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G-6-PD Deficiency Gene Dynamics in a Brazilian Population

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The population of Pôrto Alegre has been the subject of several studies with the long-range objective of understanding its genetic structure and ongoing evolution (Tondo and Salzano, 1962; Tondo et al, 1963; Salzano, 1963; Salzano and Hirschfeld, 1965; Harrison et al, 1967). The present series of two papers (Salzano et al, 1967) can be viewed as an extension of these previous investigations; but the polymorphism involved in the synthesis of glucose-6-phosphate dehydrogenase presents many challenges to the human geneticist and it was felt that a study of its incidence in the various ethnic groups of Pôrto Alegre could contribute to the elucidation of some aspects of its dynamics. In this paper we will be concerned with the penetrance of these genes in heterozygous females, with problems related to the adaptive fitness of their carriers and with the possibility of using them for estimates of gene flow between the Negro and White segments of this population.

Material and Methods

Details about demographic and genetic aspects of the population groups sampled can be obtained in the previous papers. The present population of Pôrto Alegre numbers about 700 000 persons, 14% of them showing signs of Negroid ancestry. The samples were collected at the " Instituto de Pesquisas Biológicas " of the State Department of Health, which provides free clinical examinations for the public in general and especially for government employees. In the white sample questions were asked about the parents' and grandparents' nationalities of the subjects. The results (expressed in percentages) were as follows: *Males* (102 persons): 1) Portuguese ancestry: 77.4; 2) At least two Italian grandparents: 15.7; 3) At least two German grandparents: 2.9; 4) At least two Slavic grandparents: 2.0; 5) Grandparents with other nationalities: 2.0. *Females* (141 persons): 1) Portuguese ancestry: 71.6; 2) At least two Italian grandparents: 14.2; 3) At least two German grandparents: 4.3; 4) At least two Spanish grandparents: 5.7; 5) At least two Slavic grandparents: 2.1; 6) Grandparents with other nationalities: 2.1.

The Fairbanks and Beutler (1962) spot test technique was adopted throughout

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the work. About half of the samples were tested with reagent tubes prepared in our laboratory according to the directions given by these Authors; the remainder were tested with commercially available G-6-PD field test kits. Reagent tubes either prepared by ourselves or from the kits were kept in desiccators with silica gel; this precaution was necessary because otherwise the reagents would decay markedly under the conditions prevailing in Pôrto Alegre.

The blood samples were collected by venipuncture and placed in tubes with an anticoagulant (dried solution of oxalates); the erythrocytes for G-6-PD determination were then separated by centrifugation and kept in the refrigerator until they were tested. The time elapsed from blood collection to actual testing varied from a few hours to eight days. Most samples were processed about three days after collection. No more than five samples were analyzed simultaneously. Each reagent tube had enough chemicals to test 18 samples, but to be on the safe side we decided never to test more than 15 samples with each tube.

Hemoglobin was eluted from the test paper by immersing it in distilled water for three to five minutes and blotting the strip between sheets of filter paper, the whole process being repeated four or five times until the red color due to hemoglobin disappeared. Afterwards the reagent was applied to the paper as recommended by Fairbanks and Beutler (1962) and after a two-minutes delay the color due to the formazon dye was visually estimated. A color intensity scale with four gradations could be easily made: 1) Full color development (normal G-6-PD activity); 2) Less intense coloration (intermediate " high "); 3) Weak coloration (intermediate " low "); and 4) No color development (G-6-PD activity not detected). For practical purposes, however, categories 2 and 3 were grouped together in the analysis of the data.

All tests were made at room temperature which ranged from 15 to 28° C; all samples showing a decrease of enzyme activity (categories 2, 3 and 4) were tested twice. Samples with normal activity served as controls.

Results

Incidence of G-6-PD Deficiency

Tab. 1 shows the results obtained in the 416 males and 820 females tested. As reported elsewhere (Kraus et al, 1962; Van der Sar et al, 1964) we have also found males with intermediate deficiency. The total number of deficient males (intermediate + complete) varied from 3.9% among the Whites to 15.1% among the Dark Mulattoes; the incidence in the Negroid sample in general was 12.4%. Among the females the total incidence of deficient individuals varied from 5.0% among the Whites to 18.8% among the Negroes; the general incidence in Negroids was 15.5%.

