is conceivable that naturally occurring substances (for example, *l*-amino acids) have transport mechanisms available in the body which are not present for unnatural compounds. Thus, *l*-amino acids might serve as carriers for the active isopropylhydrazide moiety through natural barriers (such as the blood-brain barrier).

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Estimation of Total Body Fat from Roentgenograms

Measurements of the thickness of the subcutaneous fat can be used for estimating man's fatness, and percentile norms for judging the relative fatness of individuals in this way have been provided (*1*). For some purposes, however, an estimate of body fat is desired in absolute rather than in relative terms. Such an absolute estimate can be made with the rather complicated methods of body water and body density measurements. This estimate, in turn, can be used to establish predictions for total fat based on the simpler measurements of subcutaneous fat (*2*). This procedure has proved satisfactory in obtaining estimating equations for total fat from skinfold measurements in young and middle-aged men (*3, 4*).

In the study described in this report the thickness of subcutaneous adipose tissue (plus skin) was measured on soft-tissue roentgenograms, taken at a distance of 72 in. between the tube and the film. No correction for triangular distortion was applied. Data are reported for four sites: (i) upper arm, at the level of the deltoid insertion (see *5*); (ii) upper arm, one-third of the distance between olecranon process and acromion; (iii) forearm; and (iv) calf, at the level of maximal width. All projections were anteroposterior. At site No. 1 the measurements were made vertically to the skin, and at the other three sites they were made vertically to the long axis of the limb. At sites No. 2, 3, and 4 both the lateral and medial thicknesses were measured and summated. Total body fat was estimated from body density, the body volume being obtained by underwater weighing with individual corrections for air remaining in the lungs and respiratory passages at the moment weight was recorded.

Middle-aged business men and professional men, participating in a longitudinal study of aging (*6*), were the subjects. The analysis was restricted to 52 men (mean age 57.1, S.D. = ± 2.7 years) whose weight did not change by more than ± 2 percent from the time of the density measurements to the time when the roentgenographic data were obtained 4 years later.

Equations for predicting body density from roentgenographic measurements and the coefficients of correlation between the two types of criteria of leanness-fatness are given in Table 1. The correlations here recorded must be considered to be slightly depressed from those that would be obtained with measurement of total fat and recording of the x-ray patterns on the same day. But the correlations in Table 1 are in the same general range as those reported when subcutaneous fat was measured with skinfold calipers (*3*). No precise comparison between the x-ray and the skinfold caliper measurements previously made is possible because of differences in the measurement sites.

The number of individuals for whom satisfactory x-ray data were available in all four sites was relatively small. Consequently, no attempt was made to relate body density to roentgenographic measurements in the form of a multiple-regression equation. This is a task for further research in which attention should be given also to some areas on the trunk, including those not readily measurable by skinfold calipers, such as the trochanteric area.

Table 1. Equations ($\hat{D} = a + bX$) for predicting body density from roentgenographic measurements (X, in millimeters) and coefficients of correlation (r) between density and roentgenographic measurements. N = size of the sample.

Site	a (intercept)	b (slope)	r	N
1. Deltoid insertion	1.07220	−0.00186	−.60	42
2. Upper arm	1.07812	−0.00219	−.75	41
3. Forearm	1.07294	−0.00309	−.76	51
4. Calf	1.06447	−0.00228	−.58	47

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Effect of Reserpine Pretreatment on Stimulation of the Accelerans Nerve of the Dog

Abstract. Pretreatment with two doses of reserpine (each 0.1 mg/kg, intraperitoneally) sensitizes the heart to the positive chronotropic action of norepinephrine and reduces the response to stimulation of the accelerans nerve. Ganglionic transmission remains unaffected. The results indicate that the presence of certain stores of peripheral sympathetic transmitter is essential for the production of tachycardia by stimulation of the accelerans nerve.

Recent experiments (*1*) show that reserpine is capable of causing a tachycardia in the heart-lung preparation of the dog by liberation of norepinephrine from its stores in the heart. Pretreatment of the dogs with reserpine prior to the isolation of the heart, by depletion of the stores of norepinephrine, prevented the positive chronotropic response of the heart-lung preparation to the challenging dose of reserpine. A dose of 0.1 mg/kg injected intraperitoneally 24 hours before the heart-lung preparation was set up was found to suffice for the pretreatment (*2*).

