



Fig. 1 Percentage of positive responses of individual homing pigeons to infrasound stimuli (40 presentations each). *a*, Chance 'positive' response levels of 22 birds when no stimulus was provided; this is the background level against which responses in other test situations should be evaluated. *b*, Responses of eight pigeons to sounds of 0.8 Hz. *c*, Responses of 14 pigeons to sounds of 1.67 Hz. *d*, Responses of five pigeons to sounds of 1.67 Hz when the tubing between the sound generator and the test chamber was closed by pinch clamps. *e*, Responses to sounds of 1.67 Hz by five pigeons whose cochleas and lagenas had been removed. *f*, Responses to sounds of 1.67 Hz by five pigeons whose columellas had been bisected. All sound amplitudes were 113.8 dB rms SPL.

(compare Fig. 1*b* and *c* with 1*a*). The response could be eliminated if two pinch clamps were placed on the tubing between the sound generator and the test chamber (Fig. 1*d*).

From preliminary experiments, we estimate the threshold (50% response) to be approximately 80 dB at 1.5 Hz, 60 dB at 5 Hz, and 50 dB at 10 Hz. These compare with published values for human thresholds of 132 dB at 1.5 Hz, 115 dB at 5 Hz and 104 dB at 10 Hz (ref. 5). The ambient noise levels inside the insulated chamber were 70 dB at 1.5 Hz (band width (bw) 10% of centre frequency), 50 dB at 5 Hz (bw 1%) and 47 dB at 10 Hz (bw 1%); inasmuch as the 50% threshold values were only slightly above these noise levels, it seems possible that the pigeons would be even more sensitive to signals of these frequencies if tested in a quieter chamber. (A more detailed study of threshold response values, down to 0.1 Hz, is in progress.)

To determine the location of the receptors responsible for infrasound detection, we tested five pigeons from which the cochleas (and lagenas) had been surgically removed, and also five pigeons with intact cochleas but with both columellas bisected so that there was no direct conduction of sound between the tympanum and the oval window. Figures 1*e* and *f* show that neither of these groups of birds respond to an infrasound stimulus of 1.67 Hz at 113.8 dB; the response levels for both groups were indistinguishable from those of normal birds in the absence of a stimulus. These results suggest that the receptors for infrasound are in the inner ear, but it is not clear which organ is responsible because removal of the cochleas and lagenas unavoidably damages the remaining portions of the labyrinth, one part of which has recently been shown to function (at least in frogs) as an auditory receptor⁸.

Unlike sounds easily audible to humans, atmospheric infrasounds can travel huge distances—often thousands of miles—without much attenuation (which is proportional to the square of the frequency). Hence such sounds could potentially provide birds with much useful information. For example, the ability of birds to sense approaching weather fronts might be aided by detection of infrasounds funnelled to earth by those fronts or by detection of the infrasound components of thunder associated with storms that are still hundreds of miles away. Localisation of several remote infrasound sources could serve as a distant landmark system for triangulation of the home site and of

the location of a release site. Properly utilised, infrasound information could thus assist in almost every aspect of avian navigation, in both homing and migration.

Whether one or more of these potential uses is actually realised is not known. There are several reasons why birds might have difficulty extracting meaningful information from infrasounds. For example, they would need to distinguish between spatially coherent infrasounds and so-called pseudosounds, which are very local pressure oscillations generated by winds and atmospheric turbulence. Further, the usual binaural comparison methods of localising sounds would be ineffective where the wavelengths are so very long (approximately 300 m for a sound of 1 Hz). Neither of these problems should be insurmountable for a bird in flight. Thus a flying bird might be able to reconstruct the spatial patterns of atmospheric infrasounds and thereby separate them from local pseudosounds. Quine and Konishi⁹ have shown that, in the usual audible range, owls can distinguish frequencies that differ by as little as 0.5–0.7%; if such discrimination holds for pigeons in the infrasound range, then doppler shifts in frequency, which for a pigeon flying at 20 m s⁻¹ toward or away from the sound source would be as high as 14%, could provide ample information for localisation. Only further research can reveal whether or not this is mere speculation.

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Genetic influences and infantile autism

In his original description of infantile autism, Kanner suggested an "inborn defect", because symptoms were often present from early infancy. Despite the rarity of a family history of autism and lack of a known increase in parental consanguinity, there are two reasons for suspecting hereditary influences: the 2% rate of autism in siblings is 50 times that of the general population¹, and a family history of speech delay is found in about a quarter of families². Reports of single pairs of twins with autism have not added much to our knowledge of genetic effects because of a bias toward reporting monozygotic (MZ) concordant pairs and because few reports contain both adequate clinical descriptions and evidence of zygosity¹. We therefore undertook a study of a systematically collected sample of 21 pairs of same-sexed twins, one or both of whom had autism as diagnosed by the criteria of Kanner³ and Rutter⁴. The

results reported here indicate the importance of hereditary influences in the aetiology of autism.

