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The genetic structure of a tribal population, the Yanomama Indians

II. Eleven blood-group systems and the ABH-Le secretor traits*

BY H. GERSHOWITZ,† M. LAYRISSE,‡ Z. LAYRISSE,‡ J. V. NEEL,‡ N. CHAGNON,† AND M. AYRES§

This paper will describe the findings with respect to 13 antigen systems of erythrocytes and saliva in 2516 Yanomama Indians living in villages located in Brazil and Venezuela. Since a principal focus of this investigation is the origin and extent of the genetic microdifferentiation encountered in a relatively undisturbed tribe, the results will be presented by village.

The Indians reported on here were drawn from a total of 46 villages, the exact location of which is shown in Ward (1972). However, there were several instances in which the number of persons sampled from a village was obviously too small to reflect gene frequencies adequately. Arbitrarily, the findings in all villages where the number sampled was less than 30 have been pooled into a 'miscellaneous' sample totalling 100 individuals. The villages contributing to that miscellaneous sample are 03 V, Y, Z, 08 G, H, K, M, P, Z. Results will be presented, then, in terms of 37 separate villages plus one 'miscellaneous' sample; the villages contributing to the miscellaneous sample are well distributed throughout the Yanomama range.

A preliminary note has been published on the gene (but not phenotype) frequencies of the first 567 individuals (10 villages) typed (Arends *et al.* 1967). The individuals reported therein have been included in this report for the sake of completeness of the presentation. Minor differences between the village numbers given in the earlier publication and in this paper result from the sampling of additional individuals in the meantime or, in the case of one village (03 J), the elimination from the sample of a group of visitors (see Arends *et al.* 1967) whose home village was later sampled.

METHODOLOGY

Specimens were collected into 15 c.c. Becton-Dickinson vacutainers containing 2·25 ml. ACD-A solution and air-lifted to a base laboratory – either the Instituto Venezuelano de Investigaciones Cientificas near Caracas, Venezuela, or the Departmento de Genetica, Universidade Federal do Para, Belem, Brazil. All bloods were kept cool from the time of collection to the time of arrival at the base laboratories some 24–72 hr later. The specimens were then subdivided and an aliquot from each was sent to the University of Michigan. Most of the blood typings for the polymorphic systems were performed in duplicate using identical antisera. Any discrepancies in the results of the two typings were reconciled by repeat typings in both laboratories on red cells preserved in glycerol at $-60°$ C. or in liquid nitrogen. The blood typing reagents with which tests were performed in duplicate included anti-A, B (group O), M, M^v, N, S, s, P_1, C, c, D, E, e, K, Fy^a, Fy^b, Jk^a, Di^a, Le^a and Le^b. Tests with the following reagents were done only in Ann Arbor: Anti-C^w, f, Mg, U, (Mu + Ht), Vw, (P + P_1), k, Kp^a, Kp^b, Js^a, Lu^a, Lu^b and Wr^a. In

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† Department of Human Genetics, University of Michigan, Ann Arbor.
‡ Instituto Venezolano de Investigaciones Cientificas, Caracas.
§ Department de Genetica, Universidade Federal do Para, Belem.

addition, testing of salivas for ABH and Le substances was likewise done only in Ann Arbor. All tests were performed in tubes with 2 % washed red cell suspensions. A more precise description of the techniques employed will be found in Gershowitz *et al.* (1967).

Gene frequencies have been calculated by maximum-likelihood procedures (MAXLIK) developed in the department of Human Genetics for the IBM 1130 (Reed & Schull, 1968). These procedures are derived from methods which assume random sampling from a large panmictic population in Hardy–Weinberg equilibrium, assumptions not valid in this type of study. (As will become apparent (Table 1), in most instances, more than half of the inhabitants of each (small) village under study have been sampled.) The error introduced into the calculations by failure to meet the assumptions of the method is unknown but probably small. The MAXLIK program includes a test for departure of the observed phenotype frequencies from those calculated on the basis of the gene frequencies derived assuming Hardy–Weinberg equilibrium. It has become abundantly apparent, however (Neel *et al.* 1964; Ward and Sing, 1970), that agreement with Hardy–Weinberg equilibrium is consistent in samples of moderate size with relatively high levels of inbreeding or assortative mating.

