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The LH-Specificity in Diabetes Mellitus : Further Observations

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ABSTRACT Blood Samples from confirmed cases of diabetes mellitus (n=571) were typed both for the ABO blood groups and the LH specificity, and compared with an appropriate control. The results indicated that as against normal individuals, the patients were overwhelmingly LH-negative ($p<0.001$) and that this difference is particularly noticeable in patients with the blood groups A and B. The implications of these results have been discussed.

INTRODUCTION

Shrivastava et al. (1979) discovered a new red blood cell membrane specificity in the seeds of *Erythrina lithosperma*, a plant belonging to the family Leguminosae. The name LH was given to this specificity to cherish the memory of Ludwik Hirszfeld, the celebrated Polish serologist. Further studies relating to the immunochemical properties of the *Erythrina lithosperma* lectin and genetics and population distribution of the LH specificity have been made by Shrivastava et al. (1979), Sehajpal and Shrivastava (1980, 1981), Reddy et al. (1981), Kaur (1983), Koley et al. (1991, 1992) and Koley and Shrivastava (1992).

The anti-LH lectin reacts with human red cells by either clumping them firmly or just weakly agglutinating them. The former type of reaction is called LH-positive and the latter, LH-negative. Since the anti-LH lectin is inhibited by some sugars (Shrivastava et al., 1979) the difference between LH-positive and LH-negative types of reaction patterns may depend upon the distribution of the cellular lectin binding receptors, similar or identical structurally to those carbohydrates. Thus it was thought that in variously glycosylated red blood cells from patients with diabetes mellitus there should be fewer receptors available on the cell surface to bind the lectin molecules and as consequence there should be more LH-negative individuals among patients with diabetes mellitus than in the normal population.

MATERIAL AND METHODS

A total of 571 blood samples of patients with diabetes mellitus (type-II) were obtained from the Varni Pathology Clinic, Sagar (Madhya Pradesh), India; 526 unrelated normal healthy individuals sampled randomly from this area acted as controls. Since sex differences are not known to exist in the LH system, the samples collected from both males and females were pooled for all analyses.

For the ABO typing, standard serological procedures were followed. The LH typing was done with anti-LH lectin prepared in our own laboratory as described by Shrivastava et al. (1979). *Exythrina lithosperma* seeds were obtained from Botanical Survey of India, Calcutta.

RESULTS AND DISCUSSION

The phenotypic distribution of the LH-types and allele frequencies in patients with diabetes mellitus and controls are given in table 1. Patients have higher frequency (42.73%) in

LH-negative type than controls (30.99%). The difference between the patients and controls in regard to the LH-types is highly significant ($p < 0.001$). In patients, the allele frequency of LH–positive type is 0.7567 and in controls it is 0.8307. Both these populations are in Hardy-Weinberg equilibrium.

Table 2 shows the distribution of the ABO blood groups along with their allele frequencies in patients and controls. Patients have slightly higher frequencies in group A (27.50%) and in group B (37.65%) than their control counterpart, while in groups O and AB, patients are less frequent (25.57% and 9.28%, respectively) than controls. There exists no significant difference ($P > 0.05$) among them. Patients have higher *A* (0.2208) and *B* (0.2869) allele frequencies than controls. Both these populations are in Hardy-Weinberg equilibrium. The ABO group wise distribution of the LH–types in patients and controls is shown in table 3. Patients have higher frequencies in A/LH^- (18.74%) B/LH^- (17.86%) and AB/LH^- (6.13%) combinations than controls.

Table 4 shows the distribution of the LH-types with blood group A in patients and

Table 1: Distribution of the LH types and allele frequencies in patients with diabetes mellitus and controls

Sample	*n*		*Phenotype frequencies*		*Allele frequencies*		*Hardy-Weinberg equilibrium*
			LH^+	LH^-	LH^+	LH^-	
Patients	571	Obs. No.	327	244			
		Obs. %	57.27	42.73	0.7567	0.2433	0.0000
Controls	526	Obs. No.	363	163			
		Obs. %	69.01	30.99	0.8307	0.1693	0.0000

$\chi^2_{(1)}$ (O individuals excluded) = 17.778, $p < 0.001$

Table 2: Distribution of the ABO types and allele frequencies in patients with diabetes mellitus and controls.