The names of twins were collected from consultants, schools and units for autistic children, the National Society for Autistic Children and two hospital twin registers. Apart from two pairs in which dermatoglyphics were used, zygosity was determined by blood grouping in all cases not markedly different in physical appearance. Case notes of the 33 pairs thus obtained were reviewed and eight pairs were rejected on diagnostic grounds. The remaining 25 pairs and their families were extensively interviewed, tested and examined at home or in hospital. Obstetric records and case notes were also studied. On the basis of all information, a further four pairs were excluded as not meeting diagnostic criteria.

The adequacy of our sampling is shown by the MZ:DZ ratio (11:10) which was about that expected for same-sexed twin pairs surviving the first year (6:7); and by the number of autistic twin pairs found. In the English twin population of school age who would meet our diagnostic criteria, between 19 and 27 autistic children would be expected and 20 were found. Our sampling of children over school age was less complete, but not likely to be biased as to zygosity or concordance since most cases came from twin registers.

The diagnoses were made from case summaries, numbered randomly to prevent sorting by pair, and from which name, exact age, and zygosity had been deleted.

At least one member of 21 same-sexed pairs of twins met the diagnostic criteria for autism. In all, the 11 MZ and 10 DZ pairs gave rise to 25 autistic children. Apart from being twins, these were similar to other reported series of autistic children with respect to sex ratio (3.4:1), social class (57% from I and II), and IQ (about 1/2 severely retarded but 1/4 with normal non-verbal IQ). Affective disorder was described in four families, but schizophrenia did not occur. One sibling suffered from autism (a rate of 2.8%). Delayed speech was reported in three families (Table 1).

Four of the 11 MZ pairs but none of the 10 DZ pairs were concordant for autism ($P=0.055$). All concordant pairs were male. Because autism is a rare condition (about 4 per 10,000 children), the MZ concordance rate is equivalent to a very high correlation in liability (over 0.9) using the multifactorial model^{9,10}.

If, using this model, it is hypothesised that autism reflects some form of continuously distributed, abnormal characteristic, the question arises whether the non-autistic co-twins showed any other abnormality. We found that very few did in the DZ pairs but in most MZ pairs the co-twins showed

Table 1 Pair-wise concordance by zygosity

	MZ pairs (n = 11)	DZ pairs (n = 10)	MZ-DZ difference (exact test)
Concordance for autism	36%	0%	$P = 0.055$
Concordance for cognitive disorder (including autism)	82%	10%	$P = 0.0015$

some form of cognitive disorder (see Tables 1 and 2).

Thus, in addition to the autistic twins, 6 non-autistic co-twins (5 MZ and 1 DZ) showed a cognitive abnormality as defined by the criteria of no phrase speech until 3 yr (three cases), IQ 70 or below (two cases), severely abnormal articulation after 5 yr (two cases), or scholastic difficulties requiring special schooling (two cases). Four non-autistic twins showed only one of the above features and none were severely handicapped. All the autistic twins met at least two of the criteria. Thus, 9 (82%) of the 11 MZ pairs were concordant for cognitive disorder/autism compared with only 1 (10%) of the DZ pairs ($P=0.0015$). The findings

Table 2 Summary of findings for MZ pairs

	First-born twin	Second-born twin
1	Autism + severe MR	Autism + severe MR
2	Autism + mild MR	Autism
3	Autism + severe MR	Autism + severe MR
4	Autism	Autism + mild MR
5	Mild MR	Autism + severe MR
6	Autism	Educational difficulties (ESN school)
7	Severe articulation defect	Autism
8	Autism + severe MR	Language disorder
9	Mild MR	Autism + severe MR
10	Autism + mild MR	Normal
11	Normal	Autism

MR, Mental retardation.
ESN, Educationally subnormal.

strengthen the suggestion of genetic determination and indicate that what is inherited is a form of cognitive abnormality which includes but is not restricted to autism.

Twins have a raised rate of perinatal difficulties leading to brain injury and it was necessary to check that the concordance was not an artefact of these. We found that it was not. In none of the four pairs concordant for autism did both twins have a history of brain injury. Furthermore, no twin with cognitive disorder in the absence of autism showed evidence of perinatal brain injury (Table 3).

Brain injury was, however, relevant to discordance

Table 3 Summary of biological hazards in discordant pairs

Hazard	MZ pairs		Non-autistic twin
	Autistic twin		
Definite	Multiple congenital anomalies		—
Possible	Neonatal convulsions		—
	Severe febrile illness		—
None	Pathologically narrow cord		—
	—		—
		DZ pairs	
Definite	Apnoea		—
	Delay second birth		—
	Delay second birth		—
	Delay second birth		—
Possible or Difference in severity	Severe haemolytic disease + apnoea		Delay second birth
	Severe haemolytic disease + apnoea		Mild haemolytic disease
None	Birth weight 1 and ¼ pounds lower		—
	Birth weight 1 and ¼ pounds lower		—
	—		—

regarding autism. We identified five features known to be associated with brain injury: kernicterus, perinatal apnoea of more than 6 min, neonatal convulsions, multiple congenital anomalies, and a second birth delayed by at least 30 min. In 6 of the 17 pairs discordant for autism, the autistic child but not the non-autistic co-twin had experienced one or more of these biological hazards in the perinatal period.