In a previous study (Gershowitz *et al.* 1970) we pointed out the discrepancies which may appear when several different methods of calculating Rh gene frequencies are compared, and demonstrated that the MAXLIK program modified to assume that only four Rh genes (R^z, R^1, R^2, R^0) were present gave results which agreed closely with those obtained by actual gene counting. However, since an examination of a large population is more likely to reveal the presence of rare genes, a total population analysis was carried out with the eight-gene MAXLIK program. Even though, then, an occasional individual village was reported by the eight-gene MAXLIK program to possess certain rare genes (notably r^y or r), *final* analyses of the villages were performed by restricting the program to those genes identified as being present in the total population study. The results reported herein for the Rh system are therefore derived by a MAXLIK program based on the assumption that only the four reported genes are present.

RESULTS

Phenotype frequencies for the traits for which variation was encountered are given in Table 1, and gene frequencies rounded off to two places because of the size of the samples in Table 2. Differences from system to system in the numbers tested in certain villages are due to various causes. For instance, an occasional blood specimen was received in somewhat poor condition, such that apparently proper typings were secured with saline agglutination reagents, but the blood would haemolyse when subjected to a Coombs test and could not be scored with reagents utilizing that test. Discrepancies in the duplicate typings were always retested whenever possible, but infrequently one of the two labs could not recover a particular specimen, or repeat typings did not, in fact, resolve the discrepancy. Such bloods were not scored for the reagent (or system) involved. Lastly, over the course of the several years duration of the study, the same village may have been visited more than once and different groups of individuals from that village subjected to slightly different testing procedures or to a different selection of typing sera. This last explanation is most obvious in the case of village 03 J.

For a variety of reasons, not all the blood specimens were always tested for all of the antisera which appeared to be yielding uniform reactions. All bloods were, however, tested and found negative with anti-A, B, anti-Mg, anti-K and anti-Wr^a. All P_2 bloods were positive with

