging from 0.125 mg/kg to 4.0 mg/kg were administered to three chimpanzees (*2*)

with highly efficient and stable schedule-controlled timing behavior, and dose-related behavioral effects were observed within the human dose range. A food reinforcement schedule requiring performance based on passage of time was selected because there is a known tendency of humans to overestimate passage of time during marihuana intoxication (*1*, *3*).

The three adult chimpanzees had been maintained for the last 5 years in a multianimal environment for primates with their behavior under control of reinforcement schedules almost identical with the one described below. Each subject was semi-isolated in its living-work compartment, the front wall of which served as a work panel. Each contained a clocklike self-identification device, a pair of transilluminated pushbuttons (A and B), red and green cue lights, and food and water delivery mechanisms.

A multilink chained schedule of food reinforcement was in effect approximately 20 hours per day. Each response chain required operation of the self-identification device by moving the clock arm to an assigned position for 5 seconds. Then a contingency requiring temporarally spaced responses was imposed. The animal first had to respond on pushbutton A, and then wait at least 60 seconds, but not longer than 90 seconds, before responding on pushbutton B in order to obtain food upon completion of the whole response chain. If a correct A-B interresponse time (A-B IRT) occurred (between 60 and 90 seconds), the first B response turned on a green cue light and the 500th B response resulted in food delivery and reinitiated the chain. However, when the preceding A-B IRT was incorrect (less than 60 seconds or greater than 90), the first B response produced a red cue light, and the 500 B responses were required to reactivate the self-identification device and hence to reinitiate the chain; there was no food delivery at the completion of the 500th response. Session durations were lengthened by imposing a 30-minute "timeout" period after completion of every tenth response chain regardless of food deliveries obtained. Data were collected to ascertain daily work outputs, response rates, reinforcement frequency, accuracy of timing performances, and the onset and time-course of Δ^9-THC activity.

