Genetic polymorphisms and racial groups in the population from the province of Pinar del Río (Cuba)

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The polymorphism of 13 genetic markers in three racial groups (whites, mulattoes and blacks) of the population of Pinar del Río province (Cuba) were investigated. Differences among phenotypic rates in 8 of the 13 markers studied was found. The allelic frequencies in the markers that showed differences were highly significant among the racial groups. The loci investigated are able to clearly differentiate one racial group from another. This finding confirm the presumption that the population of this region is not homogeneous..

Introduction

The Pinar del Río province is located in the western tip of the Island of Cuba. Its population is near 697 986 inhabitants (Census of 1981) and occupies an area of 10 924.56 Km ^{2}. The population of this province, as well as that from Cuba, is the product of migrations of different racial groups: Whites of Spanish origen, Negroes from Africa and Chinese from Asia. In 1492 this province was the least populated region in Cuba. This part of Cuba was originally inhabited by aborigines of the Guanahatabey cultural group (Carreras, 1985). The onset of Spanish colonization initially created a racial mixture between whites and aborigines. Towards the end of the XVIth century few traces of the Guanahatabey cultural group remain (Pérez de la Riva, 1972; Alonso, 1990). The Spaniards that colonized this region, during the first decade of the XVth century, were primarily from Canary Islands and Castilla (Le Riverend, 1974; Sochegui, 1980). African Negroes were brought to Cuba as slaves since the Spaniards settlement and during colonization (XVth century) (Moreno Fraginals, 1978). The number of slaves brought to Cuba during colonization is thought to be over one million individuals (Pérez de la Riva, 1970). The exact origin of this population is unknown, but it is believed that most of them were taken from places along the west coast of Africa (from Senegal to Angola) (Moreno Fraginals, 1977; Moreno, 1978; Rivero de la Calle, 1981). The racial admixture between whites and blacks created the "mulato" group. This group is a typical element in the Cuban population (Hidalgo, 1986). The Chinese people were brought to Cuba in the middle of the XIXth century from Kwangtung and Fukien provinces. Despite the great number of Chinese Coolies who arrived in Cuba (124 000) only 49 were females according to the 1899 Census

(Pérez de la Riva, 1967). Consequently, the Asian immigration left few traces on the structure of the Cuban population. This group represents a small fraction of the studied population and its possible contribution was not taken into account.

Studies of genetic markers in the Cuban population are scarce. They are limited mainly to Mas Martín et al. (1964) (ABO and RH (D) in Habana City (capital of Cuba), González et al. (1976), García et al. (1982) (GLO1), Barrios and Granda (1983) and Hidalgo (1986) in the central region of Cuba. In Pinar del Río province only our studies on the ABO (without A1 and A2 phenotypes) (Díaz, 1985) and HP systems (Díaz et al., 1995) have been performed.

The objective of this work is to investigate the polymorphism distribution of 13 genetic markers in the population of Pinar del Río province and to determine whether there exist phenotipic and allelic differences in each genetic marker studied among the three typical racial groups of this population: whites, mulattoes and blacks.

Material and methods

The sample was randomized and collected from unrelated volunteer blood donors, from Pinar del Río province Blood Banks. Among 95% donors were males and aged 20 to 40 years. All subjects were born in Pinar del Río. Their parents and grandparents had also been born in this province. The conditions of the study were communicated to all participants and each of them signed a written consent form authorizing their blood analysis.

To ensure the sample was representative of the province, the subjects were selected from each political-administrative region (municipalities) of the province. The classification of subjects into racial groups (White, Mulatto and Black) was made according to two criteria: Direct observations of morphologic characters (Hidalgo, 1986) and personal interviews where specific questions on the ancestry were asked to each of the participants by a single interviewer. We took Mulattoes and Blacks in a new group denoted as Negroids.

