age to the middle or inner ears. A tungsten electrode is advanced through the skull to a point near the cochlear aqueduct [O. W. Henson and G. D. Pollak, *Physiol. Behav.* **8**, 1185 (1972)].

12. This model deals only with the spatial information contained in the difference areas. As such, it does not provide a mechanism for localization along the vertical midline, where IID's at all frequencies will be 0 at all elevations. Such neural mechanisms have been presented [*Soc. Neurosci. Abstr.* **9**, 213 (1983)].
13. The concept of fixed spatial reference points is most plausible if the external ears are immobile. Pinna mobility in the mustache bat is limited. The very tips of the pinnae can be moved laterally, but there is very little rotation of the entire external ear. Pinna movement could have two influences on the proposed reference points: (i) the points could maintain their relative positions with respect to one another, but encode a different region of space relative to the bat's head, or (ii) their relative positions could be altered as a result of a change in pinna configuration. Whether the mustache bat moves its ears at all during flight is not known.
14. Experiments were conducted in a soundproof chamber. Hoop rotation and electrode advance were accomplished from outside the chamber. Single-unit activity was recorded with 3*M* KCl glass microelectrodes. Binaural response properties were quantified by comparing the number of impulses generated by sounds delivered to either ear alone with sounds presented to both ears. In dichotic tests, the intensity at one ear was fixed at 10 dB above threshold, while intensity at the other ear was varied in 10-dB increments. Spike counts were obtained with an on-line computer. Because of the high degree of directionality and rapid attenuation of ultrasonic frequencies, cross talk between the two ears was limited to 35 dB, as measured by cochlear microphonic threshold responses.
15. Spatial selectivity of neurons was determined by measuring threshold at each speaker position. Threshold criterion was a response to each of ten consecutive stimulus presentations.
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17. Primarily for simplicity, we have emphasized the spectral cues derived from the CF harmonics of the echolocation pulse. Also, the frequency dependence of ear directionality is most pronounced when compared at the CF harmonics. The frequency-modulated harmonics also contribute spectral cues likely to further enhance spatial resolution. Most bats emit pulses dominated by a frequency-modulated sweep, and Grinnell and Grinnell (*8*) have proposed that they obtain similar binaural spectral cues from these signals.
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Bimodal Distribution of Dopamine Receptor Densities in Brains of Schizophrenics

Abstract. *The dopamine hypothesis of schizophrenia was examined by measuring the density of dopamine receptors in the postmortem brains of 81 control subjects and 59 schizophrenics from four different countries. The densities of dopamine receptors in the tissues from the schizophrenic patients had a bimodal distribution in the caudate nucleus, putamen, and nucleus accumbens. One mode occurred 25 percent above the control density, and a second mode occurred at a density 2.3 times that of the control density for all three regions. Although almost all the patients had been medicated with neuroleptics, the two modes had the same dissociation constant for the labeled ligand used, suggesting that the neuroleptic doses were similar for the two populations of schizophrenics. The results thus provide direct evidence for two distinct categories of schizophrenia.*

The most consistent neurochemical finding relating to schizophrenia has been an increased density of dopamine receptors in postmortem brain tissue from schizophrenic patients (*1*). Although this finding has been generally confirmed (*2*), a few early reports were unable to reproduce it (*3*); subsequent research has revealed elevated densities of dopamine receptors in the brain tissues of schizophrenics treated with neuroleptics (*4*). These elevations are selective for type D_2 dopamine receptors (labeled by tritiated neuroleptics), no changes being detected in the D_1 or D_3 dopaminergic sites or other neurotransmitter receptors (*5*). Although neuroleptic treatment seems to elevate receptor density (*4*), brain tissues from neuroleptic-free schizophrenics can occasionally show markedly elevated densities (*1*). Thus, to obtain further data on the relative effects of illness and neuroleptic medication on these changes, we studied a new series of tissues under improved experimental conditions. We now report a bimodal distribution in the elevated densities of striatal dopamine receptors of neuroleptic-treated patients.

