The Black Caribs (Garifuna) of Livingston, Guatemala: Genetic Markers and Admixture Estimates

By Michael H. Crawford¹, Nancie L. Gonzalez², M. S. Schanfield^{3,4}, Dale D. Dykes⁵, K. Skradski⁵ and H. F. Polesky⁵

ABSTRACT

The Black Caribs (Garifuna) are descendants of West African and Amerindian groups from St. Vincent Island who were transplanted to the coast of Central America in 1797. The founding population, estimated at 2,500 to 5,000 persons, gave rise to 65,000 Black Caribs who presently reside in 54 fishing villages spread geographically from Stann Creek (Dangriga), Belize, to LaFe, Nicaragua. This paper documents the genetic variation observed for 24 blood group, red blood cell and serum protein systems in one of the Black Carib communities of Livingston, Guatemala. Admixture estimates, based upon Gm, suggest the following parental population contribution for Livingston: 70% African, 29% Indian and 1% European.

Livingston is a primarily Black Carib (Garifuna) maritime community of approximately 3,000 inhabitants located on the north (Atlantic) coast of Guatemala near the Belize border (see Figure 1). The Black Caribs are the descendants of escaped or shipwrecked slaves and Carib Indians who inhabited the islands of St. Vincent and Dominica in the Lesser Antilles. The newly-formed ethnic group assumed the Indian language and a large part of the culture, though some syncretism with African custom and belief are discernible. In 1797 approximately 2,000 Black Caribs on St. Vincent were forcibly deported by the British to a small island adjacent to the coast of Honduras (Taylor, 1951). From there they rapidly spread into Guatemala, Honduras and British Honduras (Belize), in all of which the Black Caribs established a series of small horticultural and fishing villages.

Historical accounts from the late 18th and early 19th centuries described Carib phenotypes in terms of considerable variability. For example, E. G. Squier, writing under the pseudonym of Samuel Bard, while reporting on the Honduran village near Trujillo, wrote: "Most are pure Indians, not large, but muscular, with a ruddy skin, and long straight hair.

Human Biology, February 1981, Vol. 53, No. 1, pp. 87-103.

¹Laboratory of Biological Anthropology, University of Kansas, Lawrence, Kansas 66045.

²University of Maryland, College Park, Maryland 20742.

³Milwaukee Blood Center, Milwaukee, Wisconsin 53233.

⁴Present address: Red Cross, National Headquarters, Washington, D.C. 20006.

⁵Minneapolis War Memorial Blood Bank, Minneapolis, Minnesota 55404.

[©] Wayne State University Press, 1981



FIG. 1. The location of a number of Belizean and Guatemalan Black Carib communities.

These were called the Red or Yellow Caribs. Another portion are very dark with curly hair, and betraying unmistakably a large infusion of Negro blood, and are called Black Caribs" (Bard, 1965, p. 317). Thomas Young similarly noted great variability among the Black Caribs. "Some are coal black, others again nearly as yellow as saffron" (Young, 1947, p. 123). Yet by 1956, Gonzalez, having visited 15 settlements extending from Trujillo to Belize City, observed considerable homogeneity among the Black Caribs, with no startling differences observable from community to community.

It is the purpose of this article to document the frequencies of erythrocyte and serum protein antigens and enzymes for the Black Caribs of Livingston and to estimate the relative proportions of West African and Indian contributions to their gene pool. While this article focuses upon Livingston Caribs, a more complete treatment of the biological affinities of Belizean, Guatemalan and Honduran Caribs are summarized in a volume under preparation (Crawford, 1980).

Methods

During the summer of 1975, a total of 206 blood specimens were collected by venipuncture at a clinic located in the meeting hall of the local Catholic church of Livingston. The blood specimens, preserved in ACD solutions, were packed in ice and were shipped for analysis within 10 days of collection to the War Memorial Blood Bank, Minneapolis, Minnesota.