In Tab. II our results are compared with other surveys performed in North, Central and South American populations. Among 2 888 Negroid males the incidence of G-6-PD deficiency (G-6-PDD) varied between 5.4 and 17.6%. The Puerto Rico

Tab. I. Incidence of G-6-PD deficiency in the four ethnic categories established

Ethnic group	Males			Females		
	N. studied	Deficients		N. studied	Deficients	
		Intermediate	Complete		Intermediate	Complete
Negroes	116	—	13	197	34	3
	%	—	11.2		17.3	1.5
Dark Mulattoes	99	2	13	225	20	11
	%	2.0	13.1		8.9	4.9
Light Mulattoes	101	2	9	257	31	6
	%	2.0	8.9		12.1	2.3
Negroids in general	316	4	35	679	85	20
	%	1.3	11.1		12.5	3.0
Whites	102	3	1	141	5	2
	%	2.9	1.0		3.6	1.4

and Baltimore samples showed unexpected low values; the latter, however, contained many related individuals; the incidence in Surinam is somewhat higher than those obtained in other places and was obtained in populations living in a malarious environment. If these three populations are not considered, the remaining frequencies show a somewhat unexpected uniformity (10-14%). Two small samples of North American white males did not show the defect but in three Brazilian groups the incidence of the deficiency among Caucasians was 3-4%. This difference is not surprising if we consider that the Northern samples were obtained among Anglo-Saxons while the Southern ones comprise individuals of predominantly Mediterranean descent. As a matter of fact, all deficient individuals of our sample had ancestors from Southern Europe (males: two of Portuguese and two of Italian ancestry; females: six of Portuguese and one of Spanish/Portuguese ancestry).

Only six surveys included women also. Among 1580 Negroid females tested the incidence of the deficiency varied between 2.8 and 15.5%. Our sample seems to be the only one which included White women.

G-6-PD Deficiency and Gene Flow

How can the results we obtained in the different racial subgroups (Tab. 1) be used for gene flow analysis? At the outset we are faced with a problem: the incidences in the different racial segments do not follow those expected according to a simple dilution model. It can be argued that this may have happened just because of sampling problems. But the discrepancy can be viewed from another angle. Thus, it is now well established that the basis of the G-6-PD deficiency in Negroes is different from the one responsible for the defect in whites (see, for instance, Beutler, 1965). The incidence of the deficiency in Negroids in general, therefore, constitutes a composite

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Tab. II. Incidence of G-6-PD deficiency in several American negro and white populations (in %)

Population	Males		Females			All	Method
	N. studied	Defic.	N. studied	Deficients			
				Intermediate	Complete		
North America							
Several Negro groups [1]	308	11.0	448	4.9	2.7	7.6	GSH St.
Chicago Negroes [2]	56	14.3	44	—	—	6.8	GSH St.
Memphis Negroes [3]	157	14.0	69	—	—	5.8	Several
Memphis Whites [3]	85	—	—	—	—	—	Several
Baltimore Negroes [4]	238	6.3	—	—	—	—	BCB
Claxton Negroes [5]	76	11.8	97	7.2	3.1	10.3	BCB
Claxton Whites [5]	44	—	47	—	—	—	BCB
Central America							
Puerto Rico Negroes [6]	56	5.4	143	—	—	2.8	BCB
Curaçao Negroes [7]	573	14.0	213	7.5	2.8	10.3	BCB
Port of Spain Negroes [8]	175	13.1	—	—	—	—	BCB
South America							
Several Negro groups, Surinam [9]	850	17.6	—	—	—	—	Sp. EA
São Paulo Negroids (L) [10]	83	9.6	—	—	—	—	BCB
São Paulo Whites (H) [10]	234	3.8	—	—	—	—	BCB
São Paulo Whites (L) [10]	323	2.8	—	—	—	—	BCB
Pôrto Alegre Negroids [11]	316	12.3	679	12.5	3.0	15.5	MTT r
Pôrto Alegre Whites [11]	102	3.9	141	3.6	1.4	5.0	MTT r

[1] Reviewed in Beutler (1960); [2] Naylor et al (1960); [3] Kraus et al (1962) – two Negro males fell in the "intermediate" glutathione group; the large number of males and females with abnormal hemoglobins indicates that the sample was not a random one; [4] Porter et al (1962) – the sample was composed of 106 unrelated males and their brothers; [5] Cooper et al (1963); [6] Suarez et al (1961); [7] Van der Sar et al (1964) – seven males showed intermediate activity; [8] Sutton (1963); [9] Pik et al (1965) – the incidence of deficient individuals varied from 7 to 20% in different regions; [10] Beiguelman et al (1966) – H = healthy persons; L = leprosy patients; [11] Present communication.