Table 1 *Blood group phenotypes of Yanomama Indians by village*

System	Phenotype	Series 03																	Series 08																	Series 11				Misc.	Total
		A	B	C	D	E	F	G	H	I	J	KP	LMN	Q	RS	T	U	W	X	ABC	D	E	F	I	J	L	N	O	Q	R	S	T	UVW	XY	ABC	D	G	HI		
MNSs	MS	—	—	—	—	1	2	—	—	—	5	8	6	9	4	5	—	7	1	—	—	1	—	—	3	7	—	—	—	2	—	1	—	9	7	3	—	—	5	86
	MSs	4	2	—	6	1	9	2	8	4	21	12	13	8	16	11	3	22	9	3	4	10	3	2	5	19	4	4	1	9	8	4	18	21	20	3	3	6	11	309
	Ms	28	12	25	41	13	7	15	21	22	24	11	2	1	13	12	3	16	36	38	5	8	5	11	5	15	33	9	20	16	8	6	30	13	9	2	21	78	29	663
	MNS	—	—	—	—	—	—	1	1	1	—	—	—	—	1	—	4	—	—	—	—	—	1	—	—	—	1	—	—	5	—	—	—	—	—	—	—	—	1	16
	MNSs	9	3	7	4	5	3	2	9	4	4	19	17	8	9	7	8	7	3	10	1	1	1	7	5	12	3	2	4	6	5	—	26	30	28	9	6	18	6	308
	MNs	28	16	17	22	31	9	15	12	15	6	14	13	4	18	16	17	11	22	91	11	16	17	28	9	13	6	12	16	12	22	12	60	22	45	13	8	18	40	757
	NS	1	—	—	1	—	—	—	—	1	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	1	—	—	—	—	1	—	5
	NSs	3	—	5	2	1	—	3	3	2	2	—	—	—	1	—	3	—	—	9	—	—	—	—	1	2	1	—	—	—	—	—	5	—	—	—	—	4	1	48
	Ns	6	10	5	—	8	4	10	—	1	—	9	14	5	7	2	6	2	4	26	10	2	4	14	4	4	2	8	7	5	21	8	21	25	42	20	—	1	7	324
		79	43	59	76	60	34	48	54	50	62	73	65	35	69	53	44	65	75	177	31	38	31	62	32	72	50	35	48	55	64	31	161	120	151	50	38	126	100	2516
P	P_1	68	36	46	43	45	15	38	33	39	58	70	61	32	65	46	41	54	54	152	28	24	24	37	25	66	39	30	47	52	55	23	141	104	119	42	28	88	91	2059
	P_2	11	7	13	33	14	18	10	21	11	4	2	1	1	3	7	2	10	21	25	3	14	7	25	7	6	11	5	1	4	9	8	20	16	32	8	10	38	9	447
		79	43	59	76	59	33	48	54	50	62	72	62	33	68	53	43	64	75	177	31	38	31	62	32	72	50	35	48	56	64	31	161	120	151	50	38	126	100	2506
Rh	CDe	48	33	35	51	32	12	28	32	36	39	52	35	17	45	29	40	32	24	96	31	32	27	55	26	55	37	25	25	25	38	14	137	102	148	50	21	72	79	1715
	CcDe	—	—	—	—	2	4	1	—	1	—	—	8	3	—	1	—	—	14	—	—	—	—	1	—	—	2	—	—	—	2	1	—	10	—	—	—	—	2	52
	cDe	—	—	—	—	—	2	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	2
	CDEe	15	4	16	23	14	11	14	20	12	9	3	1	—	—	4	—	4	10	54	—	—	—	3	5	13	10	9	11	13	22	14	22	—	—	—	—	9	39	1	385
	CDE	—	—	2	1	—	2	—	2	1	—	—	—	—	—	—	—	—	1	4	—	—	—	—	—	—	1	1	4	4	2	1	—	—	—	—	—	—	5	—	31
	CcDEe	15	4	5	1	11	3*	5	—	—	13	16	19	15	23	16	4	19	17	17	—	6	4	2	1	4	—	—	5	10	—	1	1	7	3	—	5	9	18	279
	CcDE	1	2	—	—	1	—	—	—	—	—	—	—	—	—	—	—	—	1	6	—	—	—	—	—	—	—	—	3	3	—	—	1	—	—	—	1	1	—	20
	cDE	—	—	1	—	—	—	—	—	—	1	2	1	—	1	3	—	10	5	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	2	—	—	26
	cDEe	—	—	—	—	—	—	—	—	—	—	—	1	—	—	—	—	—	3	—	—	—	—	—	—	—	—	—	—	1	—	—	—	1	—	—	—	—	—	6
		79	43	59	76	60	34	48	54	50	62	73	65	35	69	53	44	65	75	177	31	38	31	61†	32	72	50	35	48	56	64	31	161	120	151	50	38	126	100	2516
