Gene Frequencies and Microdifferentiation among the Makiritare Indians. I. Eleven Blood Group Systems and the ABH-Le Secretor Traits: A Note on Rh Gene Frequency Determinations

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Detailed knowledge of the genetic structure of human populations at any and all cultural levels remains notably sparse. Most of the current concepts (and mathematical treatments) of this structure are based on limited knowledge of civilized populations in a state of cultural, economic, and social transition. Plainly, painstaking and meticulous studies of the genetic details of a variety of populations are necessary to an understanding of such questions as the scope for natural selection and the significance of the enormous amount of genetic variability in human populations.

We are currently in midstream of an effort to characterize in all ways possible the important genetic parameters of a relatively large and undisturbed Indian tribe located in southern Venezuela and northern Brazil, the Yanomama. A recent preliminary communication has reported marked genetic microdifferentiation between the villages of these Indians (Arends et al. 1967). Although there are other reports of tribal microdifferentiation (reviewed in Neel and Salzano 1967), this case was especially striking, perhaps because of the size and lack of acculturation of the tribe.

On their northern and western tribal boundaries, the Yanomama are in contact with Makiritare, also still a relatively undisturbed tribe. When it became apparent that there was genetic exchange occurring between these two tribes, quite possibly along the pattern of much of the genetic exchange between the tribal populations of the past, our studies were expanded to the Makiritare. It will be the purpose of this series of four papers to (1) extend earlier reports (Arends and Gallango 1962, 1964, 1965; Layrisse et al. 1963; Layrisse and Wilbert 1966) on Makiritare gene frequencies, (2) record microdifferentiation among the Makiritare villages comparable to that observed among the Yanomama, (3) provide the data base against which to attempt (later) to quantitate the nature and amount of gene flow between Makiritare and Yanomama, and (4) continue the analysis begun earlier (Neel 1967, 1969) of the tribal structure responsible for this microdifferentiation. Companion papers will describe in detail an example of genetic exchange between the two tribes, with special reference to cultural factors which have implications for the fate of introduced genes

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(Chagnon et al. 1970) and explore some of the implications for evolution of this microdifferentiation (Neel and Ward 1970).

Although aspects of the language and culture of this Carib-speaking tribe have been briefly described (Koch-Grünberg 1917; Cruxent 1953; Escoriaza 1959; Fuchs 1962*a*, 1962*b*, 1964; Wilbert 1963; Francisco 1965; Barandiaran 1966; Layrisse and Wilbert 1966; Cirvieux 1968; Nothomb 1968), they have not yet been subject to intensive anthropological studies. They have remained relatively undisturbed because their traditional homeland is in the area drained by the headwaters and tributaries of such rivers as the Caura, Ventuari, and Orinoco. The number of Makiritare is roughly estimated at 2,000. Their villages are located on navigable rivers; they are superb boatmen. Their economy is based on slash-and-burn agriculture, mandioca (*Manihot esculenta*) being the staple crop. In addition, they have long been known locally as traders who may have traveled as far east as the Esequibo area of Guyana and as far west as Colombia (Koch-Grünberg 1917). Their wide-ranging travels have resulted in considerably more acculturation than is evident among their southern neighbors, the Yanomama. In the final paper of this series we will summarize aspects of their recent history and its relevance to the genetic distance between villages.

MATERIALS AND METHODS

Collection Sites

Figure 1 depicts the location of the seven Makiritare villages, designated as A through HI, which were visited in the course of field work during the first quarters of 1967, 1968, and 1969. Table 1 supplies certain information concerning each village. Layrisse and Wilbert (1966) have previously reported on the blood groups of 86 Makiritares. Of these Indians, 26 were bled at Santa Maria de Erebato, site BD, and the remaining 60 at Acanaña, site HI. Arends and Gallango (1962, 1964, 1965) and Gallango and Arends (1963) have reported on the haptoglobin, transferrin, and Gm types of the Makiritare. Two of their samples involve virtually the same individuals as those typed by Layrisse and Wilbert (1966). In addition, the material of Arends and Gallango (1962, 1965) and Gallango and Arends (1963) includes a sample from several villages on the upper Ventuari River, none of which were visited in the course of the 1967–1969 field work. It is clear that the present sample overlaps in part with these earlier samples, but it is also clear that because of the high mobility of the Makiritare, the earlier sample of villages BD and HI would include some individuals not in the more recent sample.