Methods: GSH St. = glutathione stability test; BCB = brilliant cresyl blue; Sp. EA = spectrophotometric enzyme assay; MTT r = reduction of the tetrazolium dye, MTT.

figure in which the " Negro " and " White " genes for the deficiency are present. The same reasoning can be applied to explain the excess of deficient individuals among the Dark Mulattoes: unlike the more " pure " Negroes they would also present " Negro " and " White " genes for the deficiency. Assuming for the moment that this argument is correct we could use the frequency of the male Negro subsample as representative of the " Negro " G-6-PD allele in the Brazilian population of this ethnic group. The incidence in Africa is quite variable (see, for instance, Ragab et al, 1966) but we can use the value of 20% as the frequency in the original male Africans (as was done by Workman, 1963). Whith these values we would arrive at an estimate of gene admixture of the order of 56% and an average gene flow per generation (assuming 12 generations of contact) of 4.7%. This is in accordance with previous estimates (see Salzano and Hirschfeld, 1965; Harrison et al, 1967). But the sources of error are so large that we prefer to postpone any further discussion on the uses of this gene for this purpose. We are going to start the study of the electrophoretic mobility of these variants and when these data become available it will be possible to study the problem on a much firmer basis.

G-6-PD Deficiency and Selection

There exist very little data concerning the fitness of G-6-PD gene carriers. In the study of this problem we used two different approaches. Tab. III shows the incidence of G-6-PDD in individuals who came to the collecting post because they were engaged in some kind of health treatment and in those who went there for other reasons. As can be seen there is no apparent trend of higher or lower gene frequencies in one of the two groups in the different racial segments individually or in the total sample. In Tab. IV the distribution of these genes is compared according to the ages of the individuals studied. Among the males there are no differences in the three age intervals established. Among the females, however, the difference between the incidences in the 0-29 and 30-59 age brackets is consistent in the four racial segments and statistically significant in the total sample ($\chi^2 = 6.6$; 2 d. f.; $P < 0.05$). However, the deviation is in the *opposite direction* than the one expected by postulating some kind of selective disadvantage for the G-6-PDD genes in the non-malarious environment of Pôrto Alegre.

Penetrance of G-6-PDD Genes

It is a well-known fact that the available G-6-PD methods used in population surveys detect only a fraction of female carriers of the deficient allele. Insufficient data, however, exist concerning the factors which might influence the degree of gene expression and few tests were made to compare the different methods of G-6-PDD detection. An attempt in this direction is presented in Tab. V. Using the frequency of hemizygous males deficient for G-6-PD we calculated the expected frequency of heterozygous females and compared it with the observed incidences of both inter-

Tab. III. Incidence of G-6-PD deficiency in individuals undergoing health treatment and in those who came to the collecting post for other reasons

Ethnic group and reason for examination	Males			Females		
	N. studied	Deficients		N. studied	Deficients	
		Intermediate	Complete		Intermediate	Complete
Negroes						
In health treatment	73	—	7	128	21	2
	%	—	10		16	2
Remainder	43	—	6	69	13	1
	%	—	14		19	1
Dark Mulattoes						
In health treatment	60	1	8	141	14	4
	%	2	13		10	3
Remainder	39	1	5	84	6	7
	%	3	13		7	8
Light Mulattoes						
In health treatment	56	1	5	138	19	3
	%	2	9		14	2
Remainder	45	1	4	119	12	3
	%	2	9		10	3
Whites						
In health treatment	35	1	1	49	2	2
	%	3	3		4	4
Remainder	67	2	—	92	3	—
	%	3	—		3	—
Total sample						
In health treatment	224	3	21	456	56	11
	%	1.3	9.4		12.3	2.4
Remainder	194	4	15	364	34	11
	%	2.1	7.7		9.3	3.0

mediate and complete deficiency in Pôrto Alegre females of four racial segments and in women from three other surveys.

The data of Tab. V show: (1) that factors in some way related to the racial background of the Pôrto Alegre women studied have influenced the degree of gene expression, since the estimates of penetrance vary in the different racial subgroups; (2) that the observed frequency of complete deficiency in females is too high to be considered an expression of gene homozygosis; and (3) that the Fairbanks and Beutler (1962) method we used is probably better for heterozygous detection than those used by the Authors listed in the table, since it seems to reveal about twice as many heterozygotes as the other methods.