The dopamine receptors were measured by modifications of previous methods (*6*), with a final concentration of less than 1 mg of original tissue per final milliliter of incubate. Table 1 summarizes the results. We analyzed tissue from 71 controls for the caudate nucleus, 56 for the putamen, and 47 for the nucleus accumbens. Some control subjects had received neuroleptics before death. For example, three of the Toronto subjects with Alzheimer's disease had received neuroleptics (40 mg of trifluperazine per day for over a year; 2 mg of haloperidol per day for 2 years; the third individual, an unknown amount of neuroleptics). One Los Angeles subject (control, diagnosis of drug abuse) had received perphenazine (16 mg per day) for at least 2 months. The densities of D_2 dopamine receptors for these subjects, however, were within normal limits. Almost all of the schizophrenic patients had been treated with neuroleptics (Table 1). Although 15 schizophrenic patients had not received neuroleptics in the last month, we considered only those patients who had been off neuroleptics for at least 6 months as being truly drug-free. When such information was uncertain, we assumed that the patient had taken neuroleptics.

Figure 1 indicates the bimodal pattern of the dopamine receptor densities of schizophrenics. The two caudate modes were about 13 and 127 percent higher than the control mode. Table 2 summarizes the data. The distribution of the control values for the caudate nucleus was not statistically significantly different from a normal distribution [$\chi^2(6) = 4.13, P = 0.5$]. For schizophrenics, however, caudate values differed significantly from a single normal distribution [$\chi^2(6) = 11.6, P = 0.05$].

The distribution of densities in the control putamens did not statistically differ significantly from a normal distribution [$\chi^2(6) = 5.46, P = 0.5$]. The putamens from schizophrenics had modes that were 39 and 154 percent above that of the control mode (Fig. 1), a distribution that was statistically different from a single normal distribution [$\chi^2(7) = 14.86, P < 0.05$].

The control values for the nucleus accumbens were not statistically significantly different from a normal distribution [$\chi^2(6) = 2.8, P = 0.8$]. The distribution of values for the schizophrenic accumbens tissues exhibited modes that exceeded the control mode by 25 and 102 percent and thus differed significantly from a single normal distribution [$\chi^2(8) = 13.41, P = 0.05$].

The lower modes for the tissues from schizophrenics were thus elevated from 13 to 39 percent (mean, 26 percent) above the control values. This elevation is within the range seen in rat striatum after long-term administration of neuroleptics, namely between 10 and 50 percent (*7*). It is possible, therefore, that this low-density mode represents a population of D_2 receptors that is normal but that has been elevated by the long-term administration of neuroleptics during the patient's illness.

The higher modes of 23 to 25 pmol/g (in the schizophrenic patients) represent about a 2.3-fold increase in receptor density relative to the control value.

The two modes of brain dopamine receptor densities reported here in schizophrenic patients are consistent with, but not necessarily synonymous with, the two-syndrome concept of

neuroleptics or that neuroleptics had been withdrawn months (m) or years (y) before death; ?, uncertainty whether the patient had received neuroleptics. The dissociation constants (K_d) for the control tissues were 85 ± 16 p*M* (means ± standard error of the mean) for caudate, 83 ± 16 p*M* for putamen, and 118 ± 21 p*M* for the nucleus accumbens. The K_d values for the schizophrenia tissues were 116 ± 47 p*M* for the caudate, 121 ± 41 p*M* for the putamen, and 142 ± 29 p*M* for the nucleus accumbens. Means are shown in boldface type.