Methods of Collection and Typing

Specimens were collected from every available individual in each village studied. A village pedigree was obtained in the course of the visit to the village, sufficient in most instances to establish relationships up to first cousin. As a rule, two blood specimens were obtained from each subject, one in a 15-cm^3 Becton-Dickinson Vacutainer containing 2.25 ml ACD-A solution, and one in a 20-ml Vacutainer containing no anticoagulant. When only one specimen was obtained, it was usually collected in ACD solution. Specimens were refrigerated shortly after collection, and then flown to a base lab, usually within 48 hours of collection. In Venezuela the laboratory was that of Layrisse, at the Venezuelan Institute for Scientific Investigations; in Brazil the laboratory was that of Ayres, at the Federal University of Pará, in Belém. An aliquot was removed at these laboratories and the remainder of the specimen shipped by air, refrigerated, to the Department of Human Genetics, University of Michigan. All blood typings were performed in duplicate, using identical antisera. The relatively few discrepancies were reconciled by repeat typings in both laboratories on cells preserved in glycerol at −20° C. or in liquid nitrogen.

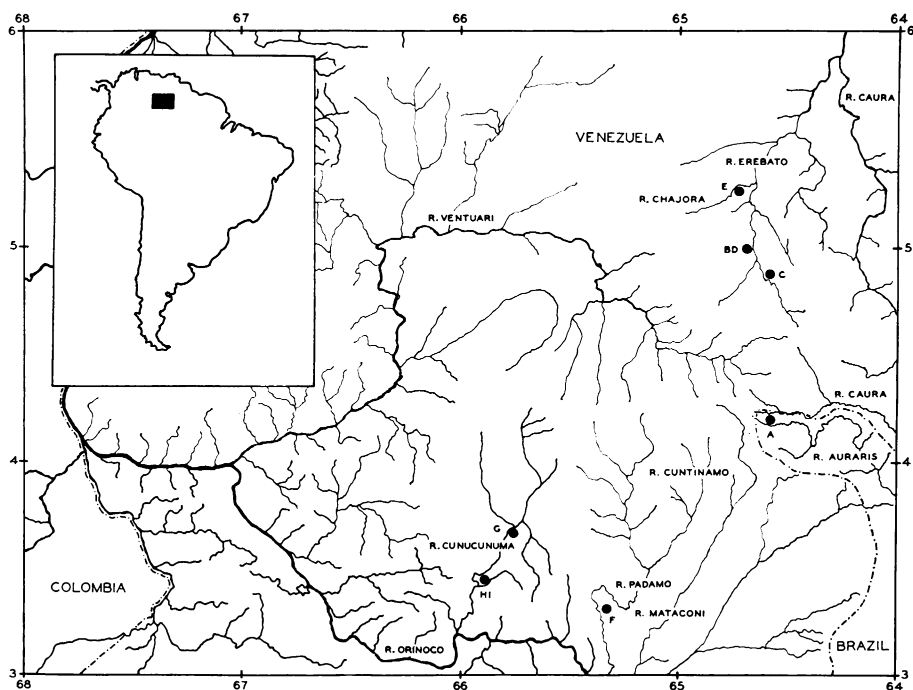

FIG. 1.—The approximate location of the seven Makiritare villages sampled in the course of this study.

