Table 2. METEOROLOGICAL OBSERVATIONS AT NEW DELHI (1951)

	Jan.	Feb.	Mar.	Apr.	May	June	July	Aug.	Sept.	Oct.	Nov.	Dec.
Relative humidity, per cent, 0830h.	74	52	53	42	35	41	63	74	63	50	50	69
Relative humidity, per cent, 1730h.	46	24	33	21	21	26	48	60	52	37	42	47
Rainfall (in.)	0.9	0.0	2.1	0.9	0.1	0.5	4.8	3.1	4.1	0.3	0.6	Nil

Small specimens, 4 in. $\times$ 2 in., of steel ($\frac{1}{8}$ in. thick) and zinc (1/20 in. thick) were exposed in the open air near the pillar for one year and their losses in weight were then determined after the corrosion products had been removed. Their corrosion-rates found in this way are compared in Table 1 with the results of similar tests made at other stations, both at home and overseas.

It is clear that the corrosive conditions at Delhi are very mild. Indeed, the zinc specimens were so little corroded that they retained much of their original polish after a year's exposure there. In my view, therefore, the lack of serious rusting of the pillar is to be attributed to the mildness of the local climate rather than to any intrinsic superiority in the corrosion resistance of the iron itself.

This conclusion is supported by a study of the meteorological data for Delhi, for which I am indebted to the Indian Meteorological Office. The figures for 1951, which fell within the experimental period, are given in Table 2.

It is known from the classical researches of W. H. J. Vernon that the relative humidity of the air is the primary controlling factor for the atmospheric corrosion of iron and that little or no rusting occurs unless the humidity exceeds 70 per cent. The records show that this critical value would only be reached at Delhi for a short time during the whole year. Presumably, too, the sulphur pollution of the atmosphere, which, as Vernon demonstrated, controls the corrosion rate when the humidity reaches the critical value for rusting, is but slight in the neighbourhood of the iron pillar.

The late Sir Robert Hadfield, who was very interested in the Delhi pillar and published an analysis of its iron, seems to have been of a similar opinion, for in one of his papers he prefaces his remarks about this and other specimens of historical interest by observing that "in dry air the action of corrosion may be very slight".

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High R^z Frequency in the Blood of Australian Aborigines

IN blood genetic surveys conducted prior to 1948 on Australian aborigines it was shown¹ that these natives possessed the gene R^z , and that its distribution in various Australian States appeared to be patchy. In all, the gene was demonstrated seven times (3 per cent) in 234 aborigines. It was found in natives of Queensland and the Northern Territory, but not in fifty-nine natives of South Australia.

One of us (J. B. B.) is at present engaged on an extensive field survey of aborigines in Western Australia and is also collecting blood samples for testing in Melbourne.

To date, the results in relation to the gene R^z are as follow:

Location	Number tested	R^1R^z	R^2R^z	R^zR^z
Cundeelee Mission	22	2	1	0
Wiluna	33	2	1	1
Cosmo Newberry	59	2	—	0
Mulga Queen	16	0	—	0
Mount Margaret	45	1	—	0
Jigalong Mission	47	7	—	1
Marble Bar	108	1	—	0
Pilgangoora	65	3	—	1
	395	18	2	3

Only seventeen samples of type Rh_1Rh_2 from Cundeelee Mission and Wiluna in a total of 134 found have been tested with anti- hr'' (anti- e) serum owing to shortage of this testing reagent. Other examples of R^2R^z probably remain undisclosed in the series. The purpose of this letter is to report finding the genotype R^zR^z in Western Australia, three examples of which occurred in 395 unmixed aborigines tested in the present survey, and the high R^z frequency averaging 6 per cent, but approaching 20 per cent in certain tribes. One previous example of R^zR^z has been reported² in the blood of an aboriginal in Queensland.

Tests have been made to date covering the $A-B-O$, $M-N-S$, Rh , P , Le^a , Fy^a , K , Lu^a and He blood groups. It has, however, been necessary to limit severely the number of tests performed with some anti-sera owing to world shortage. It can be said as a progress report that in Western Australian aborigines the percentage of $P+$ is about 25, $Fy(a+)$ is almost 100, $Le(a+)$ is about 3.5, while S of $M-N$, K , Lu^a and He antigens are absent, or rare. One example of the Rho variant presumably homozygous (CD^{ue}/CD^{ue}) has been detected in the series; the sample showed weak agglutination with some highly potent anti- Rho sera, and failed to agglutinate with others. As judged by strengths of agglutination with specially selected testing sera, it is suspected that an occasional variant occurs at the other Rh loci also. Tests made to date on 125 random adult blood samples have failed to show any evidence of the sickle-cell trait in this population.

The full blood-grouping results will be reported after the field survey concludes about the end of 1953.

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¹ Simmons, R. T., and Graydon, J. J., *Med. J. Australia*, 2, 113 (1948).
² Sanger, R., Walsh, R. J., and Kay, M. P., *Amer. J. Phys. Anthropol.*, N.S., 9, 71 (1951).