Medical Memoranda

Incest, Inbreeding, and Mental Abilities

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Two recent communications have intimated the extent of the disadvantages from which children of incestuous unions may suffer. Adams and Neel (1967) reported that of 18 offspring from unions between first-degree relatives only seven were normal, one died at 6 hours and one at 15 hours, one died at 2 months from glycogen storage disease, one had a bilateral cleft lip, and two were severely mentally retarded with seizure disorder and spastic cerebral palsy, and five mildly so (I.Q.s 50 to 70 on repeated testing); thus seven were of subnormal mental standard. Carter (1967) reported the conditions seen in 13 children of similar unions; only five were normal, three failed to reach the age of 9 years (dying at 13 months from cystic fibrosis, at 21 months from progressive cerebral degeneration with blindness, and at 8 years from a congenital heart defect respectively), and one is severely mentally retarded and four mildly so (I.Q.s 50 to 76); thus five are of subnormal mental standard, making a total in the two series of 12 out of 31.

Since incest as defined in Western society is merely the extreme and socially designated form of consanguineous union, mating between related individuals, evidence of association between less close degrees of parental consanguinity and mental abilities of offspring is of relevance to the interpretation of these findings. The results of a preliminary analysis of consanguinity and mental ability in a small closed population are presented here.

CONSANGUINITY

The most extreme form of consanguineous union that can occur in man, and which gives rise to the most highly inbred offspring, is mating between two first-degree relatives (father/daughter, brother/sister, son/mother). A child is said to be inbred if he is born of a mating between two individuals who can both trace descent from a common ancestor, so that both the genes that the child possesses at a given locus may be replicates of a single gene that was present in the common ancestor and so may be identical.

The probability that both the genes an individual possesses at a given autosomal locus are identical is measured in terms of Wright's inbreeding coefficient (F), which therefore also indicates the proportion of autosomal loci at which the individual is expected to be an identical homozygote. By this measure inbreeding can be quantified. Offspring of incestuous unions between brother and sister or between father and daughter have an inbreeding coefficient of 0.25; offspring of a marriage between two first cousins have an inbreeding coefficient of 0.0625; both of these figures will be increased if any common ancestor was himself inbred. Mating of two completely unrelated individuals produces offspring with an inbreeding coefficient of zero. It is this likelihood of increased homozygosity in inbred individuals that is responsible for the increased frequency of manifestation expected in them of simple recessive characters, and for the increased frequency of those extreme conditions which represent the tails of the distribution curves of continuously varying quantitative attributes brought about by polygenic action.

ANALYSIS OF RECORDS FROM TRISTAN DA CUNHA

From the extensive records that are available for the population of Tristan da Cunha, based largely on those collected by

Dr. L. Woolley when resident on the island in 1942, supplemented by those obtained by Dr. P. Munch during an expedition to the island in 1938 and more recently by Dr. J. Loudon, it is possible to trace the descent of every individual from the founders of the population, and so calculate his inbreeding coefficient. This has been done for every present and past member of the Tristan population (Roberts, 1967). Dr. Woolley at the same period also recorded conditions of clinical interest, among which was his assessment of the mental capabilities of each member of the then population. These he classified, by clinical appraisal over long periods, into six broad categories from normal to low-grade mental defective. The association of these assessments with the inbreeding coefficients has been examined.

Table I sets out the mean inbreeding coefficient found in each intelligence category. There seems to be a tendency for the mean inbreeding coefficient to be higher in the lower intelligence categories (column 2). That this is almost but not entirely due to there being a greater proportion of non-inbred individuals in the higher categories (column 5) is shown by comparison with the mean inbreeding coefficient calculated for the inbred alone in each category (column 6). From Table II, setting out

TABLE I.—Inbreeding and Intelligence

Intelligence Category	Mean F	Total No. of Individuals	Individuals Not Inbred		Mean F in those Inbred
			No.	%	
1. Normal ..	0.0188	35	17	48.6	0.0366
2. Slow cerebration ..	0.0391	53	11	20.8	0.0505
3. Very slow cerebration ..	0.0379	60	15	25.7	0.0517
4. Definitely inferior mental capacity ..	0.0480	45	6	13.3	0.0554
5. High-grade mental defective ..	0.0457	8	1	12.5	0.0522
6. Low-grade mental defective ..	(0.0313)	1	0	0	(0.0313)

TABLE II.—Number of Individuals by Mental Category and Inbreeding Coefficient

F	Intelligence Category						Total No. of Individuals
	1	2	3	4	5	6	
0	17	11	16	6	1	—	51
<0.03	9	18	13	12	2	—	54
<0.06	5	10	12	5	2	1	35
<0.10	3	11	16	19	2	—	51
>0.10	1	3	3	3	1	—	11

the number of individuals by intelligence category and inbreeding coefficient, the association can be seen to be highly significant at the 1% level ($\chi^2=22.85$, 9 degrees of freedom).