Vienna			Cambridge				Los Angeles				Toronto			
Age (years)	Receptor density (pmol/g)		Age (years)	Receptor density (pmol/g)			Age (years)	Receptor density (pmol/g)			Age (years)	Receptor density (pmol/g)		
	Caudate	Putamen		Caudate	Putamen	Accumbens		Caudate	Putamen	Accumbens		Caudate	Putamen	Accumbens
							Control tissues							
80	13.7	15.0	73	13.3		11.6	60	17.3		13.4	42	8.0	8.3	
76	11.5		48	15.7	14.6		66	15.4	14.9	12.8	45	6.5		
75	14.0	17.3	84			11.8	24	15.4	14.6	11.6	51	9.0	9.6	
69	12.1		84	10.2	9.8	12.4	62	14.1	14.4	14.9	39	6.4	9.3	
86	9.9		40	11.8	13.4	12.7	41	14.2	14.6	15.6	58	5.6	7.2	
72	16.1	21.2	86	10.0	9.6	9.1	37+2m	13.3	15.2	13.0		10.9	10.7	
62	13.6	20.0	65	13.3	11.8		72	12.7		7.6	70	10.8	10.4	9.5
80	16.8	16.7	72	7.8		8.2	21	13.0	12.5	12.0	52	7.4	9.6	8.9
63	6.6	13	77	8.5		10.7	43	12.8	15.6	12.5	73	9.3	11.0	10.7
79	15.4	7.0	81	9.6		10.7	17	11.5	14.4	12.3	54	8.5	7.8	6.4
65	16.7	12.0	79	10.3	13.6	10.1	57	11.0	10.6	11.3	73	11.9	14.5	12.2
71		16.6	69	9.4	9.3	11.1	56	11.4		6.9	56	7.2	11.4	
84		19.2	71	16.2		11.1	66	11.0	8.2	9.6	77+1y	10.0	12.1	
86		10.0	44	11.9			40	10.7	13.5	9.3	73+2y	8.3	11.9	10
91		16.0	72	11.0	16.7		29	7.0	6	8.5	70+2y	9.1	13.6	10.9
			83	9.0			52	10.5	10	12.4	66+1y	13.3	14	9.5
			79	13.9	14.5		47	10.2	14.2	11.6	76	11.5	12	11.3
			88	14.7			27	8.8	8.6	4.5	67	12.5		
			?	7.9			25	14.5		9.9	70	10.2		
			?	11.1			56	13.0	9	11.1		15.6		16.7
			72		14.5	8.8	23	16.5		10.2				
			68			11.6								
			78			14.2								
			35		18.9	14.5								
			104		10.9	11.9								
76	**13.3**	**15.3**	**72**	**11.3**	**13.1**	**10.9**	**44**	**12.6**	**12.3**	**11.0**	**62**	**9.6**	**10.8**	**10.6**
							Tissues from schizophrenics							
56+	12.5		53+	23.4	32.0	23.1	25+	31.0	27.7	24.6	22+	34.7	43.5	29.0
67+	15.4	20.8	80?	6.7	9.7	11.6	64+	27.7	35.2	22.2	78+	27.3	32.0	16.2
77+	10.6		27+	10.4			25+	27.6	40.2	34.7	56−	15.5	16.0	12.5
79+	13.0	14.1	77−2m	23.9		13.8	51+	26.1	26.4	18.5	70+	14.2	14.1	12.3
57+	16.8		58+	11.3		22.3	21	25.1	24.6	17.4	22−2m	25.1	23.4	23.5
62−	18.9	15.6	86−1m	11.0	10.3	15.2	20	25.0	25.2	19.6	25+	23.9	29.7	26
72−9m	8.0	13.3	56+	13.9	18.4	14.8	30+	24.2	28.0	23.1				
45+	24.0	24.3	70+	28.8	31.4	22.3	38+	22.0	20.5	22.6				
77+	23.7	25.2	67+	17.2	21.7	22.3	36+?	16.4	20.3	20.8				
78−5m	20.2	33	63?	30.0	33.5	27.8	41−?	13.5	14.5					
62+	33	27.6	56+	26.8	36.1	21.9	82+	12.9	15.8	8.8				
75−	21.5	35	44+	11.0	14.0		46+?	11.7	16.7	10.1				
73+	22.1	22.4	88−	10.2	5.7	5.8	36−		13.5					
71+		16.7	82+	10.0	13.0	13.7								
80+		23.6	68+	12.8		16.6								
60−		18.0	54+	15.2	12.5	13.6								
70−		11.3	84?	12.0										
71+		32.2	84−10y	7.9	6.9									
			65?	16.5										
			74?	20.1	26.5									
			66?	14.7	15.3									
			23+		31.0	22.5								
68	**18.4**	**22.2**	**65**	**15.9**	**20.0**	**17.8**	**40**	**21.9**	**23.7**	**20.2**	**46**	**23.4**	**23.4**	**20.0**