Fy	a+b−	34	20	35	23	26	17	17	19	15	2	18	20	6	13	24	17	30	11	75	8	9	4	17	10	8	22	19	12	13	22	14	—	—	—	—	—	—	27	607
	a+b+	30	18	21	42	27	16	19	27	31	5	41	25	19	39	23	20	28	38	77	14	20	14	28	13	37	25	14	17	33	30	14	—	—	—	—	—	—	37	842
	a−b+	14	5	3	11	6	1	12	8	4	4	14	20	10	17	6	7	7	26	23	8	8	13	17	9	26	3	2	19	9	11	3	—	—	—	—	—	—	14	340
		78	43	59	76	59	34	48	54	50	11	73	65	35	69	53	44	65	75	175	30	37	31	62	32	71	50	35	48	55	63	31	—	—	—	—	—	—	78	1789
	a+	—	—	—	—	1	—	—	—	—	—	—	—	—	—	—	—	—	—	2	1	1	—	—	—	—	—	—	—	—	1	—	137	94	123	44	38	121	20	583
	a−	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	24	26	28	6	—	5	2	91
		—	—	—	—	60	—	—	—	—	—	—	—	—	—	—	—	—	—	177	31	38	—	—	—	—	—	—	—	—	64	—	161	120	151	50	38	126	22	674
Jk	a+	53	27	48	74	38	20	30	52	31	58	58	49	31	47	44	41	46	58	138	28	37	29	58	25	60	35	30	38	47	55	26	131	95	109	45	31	86	68	1976
	a−	25	16	11	2	21	14	18	2	19	4	14	16	4	22	9	3	19	17	38	3	1	2	4	7	11	15	5	10	9	9	5	30	25	42	5	7	40	32	536
		78	43	59	76	59	34	48	54	50	62	72	65	35	69	53	44	65	75	176	31	38	31	62	32	71	50	35	48	56	64	31	161	120	151	50	38	126	100	2512
Le	a−b+	70	40	37	59	49	24	36	39	38	49	44	49	26	54	45	27	50	51	147	28	32	21	41	26	48	39	25	35	36	31	26	129	92	124	39	24	63	70	1863
	a−b−	9	3	22	17	10	9	12	15	12	13	28	16	9	14	8	17	15	24	30	3	6	10	21	6	24	11	10	13	19	33	5	32	28	27	11	14	63	30	647
		79	43	59	76	59	33	48	54	50	62	72	65	35	68	53	44	65	75	177	31	38	31	62	32	72	50	35	48	55	64	31	161	120	151	50	38	126	100	2510
ABH Sec.	Sec.	69	37	47	71	—	—	13	48	—	—	71	51	32	55	49	19	—	—	137	30	36	31	56	29	69	48	22	36	49	—	—	143	92	143	51	38	117	22	1711
	Non-sec.	1	—	—	—	—	—	—	1	—	—	—	—	—	—	—	—	—	—	2	—	—	—	—	—	—	—	—	1	—	—	—	9	—	—	1	—	—	—	15
		70	37	47	71	—	—	13	49	—	—	71	51	32	55	49	19	—	—	139	30	36	31	56	29	69	48	22	37	49	—	—	152	92	143	52	38	117	22	1726
Le Sec.	Sec.	63	35	30	57	—	—	7	38	—	—	44	37	24	44	41	13	—	—	114	27	32	21	39	23	48	38	18	26	31	—	—	129	73	119	41	25	74	20	1331
	Non-sec.	7	2	17	14	—	—	6	11	—	—	27	14	8	11	8	6	—	—	25	3	4	10	17	6	21	10	4	11	18	—	—	23	19	24	11	13	43	2	395
		70	37	47	71	—	—	13	49	—	—	71	51	32	55	49	19	—	—	139	30	36	31	56	29	69	48	22	37	49	—	—	152	92	143	52	38	117	22	1726
K	k	78	43	59	76	60	34	47‡	54	50	28	73	65	35	69	53	44	65	75	176	31	38	31	62	32	72	50	34	48	56	63	31	—	—	—	—	—	—	79	1812
Di	a−	78	43	59	76	60	34	48	54	50	62	73	65	35	69	53	44	65	63§	177	31	38	31	62	32	72	50	35	48	56	64	31	161	120	151	50	38	126	100	2504
Lu	a−b+	79	43	59	76	60	34	48	54	50	10	73	65	35	69	53	44	65	75	176	31	38	31	62	32	72	50	34	48	56	63	31	—	—	—	—	—	—	79	1794
Approximate village size		84	52	66	85	85	50	55	77	55	125	103	89	43	76	58	55	83	108	215	43	66	35	75	40	89	59	65	55	75	72	45	225	156	208	63	72	163	—	3170