Blood samples were obtained by venous puncture with anticoagulant. Plasma and cells were separated by centrifugation. Then plasma was frozen at –30º C so as to study the CHE2, PI, GC and TF systems. The red blood cells were subjected to two different processes: 1) a resuspension of erythrocytes was made to determine phenotypes of ABO (without A1 and A2), MN and P systems; 2) crioconservation, as described by Issit (1985). This was undertaken for later study of the HB, GLO1, PGM1 and G6PD systems, A1 and A2 phenotypes of ABO system and phenotypes of KELL and RH (haplotypes) systems.

The samples were transferred from the Pinar del Río Blood Bank to the Human Genetic Laboratory of The Higher Medical Science Institute of Santa Clara, Villa Clara (Cuba), at –30º C. The phenotype determination of ABO (without A1 and A2), MN and P systems were made by the method described by Levine (1954). The KELL, RH (haplotypes) and A1 y A2 phenotypes were studied by the microenzymatic technique described by The American Association of Bloods Banks (1985). The phenotype determination of Hemoglobin (HB), Glucose-6-Phosphate Dehydrogenase (G6PD) and Pseudocholinesterase (CHE2) systems were typed according to methods described by Ciscar (1972), Beutler (1966) and Heredero et al. (1974) respectively and the phenotypes of Glyoxalase 1 (GLO1) according to Taggard et al. (1978) and Palmour et al. (1980). The phenotypes of Alpha-1-antitripsin (PI) system were determined by the method described by Kueppers (1976). The phenotypes for the Group Specific Component (GC) and Transferrin (TF) were determined according to Dannewist (1985) and, the phenotypes of Phosphoglucomutase 1 (PGM1) system, were obtained by the method published by Dykes and Polensky (1981).

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Table 1. Phenotypic and allelic frequencies in each genetic system studied in Whites. $\chi 2$: Chi-Square of □oly-Weinberg equilibrium

ABO		**KELL**		**GLO1**		**PI**		**TF**	
A1	50	KK	0	1-1	27	MM	126	1-1	104
A2	20	Kk	26	2-1	80	MS	27	2-2	6
B	6	kk	281	2-2	48	SS	0	3-3	0
A1B	4	n = 307		n = 155		PI*M – 0,9117±1,6214E-2		1-3	3
A2B	0	KEL*K – 0,0423±0,00812		GLO1*1 – 0,4322±0,0281		PI*S – 0,0883±1,6214E-2		2-3	1
O	72	KEL*k – 0,9577±0,00812		GLO1*2 – 0,5657±0,0281		$\chi 2$ = 1,37; 1 d.f.; p>0,01		1-2	39
n = 152		$\chi 2$ = 0,60; 1 d.f.; p>0,01		$\chi 2$ = 0,36; 1 d.f.; p>0,01		n = 153		n = 153	
ABO*A1 – 0,1962±0,02412								TF*C1 – 0,8169±2,2104E-2	
ABO*A2 – 0,0859±0,01842		**RH**		**G6PD**		**PGM1**		TF*C2 – 0,1699±2,147E-2	
ABO*B – 0,0333±0,01038		CCDEe	1	Normals	145	1ª	62	TF*C3 – 0,0132±6,493E-2	
ABO*O – 0,6844±0,02902		CCDee	30	Deficients	3	1B	6	$\chi 2$ = 1,08; 3 d.f.; p>0,01	
$\chi 2$ = 3,19; 2 d.f.; p>0,01		CcDEe	15	G6PD*+ – 0,9797±0,0082		1ª1B	17		
		CcDee	58	G6PD*- – 0,0203±0,0082		2ª	7	**GC**	
MN		□ol	0	n = 148		2B	1	1F	4
MM	52	ccDEE	4			2ª2B	3	1S	59
MN	97	ccDEe	17	**HB**		1ª2A	39	2-2	8
NN	32	ccDee	6	AA	305	1ª2B	8	2-1F	1
n = 181		ccddee	20	AS	3	1B2A	7	2-1S	57
MN*M – 0,5552±2,6118E-2		ccddEe	1	AC	0	1B2B	3	1F-1S	24
MN*N – 0,4448±2,6118E-2		n = 152		n = 308		n = 153		n = 153	
$\chi 2$ = 1,30; 1 d.f.; p>0,01		Haplotypes		HB*A – 0,9951±7,91E-6		PGM*1ª – 0,6144±0,0278		GC*1F – 0,1078±1,7731E-2	
		Cde	0,43906±0,02867	HB*S – 0, 0049±7,91E-6		PGM*1B – 0,1274±0,0190		GC*1S – 0,6503±2,7260E-2	
P		Cde	0,00000±0,00003	$\chi 2$ = 0,007; 1 d.f.; p>0,01		PGM1*2ª – 0,2059±0,0231		GC*2 – 0,2418±0,2447	
P1	136	CDE	0,00501±0,00532			PGM*2B – 0,0523±0,0123		$\chi 2$ = 11,42; 3 d.f.; p>0,01	
P2	16	cDE	0,12495±0,02027	**CHE2**		$\chi 2$ = 9,10; 6 d.f.; p>0,01			
n = 152		cdE	0,00819±0,00822	C5+	13				
P*1	0,6755±0,0383	cDe	0,05194±0,01978	C5-	140				
P*2	0,3245±0,0383	cde	0,37279±0,02773	n = 153					
		$\chi 2$ = 1,81; 3 d.f.; p>0,01		CHE2*-	0,9566±0,0116				
				CHE2*+	0,0434±0,0116				