TABLE 1

DESCRIPTION OF VILLAGES SAMPLED

Designation (See Fig. 1)	Place	Location (Long., Lat.)	Type of Sample	Year of Sample	Size of Sample	Approximate Size of Village
A...........	Juduwaduña	4°10′ N 64°30′ W	All available	1967	71	90
BD.........	Santa Maria de Erebato	5° 0′ N 64°40′ W	All available	1968	157	176
C...........	Wasaña	4°50′ N 64°35′ W	All available	1968	43	~ 70
E...........	Chajoraña	5°15′ N 64°45′ W	All available	1968	75	82
F...........	Sharamaña	3°15′ N 65°20′ W	All available	1968	47	~ 50
G..........	Belen	3°40′ N 65°45′ W	All available	1969	72	110
HI.........	Acanaña	3°25′ N 65°55′ W	All available	1969	74	130

NOTE.—All locations are approximate, from our placement of the villages on NSAF Operational Navigation Chart ONC L-27, with location rounded to the nearest 5 feet.

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, ($P+P_1$), k, Kp^a, Kp^b, Js^a, Lu^a, Lu^b, V^w, Wr^a, and (Mu+Ht). In addition, secretor types for ABH and Le substances were also done only in Ann Arbor. All tests were performed in tubes with 2% washed red cell suspensions.

Calculation of Gene Frequencies

In this and the following two papers in the series, village gene frequencies are based on all the individuals typed from each village. Many of the individuals from whom samples were obtained were biologically related to one another. As discussed elsewhere (Neel et al. 1964), it is not feasible in such populations as these to sample with certainty only unrelated persons; our procedure is enumerative (by village), rather than sampling. Gene frequencies have been calculated by maximum-likelihood procedures developed in the Department of Human Genetics for the IBM 1130 (Reed and Schull 1968). We must stress the fact that while the gene frequencies are derived from methods which assume random sampling from a large, panmictic population in Hardy-Weinberg equilibrium, this assumption is not valid in the present study.

In the course of the calculation of the Rh gene frequencies from the data in table 2, discrepancies between the results of various methods of calculation came to light. The computer program of Reed and Schull makes no prior assumptions concerning the presence or absence of any of the eight alleles theoretically detectable with the five major antisera. Even in the absence of positive detection of a phenotype proving the presence of a particular gene, as for example, the phenotype r'r which proves the presence of the gene r', the program will assign a value for that gene frequency based on the probability of its contributions to any and all of the phenotypes to which it might contribute.

As a check on the results of this program, gene frequencies were computed by the commonly employed method of Mourant (1954), wherein, in the absence of the appropriate indicator phenotype, a particular gene is assumed not to exist in the population. For four of the seven villages examined, the results of the computer program and the Mourant procedures are in close agreement. These villages (A, BD, C, and E) are characterized by the presence of only the R^1 (*CDe*), R^2 (*cDE*), and R^o (*cDe*) and/or r (*cde*) genes. However, villages F, G, and HI have, in addition, R_z phenotypes, indicating the presence of the R^z (*CDE*) and/or r^y (*CdE*) genes. Under these circumstances the computer program, because of the varying phenotypic proportions, treats each of the three villages differently, assigning R^z to village F but r^y to villages G and HI. In addition, R^o and r are both assigned to F but only r is assigned to G and R^o to HI (table 3). (In these villages, the phenotype frequencies are such that a solution postulating the presence of R^z yields the same likelihood value as one assigning the same frequency to r^y. This aspect of MAXLIK, due to two equivalent maxima in the likelihood surface, will be discussed in a separate paper.) The method of Mourant, on the other hand, at the outset assigns r^y a zero value and, in the absence of either R^o or rr phenotypes, equates phenotypes which could be due to either the R^o or r genes to the presence of R^o. The qualitatively different results obtained by the two methods may be understood in terms of the difference in preconceptions (a maximum-likelihood computer program of this nature makes none); quantitative differences emerged, however, which necessitated further consideration.

Three other methods were then employed to explore these differences:

1. In order to determine whether the reason for the difference between the two approaches rests solely on the assumptions made, Dr. W. J. Schull developed a subroutine for the abovementioned program for the IBM 1130, which assumes r^y, r', r'', and r to be absent (only four genes present), thus in effect employing the approach of Mourant.

