TECHNICAL NOTE

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Distributions of Genetic Markers in a Nebraska Population

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ABSTRACT: Seven genetic marker systems were analyzed from liquid blood and dried bloodstain specimens submitted to the Nebraska State Patrol Crime Laboratory from various law enforcement agencies throughout Nebraska. The phenotypic and genotypic frequencies for the ABO, Lewis, esterase D (ESD), phosphoglucomutase (PGM), adenylate kinase (AK), adenosine deaminase (ADA), and haptoglobin (HP) systems were calculated. The results indicate that the phenotypic frequencies are generally in agreement with frequencies reported in other populations in the United States.

KEYWORDS: pathology and biology, genetic typing, blood, genetic marker typing, ABO blood group, Lewis blood group, phosphoglucomutase, esterase D, adenylate kinase, adenosine deaminase, haptoglobin

Genetic marker typing in blood and bloodstains, and in body fluids and body fluid stains, is employed in forensic serology laboratories for the partial individualization or exclusion of suspected depositors of stains. In order to provide investigators and courts with information about the relative occurrence of a genetic marker type or set of types, population frequency data must be available.

Genetic marker frequency data are available for a number of populations throughout the world [*1*] and in the United States [*2–4*]. It is, nevertheless, of value to have frequency data for a population that is served by a forensic science laboratory. These data add to the overall population database, enable comparison to be made with other populations in the country, and may reflect local frequencies better than data from other locations.

Frequencies for the ABO, Lewis, phosphoglucomutase (PGM), esterase D (ESD), adenylate kinase (AK), adenosine deaminase (ADA), and haptoglobin (HP) systems are reported here for a Nebraska population sample drawn from the casework of a forensic science laboratory.

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Materials and Methods

Liquid blood and bloodstains were obtained from samples submitted to the crime laboratory by various law enforcement agencies throughout all geographic regions of Nebraska. All seven marker systems were studied from casework samples (primarily from victims and suspects).

The ABO blood group was typed in whole blood by the standard slide method. The absorption-elution technique of Howard and Martin was used for grouping the ABO types from dried bloodstains [*5*]. The enzymes and the serum protein were analyzed by various techniques of electrophoresis [*6–11*]. Lewis typing was done by the Chown capillary method [*12*].

The gene frequencies were calculated by gene counting, except in the case of the ABO types, where the maximum likelihood estimation method described by Stevens was used [*13*]. Gene frequencies were not calculated for the Lewis system, as the data represent red cell Lewis types [*1*].

TABLE 1—*Frequencies of genetic markers in a Nebraska population.*

System	*n*	Phenotype	Phenotypic Frequency, *n* (%)	Gene Frequency	
ABO	1760	A	706 (40.1)	*ABO*A*	0.2477
		B	215 (12.2)	*ABO*B*	0.0804
		O	783 (44.5)	*ABO*O*	0.6719
		AB	56 (3.2)		
Lewis	818	a+b−	171 (20.9)		
		a−b+	553 (67.6)		
		a−b−	94 (11.5)		
PGM	397	1+	160 (40.3)		
		1+1−	63 (15.9)		
		1−	8 (2.0)	*PGM*1+*	0.6436
		2+1+	100 (25.2)		
		2+1−	13 (3.3)	*PGM*1−*	0.1222
		2−1+	28 (7.1)		
		2−1−	5 (1.3)	*PGM*2+*	0.1751
		2+	9 (2.3)		
		2+2−	8 (2.0)	*PGM*2−*	0.0592
		2−	3 (0.8)		
ESD	1583	1	1249 (78.9)	*ESD*1*	0.8882
		2−1	314 (19.8)	*ESD*2*	0.1118
		2	20 (1.3)		
AK	1139	1	1059 (93.0)	*AK*1*	0.9649
		2−1	80 (7.0)	*AK*2*	0.0351
		2	0 (0.0)		
ADA	1101	1	1015 (92.2)	*ADA*1*	0.9609
		2−1	86 (7.8)	*ADA*2*	0.0391
		2	0 (0.0)		
HP[a]	1052	1	185 (17.6)	*HP*1*	0.4368
		2−1	543 (51.6)	*HP*2*	0.5632
		2	318 (30.2)		
		2−1M[b]	6 (0.57)		

[a] α-chain.
[b] 2−1M combined with 2−1 in gene frequency.

Information about the race of origin of the donors was not available for most of the specimens used in this study.

Results and Discussion

Table 1 shows the distribution of genetic markers in the Nebraska population sample drawn from casework material, as described above. The phenotypic frequencies in the systems reported are generally in fairly good agreement with frequencies reported in other U.S. populations. The data were tested for deviation from Hardy-Weinberg equilibrium expectations using the chi-square goodness-of-fit test. Significant deviation from expectation was observed in the ABO data ($\chi^2 = 4.026$, $0.02 < P < 0.05$). The greatest contribution of χ^2 is attributable, however, to Group AB, the smallest class. The deviation in the HP system data was barely significant. The fact that the sample was racially mixed could explain minor deviations from equilibrium expectation in systems in which there may be significant differences in some phenotypes in different racial groups. For the most part, the frequencies resemble those of other U.S. Caucasian populations. Nebraska's population is about 95% Caucasian according to the 1980 census [*14*]. It is not known, however, how closely the population sample studied reflects the racial makeup of the general population.

ABO typing of a subsample of 442 people known to be Caucasian showed that 184 of them (41.6%) were Type A, 46 (10.4%) Type B, 201 (45.5%) Type O, and 11 (2.5%) Type AB, and the gene frequencies were 0.2533, 0.0669, and 0.6798 for Types *ABO*A*, *ABO*B*, and *ABO*O*, respectively. A subsample of 217 known Caucasians analyzed for HP type showed that 34 (15.7%) were Type 1, 107 (49.3%) Type 2-1, and 76 (35.0%) Type 2. The gene frequency of *HP*1* was 0.4032 and that of *HP*2* was 0.5968. The frequencies in both systems in these subsamples are well within Hardy-Weinberg equilibrium expectations.

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