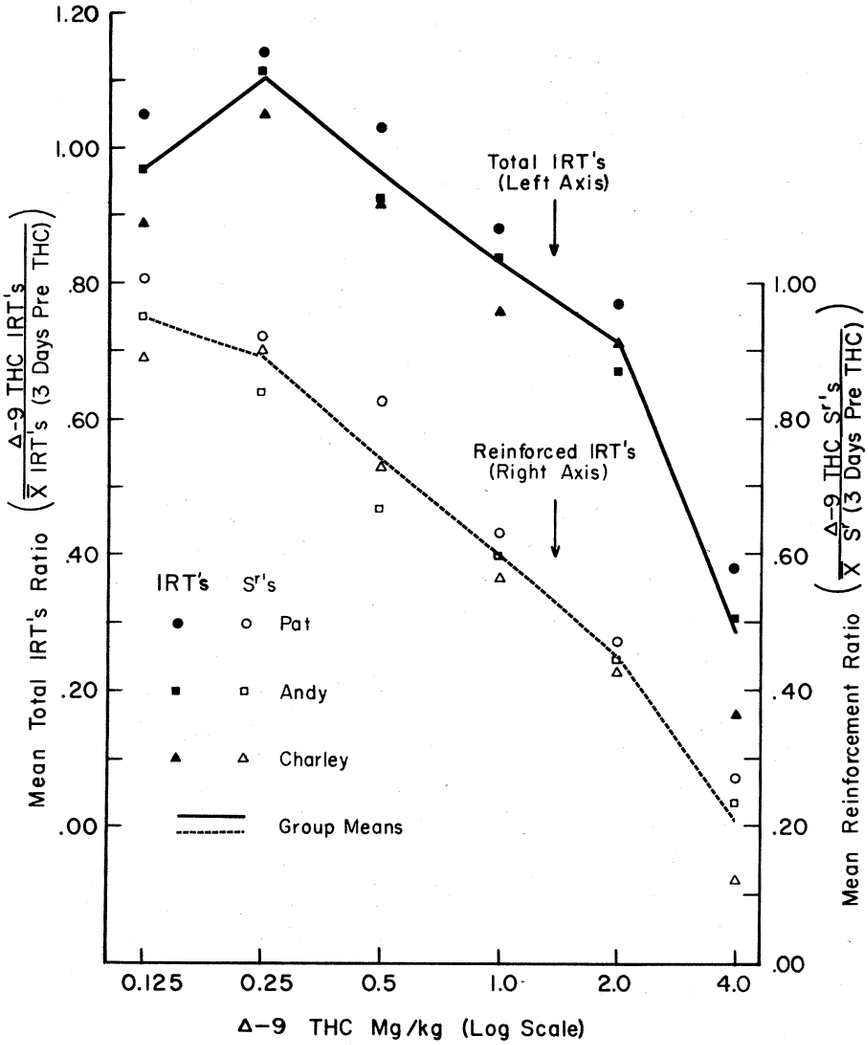

Fig. 1. Dose-related effects of Δ^9-THC on total number of A-B interresponse times (that is, daily work output) and on frequencies of those A-B IRTs, between 60 and 90 seconds' duration, which resulted in food reinforcements (S^rs) for each of the three chimpanzees. Dose levels of Δ^9-THC are shown on the log scale of the horizontal axis. The ratio values for total IRTs (left-hand axis) and for reinforced IRTs (right-hand axis) were obtained by dividing the averaged number occurring on 2 days of Δ^9-THC administrations at a given oral dose by the averaged number occurring on three placebo-control days prior to each of the drug administrations at that dose level. Ratio values for individual subjects are marked by symbols. Group mean ratio values for all three subjects are connected by the solid lines (total IRTs) and the broken lines (reinforced IRTs). The left- and right-hand axes are offset to separate the two sets of ratios.

Effects of Δ^9-THC on reinforced interresponse times (IRTs) are illustrated by the open symbols and broken-line curves in the lower portion of Fig. 1. Mean frequencies of reinforced IRTs obtained on drug days have been divided by the averaged frequencies of reinforced IRTs for the three placebo-control days prior to each day at that dose level. Values of these drug-placebo ratios of reinforced IRTs are shown on the right-hand axis of Fig. 1. Reinforced IRT frequency was suppressed increasingly as a function of drug dose. The group mean drug/placebo ratio decreased from 0.95 at the low dose, 0.125 mg/kg, to about 0.20 at the high dose, 4.0 mg/kg. Highly similar dose-dependent effects of Δ^9-THC on number of reinforced IRTs for timing performances were obtained from each of the three subjects.

These decrements in reinforced IRTs

were due, in part, to suppression of daily work output by the chimps. The total number of response chains initiated—that is, total A-B IRTs regardless of duration—was taken as a measure of daily work output. Mean work output on days of drug administration at a given dose level was compared with averaged output for three prior placebo-control days in the same manner as described above for the reinforced IRTs. Effects of Δ^9-THC on work output are illustrated by the darkened symbols and the solid line in the upper portion of Fig. 1. Values of these ratios of work output are shown on the left-hand axis. Little, if any, effect on work output was observed at doses under 1.0 mg/kg, other than possibly a slight increase in total IRTs with the 0.25 mg/kg dose. Decrements in work output, increasing with dose, did occur with doses of 1.0 mg/kg and larger. The group mean drug/placebo ratio of total IRTs dropped sharply from the high of 1.10 at the 0.25 mg/kg dose level to a low of about 0.30 at the highest dose. Prolonged breaks in performance were observed when the higher doses of Δ^9-THC took effect. The chimps appeared just to sit quietly in their cages for several hours before returning to work, if they did so at all. These breaks in performance occurred both as long intervals between response chains and long delays in completing the 500 response terminal link of the chain following an inappropriate A-B IRT. Similar effects of Δ^9-THC on work output at each dose were obtained from the individual subjects, as was the case with reinforced IRTs. However, the lower doses, as well as higher ones, resulted in decrements in number of reinforcements, but the lower doses did not decrease work output.

In addition to the decreased work output, decreased reinforced IRTs resulted also from decrements in accuracy of the timing performance. Some effects of Δ^9-THC on accuracy of timing are illustrated in Fig. 2, which shows an increasing tendency to overestimate passage of time with increasing dose of Δ^9-THC. Frequency of short A-B IRTs relative to total A-B IRTs—that is, relative frequency of A-B IRTs less than 60 seconds—was taken as a measure of the overestimation error. The three top panels of Fig. 2 illustrate the relative frequencies of overestimation errors produced by the individual subjects, and the bottom panel combines the errors in a group average, at each dose level of Δ^9-THC. Black dots connected by solid lines mark relative frequencies of IRTs less than 60 seconds in duration which occurred on days when doses were administered. Relative frequencies of such short IRTs during placebo-control sessions are shown for 3 days before (black squares connected by dotted lines) and for 3 days after (open circles connected by dashed lines) each administration of Δ^9-THC. Open triangles