The samples were tested for the following erythrocyte antigens: ABO (A, A₁, B), Kell (K, k, Kp^a, Kp^b, Js^a), MNSs (M, N, S, s, Mt^a), Rh (C, c, D, E, e), Diego (Di^a), Cartwright (Yt^a), and Gregory (Gy^a). The red cell typings were done in microtiter (U) plates using 2% suspension of washed cells by a modification of the method of Crawford, Gottman and Gottman (1970). Antisera were diluted to obtain optimal reactivity. Typings were incubated for 1 hour at room temperature (20°C). The typing plates were centrifuged in a Sorvall GLC-1 centrifuge for two minutes at 500 rpm prior to reading. Antiglobulin testing was also done in microtiter plates, after washing three times, using a 12-channel saline dispenser (Antiglobulin test rinser) and centrifuged subsequent to the addition of antiglobulin reagent and reading.

The serum protein phenotypes for transferrin (Tf), albumin (Al) and group-specific component (Gc) were determined by acrylamide slab electrophoresis and stained with amido black (Polesky, Rokala and Huff, 1975). Subtypes of transferrin were ascertained using two different buffer systems (Sutton, personal communication). Haptoglobin (Hp) and ceruloplasmin (Cp) phenotypes were determined simultaneously by electrophoresis on acrylamide slabs (McCombs and Bowman, 1969).

Stroma free hemolysates for determining erythrocyte enzyme phenotypes were obtained by washing cells three times in saline, diluting 1:1 in distilled water and centrifuging at high speed. Specimens were stored at -20° C until tested.

Adenylate kinase (AK), adenosine deaminase (ADA), 6-phosphogluconate dehydrogenase (6PGD) and acid phosphatase (AcP) were electrophoresed simultaneously on a single horizontal starch slab (Dykes and Polesky, 1976). Esterase D (EsD), malate dehydrogenase (MDH) and isocitrate dehydrogenase (ICD) were phenotyped on starch using citrate phosphate buffer system, pH 5.9, of Karp and Sutton (1967). Phosphoglucomutase (PGM) was tested using the original technique of Spencer et al. (1964). Hemoglobins were analyzed by the method described by Yunis, 1969.

Plasma specimens were shipped to the Milwaukee Blood Center and typed for the IgG immunoglobulin allotypic markers G1m (a, x, z and f), G3m (b0, b1, b3, b5, c3, c5, s and t), and the kappa light chain allotype Km(1) with the reagents listed in Table 1. In addition, selected specimens were also tested for G3m(v) and G3m(b4) and G3m(b3+b5). The plasma were diluted 1/20 and tested in microtiter plates with appropriate controls according to the methods of Schanfield (1971). The plasma was defibrinated prior to testing by heating the diluted specimens at 56°C for 15 minutes. Specimens exhibiting direct agglutinating activity were first inactivated by heat treatment at 65°C for 10 minutes and then retested.

Different methods were employed to compute gene frequencies for blood group and protein systems and Gm haplotypes. The frequencies for the Gm haplotypes were computed by gene counting. The Km^1 allelic frequency was estimated from the square root of the Km(1-) frequency. No attempt was made to correct for the interrelationship of the individuals tested. Allelic frequencies for the blood group systems are maximum likelihood estimates computed by the MAXLIK computer program of Reed and Schull (1968).

Admixture estimates for the Black Carib population based upon red blood cell markers (ABO, Rhesus, MNS, Diego, Kell blood groups systems, hemoglobin and Gm) were computed by means of the maximum likelihood sub-routine in the computer program written by Elston (1971). The gene frequencies from the Carib and Arawak Indians of Venezuela (Geerdink et al. 1974) were used to represent the Amerindian parental populations of the Black Caribs. The African component was based upon the weighted average gene frequencies of available West African population data (Mourant, Kopěc and Domaniewska-Sobczak, 1976).

RESULTS

Tables 2, 3, and 4 summarize the phenotypic frequencies of erythrocyte antigens, enzymes and proteins and serum proteins and enzymes in the sample of Black Caribs from Livingston, Guatemala. Genetic variation occurs at all loci except for Kell, Mt^a, Yt^a, Gy^a antigens and phosphoglucomutase-2 (PGM₂), malate dehydrogenase (MDH), and