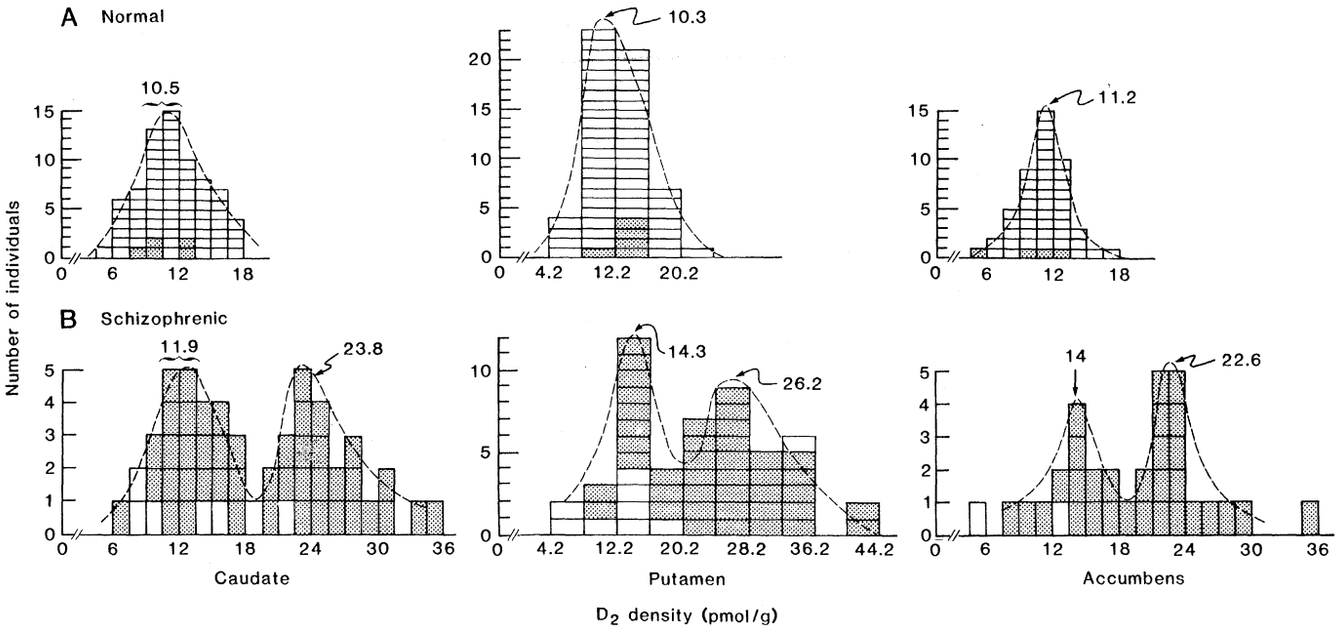

Fig. 1. Distribution of dopamine receptor densities in caudate nucleus, putamen, and nucleus accumbens tissues from control and schizophrenic patients. Each small rectangle indicates one sample of brain tissue. The stippled rectangles indicate patients that had been treated with neuroleptics; the white rectangles indicate that the subjects either never took neuroleptics or had not been taking them for at least 6 months before death.

Table 2. Mean dopamine receptor densities from control tissues and tissues from schizophrenics. Standard deviations are given in parentheses.

Group	Caudate		Putamen		Accumbens	
	Density (pmol/g)	*n*	Density (pmol/g)	*n*	Density (pmol/g)	*n*
Control	11.3 (3.1)	71	12.7 (3.4)	56	11.0 (2.34)	47
Schizophrenic	18.8 (7.3)	52	21.8 (9.85)	50	19 (6.4)	32

schizophrenia suggested by Crow (*8*). The high-density mode might represent Crow's type 1 syndrome of hallucinations and delusions associated with acute schizophrenia. The situation cannot be so simple, however, since acute schizophrenia tends to be diagnosed in patients younger than 40 years, whereas the patient ages within the high-density group ranged from 20 to 77 years. The low-density subgroup might be related to those schizophrenic patients exhibiting ventricular enlargement (*8, 9*).

The clinical records and summaries available to us in this study were not suficiently detailed for us to try to relate the low- or high-density groups to the presence or absence of tardive dyskinesia.

It is conceivable that the long-term neuroleptic treatment could have resulted in the observed bimodal distribution of receptor densities. Although the neuroleptic doses and durations were difficult to obtain and document, there is no reason to suppose that the low- and high-density subgroups of patients had received different neuroleptic doses. The residual presence of neuroleptic (administered before death) is reflected in an elevated value for the K_d of [^{3}H]spiperone, but the density (B_{max}) was unaffected. For example, the presence of 10 n*M* haloperidol as a final concentration in the incubate caused the K_d of [^{3}H]spiperone to change from 76 to 580 p*M*, whereas the density remained constant at 12.5 pmol/g (human putamen, not shown). The K_d values for the lower and higher density modes in the tissues from schizophrenic patients, however, were not significantly different, suggesting that the two modes were not a result of radically different doses of neuroleptics. For example, the K_d values of the two modes for the accumbens tissues from schizophrenics were 136 ± 25 p*M* (n = 4) and 143 ± 18 p*M* (n = 8) (mean ± standard error of the mean), calculated after the omission of a singularly high value of 676 p*M* for one patient. Likewise, the K_d values for the two modes of the schizophrenic putamens were 116 ± 13 p*M* (n = 12) and 124 ± 9 p*M* (n = 10), calculated after omitting two extremely high K_d values of 460 and 249 p*M*.

