

ERYTHROCYTE GLYOXALASE I POLYMORPHISM IN AN AFRICAN AND ENGLISH POPULATION

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1. Introduction

The glyoxalase system [1,2] comprises two enzymes: glyoxalase I, which catalyses the combination of methyl glyoxal with reduced glutathione (GSH) to form an addition compound; and glyoxalase II, which brings about the breakdown of the addition compound to give lactic acid and GSH [3]. One consequence of the functioning of glyoxalase I is the uptake of GSH, and since in human erythrocytes glyoxalase II (which reverses this) is absent [4], observing the GSH disappearance is a useful way of identifying glyoxalase I in this tissue.

Recently human erythrocyte lysates have been subjected to electrophoresis on starch gels and the glyoxalase I identified by overlaying the gel with reagents that produce a colour with GSH and an achromic region where the presence of glyoxalase I has caused the GSH to be taken up into an addition compound with methyl glyoxal [5,6]. Three common polymorphic phenotypes of glyoxalase I were found in Germany [5] and England [6]: a 'fast' phenotype in which a single fast migrating enzymic component was seen; a 'slow' type in which a single slow enzymic band was observed; and an 'intermediate' type in which three isoenzymes were seen, fast, slow, and a third isoenzyme of intermediate electrophoretic mobility. The three variant phenotypes were shown to be autosomally inherited in a simple two-allele codominant manner. The fast and slow phenotypes are the homozygous individuals and the intermediate phenotypes are the heterozygotes. The frequency of the three common phenotypes was found to be similar in the German and English populations.

We have recently had an opportunity to examine

blood samples from a Gambian population, and this communication reports a significant difference in the frequency of the glyoxalase I phenotypes between this African population and Caucasians. It is suggested that glyoxalase I may be a useful additional marker in the field of human population genetics.

2. Methods

The erythrocytes from whole blood, prevented from clotting with EDTA, were washed with saline and lysed by sonication in four volumes of water. Frozen packed red cells from The Gambia were treated with four volumes of 0.1 M mercaptoethanol (which improved the electrophoretic resolution) and sonicated. The clear haemolysates obtained after centrifugation were applied by means of filter paper inserts to starch gels made up in 0.0077 M phosphate buffer pH 6.6, and electrophoresis was carried out between buffer compartments of 0.2 M phosphate buffer pH 6.6 at a potential of about 9 V per cm gel length for 3 h. The general technique and equipment used was as described before for another enzyme [7]. The sliced gels were stained for glyoxalase I as previously described [5,6].

3. Results and discussion

200 samples from the village of Jali (The Gambia) and 200 from London were subjected to starch gel electrophoresis. The results are given in table 1, along with the data of Kömpf and Bissbort [8] on a population from South-West Germany.

150 of the London samples were assayed for red cell glyoxalase I activity. The 3 common phenotypes were found to have similar levels of enzyme activity.

Table 1

Population	Total Number			Phenotypes		Incidence of the allele determining:	
			Fast	Intermediate	Slow	'Fast'	'Slow'
Jali (Gambia)	200	No. observed	111	76	13	0.745	0.255
		No. expected	111.0	76.0	13.0		
London	200	No. observed	69	94	37	0.580	0.420
		No. expected	67.3	97.4	35.3		
Germany (S.W.)	655	No. observed	210	331	114	0.573	0.427
		No. expected	215.1	320.5	119.4		

Amongst the 200 individuals from Jali tested for red cell glyoxalase I by starch gel electrophoresis, 19 were found to be carriers of the gene for Sick Cell Haemoglobin (Hb AS phenotypes). It was noted however that whereas the frequency of Hb AS phenotypes in the Jali population as a whole was 9.5%, the frequency of Hb AS amongst those individuals of the glyoxalase 'slow' phenotype was 30.7%. The results are given in table 2. The significance of these findings will be investigated further.

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Table 2

	Total population	Glyoxalase I phenotypes		
		Fast	Intermediate	Slow
	200	111	76	13
Number of Hb AS	19	8	7	4
Frequency of AS	9.5%	7.2%	9.2%	30.7%