2. Layrisse et al. (1963) have formulated a method which has been extensively used in presenting Rh gene frequencies for Venezuelan Indians. The method is used only where Rh negative bloods are not found in the population. It differs from the Mourant procedure in that it does not employ square root methods and partitions the R_1R_2 phenotype into its two

TABLE 2

BLOOD GROUP PHENOTYPES EXHIBITING POLYMORPHISM AMONG SEVEN VILLAGES OF THE VENEZUELAN MAKIRITARE

PHENOTYPES	VILLAGE							TOTAL
	A	BD	C	E	F	G	HI	
MS	0	24	18	4	0	6	6	58
MSs	13	48	13	16	9	14	17	130
Ms	22	28	1	16	9	11	7	94
MNS	6	17	6	5	6	0	11	51
MNSs	19	26	4	12	16	16	18	111
MNs	1	6	0	15	6	14	8	50
NS	5	4	0	0	0	2	0	11
NSs	4	2	1	2	1	6	3	19
Ns	1	1	0	5	0	3	4	14
Total	71	156	43	75	47	72	74	538
P+	53	112	31	51	24	42	57	370
P−	17	45	12	24	23	30	17	168
Total	70	157	43	75	47	72	74	538
R_1R_1 (CDe)	12	27	8	7	3	15	11	83
R_1r (CcDe)	4	4	1	1	2	0	4	16
R_2R_2 (cDE)	20	46	14	26	13	20	16	155
R_2r (cDEe)	3	1	1	2	6	4	4	21
R_1R_2 (CcDEe)	32	79	19	39	14	31	34*	248
R_zR_1 (CDEe)	0	0	0	0	4	2	4	10
R_zR_2 (CcDE)	0	0	0	0	5	0	1	6
Total	71	157	43	75	47	72	74	539
Fy (a+b−)	28	94	25	38	21	....	....	206
Fy (a+b+)	35	54	15	32	22	....	....	158
Fy (a−b+)	8	8	3	5	4	....	....	28
Total	71	156	43	75	47	....	....	392
Fy (a+)	....	....	....	....	....	69	70	139
Fy (a−)	....	....	....	....	....	3	4	7
Total	....	....	....	....	....	72	74	146
Jk (a+)	42	82	32	36	30	37	28	287
Jk (a−)	29	73	11	39	17	35	46	250
Total	71	155	43	75	47	72	74	537
Di (a+)	5	84	33	29	2	14†	21†	188
Di (a−)	66	73	10	46	45	58	53	351
Total	71	157	43	75	47	72	74	539
Le (a−b+)	53	119	17	53	30	51	40	363
Le (a−b−)	18	38	26	22	17	21	34	176
Total	71	157	43	75	47	72	74	539
Le secretor	36	107	16	52	28	47	38	324
Le non-secretor	2	36	25	20	14	12	31	140
Total	38	143	41	72	42	59	69	464

* Two of these were positive with anti-f. See text.

† All of these were tested with anti-Di^b. Three in each group were negative.

contributing genotypes based on the frequencies of the four genes R^1, R^2, R^z, and R^o, which are calculated excluding the R_1R_2 bloods.

3. Since pedigrees were available for all villages, a fifth estimate of gene frequencies was obtained by pedigree analysis, assuming, in the absence of critical phenotypes, that r^y, r', r'' and r were absent. This method is identified by the term "gene counting," although, in view of the admitted frequent inability to distinguish on the basis of phenotype between such genotypes as R^1R^1-R^1r', R^2R^o-R^2r, R^2R^2-R^2r'', and R^1R^z-R^1r^y, the term is a euphemism.