* One of the three was positive with anti-f.
† The phenotype of one additional blood is uncertain. See text.
‡ One blood in addition to those itemized was positive with anti-Kp^a.
§ The other twelve bloods from this village were Di(a+).

Table 2. *Blood group gene frequencies, by village, for polymorphic systems only*

		Series 03																	Series 08																Series 11					
MNSs	Gene	A	B	C	D	E	F	G	H	I	J	KP	LMN	Q	RS	T	U	W	X	ABC	D	E	F	I	J	L	N	O	Q	R	S	T	UVW	XY	ABC	D	G	HI	Misc.	Total
	S	0·04	0·06	0·00	0·04	0·05	0·24	0·04	0·09	0·06	0·27	0·32	0·32	0·49	0·23	0·26	0·13	0·33	0·09	0·01	0·08	0·17	0·08	0·07	0·24	0·30	0·05	0·08	0·02	0·19	0·10	0·10	0·11	0·29	0·21	0·18	0·05	0·03	0·15	0·147
	s	0·60	0·49	0·63	0·75	0·50	0·47	0·50	0·65	0·66	0·62	0·33	0·23	0·20	0·44	0·48	0·33	0·50	0·69	0·50	0·40	0·55	0·49	0·42	0·38	0·44	0·79	0·49	0·63	0·51	0·36	0·45	0·46	0·29	0·27	0·20	0·77	0·78	0·54	0·489
	NS	0·08	0·00	0·10	0·05	0·02	0·00	0·06	0·11	0·08	0·03	0·00	0·00	0·00	0·02	0·00	0·12	0·00	0·00	0·05	0·00	0·00	0·02	0·00	0·03	0·03	0·05	0·00	0·03	0·07	0·00	0·00	0·05	0·00	0·00	0·00	0·07	0·09	0·04	0·028
	Ns	0·28	0·45	0·27	0·16	0·43	0·29	0·40	0·15	0·20	0·08	0·35	0·45	0·31	0·30	0·36	0·42	0·17	0·22	0·44	0·52	0·28	0·41	0·51	0·35	0·23	0·11	0·43	0·32	0·23	0·54	0·45	0·38	0·42	0·52	0·62	0·11	0·10	0·29	0·337
P	P^1	0·63	0·60	0·53	0·34	0·51	0·26	0·54	0·38	0·53	0·75	0·83	0·87	0·83	0·79	0·64	0·78	0·60	0·47	0·62	0·69	0·39	0·52	0·36	0·53	0·71	0·53	0·62	0·86	0·73	0·62	0·49	0·65	0·63	0·54	0·60	0·49	0·45	0·70	0·578
Rh*	R^z	0·10	0·07	0·17	0·16	0·13	0·24	0·15	0·22	0·14	0·07	0·02	0·01	0·00	0·00	0·01	0·00	0·03	0·10	0·09	0·00	0·00	0·00	0·02	0·08	0·09	0·12	0·16	0·23	0·22	0·20	0·26	0·07	0·00	0·00	0·00	0·13	0·20	0·01	0·094
	R^1	0·80	0·86	0·77	0·83	0·75	0·60	0·79	0·78	0·85	0·81	0·84	0·75	0·74	0·82	0·74	0·95	0·67	0·58	0·74	1·00	0·92	0·94	0·95	0·91	0·88	0·86	0·84	0·69	0·65	0·78	0·71	0·92	0·92	0·99	1·00	0·74	0·76	0·89	0·823
	R^2	0·10	0·07	0·06	0·01	0·10	0·03	0·05	0·00	0·00	0·12	0·14	0·17	0·22	0·18	0·21	0·05	0·30	0·20	0·07	0·00	0·08	0·06	0·02	0·01	0·03	0·00	0·00	0·08	0·12	0·00	0·02	0·01	0·03	0·01	0·00	0·13	0·04	0·09	0·070
	R^0	0·00	0·00	0·00	0·00	0·02	0·13	0·01	0·00	0·01	0·00	0·00	0·07	0·04	0·00	0·01	0·00	0·00	0·12	0·00	0·00	0·00	0·00	0·01	0·00	0·00	0·02	0·00	0·00	0·01	0·02	0·01	0·00	0·05	0·00	0·00	0·00	0·00	0·01	0·014
Duffy	Fy^a	0·63	0·67	0·77	0·58	0·67	0·74	0·55	0·60	0·61	0·41	0·53	0·50	0·44	0·47	0·67	0·61	0·68	0·40	0·65	0·50	0·51	0·35	0·50	0·52	0·37	0·69	0·74	0·43	0·54	0·59	0·68	0·61§	0·53§	0·57§	0·65§	1·00§	0·80§	0·58	0·575
Kidd	Jk^a	0·43	0·39	0·57	0·84	0·40	0·36	0·39	0·81	0·38	0·75	0·56	0·50	0·66	0·44	0·59	0·74	0·48	0·52	0·54	0·69	0·84	0·75	0·75	0·53	0·61	0·45	0·62	0·54	0·60	0·62	0·60	0·57	0·54	0·47	0·68	0·57	0·44	0·43	0·538
Le Sec.	Le†	0·66	0·74	0·39	0·53	0·59	0·48	0·50	0·47	0·51	0·55	0·38	0·50	0·49	0·54	0·61	0·38	0·52	0·43	0·59	0·69	0·60	0·43	0·42	0·57	0·42	0·53	0·47	0·48	0·41	0·28	0·60	0·55	0·52	0·58	0·53	0·39	0·29	0·45	0·492
Le Sec.	Le‡	0·68	0·77	0·40	0·56	—	—	0·32	0·53	—	—	0·38	0·48	0·50	0·55	0·60	0·44	—	—	0·58	0·68	0·67	0·43	0·45	0·55	0·45	0·54	0·57	0·45	0·39	—	—	0·61	0·55	0·59	0·54	0·42	0·39	0·70	0·522

* R^z = CDE, R^1 = CDe, R^2 = cDE, R^0 = cDe.

† Gene frequencies derived from red cell phenotypes, i.e. $Le = 1 - \sqrt{\frac{Le(a-b-)}{N}}$.

‡ Gene frequencies derived from secretory phenotypes, $Le = 1 - \sqrt{\frac{le\ le}{N}}$.