Chain		Notat	tion ¹		
Chain type		Alphameric	Numeric	Agglutinator	Coat
IgG 1	Glm	f	3	Sta	Dan
0		Z	17	Pon or Ree	Dwi
		a	1	Pan	Dwi
		x	2	Dev or Max	Pet
IgG 3	G3m	b0	11	Tol	Hun
-		bl	5	Ble or Tol	Hun
		b3+b5	10 + 13	Pla ^{2,3}	Hun
		b3	13	Log	Hun
		b 4	14	G84 ^{2,3}	Hun
		b5	10	Ste	Hun
		c3	6	Alf	522
		c3	6	3200 ^{2,3}	522
		c5	24	Hod or And	522
		g	21	B755 ^{2.3}	Sul
		g	21	R-Hu	Sul
		g	21	Leh	Sul
		S	15	Gai	Puh
		t	16	Ros	Puh
		v	27^{2}	Ray	Sul
Kappa	Km	1	1	Cla	511
••		1	1	Sim	511

Reagents Used for Immunoglobulin Allotyping in this Study

¹Notation recommended by WHO workshop in immunoglobulin allotypes July 1974 (W.H.O. Committee, 1976). Alphameric notation will be used throughout this report.

²Not all specimens tested for this specificity.

³Generously provided by Prof. A.G. Steinberg, Ms. D. Brazier, Dr. S. Litwin and Dr. E. Van Loghem.

isocitrate dehydrogenase (ICD) erythrocyte enzymes. In addition, none of the loci deviate significantly from Hardy-Weinberg expectations.

The gene frequencies for the erythrocyte antigens are summarized in Table 5. In the ABO systems, the Livingston population exhibits a relatively high frequency of the B allele compared to other Black Carib populations, while the 0 allele is less frequent than among Black Caribs of Belize (Crawford, 1980; Weymes et al. 1980; Firschein, 1961). In the Rhesus system, the frequency of the cDe chromosomal segment reflects

	L	ivingston
System and phenotype	No.	Proportion
A_1A_2BO :		
0	112	0.549
\mathbf{A}_{1}	23	0.113
A_2	9	0.044
В	55	0.270
A ₁ B	2	0.010
$A_2 B$	3	0.015
Total	204	1.000
Rhesus:		
CcDe	49	0.239
cDe	68	0.332
cDEe	38	0.185
CcDEe	26	0.127
CDEe	9	0.044
cde	9	0.044
CDe	4	0.019
cDE	2	0.010
Total	205	1.000
MNSs:		
MS	3	0.015
MSs	17	0.084
Ms	20	0.099
MNS	5	0.025
MNSs	27	0.134
MNs	59	0.292

Distribution of Red Cell Antigen Phenotypes among the Black Caribs of Livingston, Guatemala

the considerable African contribution to the Black Carib gene pool. The Amerindian contribution to the Black Carib gene pool is reflected by the presence of the Diego (Di^a) allele.

Unfortunately, Duffy antisera were not available at the time of this study, thus the incidence of the phenotype Fy(a-b-) is unknown. However, the FyFy phenotype was detected in a subsequent population of Black Caribs (Dangriga) and the Fy allele was found to have a frequency of 84.66% (Crawford, 1980).

	L	ivingston
System and phenotype	No.	Proportion
NS	5	0.025
NSs	18	0.089
Ns	48	0.238
Total	202	1.000
Diego:		
Di(a+)	8	0.040
Di(a-)	193	0.960
Total	201	1.000
Kell:		
Kk	0	0
kk	202	1.000
Total	202	1.000
Mta:		
Mt(a+)	0	0
Mt(a-)	202	1.000
Total	202	1.000
Yta:		
Yt(a+)	202	1.000
Yt(a-)	0	0
Total	202	1.000
Gya:		
Gy(a+)	202	1.000
Gy(a-)	0	0
Total	202	1.000

Table 2 (continued)Distribution of Red Cell Antigen Phenotypes among theBlack Caribs of Livingston, Guatemala

A further indicator of Amerindian admixture is the presence of Albumin Mexico, which occurs at the incidence of 0.5% in Livingston (Table 6). The presence of the transferrin variant D_1 is further documentation for an African component in the Black Carib gene pool. The acid phosphatase locus exhibits characteristic African pattern with a low frequency of the allele P^A , high frequency of the P^B allele and the absence of the P^C allele. The P^C allele has a frequency of approximately 5% in Europe. The P^R allele, normally rare or absent elsewhere in the world, has a frequency

