Progeria in twins

Sir,

Viegas, Souza, and Salzano (1974) reported identical twins with progeria, a most important observation. However, they implicate autosomal recessive inheritance for progeria, despite the fact that the great majority of patients with progeria represent a sporadic occurrence in an otherwise normal family (DeBusk, 1972). Our recent data (Jones *et al*, 1975) showed older paternal age, a known factor associated with fresh gene mutation for autosomal dominant disorders, in fathers of progeria individuals. The mean paternal age for 18 such fathers was 34.1 years. The father of the Viegas *et al* patient was 38 years at the birth of these affected identical twins. Though genetic heterogeneity may exist for the phenotype of progeria, the present data favour the concept that most cases are the result of a single fresh mutant gene aetiology.

Yours, etc,

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Chromosome abnormalities in Southwest American Indian patients

Sir,

As authors such as Thorburn (1971) and Hungerford (1963) have shown, increasing interest has been shown in the occurrence and incidence of chromosomal aberrations in different populations and racial groups. However, there are few data from many populations. For example, Cornu *et al*'s (1968) reported cases of trisomy syndromes have dealt mainly with children of Caucasian ancestry, though frequency of Down's syndrome has also been estimated for Negroes and Orientals by Wagner (1962) and Lejeune (1964). The need for further genetic evaluation of varied populations is indicated, since the distribution of chromosomal abnormalities such as trisomy syndromes appears to differ with respect to geography (space) and time, as Day (1966) has shown. The purpose of this report is to present additional data of this nature obtained from American Indian patients from New Mexico and Arizona. These groups are highly endogamous as a result of inbreeding according to Hewitt and Stewart (1970), and consequently form population isolates of special interest. We were unable to find any reports describing the occurrence of any type of chromosomal aberration among similar Indian groups, and, in particular, with reference to Down's syndrome.

Chromosome analysis was performed on an Indian group of 42 patients by a modified method of Moorhead *et al* (1960), and compared against a control group of 270 Caucasian patients. Both groups represent patients referred to the Cytogenetics Laboratory at the University of New Mexico School of Medicine over a period of 5

TABLE

COMPARISON OF CHROMOSOMAL ABNORMALITIES IN AMERICAN INDIAN AND CAUCASIAN PATIENT GROUPS

Patient or Karyotype Classification	Caucasian Totals		Indian Totals		No. of Cases				
	No. of Cases	% Total	No. of Cases	% Total	Navajo	Pueblo	Zuni	Apache	Tribe Unknown
Normal	156	58	26	62	10	10	1	1	4
Total chromosomal abnormalities	114	42	16	38	8	3	1	1	3
Trisomy G	56	21	8	20	3	2	1		2
Trisomy D	2	0.9	1	2.5	1				
Trisomy E	3	1.4	1	2.5	1				
Klinefelter's (XXY)	12	5.4	1	2.5					1
'Superfemale' (XXX)	—	—	1	2.5	1				
Turner's (XO)	6	2.7	1	2.5	1				
Testicular feminization (phenotypic female, XY)	—	—	1	2.5	1				
Marker chromosome	5	2.3	1	2.5				1	
Philadelphia chromosome	15	6.8	1	2.5		1			