Tab. IV. Distribution of G-6-PD deficiency phenotypes according to age

Ethnic group	Age	Males			Females		
		N. studied	Deficients		N. studied	Deficients	
			Intermediate	Complete		Intermediate	Complete
Negroes *	0-29	53	—	5	79	11	1
		%	—	9		14	1
	30-59	56	—	7	97	19	2
		%	—	12		20	2
	60 and	6	—	1	12	4	—
	more	%	—	17		19	—
Dark Mulattoes	0-29	46	1	7	106	8	4
		%	2	15		7	4
	30-59	45	1	5	103	11	7
		%	2	11		11	7
	60 and	8	—	1	16	1	—
	more	%	—	12		6	—
Light Mulattoes	0-29	52	—	5	133	15	1
		%	—	10		11	1
	30-59	42	1	4	109	13	5
		%	2	10		12	5
	60 and	7	1	—	15	3	—
	more	%	14	—		20	—
Whites	0-29	49	1	—	62	2	—
		%	2	—		3	—
	30-59	49	2	1	64	3	1
		%	4	2		5	2
	60 and	4	—	—	15	—	1
	more	%	—	—		—	7
Total	0-29	200	2	17	380	36	6
		%	1.0	8.5		9.5	1.6
	30-59	192	4	17	373	46	15
		%	2.1	8.9		12.3	4.0
	60 and	25	1	2	67	8	1
	more	%	4.0	8.0		11.9	1.5

* The age of one individual from this ethnic group is unknown.

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Tab. V. Calculation of some genetic parameters of the groups studied and of three other populations

Ethnic group	Frequency of hemizygous males deficient for G-6-PD	Expected frequency of heterozygous females	Observed frequency of intermediate deficiency in females	Postulated degree of dominance (Obs./Exp.) (D^1)	Expected frequency of homozygous females	Observed frequency of complete deficiency in females	Frequency considering all deficient females as heterozygotes	Postulated degree of dominance (Obs./Exp.) (D^2)
Porto Alegre [1]								
Negroes	0.112	0.198	0.173	0.874	0.013	0.015	0.188	0.950
Dark Mulattoes	0.151	0.256	0.089	0.348	0.023	0.049	0.138	0.539
Light Mulattoes	0.109	0.194	0.121	0.624	0.012	0.023	0.144	0.742
Negroids in general	0.124	0.218	0.125	0.573	0.015	0.030	0.155	0.711
Whites	0.039	0.074	0.036	0.487	0.002	0.014	0.050	0.676
Several US Negro groups [2]	0.110	0.196	0.049	0.250	0.012	0.027	0.076	0.388
Claxton Negroes, USA [3]	0.118	0.208	0.072	0.346	0.014	0.031	0.103	0.495
Curaçao Negroes [4]	0.140	0.240	0.075	0.313	0.020	0.028	0.103	0.429

[1] Present communication; [2] Reviewed in Beutler (1960); [3] Cooper et al (1963); [4] Van der Sar et al (1964).

Discussion

It is our opinion that probably due to the usefulness of the G-6-PD polymorphism for studies of gene action and linkage, population studies of these variants were somewhat neglected. Many problems in the latter field, however, remain open for studies. Simultaneous investigations in many populations of G-6-PD activity and electrophoretic mobility of the enzyme are needed to clarify some problems of distribution and others as well. For instance, are the males in whom only " intermediate " G-6-PD activity was detected carriers of a different G-6-PDD allele or are there genetic and/or environmental factors which are responsible for the difference in gene expression?

The difficulties we encountered in applying our data to gene flow models were already stressed. But it should be pointed out that a combined approach to this problem, studying G-6-PD activity and electrophoretic mobility as suggested above, can furnish some valuable data for the elucidation of gene flow problems. The apparently high incidence of deficiency we found among Dark Mulatto males can be explained in several ways. Assuming for the moment that it was due to a combination of " Negro " and " White " genes for deficiency (those related to the electrophoretic bands A and B), it is possible to suggest that we are dealing again with problems of gene expression. No studies exist concerning the manifestation of different G-6-PDD alleles in persons of a racial background different from the one in which the variants originated. Genes normal in a predominantly Caucasian genotype can function in an abnormal way in another genetic background with or without the production of a slightly different enzyme. The only indication we have showing that this may be so is the report by Marks et al (1961) that the G-6-PD purified enzyme of a deficient Caucasian-Negro male showed properties of the catalytic site different from those of one deficient Caucasian male and four deficient Negro males.