. **Table 2**. Phenotypic and allelic frequencies in each genetic system studied in olymorph. χ2: Chi-Square of oly-Weinberg equilibrium

ABO
A1 30
A2 15
B 7
A1B 2
A2B 0
O 56
n = 110
ABO*A1 – 0,1576±0,02569
ABO*A2 – 0,0852±0,02104
ABO*B – 0,0418±0,01364
ABO*O – 0,7152±0,03260
χ2 = 1,05; 2 d.f.; p>0,01

MN
MM 42
MN 81
NN 30
n = 141
MN*M – 0,5392±2,8495E-2
MN*N – 0,4608±2,8495E-2
χ2 = 0,65; 1 d.f.; p>0,01

P
P1 77
P2 33
n = 110
P*1 – 0,4523±0,0398
P*2 – 0,5477±0,0398

KELL
KK 1
Kk 20
kk 201
n = 222
KEL*K – 0,0495±0,01029
KEL*k – 0,9505±0,01029
χ2 = 0,42; 1 d.f.; p>0,01

RH
CCDEe 1
CCDee 15
CcDEe 10
CcDee 32
ol 0
ccDEE 1
ccDEe 20
ccDee 23
ccddee 7
ccddEe 1
n = 110
Haplotypes
Cde 0,32810±0,03223
Cde – 0,00000±0,00005
CDE – 0,00826±0,00863
cDE – 0,13213±0,02681
cdE – 0,01415±0,01521
cDe – 0,26731±0,04528
cde – 0,25003±0,04485
χ2 = 4,27; 3 d.f.; p>0,01

GLO1
1-1 10
2-1 64
2-2 42
n = 112
GLO1*1 – 0,3750±0,0320
GLO1*2 – 0,6250±0,0320
χ2 = 3,11; 1 d.f.; p>0,01

G6PD
Normals 95
Deficients 9
G6PD*+ – 0,9135±0,0195
G6PD*- – 0,0865±0,0195
n = 104

HB
AA 217
AS 6
AC 0
n = 223
HB*A – 0,9865±5,45E-3
HB*S – 0, 0134±5,45E-3
χ2 = 0,035; 1 d.f.; p>0,01

CHE2
C5+ 4
C5- 107
n = 111
CHE2*- – 0,9818±0,0089
CHE2*+ – 0,0182±0,0089

PI
MM 101
MS 9
SS 1
PI*M – 0,9505±1,4564E-2
PI*S – 0,0495±1,4564E-2
χ2 = 2,47; 1 d.f.; p>0,01
n = 111

PGM1
1ª 50
1B 3
1ª1B 20
2ª 3
2B 0
2ª2B 1
1ª2A 23
1ª2B 6
1B2A 5
1B2B
n = 112
PGM*1ª – 0,6652±0,0315
PGM*1B – 0,1429±0,0233
PGM1*2ª – 0,1562±0,0242
PGM*2B – 0,0357±0,0123
χ2 = 0,63; 6 d.f.; p>0,01