§ Fy^a calculated from tests with anti-Fy^a only.

anti-(P + P_1). One hundred and fifty-one R_1R_2 bloods were tested with anti-f; three were found to be positive (see Table 1). The other reagents yielding constant and invariant results, not listed in Table 1, and the village populations tested with them were:

		Village
Anti-C^w (C + bloods only)	All negative	03 A–08 T
Anti-U	All positive	03 A–08 T
Anti-K	All positive	03 A–08 T
Anti-Kp^a	All negative but one	03 A–08 T
Anti-Kp^b	All positive	03 A–08 T
Anti-Vw	All negative	03 A–08 T
Anti-Js^a	All negative	03 K–11 HI and
Anti-(Mu + Ht)	All negative	03 K–08 N

The sequences of villages are defined by the first row of Table 1.

Two sets of gene frequencies are presented for *Le* in Table 2. Each is based on a different test procedure, one employing red-cell phenotypes and one saliva phenotypes. Since individuals with the red-cell phenotype Le(a – b –) are presumed to lack the *Le* gene, both the red cell typings and the saliva typings yield information as to the frequency of *Le*. The differences in the two sets of *Le* gene frequencies obtained by the two tests are small; differences of the order of 0·05–0·10 are usually associated with villages wherein the number of persons whose saliva was typed was only a small proportion of the number whose bloods were typed; these differences probably reflect sampling error. In village HI, however, 63 out of 126 specimens of red cells were classified as Le(a – b –) whereas 43 out of 117 saliva specimens were found to lack the Le antigens; the difference yields a χ^2 of 4·3. It is difficult to see how the red-cell phenotypes might be incorrect; not a single discrepancy was noted in the Lewis typings between the two laboratories. Eleven individuals were, however, typed as H and Le secretors and as Le(a – b –); the difference between the two methods deriving the *Le* frequency disappears if these eleven individuals are assumed to be Le(a – b +).

Due to limitations in the amount of anti-Xg^a available, only village 08 ABC was tested for the frequency of Xg^a antigen. Among the 99 males tested, 59 were Xg(a +), and among the 65 females tested, 41 were positive. The Xg^a frequency based on males alone is 0·60, but on females, 0·39. Conventional Hardy–Weinberg analysis yields a χ^2 of 21·2 when the Xg^a frequency derived from males is used to calculate the expectation in females, and a χ^2 of 17·7 if the Xg^a gene frequency derived from the female data is used to calculate expectation in males. Although interpretation of these data is difficult, the existence of polymorphism in the Xg system in this population can be accepted.

Table 1 calls attention to the fact that one blood in village 03 G was found to react with anti-Kp^a. This particular blood was tested twice while still fresh, and retested 7 months later, after storage in liquid nitrogen, with the same positive results. The amount of blood available was insufficient for an absorption to be performed. The parents of this 35-year-old female were deceased. Her husband and two children were negative. No other blood reacting with anti-Kp^a has been found among almost 2500 Yanomama, although since the members of this population are interrelated in so many different ways (Chagnon *et al.* 1970), one might have expected to have found other instances of the antigen. The origin of the Kp^a antigen in this individual may well have been mutational although clear documentation is obviously unavailable.

One other exceptional blood is referred to in Table 1. The blood of a 5-year-old male was found not to react, in *both* laboratories, with the anti-C, c, E and e sera used in the routine screening

program. It did react with anti-D, but unlike the usual 'Rh deletions' (Race & Sanger, 1968) it did not react in saline with incomplete anti-D. In fact, reactions with a variety of anti-D antisera were weaker than a normal R_0 control. In further tests, 11 anti-C sera, 12 anti-c, 10 anti-e and 24 anti-E sera failed to react with this blood. Three anti-C sera reacted weakly. The parents of this child could not be identified but his grandparents (from only one lineage) and some aunts and uncles and a sib were all R_1R_1 with no evidence of any abnormality in their typings. Frozen erythrocytes were not available and no further information could be obtained.

Table 1 shows that of the 1726 salivas tested, 15 were thought to be from H non-secretors. With the exception of the Xavante (Neel *et al.* 1964), South American Indians have been found uniformly to be H secretors (Salzano, 1964), and thus these 15 salivas are critical in determining whether the *se* gene truly exists in this population, in contra-distinction to most other South American Indian tribes. However, among the 15, 12 are simultaneously Le non-secretors and only 3 are Le secretors. A heterogeneity χ^2 test comparing this distribution of Le secretors with that found among all other salivas tested (all of which were H secretors) yields, with 1 D.F., the highly significant χ^2 of 27·97. It is also pertinent to point out that the red-cell phenotype of each of the three H-non-secretor, Le-secretors was Le(a − b +). This suggests that the salivas had lost H substance through enzymic degradation (i.e. improper heat inactivation). It is presumed that all the salivas of the 12 H, Le − non-secretors have lost H activity; some may have also lost Le activity since their red-cell phenotypes are also Le(a − b +). We must conclude that the Yanomama, too, are uniformly H secretors. Other occasional discrepancies between red cell and secretor phenotypes similar to those previously reported among Xavante Indians (Gershowitz *et al.* 1967) will be presented and discussed in a subsequent paper.