Other experimental evidence indicates that pretreatment with reserpine abolishes the stimulant action of nicotine on isolated rabbit atria (*3*); in this preparation nicotine acts presumably on ganglion cells or chromaffine tissue situated in the heart wall, or on both, and thus liberates sympathin. Reserpine has also been found to reduce the norepinephrine content of sympathetic ganglion cells (*4*).

The experiments described in this report (*5*) were undertaken in order to

study the effect of reserpine-induced depletion of the norepinephrine stores of the heart on the response of the heart rate of stimulation of the accelerans nerve. The ganglion cells of these sympathetic fibers are located in the stellate ganglion and thus differ from the nicotine-sensitive structures of the heart wall. By applying both pre- and postganglionic stimulation, we have tried to establish whether the changes caused by reserpine are fully or partly due to its ganglionic effects (*4*).

The reserpine pretreatment was carried out with two doses of 0.1 mg/kg each, given intraperitoneally 48 and 24 hours prior to the experiment. Fourteen dogs (seven controls and seven pretreated) were anesthetized with 35 mg of sodium pentobarbital per kilogram (some of the pretreated animals required less); the chest was opened, and the preganglionic fibers of the right accelerans nerve were prepared after their central connections had been cut. Electrical stimulation (25 shocks, of 0.7 msec duration, per second) was applied for 10 seconds; the strength of stimulation was increased until a maximal acceleration of the heart beat was obtained. The postganglionic fibers were then prepared at a distance of about 8 mm from the stellate ganglion, and supramaximal stimulation was applied. In order to ensure that the bipolar platinum electrodes were functioning the left vagus was also stimulated. Stimulation of the vagus as well as of the pre- and postganglionic fibers of the right accelerans nerve was repeated after the intravenous injection of 1 mg of atropine sulfate per kilogram, which abolished the response to vagal stimulation. Finally, the response of the heart rate to the intravenous injection of increasing amounts of norepinephrine bitartrate (0.075 to 7.5 μg/kg free base) was determined. The heart rate was read from electrocardiogram tracings recorded with a Grass ink-writing oscillograph.

Pre- or postganglionic stimulation of the accelerans nerve caused pronounced tachycardia in five of the seven control animals, but the response of the heart was rather small in two (C5 and C6) (Table 1). These two animals, however, were found to be relatively insensitive to injections of norepinephrine (Table 2). There was neither a significant difference between the response of the heart rate to pre- and postganglionic stimulation, nor did the administration of atropine cause a significant change in the response of the heart. The mean response of the heart to pre- or postganglionic stimulation of the accelerans nerve (before or after atropine) was equivalent to that caused by the injection of more than 4.9 μg of norepinephrine per kilogram; the reduced response of dogs C3 and C6 to the second period of preganglionic stimulation was probably due to some damage to the nerve fibers.

Six of the pretreated animals were in good condition; only one (R4) suffered from severe diarrhea. The initial heart rate of the reserpine-pretreated animals was significantly lower than that of the controls ($P < 0.001$). The initial blood pressure was also lower, though not significantly; this scatter of results may have been due to the fact that some of the pretreated animals required a smaller dose of sodium pentobarbital.

Pretreatment with reserpine abolished or reduced the response of the heart rate to stimulation of the right accelerans nerve; the difference between the two groups of animals was highly significant ($P < 0.001$). Pretreatment with reserpine did not affect the transmission of maximal impulses through the stellate ganglion, since there was no significant difference between the response of the heart rate to pre- and postganglionic stimulation. Atropine likewise failed to change the response of the heart. Injections of norepinephrine, on the other hand, were found to have an increased action in pretreated animals; the difference between the two groups of animals was highly significant ($P < 0.001$). The increased response of the heart to norepinephrine cannot be fully explained by the lower initial heart rate of the pretreated animals, because these were found to respond to such small doses of norepinephrine as failed to elicit tachycardia in the control animals. Comparison of the positive chronotropic effect of stimulation of the accelerans nerve with that of intravenous injections of norepinephrine shows that, in the pretreated animals, nerve stimulation caused a heart rate increase equivalent to that of an injection of less than 0.18 μg of norepinephrine per kilogram (controls: more than 4.9 μg/kg).