	L	ivingston
System and Phenotype	No.	Proportion
Albumin:		
AA	203	0.990
AMe	2	0.010
Total	205	1.000
Haptoglobin:		
1-1	73	0.358
2-1	97	0.475
2-2	30	0.147
2-1M	4	0.020
Total	204	1.000
Transferrins:		
CC	195	0.947
CD_1	11	0.053
Total	206	1.000
Ceruloplasm:		
AB	9	0.044
BB	196	0.956
Total	205	1.000
Group Specific Component:		
1-1	43	0.210
1-2	161	0.790
2-2	0	0.000
Total	204	1.000
Esterase D (EsD):		
1-1	166	0.834
2-1	31	0.156
2-2	2	0.010
Total	199	1.000
Malate Dehydrogenase (MHD):		
1-1	205	1.000
Isocitrate Dehydrogenase (ICD):		
1-1	205	1.000
Hemoglobin (Hb):		
AA	172	0.852
AS	29	0.143
SS	1	0.005
Total	202	1.000

Distribution of Serum Protein Phenotypes in the Black Carib Population of Livingston, Guatemala

	Livingston			
System and Phenotype	No.	Proportion		
Phosphoglucomutase 1 (PGM):				
1-1	154	0.774		
2-1	42	0.211		
2-2	3	0.015		
Total	199	1.000		
Phosphoglucomutase 2:				
1-1	200	1.000		
Acid Phosphatase (AP):				
AA	6	0.030		
AB	58	0.286		
BB	122	0.601		
AR	1	0.005		
BR	16	0.079		
Total	203	1.000		
Adenosine Deaminase (ADA):				
1-1	204	0.997		
2-1	0	0.000		
2-2	1	0.003		
Total	205	1.000		
6-Phosphogluconate				
Dehydrogenase (6PGD):				
AA	198	0.966		
AC	7	0.034		
Total	205	1.000		
Adenylate Kinase (AK):				
1-1	198	0.975		
2-1	5	0.025		
Total	203	1.000		
Esterase D (EsD):				
1-1	166	0.834		
2-1	31	0.156		
2-2	2	0.010		
Total	199	1.000		
Malate Dehydrogenase (MDH):				
1-1	205	1.000		
Isocitrate Dehydrogenase (ICD):				
1-1	205	1.000		
Hemoglobin (Hb):				
AA	172	0.851		
AS	29	0.143		
SS	1	0.005		
Total	202	1.000		

Distribution of Red Cell Enzyme and Hemoglobin Phenotypes in the Black Carib Population of Livingston, Guatemala

System	Allele	Frequency
$A_1 A_2 BO$	0	0.744
	\mathbf{A}_{1}	0.064
	A_2	0.032
	В	0.160
MNSs	MS	0.110
	Ms	0.314
	NS	0.108
	Ns	0.468
Rh	CDE	0.034
	cDE	0.145
	cDE	0.508
	CDe	0.091
	CdE	0.000
	Cde	0.121
	cdE	0.000
	cde	0.101
Diego	Di ^a	0.020
<u> </u>	Di ^b	0.980
Kell	k	1.000
	K	0.000
Martin	Mt ^a	0.000

Distribution of Blood Group Frequencies in Livingston Caribs

varying from 2 to 25% in African with the latter percent found among the Khoisan peoples (Mourant, Kopěc and Domaniewska-Sobczak, 1976). The Black Carib frequency of the P^{R} allele (4%) further lends credence to their West African origins.