The G-6-PDD gene carriers should have a fitness close to one and our failure to demonstrate differences of incidence in groups of people with minor health problems and normals came as no surprise. The age analysis did not reveal any departure in frequency compatible with selective problems. However, the excess of deficient individuals among women of ages 30-59 compared with those of ages 0-29 is interesting and deserves further comment. In the Negroids it could be due to the larger amount of admixture which might exist in the younger generation; but the trend is also present among white women; it is tempting to relate this, in some way, with penetrance modifiers which would act differentially in the two age intervals. We would also like to emphasize again the need for further studies on the penetrance factors in females which are in some way related to racial background in our sample. The G-6-PD polymorphism already furnished extremely valuable data for the knowledge of the human genotype; additional population studies may be quite revealing in that connection.

Summary

Studies on the glucose-6-phosphate dehydrogenase activity of erythrocytes from 416 males and 820 females are reported. The total number of deficient males (intermediate + complete) varied from 3.9% among the whites (102 persons) to 15.1% among Dark Mulattoes (99 individuals); the incidence in the Negroid sample in general (316 persons) was 12.4%. The data obtained among the women were utilized to study problems of penetrance of the G-6-PDD genes. Results obtained in both sexes were employed to verify, unsuccessfully, possible selection differentials through age analysis and the comparison of the gene incidences of people affected with minor ailments and controls. Problems of gene flow analysis in populations like this one in which the " Negro " and " White " forms of the deficiency are present were stressed.

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References

Beiguelman B., Pinto W. Jr., Dall'Aglio F. F., Da Silva E., Vozza J. (1966). Deficiência de G-6-PD e lepra. Ciência e Cultura, **18:** 95-96.

Beutler E. (1960). Drug-induced hemolytic anemia (primaquine sensitivity). *In:* The Metabolic Basis of Inherited Disease. J. B. Stanbury, J. B. Wyngaarden & D. S. Fredrickson Eds., McGraw Hill, New York.

— (1965). G-6-PD deficiency. *In:* The Metabolic Basis of Inherited Disease. J. B. Stanbury, J. B. Wyngaarden & D. S. Fredrickson Eds., McGraw Hill, New York.

Cooper A. J., Blumberg B. S., Workman P. L., McDonough J. R. (1963). Biochemical polymorphic traits in a U.S. White and Negro population. Amer. J. Hum. Genet., **15:** 420-428.

Fairbanks V. F., Beutler E. (1962). A simple method for detection of erythrocyte G-6-PD deficiency (G-6-PD spot test). Blood, **20:** 591-601.

Harrison G. A., Owen J. J. T., Da Rocha F. J., Salzano F. M. (1967). Skin colour in Southern Brazilian populations. Hum. Biol., **39:** 21-31.

Kraus A. P., Neely C. L., Carey F. T., Kraus L. M. (1962). Detection of deficient erythrocyte regeneration of reduced triphosphopyridine nucleotide from glucose-6-phosphate. Evaluation of a rapid screening test. Ann. Intern. Med., **56:** 765-773.

Marks P. A., Gross R. T., Banks J. (1961). Evidence for heterogeneity among subjects with G-6-PD deficiency. Abstract, 2nd Internat. Congr. Hum. Genet., Excerpta Med. Int. Congr. Series, **32:** 60-61.

Naylor J., Rosenthal I., Grossman A., Schulman I., Hsia D. Y. Y. (1960). Activity of G-6-PD in erythrocytes of patients with various abnormal hemoglobins. Pediatrics, **26:** 285-292.

Pik C., Loos J. A., Jonxis J. H. P., Prins H. K. (1965). Hereditary and acquired blood factors in the Negroid population of Surinam. II. The incidence of haemoglobin anomalies and the deficiency of G-6-PD. Trop. Geogr. Med., **1:** 61-68.