DISCUSSION

The present findings confirm and extend the earlier observations concerning genetic microdifferentiation in the Yanomama Indians. They also confirm the findings in the Makiritare Indians (Gershowitz *et al.* 1970). To cite only a few examples from the present experience, in villages in which 50 or more individuals were typed (i.e. a minimum of 100 genes), *MS* frequencies vary from 0·0 to 0·33, *NS* from 0·11 to 0·62, P_1 from 0·36 to 0·87, R_2 from 0·0 to 0·22, Fy^a from 0·40 to 0·77, and so on. Fig. 1 summarizes the range in gene frequencies for each system. As in the past, we refrain from testing the statistical significance of these differences by the usual techniques, inasmuch as the 'sample' not only includes related persons, but also comprises such a high proportion of the total inhabitants of a village as to render this almost an enumerative procedure. The primary factor in the origin of these large differences has been felt to be the nature of the fission process whereby new villages originate from established villages (Arends *et al.* 1967), with the differential (increased) fertility of headmen now also seen as playing a role (Chagnon *et al.* 1970).

Taken at face value, the differences in gene frequencies between Yanomama villages are as great as the differences between tribes of American Indians, as summarized by Salzano (1957), Layrisse & Wilbert (1966) and Post, Neel & Schull (1968). Thus, for example, the frequency of *Ns* in the American Indian ranges from 0 to 0·5 while the Yanomama inter-village range is 0·1 to 0·6. The intertribal ranges and Yanomama inter-village ranges are the same for R_2 (0–0·2), Jk^a (0·4 to 0·8) and Fy^a (0·2–1·0), although in the case of Fy^a one tribe is listed as having a frequency of zero. Some of these intertribal differences, such as the wide range in I^A and Di^a

frequencies, are clearly valid. On the other hand, in the light of the present experience, one is forced to question to what extent these differences are only apparent, depending on the region in the tribal distribution from which the sample was drawn. An extreme example of the non-representedness of a single village is given in the case of village 03 X. This differs from all the other Yanomama villages listed in the presence of the Di^a antigen. The absence of the Di^a antigen in the Yanomama was suggested by the first studies of this tribe (Layrisse, Layrisse & Wilbert, 1962). The origin of this one exceptional situation has been described by Chagnon *et al.* (1970). However, from the findings of another group of investigators (Matson *et al.* 1968), sampling *only* a mixture of Yanomama villages in this vicinity, one would be forced to conclude that a 'modest' frequency of the Di^a antigen characterized the whole Yanomama population.