The Black Caribs of Livingston are polymorphic for all common Black African haplotypes: $Gm^{z,a;b0,1,3,4,5,u,v}$ (hereafter referred to as $Gm^{z,a;b)}$, $Gm^{z,a;b0,1,c3,5,u}$ (hereafter referred to as $Gm^{z,a;b,c3,5}$), $Gm^{z,a;b0,1,4,5,c3,uv}$ (hereafter referred to as $Gm^{z,a;b,c3}$) and $Gm^{z,a;b0,3,5,s,v}$ ($Gm^{z,a;b,s}$). In addition, a low frequency of the European haplotype, $Gm^{f;b0,1,3,4,5,u,v}$ (hereaftter referred to as $Gm^{f;b}$) was observed in this population (2.1%), while the haplotypes $Gm^{z,a;g,u,v}$ (hereafter referred to as $Gm^{z,a;g}$) and $Gm^{a,z,x;g,u,v}$ (hereafter referred to as $Gm^{z,a,x;g}$) which occur in both European and Amerindians are distributed polymorphically (Table 7). Two rare African haplotypes, $Gm^{z,a;g,b0,1,3,4,5,u,v}$ (commonly referred to as $Gm^{z,a;g,b}$) and $Gm^{z,a,x;b0,1,3,4,5,u,v}$ ($Gm^{z,a,x;b}$), were detected in low frequency (Table 8).

System	Allele	Frequency
Albumin	Al ^A	0.995
	Al ^{Mex}	0.005
Haptoglobin	Hp^1	0.596
	Hp^2	0.395
	Hp^{2m}	0.010
Transferrin	Τſ °	0.973
	Tf D1	0.027
Phosphoglucomutase 1	PGM-1 ¹	0.879
	PGM-1 ²	0.120
Phosphoglucomutase 2	PGM-2 ¹	1.000
Acid Phosphatase	p ^A	0.175
	р ^в	0.783
	p ^R	0.042
Ceruloplasmin	Ċp ^A	0.022
	Срв	0.978
Adenosine deaminase	ADA ¹	1.000
	ADA ²	0.000
Group-specific component	Gc1	0.895
	Gc ²	0.105
6-Phosphogluconate dehydrogenase	PGD ^A	0.983
	PGD ^c	0.017
Adenylate kinase	AK ¹	0.988
	AK ²	0.012
Esterase D	EsD ¹	0.912
	EsD ²	0.088
Malate dehydrogenase	MDH ¹	1.000
	MDH ²	0.000
ICD	ICD ¹	1.000
	ICD^2	0.000
Hemoglobin	Hb ^A	0.923
-	Hb ^s	0.077

Distributions of Red Blood Cell and Serum Gene Frequencies in Livingston Caribs

ADMIXTURE ESTIMATES

Table 9 compares the admixture estimate for the Black Caribs of Livingston based only upon Gm with that (estimated proportions of the gene pool) computed using seven distinct blood group and protein systems. A maximum likelihood estimate, using a triracial ancestry model of the Black Caribs, suggests that 75.2% of the gene pool is of West African

Т	้ล่	b	le	7
	u	0.		

Immunoglobulin Phenotypes in Black Caribs of Livingston, Guatemala

Phenotype Glm G3m	Observed numbers	Proportion	
z, a; b0, 1, 3, 4, 5, v	43	0.230	
z, a; b0, 1, 3, 4, 5, c3, 5, v	24	0.128	
z, a; b0, 1, 3, 4, 5, c3, v	13	0.069	
z, a; b0, 1, 3, 4, 5, s, v	5	0.027	
f, z, a; b0, 1, 3, 4, 5v	4	0.021	
z, a; g, b0, 1, 3, 4, 5, v	19	0.102	
z, a, x; g, b0, 1, 3, 4, 5, v	31	0.166	
z, a; b0, 1, c3, 5	3	0.016	
z, a; b0, 1, 4, 5, c3, 5, v	2	0.011	
z, a; b0, 1, 3, 5, c3, 5, s, v	1	0.005	
z, a; g, b0, 1, c3, 5, v	6	0.032	
z, a, x; g, b0, 1, c3, 5, v	3	0.016	
z, a; b0, 1, 4, 5, c3, v	1	0.005	
z, a; b0, 1, 3, 4, 5, c3, s, v	1	0.005	
z, a; g, b0, 1, 4, 5, c3, v	3	0.016	
z, a, x; g, b0, 1, 4, 5, c3, v	3	0.016	
z, a; g, b0, 3, 5, s, v	1	0.005	
z, a, x; g, b0, 3, 5, s, v	3	0.016	
z, a; g, v	6	0.032	
z, a, x; g, v	9	0.048	
z, a, x; b0, 1, 3, 4, 5, v	3	0.032	
z, a, x; b0, 1, 3, 4, 5, c3, v	1	0.005	
z, a; g, b0, 1, 3, 4, 5, c3, 5, v		0.011	
Total	187	1.000	
χ^2 11.94, df 11, p>.05			
K m(1 +)	120	0.642	
Km(1-)	67	0.358	
Total	187	1.000	