TF
1-1 93
2-2 1
3-3 0
1-3 0
2-3 0
1-2 17
n = 111
TF*C1 – 0,9144±1,8775E-2
TF*C2 – 0,1699±1,8775E-2
χ2 = 8,03E-2; 1 d.f.; p>0,01

GC
1F 34
1S 19
2-2 1
2-1F 9
2-1S 3
1 1F-1S 45
n = 111
GC*1F – 0,5495±3,3392E-2
GC*1S – 0,3873±3,2695E-2
GC*2 – 0,0630±1,6314E-2
χ2 = 2,46; 3 d.f.; p>0,01

. **Table 3**. Phenotypic and allelic frequencies in each genetic system studied in Blacks. χ2: Chi-Square of oly-Weinberg equilibrium

ABO		**KELL**		**GLO1**		**PI**		**TF**	
A1	19	KK	2	1-1	7	MM	98	1-1	99
A2	11	Kk	25	2-1	48	MS	7	2-2	0
B	21	kk	186	2-2	51	SS	0	3-3	0
A1B	0	n = 213		n = 106		PI*M – 0,9667±0,0123		1-3	0
A2B	0	KEL*K – 0,0681±0,0122		GLO1*1 – 0,2924±0,0312		PI*S – 0,0333±0,0123		2-3	0
O	56	KEL*k – 0,9319±0,0122		GLO1*2 – 0,7075±0,0312		χ2 = 0,10; 1 d.f.; p>0,01		1-2	6
n = 107		χ2 = 1,19; 1 d.f.; p>0,01		χ2 = 0,84; 1 d.f.; p>0,01		n = 105		n = 153	
ABO*A1 – 0,0936±0,02042								TF*C1 – 0,9715±0,0114	
ABO*A2 – 0,0596±0,01728		**RH**		**G6PD**		**PGM1**		TF*C2 – 0,0285±o,0114	
ABO*B – 0,1045±0,02151		CCDEe	1	Normals	88	1ª	42	χ2 = 7,73E-2; 1 d.f.; p>0,01	
ABO*O – 0,7420±0,03121		CCDee	3	Deficients	15	1B	2		
χ2 = 4,52; 2 d.f.; p>0,01		CcDEe	3	G6PD*+ – 0,8544±0,0246		1ª1B	23		
		CcDee	15	G6PD*- – 0,1456±0,0246		2ª	4	**GC**	
MN		ol	1	n = 103		2B	0	1F	49
MM	40	ccDEE	1			2ª2B	1	1S	5
MN	67	ccDEe	28	**HB**	1ª2A	21	2-2	2	
NN	34	ccDee	49	AA	195	1ª2B	7	2-1F	6
n = 141		ccddee	6	AS	15	1B2A	4	2-1S	3
MN*M – 0,5213±0,0297		ccddEe	0	AC	1	1B2B	1	1F-1S	40
MN*N – 0,4787±0,0297		n = 107		n = 211		n = 105		n = 105	
χ2 = 0,32; 1 d.f.; p>0,01		Haplotypes		HB*A – 0,9620±9,29E-3		PGM*1ª – 0,6429±0,0330		GC*1F – 0,6857±3,2034E-2	
		Cde 0,09911±0,02631		HB*S – 0, 0355±9,01E-3		PGM*1B – 0,1524±0,0248		GC*1S – 0,2523±2,9974E-2	
P		Cde 0,01888±0,01803		HB*C – 0,0023±2,36E-3		PGM1*2ª – 0,1619±0,0254		GC*2 – 0,0619±1,6629E-2	
P1	78	CDE – 0,00816±0,00009		χ2 = 0,32; 1 d.f.; p>0,01		PGM*2B – 0,0428±0,0139		χ2 = 8,12; 3 d.f.; p>0,01	
P2	29	cDE – 0,15071±0,02533				χ2 = 1,97; 6 d.f.; p>0,01			
n = 107		cdE – 0,00000±0,00004		**CHE2**					
P*1 – 0,4795±0,0412		cDe – 0,48289±0,05009		C5+	3				
P*2 – 0,5205±0,0412		cde – 0,24023±0,04666		C5-	103				
		χ2 = 7,96; 3 d.f.; p>0,01		n = 105					
				CHE2*- – 0,9856±0,0082					
				CHE2*+ 0,0144±0,0082					