Sample	*n*		*Phenotype frequencies*				*Allele frequencies*			*Hardy-Weinberg equilibrium*
			O	*A*	*B*	*AB*	*A*	*B*	*O*	
Patients	571	Obs. No.	146	157	215	53				
		Obs. %	25.57	27.50	37.65	9.28	0.2208	0.2869	0.4922	3.6706
Controls	526	Obs. No.	143	139	195	49				
		Obs %	27.19	26.43	37.07	9.32	0.2094	0.2784	0.5120	1.7382

$\chi^2_{(3)} = 0.412$, $p > 0.05$

Table 3: ABO group-wise distribution of the LH types in patients with diabetes mellitus and controls

Sample	*n*		*O*		*A*		*B*		*AB*	
			LH^+	LH^-	LH^+	LH^-	LH^+	LH^-	LH^+	LH^-
Patients	571	Obs. No.	146	—	50	107	113	102	18	35
		Obs. %	25.57	—	8.76	18.74	19.79	17.86	3.15	6.13
Controls	526	Obs. No.	143	—	72	67	129	66	19	30
		Obs %	27.19	—	13.69	12.74	24.53	12.55	3.61	5.70

Table 4: Distribution of the LH types in patients with diabetes mellitus and controls, with blood group A

Sample	*n*		*LH⁺*	*LH⁻*	*Hardy-Weinberg equilibrium*
Patients	157	Obs. No.	50	107	
		Obs.%	31.85	68.15	0.0000
Controls	139	Obs.No.	72	67	
		Obs. %	51.80	48.20	0.0000

$\chi^2_{(1)}$ 12.11, P<0.001

controls. There are more LH-negative individuals (68.15%) in patients than controls (48.20%) and the difference is highly significant (p <0.001), although both the populations are in Hardy-Weinberg equilibrium.

Distribution of the LH-types with blood group B in patients and controls is given in table 5. There are more LH-negative individuals in patients (47.44%) than controls (33.85%) and the difference is significant statistically (P<0.01) However, patients and control populations are in Hardy-Weinberg equilibrium.

Table 5: Distribution of the LH types in patients with diabetes mellitus and controls, with blood group B

Sample	*n*		*LH⁺*	*LH⁻*	*Hardy-Weinberg equilibrium*
Patients	215	Obs. No.	113	102	
		Obs. %	52.56	47.44	0.0000
Controls	195	Obs. No.	129	66	
		Obs %	66.15	33.85	0.0000

$\chi^2_{(1)}$ = 7.814, P <0.01

Table 6 : Distribution of the LH types in patients with diabetes mellitus and controls, with blood group AB

Sample	*n*		*LH⁺*	*LH⁻*	*Hardy-Weinberg equilibrium*
Patients	53	Obs. No.	18	35	
		Obs.%	33.96	66.04	0.0000
Controls	49	Obs. No.	19	30	
		Obs %	38.78	61.22	0.0000

$\chi^2_{(1)}$ = 0.255, P>0.05

The distribution of the LH-types with blood group AB in patients and controls is shown in table 6. Although the patients have more LH-negative individuals (66.04%) but the difference is not statistically significant (P>0.05). Both these populations, once again, are in Hardy-Weinberg equilibrium.

We generated our hypothesis on the assumption that excessive *in vivo* glycosylation of erythrocyte membrane should offer less number of attachment sites to the carbohydrate-rich reactive component of the anti-LH lectin than would normal erythrocytes, and therefore, in patients with diabetes mellitus, there should be more LH-negative individuals than in the normal population. The results indicate that as against normal individuals the patients with this disease are overwhelmingly LH-negative (P<0.001) and also that this difference is particularly noticeable in patients with the blood groups A and B. A significant preponderance of LH- negative type in diabetic patients, both with blood group A and with blood group B allows us to conclude that the LH system has more than a chance association with diabetes mellitus. We did not consider O individuals since they were all LH- positive. The AB cells react weakly with the anti-LH lectin in the order $O > A_2 > B > A_1 > AB$, and this might explain why we did not find any significant difference in the distribution of the LH types in patients and controls with blood group AB.

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REFERENCES

Kaur, K.: *The LH Red Blood Cell Membrane Specificity in Man and Selected Animals*. Ph.D. Thesis (Unpublished), Punjabi University, Patiala (1983).

Koley, S., Jain, R. and Shrivastava, P.K.: The LH-specificity in diabetes mellitus. pp 183-187. In: *New Horizons in Human Biology*. I.J.S. Bansal et al. (Eds.). Today and Tomorrow's Printers & Publishers, New Delhi (1991).

Koley, S. and Shrivastava, P.K.: The distribution of the ABO and LH blood groups in some central Indian populations. *J. Hum. Ecol., 3* : 239-242 (1992).

Koley, S., Jain, R. and Shrivstava, P.K.: Serum antibody titres against LH-positive and LH-negative red blood cells. *South Asian Anthropologist, 13:* 43-45 (1992).

Reddy, V.R., Naidu, G.P. and Shrivastava, P.K.: The LH red blood cell membrane specificity in three south Indian populations. *Acta Anthropogenet., : 5 :* 189-194 (1981).

Sehajpal, P.K. and Shrivastava, P.K.: Genetics of the LH red blood cell membrane specificity. *Acta Anthropogenet., 4 :* 85-88 (1980).

Sehajapal, P.K. and Shrivastava, P.K.: Distribution of LH types in some north Indian populations. *Acta Anthropogenet., 5* : 181-187 (1981).

Shrivastava, P.K., Sehajpal, P.K. and Kaur, H.: The LH system: A new red blood cell membrane specificity in man. *Acta Anthropogenet., 3 :* 83-92 (1979).