The results of these analyses are shown in table 3. It is obvious that three of the four methods which assume only four genes to be present (identified in the table as the computer-4 gene, Layrisse, and "gene counting" methods) give results which are in close agreement with

TABLE 3

Rh GENE FREQUENCIES* OBTAINED FOR THREE INDIAN VILLAGES BY FIVE METHODS

METHOD	GENES						df	χ^2	P
	CDE (R^z)	*CDe* (R^1)	*cDE* (R^2)	*cDe* (R^o)	*CdE* (r^y)	*cde* (r)			
Village F ($N=47$):									
Computer-8 gene....	.105	.267	.533	.050	....	.045	5	2.09	.9–.8
Mourant............	.168	.204	.470	.158	....	....	5	5.72	.5–.3
Computer-4 gene....	.105	.267	.533	.095	....	....	5	2.09	.9–.8
Layrisse............	.116	.256	.522	.106	....	....	5	2.02	.9–.8
Gene counting.......	.096	.277	.542	.085	....	....	5	2.54	.8–.7
Village G ($N=72$):									
Computer-8 gene....	....	.437	.521	....	.014	.028	6	6.35	.5–.3
Mourant............	.030	.421	.505	.044	....	....	5	5.81	.4–.3
Computer-4 gene....	.015	.437	.520	.028	....	....	5	6.18	.3–.2
Layrisse............	.015	.436	.520	.029	....	....	5	5.98	.4–.3
Gene counting.......	.014	.438	.520	.028	....	....	5	6.25	.3–.2
Village HI ($N=74$):									
Computer-8 gene....	....	.430	.478	.056	.036	....	6	3.52	.8–.7
Mourant............	.070	.396	.443	.091	....	....	5	6.82	.3–.2
Computer-4 gene....	.036	.430	.478	.056	....	....	5	3.52	.7–.5
Layrisse............	.042	.424	.472	.062	....	....	5	3.37	.7–.5
Gene counting.......	.047	.419	.466	.068	....	....	5	3.61	.7–.5

* Although we believe that the small samples involved do not warrant presenting gene frequencies to three decimal places, we have done so here in order to introduce greater precision into the comparison of χ^2 values.

each other. The fifth method, computer-8 gene, assigns frequencies to genes whose existence is questioned by the failure to detect indicator phenotypes. Thus, a dilemma is posed: which genes and which frequencies shall be accepted as proper descriptions of the population? A traditional approach to this question contrasts the goodness-of-fit of the observed phenotype frequencies with those predicted by the derived gene frequencies, as by the use of χ^2. On this basis, there is no clear superiority of one method over the others. However, with respect to the methods which assume only four genes to be present, the Mourant procedure, both by inspection and by *P* values, tends to yield results which differ from the other three. We conclude that it is inappropriate for these populations.

It must again be emphasized that both the determination of the gene frequencies (aside from the gene-counting method) and the tests for goodness-of-fit assume Hardy-Weinberg equilibrium. The conditions of Hardy-Weinberg equilibrium are clearly not met by the population of an Indian village (see Neel et al. 1964). In addition, our observations are based on a virtually complete enumeration of a village of interrelated persons (rather than inde-

pendent sampling from a large universe). There is thus an element of approximation in each step of the traditional process: the derivation of gene frequencies, the comparison of observed to expected phenotypes, and the tests of significance. For the present, in considering the Rh gene frequencies of American Indians, one must bear in mind these possibilities for error. From the pragmatic viewpoint, it is noteworthy (table 3) that if R^z is viewed as the sum of the (indistinguishable) $R^z + r^y$ genes, and R^o as the sum of the (indistinguishable) $R^o + r$ genes, all methods (except Mourant's) yield essentially the same conclusions, whatever the biases may be. We have therefore chosen to present the Rh gene frequency data as determined by the gene-counting method.

The comparison between the results of these methods cannot be made for a non-Indian population, since, on the one hand, for no non-Indian population is there reasonable justification to assume the presence of only these four genes and, on the other, we know of no non-Indian population in which the phenotypes permit comparable accuracy in gene counting procedures. It should not be assumed that the Mourant procedure will be as inefficient for the analysis of all populations; we have no evidence on this point. It will be noted that only the eight-gene computer program assigns a frequency to *r;* this gene has now been identified in eight Indian populations (see Gershowitz et al. 1967), and the propriety of excluding it from calculations until its presence is proven by the critical rr phenotype is a moot point. To sum up, it is apparent that with respect to the Rh system, the gene frequencies derived from a set of data may be greatly influenced by the assumptions one makes concerning the genes present in a population and the manner in which these assumptions are implemented.