Table 1. Response of the heart to nerve stimulation in normal (C) and reserpine-pretreated (R) dogs. *a*, Increase in heart rate (beats per minute); *b*, dose of norepinephrine (micrograms per kilogram, intravenous) causing equivalent increase in heart rate as calculated from Table 2 (with exception of R1 for which other doses of norepinephrine were chosen for the dose-response curve).

Dog	Before atropine						After atropine			
	Initial conditions		Preganglionic stimulation		Postganglionic stimulation		Preganglionic stimulation		Postganglionic stimulation	
	Heart rate (beat/min)	Blood pressure (mm-Hg)	*a*	*b*	*a*	*b*	*a*	*b*	*a*	*b*
					Normal dogs					
C1	161				78					
C2	148	134	90	> 7.5	84	> 7.5	69	> 7.5	61	> 7.5
C3	152	110	84	> 7.5	75	> 7.5	35	2.0	64	> 7.5
C4	175	106	108	> 7.5	98	> 7.5	76	> 7.5	73	> 7.5
C5	185	150	68	> 7.5	51	> 7.5	48	> 7.5	25	> 7.5
C6	149	136	17	5.1	29	> 7.5	2	0.9	20	6.1
C7	146	126	65	4.7	89	> 7.5	55	3.9	78	6.5
Mean	159.4	127.0	71.8	> 6.6	72.0	> 7.5	47.5	> 4.9	53.5	> 7.1
S.E.	± 5.7	± 6.8	± 12.7		± 9.1		± 10.9		± 10.1	
					Reserpine-pretreated dogs					
R1	100		0	< 0.08	13	0.26	13	0.26	22	0.46
R2	138	114	10	0.10	7	0.09	14	0.12	16	0.14
R3	103	99	3	0.12	0	< 0.08	1	< 0.08	0	< 0.08
R4	69	52	2	0.09	0	< 0.08	2	0.09	4	0.11
R5	110	104	1	< 0.08	2	< 0.08	0	< 0.08	3	< 0.08
R6	122	124	17	0.36	20	0.38	15	0.35	13	0.29
R7	118	119	4	< 0.08	2	< 0.08	4	< 0.08	13	0.08
Mean	108.6	102.0	5.3	< 0.13	6.3	< 0.15	7.0	< 0.15	10.1	< 0.18
S.E.	± 8.2	± 10.7	± 2.3		± 2.9		± 2.5		± 3.0	
P	< 0.001	> 0.05	< 0.001		< 0.001		< 0.01		< 0.01	

Table 2. Response of the heart to intravenous injections of norepinephrine in normal (C) and reserpine-pretreated (R) dogs after intravenous administration of atropine (1 mg/kg).

Dog	Increase in heart rate (beats/min) after intravenous administration of norepinephrine (μg/kg)				
	0.075	0.225	0.75	2.25	7.5
			Normal dogs		
C2	0	0	8	30	60
C3	0	0	12	48	60
C4	0	0	18	46	72
C5	0	0	0	8	14
C6	0	0	1	7	23
C7	0	11	38	44	83
Mean	0	1.8	12.8	30.5	52.0
			Reserpine-pretreated dogs		
R2	4	30	52	89	102
R3	2	5	26	69	108
R4	1	12	19	40	90
R5	5	13	32	72	106
R6	4	8	38	80	96
R7	13	32	41	88	111
Mean	4.8	16.7	34.7	73.0	102.2
P			< 0.001		

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Influence of Social Interactions on Learning Rates in Birds

That learning may be influenced by the behavior of an individual's congeners is fairly obvious (*1*). Relatively little evidence exists, however, demonstrating the role of observational learning among animals in their normal environment, though such evidence as there is indicates that imitational behavior may play a major role in the ontogeny of species-specific response patterns (*2*).

Previous work has shown, for example, that the learning of feeding and avoidance responses by greenfinches (*Chloris chloris*) is greatly affected by the presence of a second individual (*3*). While single birds learned a discrimination rapidly, as did birds which had been trained after having been allowed to observe an already trained bird performing, birds which were being trained in the presence of a naive partner required considerably longer. Finally, when well-trained birds observed the performances of untrained partners, their own performances, which had previously been correct, repeatedly fluctuated to random, or nondiscriminatory, levels. Behavioral data suggested that temporary effects such as social facilitation were not involved, but, rather, that the sight of another bird feeding can actually serve as an unconditioned stimulus potent enough to overcome the negatively reenforcing effect of noxious food.