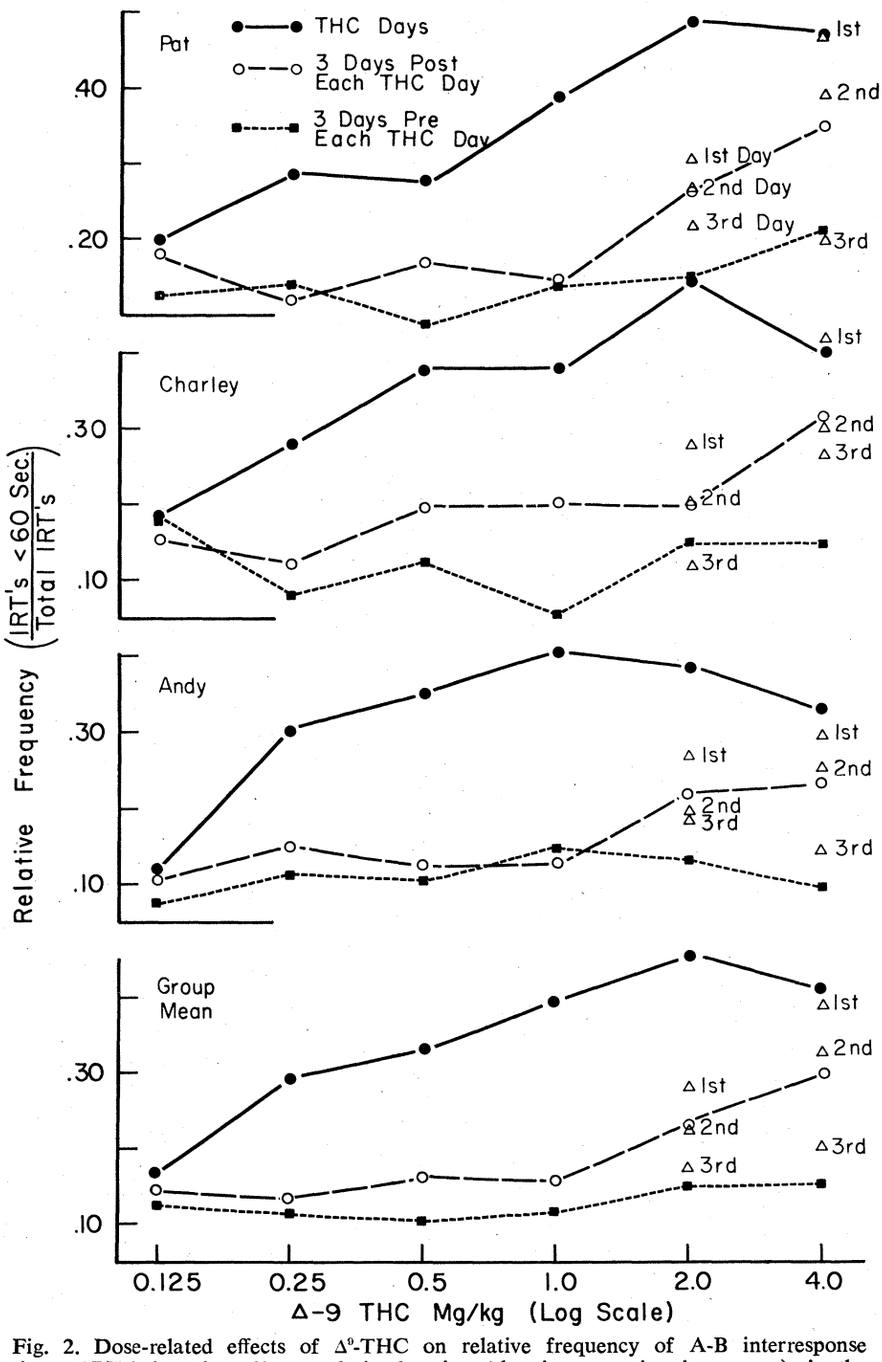

Fig. 2. Dose-related effects of Δ^9-THC on relative frequency of A-B interresponse times (IRTs) less than 60 seconds in duration (that is, overestimation errors), in the timing performances of three chimpanzees. Dose levels are shown on the log scale of the horizontal axis. The numbers of IRTs under 60 seconds in duration are divided by total IRTs regardless of duration. Relative frequencies of these overestimation errors occurring on days when oral doses of Δ^9-THC were administered (black circles connected by solid lines) are compared with relative frequencies occurring during placebo-control sessions 3 days before (black squares connected by dotted lines) and 3 days after (open circles connected by dashed lines) each day of drug administration. Relative frequencies occurring on individual first, second, and third days after administrations of the two highest dose levels are marked by numbered open triangles. Results obtained from individual subjects are shown in the three top panels. Results obtained with all three subjects are combined for the group mean results shown in the bottom panel.