In this study, Rh gene frequencies were obtained insofar as possible by direct-counting procedures based on serology and pedigree analysis. Under the assumption that only the genes R^1, R^2, R^z, and R^o are present, the only phenotype for which (in the absence of testing with anti-f) recourse to pedigree information is necessary in the assignment of genotype is R_1R_2. In villages A, BD, C, E, and F, where the initial typing did not include tests with anti-f, 160 of the 183 R_1R_2 individuals could be assigned a genotype on the basis of pedigree data. The remaining 23 were retested with anti-f on blood specimens which had been frozen in liquid nitrogen. All of them (11 in village A, 9 in village B, and 3 in village F) were negative and so they were assigned the R^1R^2 genotype. All R_1R_2 individuals in village G and HI were tested on fresh blood specimens with anti-f, and two positives were found in village HI as noted in table 2. The phenotype R_zr has been omitted from the table because, had it been included, the assignment of bloods to the R_1R_2 phenotype would have presumed identical testing procedures for all of them and this was not the case. Thus with these assumptions and tests, all individuals can be assigned a genotype.

RESULTS AND DISCUSSION

Table 2 presents the data for those markers which are "informative," that is, useful in characterizing villages, and table 4 itemizes the number of specimens tested for those markers which were invariant. It will be seen in table 2 that, for a given village, the same number of bloods may not have been tested for each system. As noted earlier, discrepancies between the two laboratories were resolved by repeat typings. Where this was not possible, and the discrepancy remained, no result is reported. The numbers reported for the studies on saliva represent the actual number of specimens collected.

Table 5 presents gene frequencies for the seven polymorphic blood group systems. The four invariant systems have been omitted from the table. The *Se* gene of the ABH secretor system, *k* of the Kell system, Lu^b of the Lutheran system, *Wr* of the Wright system, and I^o of the ABO system all had frequencies of 1.00. Among the polymorphic systems, the intervillage variation is often striking: the frequency of the *MS* gene ranges from .14 to .67; R^z has a frequency as high as .1 in one village, but is

TABLE 4

NUMBER OF MAKIRITARE SPECIMENS TESTED WHEREIN THE RESULTS OF SUCH TESTS WERE UNIFORM

System	Type	Village							Total
		A	BD	C	E	F	G	HI	
ABO	O	71	157	43	75	47	72	74	539
MN	U+, V^w−	71	157	43	74	47	......	......	392
	Mg−	71	157	43	74	47	72	74	538
	(Mu+Ht)+	71	157	43	74	......	......	......	345
P	$(P+P_1)$*	17	43	12	24	23	30	17	166
Rh	C^w−†	48	110	28	47	23	48	55	359
Kell	K−Js^a−	71	157	43	74	47	72	74	538
	k+Kp^a−Kp^b+	71	157	43	74	47	......	......	392
Lutheran	Lu(a−b+)‡	71	157	43	74	47	......	......	392
Wright	Wr^a−	71	157	43	74	47	72	74	538
ABH secretion	Secretor	38	143	41	72	42	59	69	464

* Only bloods positive to anti-P_1 were tested.
† Only bloods positive to anti-C were tested.
‡ All bloods were tested with both anti-Lu^a and anti-Lu^b.