origin, 22.4% is Amerindian, and 2.4% European. The Gm based estimate of admixture is similar to those based upon all seven loci—with two minor exceptions. The Spanish and African proportions are slightly inflated and the Amerindian component is reduced in the maximum likelihood estimate when compared to m values based upon Gm. Crawford et al. (1976) discuss some of the biases of the maximum likelihood estimates when this method is applied to a triracial hybrid population in Mexico.

98

						ł	Iaplotyp	be			
Population	N	IgGl IgG3	z,a b	z,a b,c3,5	z,a b,c3	z,a b,s	f b	z,a g	z,a,x g	z,a b,s,t	Km ¹
Livingston ¹	187		0.495	0.118	0.067	0.029	0.011	0.134	0.131		0.401
Bantu ²	115		0.613	0.287	0.030	0.065					0.433
English (USA) ²	146		0.006				0.695	0.202	0.093	0.003	0.097
Carib (Surinam) ⁴	257		0.060	0.085	0.045	0.021	0.021	0.534	0.227	0.006	0.365
Arawak (Surinam) ⁴	194		0.052	0.016		0.003	0.010	0.656	0.261	0.003	0.380

A Comparison of Immunoglobulin Haplotype Frequencies of Black Caribs from
Livingston, Guatemala with other Populations

¹This study

²Van Loghem (1978)

³Stevenson and Schanfield (in preparation)

⁴Geerdink et al. (1974)

One measure of the relative fit of admixture models is the degree of agreement between the observed and expected values of gene frequencies in the hybrid group (Crawford et al. 1976). This test is based upon the multiplication of the estimated contributions from each parental population by the frequency of the gene in that population. The sum of the two products in a biracial hybrid (or three in a triracial) is the expected frequency of a gene, and can be compared with the observed value of that gene frequency in the hybrid group and tested by chi-square. An average

Table 9

		Parenta	l Population's Cont	ribution	
Population	Marker	African	Indian	Spanish	
	System	%	%	%	
Livingston	Gms	70.0	29.0	1.0	
Livingston	7 loci*	75.2	22.4	2.4	

A Comparison of Admixture Estimates for Livingston Black Caribs, Based upon Immunoglobulins and other Blood Markers

*Systems used are: ABO, Rh, MNS, Diego, Kell, Hemoglobin, and Gm.

chi-square value for all genes is obtained following the method of Pollitzer (1964).

Most of the maximum likelihood based models of biracial or triracial origins result in approximately the same average chi-square values (Table 10). The lowest total chi-square value detected for the Livingston Black Carbis, 7.16, occurs with Spanish, West African and Arawak Indian parental populations. Four of the seven loci tested deviated significantly from expectation-if gene flow alone was responsible for the observed gene frequencies in the hybrid population. The four loci, ABO, Rhesus, Hemoglobin and Gm, have all been proposed as systems upon which natural selection operates (Workman, 1968). Interestingly, the loci which deviate significantly among a Black Carib population, found in a malarial region, are different from those that deviate in the altiplano of Mexico (Crawford, 1976). Of the 15 loci tested in Tlaxcala, only Duffy, Group Specific Components and Lewis deviate significantly from admixture expectation. Of these three loci, evidence exists for the operation of selection on Gc and particularly the Duffy FyFy phenotype (Miller et al. 1975). While we recognize the difficulties associated with the possible errors in the hypothesized parental gene frequencies (Mandarino and Cadien, 1974) and the necessity of a large number of loci for an accurate estimate

Table 10

Locus	Degrees of Freedom	Parental Populations		
		European, African Arawak/Vencarib	European, African Arawak	African Vencarib/Arawak
АВО	2	10.84**	6.43*	7.09*
Rhesus	5	49.95**	29.18**	32.83**
MNSs	3	5.37	6.62	5.46
Diego	1	0.32	0.04	0.01
Kell	1	0.21	0.39	0.24
Hemoglobin	1	3.85*	7.64**	6.62*
Gm	7	38.20**	46.28**	37.66**
Total	20	8.54	7.16	8.54

Comparison of Chi-Square Values for Different Methods of Calculating Proportion of Admixture

**p < 0.01

*p < 0.05

of m (Adams and Ward, 1973), the observed patterns in both Tlaxcala and Livingston are suggestive of the possible action of selection.