COMMENT

Full allowance has to be made for the subjective nature of the classification of mental ability in interpreting these preliminary findings, which are being followed up by Dr. Keir, using the results of objective tests of intelligence. This association could mean that some of the worse mental conditions in this population are due to inbreeding so far; but the alternative explanation is also reasonable—that those of poor mental capacity tend to breed more closely if opportunity offers. Of these two explanations the second is supported in Adams and Neel's (1967) series by some of their father/daughter, but not by their 12 brother/sister, incestuous unions. In all except one of the latter the matings were between sibs of normal intelligence (I.Q.s 90 to 120, with mean values 110 in males and 103 in females), and the occurrence of mental retardation in four of

their offspring suggests an inbreeding effect. There is further supporting evidence from Japan, where Schull and Neel (1965) examined seven various indicators of school attainment—for example, performance in language or science—in relation to inbreeding, taking into account age, school grade, and socioeconomic status. They observed a consistent and significant depression in performance in all seven indicators with inbreeding; by comparison with the outbred mean there was a reduction of 1% in performance per increase of 1% in F. It seems that, like physical dimensions, performance in neuromuscular tests, and other quantitative characters, mental capabilities may well be particularly susceptible to the effects of inbreeding.

The view was long since expressed that intelligence is under polygenic control, and there is much evidence to support it. On the other hand, Morton's (1960) analysis of Penrose's data suggested to him the influence of major autosomal recessive genes in an appreciable proportion of cases of severe mental defect. The present findings, which relate to less severe conditions, also suggest that some of the genes tending to lower

mental capabilities are recessive. It seems more reasonable, however, to interpret the results as indicating not control by major recessive genes but polygenic control with directional dominance, a suggestion which carries wide implications. But since the highest category here employed was "normal" and exceptionally able individuals were not distinguished, the data do not establish it. For this purpose information on the relative numbers of offspring of consanguineous unions who are above as well as below average in ability is required.

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Tongue Pain with Giant Cell Arteritis

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Local effort pain of the tongue is described as a feature of giant cell arteritis in this case report.

CASE REPORT

A previously fit 78-year-old man woke with severe neck pain which radiated over his head and was attributed to cervical spondylosis. Two weeks later he began to experience increasing malaise and pain in the tongue. This was felt diffusely at the back of his tongue and was described as a tired ache; on one occasion it followed a prolonged period of talking, but otherwise occurred only on eating, and had prevented him from taking solid food for four weeks when he was referred to hospital on 8 March 1967. It was found that eating solid food was limited after two minutes by onset of the pain, which then after a further half-minute gradually faded over the next five minutes. The pain could also be reproduced by rapid repeated protrusion of the tongue, but not by drinking or by repeated jaw movements alone.

The tongue and the temporal and occipital arteries appeared clinically normal and there were no bruits over the carotid arteries. Neck movements were limited by cervical spondylosis. There were no other abnormal findings on physical examination.

Biopsy of the right temporal artery was performed. The histological features were those of giant cell arteritis, with thrombosis, and recanalization of the lumen. Haemoglobin was 14.8 g./100 ml., white blood cell count 13,000/cu. mm. with a normal differential count, and erythrocyte sedimentation rate 34 mm. in one hour (Wintrobe).

Corticosteroid therapy was started. One week later the malaise and tongue pain had resolved.

COMMENT

Giant cell arteritis is a widespread vascular disorder (Meadows, 1966). Pain in the jaw muscles on chewing has been regarded as a typical feature of the disease (Robertson, 1952; Meadows, 1954; Ross Russell, 1959), and Meadows (1966) has noted that pain may radiate to the tongue.

Missen (1961) found histological evidence of lingual arteritis in all of five patients whose tongues had appeared normal. However,

clinical evidence of tongue ischaemia is uncommon. Gangrene of the anterior part of the tongue has been reported in five cases (Howard and Cremin, 1959; Wise, 1959; Brearley and Macdonald, 1961; Pitt *et al.*, 1961; McGill, 1961; Davis and Davis, 1966). Swelling of the tongue (Wolinetz *et al.*, 1948; Cornil *et al.*, 1949), a sensation that the tongue was too big (Meadows, 1954), and constant pain or burning of the tongue (Bowers, 1940; Post, 1949; Olivo and Spizzo, 1951) have also been noted, but local effort pain of the tongue does not appear to have been previously described.

Early recognition and treatment of giant cell arteritis is a matter of urgency. The present case has therefore been reported to show that angina of the tongue may be a presenting symptom of the disease, histological confirmation of which can be obtained from biopsy of a clinically normal temporal artery (Alestig and Barr, 1963).

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