It remains to be established experimentally, in rats, whether long-term neuroleptics would result in a bimodal distribution of elevated dopamine receptor densities. And if the high-density subgroup represents a genetically different population, one might expect to observe a bimodal distribution of schizophrenic dopamine receptor densities in other tissues (*10*).

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Siblicidal Aggression and Resource Monopolization in Birds

Abstract. *In Texas, great egret* Casmerodius albus *chicks attack younger nestmates, often fatally (siblicide). By contrast, the young of neighboring great blue herons* Ardea herodias *seldom strike or kill siblings. These interspecific differences seem related to prey size: only fish provided by egret parents are small enough for chicks to monopolize (a process facilitated by aggression). Experimentally cross-fostered heron chicks raised on small prey by egret parents became siblicidal, but the reverse procedure of cross-fostering egret chicks did not reduce aggression or siblicide.*

Siblicide, fatal aggression among siblings (*1, 2*), is a taxonomically widespread phenomenon that can occur prenatally (*3*) or postnatally (*4*). Because it involves the killing of close kin, inclusive fitness theory predicts that siblicide would evolve only when the principal's own survival is seriously jeopardized. Although such risk could be expected when critical resources are currently or prospectively inadequate for all broodmates to survive (*5*), little is known about the way in which such resources affect sibling aggression. I have proposed that the degree to which small food (*6*) can be monopolized profitably is an important ecological determinant of sibling aggression and, therefore, of siblicide (*7*). I now partially confirm this prey size hypothesis by a cross-fostering experiment involving two species of colonial Ardeidae (Aves, Ciconiiformes).

I studied great egrets (*Casmerodius albus*) and great blue herons (*Ardea herodias*) on three islands in Lavaca Bay, Texas (30°39′N, 96°34′W) during the summers of 1979, 1980, 1981, and 1982. Both species are piscivorous and monogamous, with males and females sharing all aspects of parental care. Incubation typically begins after the first egg is laid, so the 1- to 2-day interval between the laying of successive eggs produces comparable hatching intervals. Both species frequently experience brood reduction, but the demise of the youngest chick is effected differently between species. Whereas siblicide is the apparent cause of death in many, perhaps most, egret brood reductions, it was significantly rarer in heron nests (*8*).

During the first month, aggression in egret broods is on the average 18 times higher than aggression in heron broods, most of which do not fight at all (Table 1). Egret nestmates quickly form stable, age-dependent dominance hierarchies that confer distinct feeding advantages to

Table 1. Fighting rates of broods (three and four chicks) of great egrets and great blue herons in Texas. All data are from the first 25 days after the completion of hatching and are expressed as means ± 1 standard deviation.

Brood size	Natural broods		Foster broods*	
	Broods (No.)	Fighting rates†	Broods (No.)	Fighting rates†
Great egrets				
Three chicks	9	1.46 ± 1.32	9	1.09 ± 1.11
Four chicks	2	1.42 ± 0.16	1	0.69
Pooled	11	1.45 ± 1.22	10	1.05 ± 1.05
Great blue herons				
Three chicks	4	0.08 ± 0.17	7	1.51 ± 0.75
Four chicks	9	0.08 ± 0.14	2	0.38 ± 0.29
Pooled	13	0.08 ± 0.14	9	1.26 ± 0.83

*Egret chicks raised by heron adults and heron chicks raised by egret adults (see text). †Fighting rates were standardized across brood sizes by $F_t/N_d/N_r$, where F_t is the total fights, N_d is the number of possible dyads, and N_r is the number of days recorded. The number of possible dyads varies with brood size: three-chick broods have three possible dyads, and four-chick broods have six. Fighting rates between species show significant differences [$P < 0.001$, analysis of variance (ANOVA) with Scheffé test]. Rates between brood sizes within each species were not significantly different (*17*).