PORTER I. H., SCHULZE J., MCKUSICK V. A. (1962). Genetical linkage between the loci for G-6-PD deficiency and colour-blindness in American Negroes. Ann. Hum. Genet., **26:** 107-122.
RAGAB A. H., EL-ALFI O. S., ABBOUD M. A. (1966). Incidence of G-6-PD deficiency in Egypt. Amer. J. Hum. Genet., **18:** 21-25.
SALZANO F. M. (1963). Blood groups and gene flow in Negroes from Southern Brazil. Acta Genet. Basel, **13:** 9-20.
— HIRSCHFELD J. (1965). The dynamics of the Gc polymorphism in a Brazilian population. Acta Genet. Basel, **15:** 116-125.
— LEWGOY F., TONDO C. V., DA ROCHA F. J. (1967). G-6-PD deficiency and abnormal hemoglobins in a Brazilian population. A.Ge.Me.Ge. (In press).
SUAREZ R. M., OLAVARRIETA S., BUSO R., MEYER L. M., SUAREZ R. M. Jr. (1961). G-6-PD deficiency among certain Puerto Rican groups. Bol. Asoc. Med. Puerto Rico, **53:** 41-48.
SUTTON R. N. P. (1963). Erythrocyte G-6-PD deficiency in Trinidad. Lancet, **1:** 855.
TONDO C. V., SALZANO F. M. (1962). Abnormal hemoglobins in a Brazilian Negro population. Amer. J. Hum. Genet., **14:** 401-409.
— MUNDT C., SALZANO F. M. (1963). Haptoglobin types in Brazilian Negroes. Ann. Hum. Genet., **26:** 325-331.
VAN DER SAR A., SCHOUTEN H., STRUYER BOUDIER A. M. (1964). G-6-PD deficiency in red cells. Incidence in the Curaçao population, its clinical and genetic aspects. Enzymologia, **27:** 289-310.
WORKMAN P. L., BLUMBERG B. S., COOPER A. J. (1963). Selection, gene migration and polymorphic stability in a U. S. White and Negro population. Amer. J. Hum. Genet., **15:** 429-437.

RIASSUNTO

Sono descritti gli studi sulla attività della glucosio-6-fosfato deidrogenasi eritrocitaria di 416 uomini e 820 donne. Il numero totale degli uomini deficienti (attività intermedia + deficienza completa) varia da 3.9% tra i bianchi (102 persone) a 15.1% tra i mulatti scuri (99 individui); l'incidenza nel campione negroide in generale (316 persone) è del 12.4%. I dati ottenuti nelle donne sono stati utilizzati per studiare i problemi sulla penetranza dei geni per la G-6-PDD. I risultati ottenuti in entrambi i sessi sono stati usati per verificare (senza successo) possibili differenze selettive attraverso l'analisi dell'età e paragonando le incidenze geniche in persone colpite da malattie non gravi e controlli. Sono discussi i problemi dell'analisi della deriva genetica che sorgono in popolazioni come questa, in cui sono presenti le forme « Nera » e « Bianca » di tale deficienza.

RÉSUMÉ

L'Auteur présente une étude sur l'activité de la glucose-6-phosphate deshydrogénase des érythrocytes de 416 hommes et 820 femmes. Le nombre total d'hommes avec déficience (activité partielle + déficience totale) a varié de 3.9% pour les blancs (102 individus) à 15.1% pour les mulâtres foncés (99 individus); le pourcentage dans l'échantillon noir en général (316 individus) fut du 12.4%. Les résultats obtenus pour les femmes ont été utilisés pour étudier les problèmes de pénétrance des gènes pour la G-6-PDD. Les résultats obtenus pour les deux sexes ont été utilisés (sans succès) pour montrer les possibles différences de sélection par l'analyse des âges et par la comparaison du pourcentage des gènes entre les personnes atteintes de maladies sans gravité et les témoins. On a discuté les problèmes de l'analyse du flux génique qui surgit dans une telle population dans laquelle sont présentes les formes « blanche » et « noire » de cette déficience.

ZUSAMMENFASSUNG

Verf. beschreiben Beobachtungen der Wirkung von Glukose-6-Phosphat Deshydrogenase der Erythrocyten an 416 Männern und 820 Frauen. Die Zahl der Männer mit ungenügender Aktivität (mittlere + ungenügende Wirkungen) schwankte zwischen 3.9% bei Weissen (102 Personen) und 15.1% bei dunklen Mulatten (99 Individuen); das Vorkommen bei Negern war generell 12.4%. Die bei Frauen erhaltenen Angaben wurden benutzt, um das Problem der Penetranz von G-6-PDD Genen zu untersuchen. Dann verwendeten Verf. die Resultate beider Geschlechter um, leider erfolglos, die möglichen Unterschiede der Selektion durch Analyse des Alters und Vergleich des genetischen Vorkommens bei Leicht-Kranken und Kontrollen nachzuprüfen. Im Vordergrund standen die Probleme des genetischen Flusses, die bei Populationen, wie dieser, auftauchen und bei welchen die Formen ungenügender Funktion der « Neger » und der « Weissen » zusammentreffen.

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