TABLE 5

BLOOD GROUP GENE FREQUENCIES AMONG THE VENEZUELAN MAKIRITARE

System and Gene	Village							Total
	A	BD	C	E	F	G	HI	
MNSs:								
MS	.14	.38	.67	.24	.18	.24	.35	.32
Ms	.53	.42	.20	.45	.50	.40	.31	.40
NS	.27	.15	.10	.08	.22	.12	.14	.15
Ns	.06	.05	.04	.23	.10	.24	.20	.13
P:								
P^1	.51	.46	.47	.43	.30	.35	.52	.44
P^2	.49	.54	.53	.57	.70	.65	.48	.56
Rh:								
CDE	0	0	0	0	.10	.01	.05	.02
CDe	.42	.44	.42	.36	.27	.44	.42	.41
cDE	.53	.54	.56	.62	.54	.52	.47	.54
cDe	.05	.02	.02	.02	.09	.03	.06	.03
Duffy:								
Fy^a	.64	.78	.76	.72	.68	.80*	.77*	.73
Fy^b	.36	.22	.24	.28	.32	.20	.23	.27
Kidd*:								
Jk^a	.36	.31	.49	.28	.40	.30	.21	.32
Jk^b	.64	.69	.51	.72	.60	.70	.79	.68
Diego*:								
Di^a	.04	.32	.52	.22	.02	.10	.15	.19
Di^b	.96	.68	.48	.78	.98	.90	.85	.81
Lewis secretor:								
Le	.77	.50	.22	.47	.42	.55	.33	.45
le	.23	.50	.78	.53	.58	.45	.67	.55

NOTE.—Gene frequencies, for all systems except Rh, were calculated by the maximum likelihood program of Reed and Schull (1968). Gene frequencies given for the Rh system were obtained by direct gene counting.

* Only one antigen of the system was tested.

completely absent from others; and Di^a and Le also show wide ranges in frequency. Despite this wide range in village gene frequencies, the estimates based on the total samples agree quite well with the previous estimate, based on 86 individuals, of Layrisse and Wilbert (1966). They cite the following frequencies: $MS = .35$, $Ms = .34$, $NS = .18$, $Ns = .13$, $R^z = .02$, $R^1 = .38$, $R^2 = .52$, $R^o = .08$, $P^1 = .42$, $Fy^a = .81$, $Jk^a = .33$, $Di^a = .16$, and $K = 0$. All bloods were Le(a−).

The gene frequencies obtained for the total sample (seven villages pooled) were also used in a comparison with the published gene frequencies for South American Indians. However, the present study reveals the need for caution in such comparisons. Consider only the Diego system. The frequency given for the total in table 5 is .19. However, two of the villages are characterized by Di^a frequencies of .02–.04 and another of .52. If our sample had been restricted to the usual two or three villages, the estimation of Diego frequencies might well have varied by a factor of 4. To what extent microdifferentiation of this type influences some of the estimates on tribal gene frequencies in the literature is unclear; the possibilities are greatest when the groups are relatively unacculturated and retain the tribal population structure.

Despite these village differences, as a tribe, the Makiritare possess the genetic criteria characterizing the American Indian given in Boyd (1963) and Neel and Salzano (1964), namely, high frequencies of I^o, L^m, R^2, Fy^a, Di^a, low frequencies of r and R^o, and absence of K, Le (a+), and Lu^a. Two points are worth special mention. One is that all the villages tested are characterized by R^2 frequencies at the high end of the range reported for American Indians (only 11 of the 99 American Indian tribes reported in Post et al. (1968) possess R^2 gene frequencies higher than the .54 reported here) and the Jk^a frequencies for all the villages are at the low end of the range (only nine of 57 tribes in the Post et al. [1968] report have Jk^a frequencies below .32). In addition, the Makiritare are all ABH secretors; American Indians are characterized by very high frequencies of the *Se* gene (Salzano 1964) with only one notable exception, the Xavante of Brazil (Neel et al. 1964).

SUMMARY

Phenotype and gene frequencies for 11 blood group systems and for the ABH-Le secretor systems are reported for 539 Makiritare Indians of Venezuela, located in seven villages. The gene frequencies of the total sample fall well within the ranges published for the American Indian although R^2 is at the high end and Jk^a at the low end of the ranges for these genes. However, village gene frequencies are shown to vary widely from the mean population frequency, and the difficulty of comparing population gene frequencies in the face of village heterogeneity is discussed. A case demonstrating the relative inefficiency, under certain circumstances, of a widely used method for determining Rh gene frequencies is documented.

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