Additional Data on PGP (EC 3.1.3.18) Polymorphism in Düsseldorf

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SUMMARY

Blood samples from 1582 unrelated German individuals in the Düsseldorf area were studied for phosphoglycolate phosphatase polymorphism. The number of the observed phenotypes did not diverge from the expected values (Hardy-Weinberg).
The gene frequencies were calculated as: PGP^1 .8606 PGP^2 .0964 PGP^3 .0424 . The assumed autosomal codominant mode of inheritance was confirmed by the analysis of 688 mother child data, including 501 "critical" mother child pairs. The assumption that there is a lower PGP^3 frequency in the Düsseldorf area could not be confirmed.
Several other PGP studies are discussed and compared.

INTRODUCTION

Phosphoglycolate phosphatase (PGP) was first described in plant tissues (*Richardson and Tolbert, 1961; Randall, Tolbert and Gremel, 1971*).
In 1977 *Badwey* described the enzyme activity in human erythrocytes. It remains conceivable that PGP activity plays some part in the regulation of oxygen transfer to and from haemoglobin (*Rose, 1976; Rose and Liebowitz, 1970*).
There is no evidence to suggest that different electrophoretic variants of PGP are associated with different levels of activity but day-to-day variation in PGP activity even in the same individual has been reported (*Turner and Hopkinson, 1981*).
The results of several authors indicate the localization of the gene for human PGP on the chromosome 16 (*Weil et al., 1979; Povey et al., 1980; Gal et al., 1986*).
Six phenotypes - due to three alleles PGP^1, PGP^2 and PGP^3 - can be separated by electrophoresis. A rare allele was described as $PGP^{Sumatra}$ (*Henke et al., 1985*). *Weber* reported about a "silent" allele PGP^0.
Close linkage between PGP and the markers ABO, acP, ADA, PGM_1, PGM_3, GLO and HLA could be ruled out. Close linkage to other markers could not be proved (*Siebert et al., 1980*)(References see 3).

MATERIALS AND METHODS

Blood samples of 1582 unrelated and apparently healthy Germans living in the Düsseldorf area were investigated. 688 mother child pairs were analysed.

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The PGP phenotypes were demonstrated by means of starch gel electrophoresis. The method was described by *Barker and Hopkinson, Martin et al.* (References see 3).

RESULTS

The patterns of PGP seem to be more indistinct than zymograms of other enzymes - similiar to the isozyme patterns of GPT after starch gel electrophoresis. However, a reliable determination is possible.
Table 1 shows the distribution of phenotypes in our study. We found a good agreement between the observed and expected values according to the *Hardy-Weinberg* law, the gene frequencies were calculated as: PGP^1 .8606 PGP^2 .0964 and PGP^3 .0424 .We analysed the distribution of phenotypes in 688 mother child pairs. As expected we did not find any mother child exclusion. This study includes 502 "critical" mother child pairs (Tab.2)
We combined this study and the study of *Henke et al.* (2). Now the combined studies on PGP polymorphism in Düsseldorf include 2145 individuals
Combining 9 studies on PGP polymorphism in Germany the combined studies include 10524 individuals and show the distribution of phenotypes in Germany. There is a good agreement between observed and expected values. The combined gene frequencies were calculated as: PGP^1 .8551 PGP^2 .1059 and PGP^3 .0390.

DISCUSSION

Though the PGP typing by means of starch gel electrophoresis may not have been optimal there are no difficulties for a proficient examiner to distinguish the different phenotypes. But it is absolutely necessary to use a wellknown PGP^{1-3} as control .
When adding the studies of other authors data of 10524 examined individuals have been published in Germany. Because of its AVACH-value (more than 12 %) PGP is a useful marker in paternity testing
Our investigation of 688 mother child pairs agrees with the data reported by other authors (Referances see 3). Thus our results confirm the formal hypothesis, that PGP polymorphism is controlled by three codominant alleles at an autosomal locus. The analysis of the "critical" mother child pairs did not show any deviation from *Mendelian* proportions - such as reported by other authors. Only in one case a "silent" allele was found (*Weber*, 1986).
Henke (2) reported about a low frequency of PGP^3 in Düsseldorf, significantly lower than in Bonn (1). We cannot support this suggestion. The PGP^3 frequency reported by *Brink* et al. is high - possibly due to the small number of investigated cases - but the PGP frequencies in Düsseldorf are very similiar to those in other countries of Germany.

Table 1 *PHENOTYPE AND GENE FREQUENCIES OF PGP IN THE DÜSSELDORF POPULATION*

	observed		*expected*	
Phenotype	*n*	*%*	*n*	*%*
PGP ^{1}	1172	74.08	1171.7	74.07
PGP $^{2-1}$	261	16.50	262.5	16.59
PGP ^{2}	18	1.14	14,7	.93
PGP $^{3-2}$	8	.51	13.1	.83
PGP $^{3-1}$	118	7.46	117.0	7.40
PGP ^{3}	5	.32	2.9	.18
total	1582	100.01	1581.9	100.00

GENE FREQUENCIES: PGP^{1} .8606 PGP^{2} .0964 PGP^{3} .0424

Table 2 *PGP PHENOTYPES IN 688 MOTHER-CHILD PAIRS IN DÜSSELDORF*

	Child						
Mother	*1*	*2-1*	*2*	*3-2*	*3-1*	*3*	*n*
1	431	44			16		491
2-1	50	57	6	3	2		118
2		10					10
3-2		1			5		6
3-1	27	5		2	28		62
3					1		1

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