mark the relative frequencies of short IRTs which occurred on individual first, second, and third days after administration of the two highest doses. The combined results shown in the bottom panel of Fig. 2 are representative of the results obtained with the individual subjects shown in the upper panels. Relative frequencies of overestimation errors were low and stable on placebo-control days prior to Δ^9-THC administrations (black squares). When Δ^9-THC was given, overestimation errors increased in relative frequency with increasing dose level (black circles). This effect of Δ^9-THC on accuracy of timing performance diminishingly perseverated on days following drug administrations (open circles). Relative frequencies of overestimation errors were elevated up to 3 days after administrations of the higher doses of Δ^9-THC (numbered open triangles). The other timing error, A-B IRTs longer than 90 seconds in duration, were typically low in relative frequency, never exceeding 10 percent of the work output. The drug had no observable effect on this type of timing error.

These data are important for several reasons. The fact that doses of 1.0 mg/kg and larger decreased the frequency of food-reinforced operant behavior confirms effects that have been observed with other species (*4*). The finding that high doses of the drug continue to exert an effect for up to 3 days also is of interest in view of biochemical data indicating that metabolites of Δ^9-THC persist in the body for up to a week after ingestion (*5*). Finally, the fact that drug effects were obtained at the 0.25 mg/kg dose level, well within the effective dose range for humans, and the fact that the change in timing behavior appears to confirm data on humans, both suggest that the chimpanzee may be a useful animal in studies of marihuana.

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Attentiveness to Sensory Stimuli: Central Control in Locusts

Abstract. *Wind-angle changes evoke yaw-correcting deflections of the abdomen in tethered locusts, but only if the wings are beating. In flight, the central neuronal flight motor drives abdominal motoneurons in rhythmic bursts. Wind angle inputs, which are inadequate alone to drive these motoneurons, alter the number of spikes per burst, and the alterations are reciprocal in opposite nerves. Burst frequency and phase are unaffected.*

How an animal determines at any moment whether or not to respond to a given sensory stimulus is a long-standing problem in the study of animal behavior. In the past, this role of controlling responsiveness has often been relegated to an ill-defined "central excitatory state" or level of "arousal." In the desert locust (*Schistocerca gregaria* ph. *gregaria*), this process can be studied quantitatively in individually identified motoneurons.

A tethered locust responds to head-on wind from a miniature wind tunnel by lifting its abdomen, legs, and antennae into flight posture and beating its wings as in normal flight (*1*). Pivoting the wind tunnel in a horizontal arc about the head mimics most of the aerodynamic conditions of yaw in free flight. This stimulus, which the locust monitors with a bank of delicate cephalic wind-receptor hairs (*2*), evokes a complex yaw-correcting maneuver involving the wings (*3*), abdomen, legs, and head (*4*). If the locust spontaneously ceases flying in the maintained wind stream, none of these movable members respond to wind-angle changes, but all movements reappear in full measure the instant flight spontaneously resumes (*4*).

We have studied the motor mechanisms involved in yaw-correcting movements, and have focused on the switch-like mechanism whereby the insect attends to a flight-relevant stimulus (wind-angle change) during flight and apparently ignores the same stimulus during flightless intervals. In studying the abdomen's response—a lateral, rudderlike deflection determined by the wind angle (*4*)—we find that during flight, the central neuronal flight motor, known to coordinate the patterned wing movements, also drives abdominal motoneurons in synchronous impulse bursts. In a head-on wind, the bursts of both sides are of approximately equal strength and cause balanced rhythmic contractions of opposite abdominal muscles, which lift the abdomen into flight posture and cause a slight vertical vibration. Wind-angle changes modulate, in a bilaterally reciprocal fashion, the number of impulses per motoneuron burst and thereby unbalance the contractions of the two sides and cause a lateral deflection. The wind input, while sufficient to modulate ongoing neural activity, is inadequate by itself to activate the motoneurons.

During flight, the abdomen undergoes high-frequency, low-amplitude vibrations in the vertical plane. Mechanical recordings of this movement (Fig. 1A) show that the vibrations are synchronous with the wingbeat. To determine whether this vibration originates in the abdominal musculature or results passively from mechanical coupling to the vibrating thorax, we isolated the abdomen from the