In the experiments described in this report (*4*) the speed with which adult, wild-trapped greenfinches learned to discriminate between a palatable and unpalatable food source was compared for single birds and heterosexual pairs. These birds were housed in wire-mesh aviaries of volume approximately 110 ft,[3] and located on the edge of a wood, in conditions approximating the natural habitat of the species. Food and water were available *ad libitum*, and adequate plant cover and perches were provided. In addition, in each cage was placed a sprig of box (*Buxus* sp.) and ivy (*Hedera* sp.) to whose leaves were glued sunflower seeds or sunflower seeds with moist aspirin replacing the kernel. There were six seeds on each sprig, single birds receiving one pair of sprigs (box and ivy), paired birds receiving twice that number. The sprigs were replenished twice daily. For half of the birds the ivy served as the source of the noxious food (aspirin filled seeds); for the other half of it was box. An error was scored as a failure to take a palatable seed or as the seizing of an unpalatable one. The criterion for seizing was the splitting of a seed or its removal. After the first few trials, virtually all errors were due to taking the incorrect seed rather than to failure to feed.

Because weather conditions and day length were known to affect motivation and hence learning speed, the only valid comparisons that can be made are between paired and single birds which were tested simultaneously. The data given in Table 1, when presented in this fashion, show a striking parallel with those achieved in the previous experiments, which were conducted in a more artificial fashion (*3*). In eight instances the single birds, whether male or female, learned with considerably greater rapidity than the paired birds, who, in several instances, failed to learn the discrimination altogether. In the single instances where the pair learned as rapidly as the single control, observations established that only the female was, in fact, responding. In all other pairs, both birds simultaneously participated in the feeding, at least during the observation period.

This interference with discrimination learning in social situations can fail to be maladaptive only among species whose feeding responses are so conservative as to virtually eliminate the likelihood of their feeding on some unsuitable or noxious food. As earlier work has suggested (*3*), the observation of a partner's feeding response is powerful enough a stimulus, even after delays of 24 hours, to overcome previously established avoidance behavior. Presumably this will not be true of species with a more varied diet or a more opportunistic feeding habit than greenfinches. One might also expect a different situation in species which remain in flocks throughout the year. Investigation of these possibilities should be of considerable value in an elucidation of the relation between learning processes and social organization.

Table 1. Number of errors on successive trials for paired and single greenfinches. The differences between paired and single birds were significant ($p = .05$ or less). Trials were discontinued as soon as one group was discriminating with an accuracy of 11 or less errors in three consecutive trials, this number of errors representing a significant level of discrimination ($p = .05$ or less). The errors made by the single bird were doubled, in order to make direct comparisons possible. The minus signs (−) refer to trials on which the birds did not take either palatable or unpalatable seeds.

Bird	Errors
Pair 1	12, −, 14, 9, 3, 6*
Male 1	−, −, 6, 0, 2, 0
Pair 2	12, 13, 9, 9, 9, 9, 10, 8, 8
Male 2	6, 10, 12, 12, 10, 12, 4, 2, 2
Pair 3a	−, −, 9, 6, 7, −, 6, −, 4, 2, 0, 3, 3, 0, 0†
Pair 3b	11, 8, 12, 7, 12, 7, 7, 6, 10, 6, 8, 4, 1, 11, 7
Male 3	−, −, 16, 2, 12, −, −, −, 4, 2, 4, 0, 4, 0
Pair 4	11, 7, 11, 7, 5, 7, 1, 7, 4, 11, 5, 6, 2, 1, 0
Male 4	−, 12, 6, 4, 0, 2, 2, 0
Pair 5	−, −, 12, −, 15, 12, 5, 9, 11, 9, 8, 12, 11, 12
Female 5	12, 10, 8, 4, 0, 0, 0, 0
Pair 6	15, 12, 10, 8, 12, 11, 11, 9
Male 6	−, 16, 6, 2, 2, 6, 2, 2
Pair 7	−, −, 12, 11, 8, 11, 7, 14, 12, 8
Female 7	10, −, −, 6, 2, 6, 0, 6, 0, 0
Pair 8	12, 12, 12, 12, 12, 12, 12, 12
Male 8	8, 6, 0, 4, 10, 4, 6, 4

* Escaped. † Only the female responded.

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