A wider definition of biological hazard was then applied to the 11 cases not differentiated on the stricter criteria. Using the wider definition (lower birth weight by 1 pound or more, pathological narrow umbilical cord, a more severe kernicterus with apnoea and a severe febrile illness—possibly encephalitis—just before onset), a further six discordant pairs were differentiated. Thus, in 12 of 17 pairs discordant for autism, the autistic member probably or possibly suffered a brain injury, whereas in none of the discordant pairs did this occur only in the non-autistic member.

To summarise the results: the markedly higher rate of concordance in MZ, compared with DZ, pairs for both autism and cognitive abnormalities indicates the importance of genetic factors in the aetiology of autism. Indeed, the concordance rates in conjunction with the very low prevalence of autism in the general population point to a strong genetic determination. Nevertheless, the finding of an association between biological hazards in the perinatal period and autism also demonstrates the important role of environmental influences for the causation of autism (but not cognitive disorder).

In some cases, genetic factors seem to be both necessary and sufficient causes. Among the eight autistic children in the four concordant MZ pairs, biological hazards (even using the broader criteria) occurred in only one child. He was more severely affected than his twin. On the other hand, brain injury alone seems to be a sufficient cause in some cases. In one of the two completely discordant MZ pairs, the autistic twin had experienced neonatal convulsions, whereas the non-injured co-twin was normal in every way. The importance of brain damage is also indicated by the finding in other studies of autism in children with congenital rubella or infantile spasms.

Finally, some cases seem to result from the combination of an inherited cognitive defect plus brain damage. In three of the five MZ pairs discordant for autism but concordant for cognitive deficit, the autistic child had experienced a biological hazard whereas the cognitively impaired non-autistic co-twin had not.

Determining the mode of inheritance is difficult since autistic children rarely reproduce. It would be helpful to know what happens to the offspring of the non-autistic twins with cognitive impairment, but no such information is available.

In summary, we conclude that this systematic study of 21 same-sexed twin pairs in which at least one twin showed the syndrome of infantile autism, indicates the importance of a genetic factor in the aetiology of autism. It also indicates the importance of brain injuries, especially during the perinatal period, which may operate either by themselves or in combination with a genetic predisposition involving language. Both the mode of inheritance and exactly what is inherited remain uncertain.

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Anti-fouling role of antibiotics produced by marine algae and bryozoans

A GRADIENT of antibacterial activity along fronds of the large brown seaweed *Laminaria*¹ runs opposite to gradients of density of bryozoans and spirorbid tube worms settled on the fronds^{2,3}. Bryozoan colonies attached to a *Laminaria* frond grow towards the younger region⁴, where antibiotic production is lowest. It has been suggested that tannins produced by branch tips of *Sargassum* have an anti-fouling role⁵, but we have seen no other clear evidence of such an effect, although many marine plants and animals remain remarkably free from encrustation by other organisms⁶. We have now found further evidence in several examples of differential settlement by spirorbid worms on algae and by a bryozoan on another species of bryozoan.

We had noticed that holdfast branches of *Laminaria digitata* (Huds.) Lamour on Pembrokeshire and Plymouth shores bear populations of *Spirorbis inornatus* L'Hardy and Quiévreux, always attached to the sheltered inner sides. The same spirorbid settles abundantly on *Himantalia elongata* (L.) S. F. Gray, but only on the undersides of the button-like expansions which protect the attachment points of the alga. In laboratory experiments, which followed the usual procedures⁷ and will be reported in detail elsewhere, we found that settling *Spirorbis* larvae invariably select those parts of the two algae, irrespective of the contours of the surfaces and of their orientation to light and gravity. We tested these two sites for differential production of antibiotics.

Disks 5 mm in diameter cut from the surfaces were tested by standard procedures⁸ against *Staphylococcus aureus*, strain NC1B8244. This grew readily or formed indistinct clouded zones around surfaces normally shaded and colonised abundantly by epibionts (Fig. 1a and c), but left

Fig. 1 Growth (a, c and e) and inhibition (b, d and f) of *Staphylococcus aureus* round pieces cut from inner surface of *Laminaria* holdfast branch (a); outer surface of latter (b); lower surface of *Himantalia* button (c); upper surface of latter (d); growing edge of *Flustra foliacea* colony (e); old part latter (f). The scale lines represent 5 mm.

