

Additional Data on PGP Genetic Transmission in Family Groups

B. Caeiro and T. A. Varela

Dept. of Anthropology, Faculty of Biology, University of Galicia, Santiago de Compostela, Spain

Summary. PGP (phosphoglycolate phosphatase, EC 3.1.3.18.) genetic studies were performed in 188 families including a total of 415 offspring in the Galician population. The results are in agreement with the formal hypothesis of three codominant alleles at an autosomal locus. No silent alleles have been observed.

Key word: Phosphoglycolate phosphatase, genetic study

Zusammenfassung. Genetische Studien der PGP (Phosphoglykolat-Phosphatase EC 3.1.3.18.) wurden an 188 Familien der Bevölkerung Galiziens durchgeführt, welche eine Gesamtzahl von 415 Kindern aufwiesen. Die Resultate stehen im Einklang mit der Hypothese der Existenz dreier Allele an einem autosomalen Locus.

Schlüsselwort: Phosphoglykolat-Phosphatase, genetische Studie

Introduction

The enzyme phosphoglycolate phosphatase (PGP EC 3.1.3.18.) was initially detected in human red cells by Badwey in 1977. Further studies have shown that the enzyme represents an electrophoretic polymorphism genetically controlled (Barker and Hopkinson 1978), due to the existence of an autosomal locus presumably located on chromosome 16 (Povey et al. 1980).

The aim of this work is to provide additional data on PGP genetic transmission in familial groups in regard of the small number of families hitherto analyzed and because of the great value of genetic markers for paternity testing.

Materials and Methods

One hundred eighty-eight unrelated families including a total of 415 offspring from western Galicia were tested. Blood cells were washed with 0.9% saline and stored at -20° C for up to 2 months. Hemolysates were obtained by freezing and thawing of erythrocytes in three consecutive cycles.

Offprint requests to: Dr. B. Caeiro (address see above)

Table 1. Distribution of PGP phenotypes in 188 families from Galicia. Expected values are put in parentheses

Matings	Families	Children	Phenotypes of children	f children						İ
	No.	No.	1-1	2-1	3-1	2-2	3-3	3-2	DF	χ^{5}
I-1 × I-1	130	290	290 (290.0)	1	i	1	I	I	1	1
$2-1 \times 1-1$	32	72	33 (36.0)	39 (36.0)	1	ļ	ļ	ı	ч	0.5
3-1 × I-1	17	38	21 (19.0)	.	17 (19.0)	1	1	1	-	0.42
$2-1 \times 2-1$	4	6	1 (2.25)	5 (4.50)		3 (2.25)	l	I	2	1.00
$2-1 \times 3-1$	1	2	(0.5)	(0.5)	1 (0.5)	,	ļ	0 (0.5)	en	1.50
3-1×3-1	Н	4	(1.00)	.	(2.00)	1	2 (1.00)	.	2	2.00
Total	188	415	346	44	20	3	2	0		

Table 2, Pooled PGP family data from three publications: Barker and Hopkinson (1978), Amorim et al. (1980), and this study. Expected values are put

Matings	Families	Children	Phenotypes	Phenotypes of children						
3	No.	No.	1-1	2-1	3-1	2-2	3-3	3-2	DF	χ^2
$1-1 \times 1-1$	236	578	578 (578)	l	ļ			I	1	
$1-1 \times 2-1$	69	174	88 (87)	86 (87)	1	1	1	I	-	0.02
$1-1 \times 2-2$	'n	10	1	10 (10)	1		I	I		1
$1-1 \times 3-1$	40	68	50 (44.5)	Ì	39 (44.5)		1	1	1	1.36
2-1×2-1	6	21	2 (5.25)	11 (10.5)	1	8 (5.25)	.		2	3.47
3-1×3-1	4	oc	(2) 22	1	4 (4)	I	6 (2)		2	0.00
1-1×3-2	ī	7	ì	4 (3.5)	3 (3.5)			l	Ţ	0.14
$2-1 \times 3-1$	4	10	4 (2.5)	1 (2.5)	4 (2.5)	1		1 (2.5)	m	1.80
Total	368	768	724	112	50	8	2			

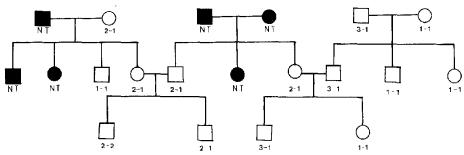


Fig. 1. Segregation of PGP phenotypes from three families carrying the three alleles. NT = not tested

Typing of hemolysates was made with horizontal starch gel electrophoresis for 18 h at 4 V/cm, Staining was carried out according to Barker and Hopkinson (1978) with modifications.

Results and Discussion

Table 1 shows the segregation of PGP phenotypes in 188 families.

The analysis of the results indicates that the phenotypes are due to the occurrence of three codominant alleles (PGP¹, PGP², PGP³) at an autosomal locus. This is in agreement with the hypothesis previously established (Amorim et al. 1980; Barker and Hopkinson 1978; Siebert et al. 1980) since there are no exceptions to this hypothesis from all the offspring examined, and the segregation ratios are in keeping with Mendelian expectations (Table 2).

Figure 1 represents the genetic transmission of the three PGP alleles in three family groups.

Chances of excluding paternity using PGP in the Galician population were 0.0684 (Rey, pers. communic.). No silent allele has been detected in any previous study. However, this does not indicate its absence because a high percentage (nearly 70%) of parents examined displayed homozygous phenotype combinations.

References

Amorim A, Siebert G, Ritter H, Kömpf J (1980) Formal genetics of phosphoglycolate phosphatase (PGP): Investigation on 272 mother-child pairs. Hum Genet 53:419-420

Badwey JA (1977) Phosphoglycolate phosphatase in human erythrocytes. J Biol Chem 252: 2441-2443

Barker RF, Hopkinson DA (1978) Genetic polymorphism of human phosphoglycolate phosphatase (PGP). Ann Hum Genet 42:143-151

Povey S, Jeremiah SJ, Barker RF, Hopkinson DA, Robson EB, Cook PJL, Solomon E, Bobrow M, Carrit B, Buckton KE (1980) Assignment of the human locus determining phosphoglycolate phosphatase (PGP) to chromosome 16. Ann Hum Genet 43:241-248

Siebert G, Amorim A, Kömpf J (1980) Human phosphoglycolate phosphatase (PGP) EC 3.1.3.18. Analysis. Hum Genet 53:412-423