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Serogenetic Characterisation of the Bhil Tribe of Rajasthan

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ABSTRACT Baseline data are presented on A1A2BO Rhesus blood groups; PGM1, EsD, AK, ADA, GLO1, and ACP1 isozymes ; haptoglobin and haemoglobin variants investigated in an endogamous tribal group of Bhils from Banswara district of Rajasthan. Except for the haplotypes of Rhesus and the alleles of PGM1, ACP1 and ADA systems, distribution of alleles are in genetic equilibrium.

INTRODUCTION

The distribution of eight serogenetic marker systems *viz* : A1A2BO, Rhesus, PGM1, ESD, AK, ADA, GLO1, ACP1, HP and haemoglobin variants investigated in an endogamous tribal group of Bhils from Banswara district of Rajasthan is reported in the present paper. The Bhil constitute 39 per cent of the total tribal population of Rajasthan, a north-western state of India. The word "Bhil" is believed to have been derived from the Dravidian word "Bhilu" meaning bow. Bhils exhibit peculiar settlement pattern by living in widely spread hutments. They are found scattered throughout the state of Rajasthan with more concentration in the district of Banswara, Udaipur and Dungarpur. Majority of the Bhils believe in Hindu religion and are engaged in agricultural activities.

MATERIAL AND METHODS

The data for the present study were collected at random from a total of 92 apparently healthy and not closely related individuals of either sex hailing from the Bhils of Banswara district, Rajasthan. Whole blood samples by venipunture were collected in EDTA-vials, kept in icebox and transported to the laboratory within 3-4 days.

Red cells were tested for A1A2B0 and Rhesus blood group systems using Orthodiagnostics, USA/Diamed, AG, Switzerland make anti-A, -B,-C,-C, -D, -E, - e sera, and anti-A1 and anti-H lectins following manufacturers' instructions and standard serological techniques.

The isozymes of PGM1 and EsD were typed together in agarose gel following the method of Wraxall and Stolorow (1986). Similarly, AK and ADA were typed together in agarose gel following Murch et al. (1986).

ACP1 isozymes were typed as described by Wraxal and Emes (1976), but in agarose gel. For GLO1 typing mixed agarose-starch gel technique of Scott and Fowler (1982) was used, and before staining Haemoglobin pattern was observed on the cathodal portion of the gel. Hp typing was done in thick starch gel according to Smithies (1955).

The allelic/haplotype frequency computation were done using formulae given by Mourant et al. (1976) except for the A1A2B0 system for which the formulae derived by Yasuda (1984) were used.

RESULTS AND DISCUSSION

Phenotypes and allele/haplotype frequency data are presented in table 1. Alleles *O*, *B*, *A1* and *A2* among the Bhils of present study occur with frequencies of 60.2%, 23.1% 14% and 2.7%, respectively. Among the Bhils of Panchmahal in the adjoining state of Gujarat these frequencies were reported to be 53.25%, 25.64%, 20.32% and 0.79%, respectively (Vyas et al., 1962). Thus, in comparision to the Bhil of Gujarat, the Bhil of Rajasthan are characterised by higher frequency of *O* and lower frequency of *A* allele. However, frequencies observed in the present study are very close to the average frequencies of *O*,*B*,*A1* and *A2* alleles computed for the pooled data of north Indian populations (Kushwaha and Chahal, 1994) and are within the

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Table 1: Phenotype and allele/haplotype frequencies of serogenetic marker systems investigated among Bhil tribe of Banswara district, Rajasthan

System/Phenotype	*Observed Number*	*Expected Number*	*Allele/Haplotype Frequency*	χ^2 *value*
A1A2BO				
O	33	33.314		
A1	18	18.039		
A2	3	3.029	*ABO* A1* = 0.140	
B	29	30.512	*ABO* A2* = 0.027	χ^2_3 = 3.12
A1B	6	5.968	*ABO* B* = 0.231	0.30>P>0.20
A2B	3	1.138	*ABO* O* = 0.602	
Total	92	92.000	1.000	
Rhesus				
CCDEE	3	0.161		
CCDEe	4	3.857		
CCDee	24	23.147		
CCddEE	1	0.000		
CcDEE	2	1.517		
CcDEe	13	20.210		
CcDee	21	23.940	*CDE* = 0.0418	χ^2_6 = 58.2
ccDEE	2	3.581	*CDe* = 0.5016	P<0.01
ccDEe	11	9.411	*cDE* =0.1973	
ccDee	8	4.490	*cDe* = 0.1239	
ccddee	3	1.686	*cde* = 0.1354	
Total	92	92.000	1.0000	
Acid Phosphatase (ACP1)				
ACP A	10	5.444		
ACP AB	22	31.111	*ACP 1*A* = 0.259	χ^2_1 = 6.947
ACP B	49	44.445	*ACP 1*B* = 0.741	0.05 > P > 0.02
Total	81	81.000	1.000	
Adenylate Kinase (AK)				
AK 1	64	64.0		
AK 1-2	16	16.0	*AK * 1* = 0.889	χ^2_1 = 0.00
AK 2	1	1.0	*AK * 2* = 0.111	P > 0.99
Total	81	81.0	1.000	
Adenosine Deaminase (ADA)				
ADA 1	55	51.690		
ADA 1-2	21	27.620	*ADA * 1* = 0.789	χ^2_1 = 4.77
ADA 2	7	3.690	*ADA * 2* = 0.211	0.05 > P > 0.02
Total	83	83.000	1.000	
Esterase D (ESD)				
ESD 1	52	51.525		
ESD 1-2	26	26.951	*ESD * 1* = 0.793	χ^2_1 = 0.102
ESD 2	4	3.524	*ESD * 2* = 0.207	0.80 > P > 0.70
Total	82	82.000	1.000	
Glyoxalse I (GLO 1)				
GLO 1 1	7	8.028		
GLO1 1-2	37	34.944	*GLO 1*1* = 0.315	χ^2_1 0.280
GLO1 2	37	38.028	*GLO 1*2* = 0.685	0.70 > P > 0.50
Total	81	81.000	1.000	
Phosphoglucomutase 1 (PGM1)				
PGM 1 1	51	46.695		
PGM 1 1-2	21	29.611	*PGM 1*1* = 0.759	χ^2_1 = 6.85
PGM 1 2	9	4.694	*PGM 1 * 2* = 0.241	P < 0.01
Total	81	81.000	1.000	