DISCUSSION

The ethnohistory of the Black Caribs (Garifuna) indicates that the population arose from the hybridization of the Carib Indians and West Africans on St. Vincent Island. The genetic admixture estimates confirm the presence of African and Carib (Amerindian) alleles in the gene pool, but also reveal some Spanish or European gene flow (1 - 2.4%). While a portion of this admixture is clearly the result of the temporary residence of Europeans in Livingston, some may be due to liaisons between Black Caribs and Creoles. The Creoles are individuals or populations who are the result of European and African mixture and often speak a Creole language, which in the Livingston area is usually based upon English.

Of particular ethnohistorical interest is that the "best fit" of various triracial and biracial models of admixture came with an Arawak parental population representing the Indian component. This finding can in part be explained by the fact that the Arawak Indians were in control of the West Indies until the arrival of the Carib Indians in the late 14th century. According to oral tradition, the Caribs set out from the coast of Venezuela moving north. Steward and Faron (1959) state that the Caribs conquered the Arawak of the Lesser Antilles, killing the men and taking the women as wives. The archeological evidence suggests a gradual deterioration of Arawak pottery after the Carib invasion. However, no new pottery styles or motifs are evident immediately following the invasion. Since Carib pottery is generally of poorer quality than any made by Arawak, and since women generally made the pottery in the Americas, it is not surprising that Arawak pottery features and motifs persisted for some time after the Carib invasion. Judging from the biological evidence the Arawak contributed genetically more to the Black Caribs than did the Venezuelan Caribs. This suggests that the Indian component was largely female Arawak (and some surviving males) and smaller numbers of conquering Carib males.

ACKNOWLEDGEMENTS

This study was supported in part by grants from the National Institute of Dental Research DE04115-02 and from the Research and Education Trust of the Milwaukee Blood Center. During the analysis of these data the senior author was on a PHS Research Career Development Award K04 DE028-02. We would like to acknowledge the technical assistance of Ms. Victoria Enciso and Mr. R. Baylarian. Ms. Ruth Byleen assisted in the preparation of the manuscript. We thank Father Anthony Briskey for the use of the church facilities in Livingston.

Received: 21 January 1980.

LITERATURE CITED

- ADAMS, J. P. AND R. H. WARD 1973 Admixture studies and the detection of selection. Science, 180: 1137-1143.
- BARD, S. A. 1965 Waikna; or adventures on the Mosquito Shore. Gainesville, Florida (a facsimile of the 1855 edition).
- CRAWFORD, M. H. 1976 The Tlaxcaltecans: prehistory, demography, morphology and genetics. University of Kansas Publications in Anthropology, #7, Lawrence.
- CRAWFORD, M. H. (ed.) 1980 Population biology and culture history of the Black Caribs (Garifuna) of Central America. Manuscript.
- CRAWFORD, M. H., F. E. GOTTMAN AND C. A. GOTTMAN 1970 Microplate system for routine use in blood bank laboratories. Transfusion, 10: 258-263.
- CRAWFORD, M. H., P. L. WORKMAN, C. MCLEAN AND F. C. LEES 1976 Admixture estimates and selection in Tlaxcala. *In:* The Tlaxcaltecans: prehistory, demography, morphology and genetics, M. H. Crawford (ed.). University of Kansas Publications in Anthropology, #7, pp. 161-168.
- DYKES, D. D. AND H. F. POLESKY 1976 The usefulness of serum protein and erythrocyte enzyme polymorphisms in paternity testing. Am. J. Clin. Path. 65: 982-986.
- ELSTON, R. C. 1971 The estimation of admixture in racial hybrids. Ann. Hum. Genet. 35: 9-17.
- FIRSCHEIN, I. L. 1961 Population dynamics of the sickle-cell trait in the Black Caribs of British Honduras, Central America. Am. J. Hum. Genet. 13: 233-254.
- GEERDINK, R. A., L. E. NIJENHUIS, E. VANLOGHEM AND E. LI FO SJOE 1974 Blood groups and immunoglobulin groups in Trio and Wajana Indians from Surinam. Am. J. Hum. Genet. 26: 45-53.
- GHIDINELLI, A. 1976 La familia entre Los Caribes Negros, Ladinos y kekchies de Livingston. Guatemala Indigena XI, Nos. 3-4. Guatemala.
- GONZALEZ, N. L. 1969 Black Carib household structure. Seattle.
- KARP, G. W. AND H. E. SUTTON 1967 Some new phenotypes of human red cell acid phosphatase. Am. J. Hum. Genet. 19: 54-62.
- MANDARINO, L. AND J. D. CADIEN 1974 Use of ranked migration estimates for detecting natural selection. Am. J. Hum. Genet. 26: 108-110.
- MCCOMBS, M. L. AND B. H. BOWMAN 1969 Demonstration of inherited ceruloplasmin variants in human serum by acrylamide electrophoresis. Texas Rep. Biol. Med. 27: 769-772.
- MILLER, L. H., S. J. MASON, J. A. DVORAK, M. H. MCGINNIS AND I. K. ROTHMAN 1975 Erythrocyte receptors for (Plasmodium knowlesi) Malaria: Duffy blood group determinants. Science, 189: 561-562.