The homogeneity of phenotype frequencies among racial groups, were tested by the chi-square test of homogeneity (X^2). The significance level was chosen at $p<0,01$ (negroids were excluded from this comparison). The allelic frequency estimates were obtained by maximun likelihood method. In systems with one completely dominant allele we assumed that the Hardy-Weinberg equilibrium was reached. In the estimation of haplotype frecuencies of the RH system five sera were employed: anti-C, anti-c, anti-D, anti-E and anti-e, considering the simultaneous presence of Cde and CDE.

The Hardy-Weinberg equilibrium condition was calculated according to standard procedures described by Smith (1970). A $p<0,01$ level of significance with one degree of freedom was used. For those loci with more than two alleles, the expected genotypic proportions were obtained by means of an obvious extension of the Hardy-Weinberg law. In those cases, the degrees of freedom were calculated as the difference between the number of alleles and the number of possible phenotypes (Workman et al., 1963).

Results

The results of phenotypic and allelic frequencies of all genetic systems are presented in Tables 1 to 4 for whites, mulattoes, blacks and negroids in general respectively. The χ^2 test for homogeneity of phenotypes among racial groups was highly significant ($p<0,01$) in the ABO, P, RH (haplotypes), TF, GC, G6PD, HB and GLO1 (Table V). The χ^2 (H-W) test was not significant for all the racial groups and for all the genetics markers (Tables I to IV).

Discussion

If all genes with a frequency higher than 0,01 are considered polymorphic (Harris, 1980), it can be stated. That the according to our study hemoglobin is not polymorphic in the white and black racial groups. Moreover, certain systems such CHE2 and TF show polymorphic borderline frequencies. The differences observed in allelic frequencies between the different racial groups are large. Thus, the Pinar del Río population is not homogeneous. These findings are in agreement with previous results obtained by González et al. (1976); Hidalgo (1986); García et al. (1982); Barrios y Granda (1983) in other Cuban populations. Upon analyzing the heterogeneity between the different racial groups it was observed that among the 13 systems most of them showed significant differences. If, data about HP (haptoblobin) system obtained by Díaz et al. (1995) in the same population, are included, we can see that out of 14 genetic systems in that population, nine of them showed highly significant differences. This racial heterogeneity has been maintained by more than 17 generations since the Spaniards` arrival to Cuban Islands. Perhaps assortative mating maintains these groups apart. The non-significant deviation from the Hardy-Weinberg equilibrium obtained by the goodness-of-fit test between the observed and the expected phenoptypic frequencies suggests that the conditions of random mating, homogeneity of subsamples, and absence of selection are roughly fulfilled in each group. These findings affirm our hypothesis about the existence of differences between racial groups in Pinar del Río. Taken together these loci discriminate well among the racial groups examined here (64,4% of these polymorphisms studied in our population showed significant differences).

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Resumen

El polimorfismo de 13 marcadores genéticos de tres grupos raciales (blancos, mulatos y negros) de la población de la provincia de Pinar del Río (Cuba) ha sido analizada. Se encontraron diferencias en los índices fenotípicos en 8 de los 13 marcadores estudiados. En los marcadores en los que se encontraron diferencias, las frecuencias alélicas eran muy distintas, por lo que los loci analizados discriminan clara-

mente entre en los grupos raciales analizados. Este resultado confirma la hipótesis de que la población de esta región no es homogénea.

Palabras clave: polimorfismos genéticos, grupos raciales, Cuba

Polimorfismos genéticos y grupos raciales en la población de la provincia de Pinar del Río (Cuba)