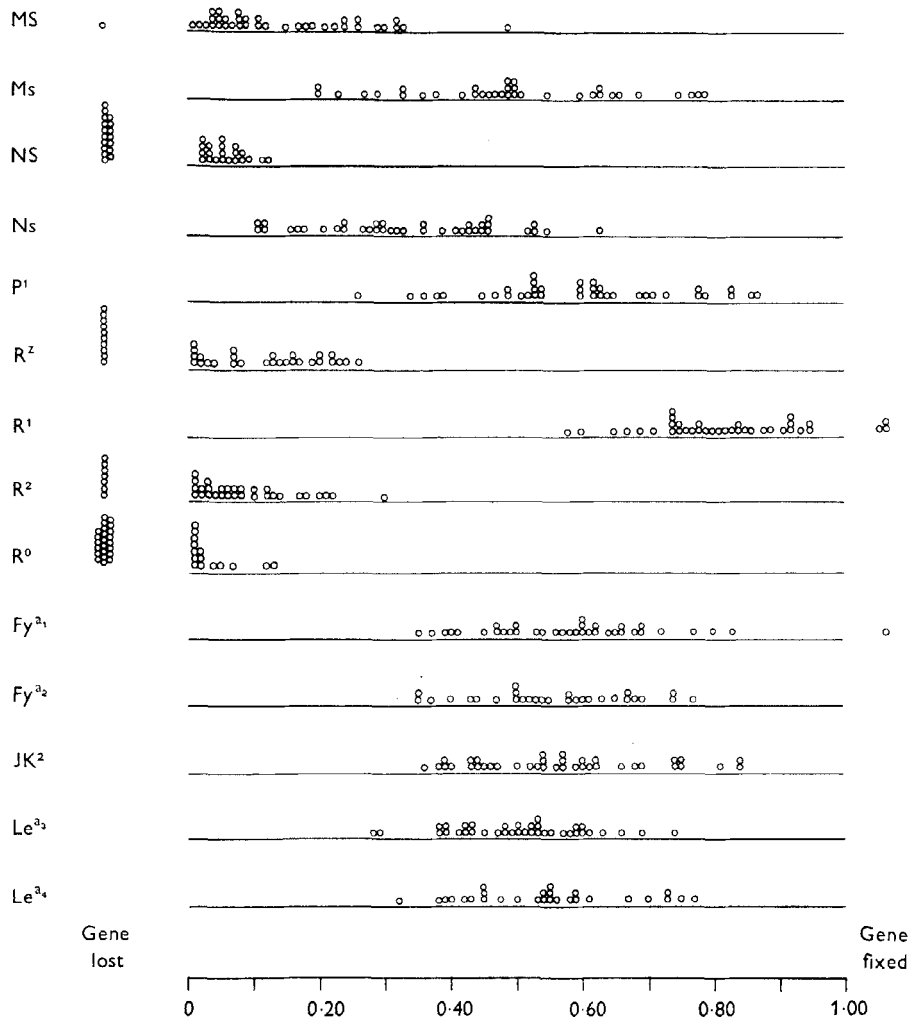

Fig. 1. The distribution of 12 gene frequencies in 37 Yanomama villages (see addenda p. 269 for footnotes).

Especially noteworthy in the present data are villages in which one allele in a system has been lost, or another has gone to fixation. This is especially striking for the Rh system. As long as these villages remain a part of the migration matrix interconnecting Yanomama villages, this is a temporary situation. On the other hand, should any one of such villages by force of events

become the nucleus for a new tribe (with low inter-tribal migration rates), then the result of an essentially chance event could become perpetuated (cf. Neel, 1969).

We have earlier presented a preliminary 'phylogeny' of 12 Indian tribes of Central and South America, and commented on some of the peculiarities in the apparent relationships which emerged (Fitch & Neel, 1969). It would seem desirable, before much further thought is expended on these peculiarities, to resample many of these tribes, in another part of their distribution. Unfortunately, the extreme and erratically distributed decimations to which most Indian tribes have been subject may present a severe impediment to the reconstruction of tribal relations (cf. Neel & Salzano, 1964). Where all but a minor fraction of a tribe has disappeared we can never be certain how 'typical' that remnant is of the total tribe. Basic relationships may thereby be obscured. Thus, valuable though this new approach appears to be, the ultimate appraisal of tribal relationships rests with the synthesis of all the data – linguistic and ethnographic as well as genetic.

In subsequent papers we shall manipulate these village differences in a variety of ways, as we explore their implications for population structure. At this juncture we wish only to point out that the existence of extensive demographic data on this same group renders possible a clear confrontation between various direct and indirect measures of inbreeding, effective population size, and selection.

SUMMARY

The results of tests for 11 red cell antigen systems (ABO, MNS, P, Rh, K, Fy, Jk, Le, Lu, Di and Wr) are presented for 2516 Yanomama Indians of Northern Brazil and Venezuela (distribution, lat. 1–5° N, long. 62–65° 30′ W). The secretor status, in relation to the salivary antigens Le and H, was determined for 1726 of them. Although the average gene frequencies for the tribe are well within the range of values reported for South American Indian tribes, individual villages vary widely in their gene frequencies; for example, the *MS* gene varies from 0·00 to 0·33, *NS* from 0·11 to 0·62, P_1 from 0·36 to 0·87, R^2 from 0·00 to 0·22, and Fy^a from 0·40 to 0·77. The data support the contention that extensive micro-heterogeneity may characterize what is believed to be a single population. The inter-village range in gene frequencies encountered in this study of the inhabitants of 37 villages is as great, for some blood group genes, as the ranges previously reported for inter-tribal differences.

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ADDENDA: FOOTNOTES TO FIGURE I

1. Fy^a calculated from tests with anti-Fy^a only.
2. Fy^a calculated in villages tested with both anti-Fy^a and anti-Fy^b.
3. Le^a calculated from red cell phenotypes.
4. Le^a calculated from saliva phenotypes.