Table 1: Contd.....

System/Phenotype	*Observed Number*	*Expected Number*	*Allele/Haplotype Frequency*	χ^2 *value*
Haptoglobin (HP)				
HP 1	6	4.396		
HP 1-2	28	31.209	*HP * 1* = 0.220	χ^2_1 = 0.962
HP 2	57	55.395	*HP * 2* = 0.780	0.50>P>0.30
Total	91	91.000	1.000	
Haemoglobin (Hb)				
AA	92	1.000	*Hb* A* = 1.000	
Total	92	1.000	1.000	

ranges reported for various Indian population groups (Bhasin et al., 1992).

In Rhesus system (D,C,c,E,e) 11 phenotypes were detected out of the 18 possible phenotypes. Haplotype *CDe* occurs with highest frequency of 50.16% followed by *cDE* (19.73%) against 67.3% and 1.4%, respectively reported for the Bhil of Gujarat (Vyas et al., 1962). Frequency of *cDe* in present study and among the Bhil of Gujarat are quite close, 12.39% and 12%, respectively. Frequencies of cde among the Bhil of Rajasthan and Gujarat are 13.54% and 13.3% and that of *CDE* is 4.18% and 2.3%, respectively. The frequency of all the haplotypes detected in the present study are within the general ranges reported for north Inidna populations (Bhasin et al., 1992).

In the ACP1 system, only three phenotypes were detected. Allele *ACP*1* occurs with the frequency of 25.9% against 21.65% reported for the Bhil from Bombay (Mukherjee et al., 1979) and 19.9% for the Bhil from Jhabua district of Madhya Pradesh (Papiha et al., 1978), and is very close to the frequency (24.7%) computed for north Indian populations (Kushwaha and Chahal, 1994). Allele *C* is absent in the present as well as in the two other samples of Bhil tribe investigated so far.

Frequency of *AK*2* allele among the Bhil of present study is 11.1% against 10% reported for Bhil of Udaipur district of Rajasthan (Papiha et al., 1982), 4.1% for the Bhil of Madhya Pradesh (Papiha et al., 1978) and the average frequency (7.5%) for north Indian populations (Kushwaha and Chahal, 1994).

In the present study *ADA*2* allele is 21.1% against 11% among the Bhil of Udaipur district of Rajasthan (Papiha et al., 1982), 7.9% in the Bhil of Madhya Pradesh (Papiha et al., 1978) and average frequency of 12% reported for north Indian populations (Kushwaha and Chahal, 1994).

*ESD*2* allele in present study is 20.7% whereas among the Bhil of Udaipur it was reported 34% (Papiha et al., 1982), for the Bhil of Maharashtra 39.3% (Mukherjee et al., 1979) and for the Bhil of Madhya Pradesh 26.2% (Papiha et al., 1978). The average frequency being 26.1% in north Indian populations (Kushwaha and Chahal, 1994).

Frequency of *GLO*1* allele is 31.5% against the mean frequency of 26.35% in north Indian populations (Kushwaha and Chahal, 1994).

Among the Bhil of present study the *PGM*2* allele is 24.1% against 29% in the Bhil of Udaipur (Papiha et al., 1982) and 29.6% among the Bhil of Madhya Pradesh (Papiha et al., 1978), and the mean frequency of 29.4% in north Indian populations (Kushwaha and Chahal, 1994).

*HP*1* allele occurs with 22% frequency against 17.5% in the Bhil of Bombay (Mukherjee et al., 1979) and 10.3% in Bhil of Madhya Pradesh (Papiha et al., 1978) and the average frequency of 16.7% in north Indian populations (Kushwaha and Chahal, 1994). As for Hb pattern, all the samples were found to be AA and no variant was detected

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