- MOURANT, A. E., A. C. KOPĚC AND K. DOMANIEWSKA-SOBCZAK 1976 The distribution of the human blood groups and other polymorphisms. Oxford University Press, London.
- POLESKY, H. F., D. ROKALA AND T. HUFF 1975 Serum proteins in paternity testing. In: Paternity Testing, H. F. Polesky (ed.). American Society of Clinical Pathologists. Chicago, The Division of Educational Media Services, pp. 30-44.
- POLLITZER, W. S. 1964 Analysis of a tri-racial isolate. Human Biol. 36: 362-373.
- REED, T. E. AND W. J. SCHULL 1968 A general maximum likelihood estimation program. Am. J. Hum. Genet. 20: 579-580.
- SCHANFIELD, M. S. 1971 Population studies on Gm and Inv antigens in Asia and Oceania. University Microfilms, Ann Arbor.
- SCHANFIELD, M. S., H. H. FUDENBERG, M. H. CRAWFORD AND K. TURNER 1978 The distributions of immunoglobulin allotypes in two Tlaxcaltecan populations. Ann. Hum. Biol. 5: 577-590.
- SOLIEN, N. L. (same as Gonzalez) 1959 West Indian characteristics of the Black Carib. Southwestern J. Anthropol. 15: 303-307.
- SPENCER, N., D. A. HOPKINSON AND H. HARRIS 1964 Phosphoglucomutase polymorphism in man. Nature, 204: 742-745.
- STEWARD, J. H. AND L. C. FARON 1959 Native peoples of South America. McGraw-Hill, New York.
- TAYLOR, D. 1951 The Black Carib of Honduras. Viking Fund Publication in Anthropology, No. 17, Wenner-Gren Foundation, New York.
- VANLOGHEM, E., L. SALIMONU, A. WILLIAMS, B. O. OSUNKOYA, A. M. BOYD, G. LELANGE AND L. E. NIJENHUIS 1978 Immunoglobulin allotypes in African populations. I. Gm-Am haplotypes in a Nigerian population. J. Immunogenetics, 5: 143-147.
- WEYMES, H., D. TILLS, A. WARLOW, A. C. KOPĚC, J. M. LORD, R. G. HUNTSMAN AND R. CUSTODIO 1980 Blood groups and haemoglobin and plasma protein polymorphism in Black Carib populations. Manuscript.
- WHO Committee on human immunoglobulin allotypes 1976 Review of the notation for the allotypic and related markers of human immunoglobulins. Europ. J. Immunol. 6: 599-601.
- WORKMAN, P. L. 1968 Gene flow and the search for natural selection in man. Human Biol. 40: 260-279.

YOUNG, T. 1947 Narrative on a residence on the Mosquito Shore. London.

YUNIS, J. 1969 Biochemical methods in red cell genetics. Academic Press, New York.