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Genetics of the LH-specificity: Further Observations II

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INTRODUCTION

A new red blood cell membrane specificity in the seed extracts of *Erythrina lithosperma*, a legume plant, was reported by Shrivastava et al. (1979) and was called LH by them. The name was given to this specificity to cherish the memory of Ludwik Hirszfeld, the celebrated Polish serologist. 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 (LH+) and the latter LH-nagative (LH-). Subsequent studies relating to the immunochemical properties of the anti-LH lectin, and genetics and distribution of LH phenotypes have been made by Shrivastava et al. (1979), Sehajpal and Shrivastava (1980), Reddy et al. (1981), Kaur (1983), Koley and Shrivastava (1992) and Koley et al. (1995).

Since LH-specificity has shown promise of being a useful serological tool we thought it fit to check its reported (Sehajpal and Shrivastava, 1980 and Koley and Shrivastava, 1992) pattern of inheritance in the Central Indian context, considering large number of families.

MATERIALS AND METHODS

A total of 828 blood samples from 199 families including 398 parents and 430 offspring (239 males and 191 females) were collected from five districts of Madhya Pradesh, India viz. Sagar, Tikamgarh, Damoh, Chattarpur and Panna in the year 1988 to 1991. Seeds of *Erythrina lithosperma* were obtained from Botanical Survey of India, Calcutta. For ABO typing standard serological procedures were followed. The LH typing was done with the anti-LH lectin prepared as described by Shrivastava et al. (1979).

RESULTS AND DISCUSSION

Table 1 shows the different parental mating combinations and the LH types of the children in this material. From this table it is clear that the LH types are distributed almost equally in the two sexes and if under genetic control the genes responsible for determination of the LH types must be borne on one of the autosomes. In the mating combination LH-positive x LH positive, all the offspring examined are LH-positive. In the mating combinations LH-positive x LH negative, the ratio between the two LH types among the offsprings is about 1:1. It suggests strongly that the majority of parents with the blood type LH-negative are heterozygotes. In the mating combinations, LH-negative x LH-negative, the distribution of LH types among offsprings once again suggests greater number of heterozygotes than homozygotes among parents.

Studying the pattern of transmission of the LH types in individual families, it can be stated firmly that the mode of inheritance is an autosomal dominant type, with the gene controlling the LH-negative phenotype clearly dominant over its allele responsible for the LH-positive type.

The pedigrees were analysed also for the transmission of the ABO blood type, as both the LH and the ABO systems seem to be associated.

As many as 23 parental mating combinations were recorded based on the ABO and LH types. These were represented among 430 offsprings in 199 families (table 2,3,4,). The analysis was made only to recheck the transmission of LH types in association with the transmission of the ABO blood types. The major outcome of this twin analysis is that in a relatively large number

Table 1: Parental mating combinations and LH types in offspring

Mating combination	*Number of families*	*Number of offspring*		*LH⁻*		*LH⁺*	
		Male	*Female*	*Male*	*Female*	*Male*	*Female*
$LH^+ \times LH^+$	78	98	65	98	65	-	-
$LH^- \times LH^-$	92	100	102	60	70	40	32
$LH^+ \times LH^-$	29	41	24	07	05	34	19
Total	199	239	191	165	140	74	51

Table 2: Parental mating combination LH⁺ x LH⁺ and LH types in offspring

Mating combination	*Number of families*	*Number of offspring*	*Offspring phenotype*
O/LH⁺ x O/LH⁺	13	29	O/LH⁺ = 29
O/LH⁺ x A/LH⁺	05	08	B/LH⁺ = 5; A/LH⁺= 3
O/LH⁺ x B/LH⁺	32	65	A/LH⁺ = 34; O/LH⁺=31
O/LH⁺ x AB/LH⁺	01	02	A/LH⁺ = 1; B/LH⁺= 1
A/LH⁺ x A/LH⁺	02	04	A/LH⁺ = 2; O/LH⁺= 2
A/LH⁺ x B/LH⁺	06	13	A/LH⁺ = 7; B/LH⁺= 6
B/LH⁺ x B/LH⁺	17	38	B/LH⁺ = 31; O/LH⁺= 7
B/LH⁺ x B/LH⁺	02	04	A/LH⁺ = 3; AB/LH⁺= 1
Total	78	163	

Table 3: Parental mating combination LH⁺ x LH⁻ and LH types in offspring

Mating combination	*Number of families*	*Number of offspring*	*Offspring phenotype*
O/LH⁺ x A/LH⁻	22	49	A/LH⁻=25; O/LH⁺= 21; A/LH⁺=3
O/LH⁺ x B/LH⁻	15	30	O/LH⁺=15; B/LH⁺= 2;B/LH⁻=13
O/LH⁺ x B/LH⁻	07	15	A/LH⁻=5; B/LH⁻= 5;B/LH⁺=5
A/LH⁺ x AB/LH⁺	07	15	A/LH⁻ = 8; O/LH⁺= 5; A/LH⁺=2
A/LH⁺ x AB/LH⁻	02	04	A/LH⁻=3; AB/ LH⁺=1
B/LH⁺ x A/LH⁻	20	46	B/LH⁺=13; O/LH⁺=16; A/LH⁺=2 AB/LH⁺= 4; AB/LH⁻=11
B/LH⁺ x B/LH⁻	13	29	B/LH⁺=8; B/LH⁻= 14;O/LH⁺=7
B/LH⁺ x AB/LH⁻	05	13	B/LH⁺=7; AB/LH⁻= 5;B/LH⁻=1
AB/LH⁺ x A/LH⁻	01	01	A/LH⁻=1
Total	92	202	

of cases the analysis of LH typing will help greatly in the identification of the parental ABO genotypes.

In the conclusion of our observation on genetics of the LH-specificity, it may be noted that the LH-specificity is not age dependent; it is a permanent characteristic of an individual; it is not a sex linked trait; the gene responsible for the manifestation of the LH-negative type was dominant over its allele responsible for the determination of the LH+ type. Our experiments strongly support the findings of Sehajpal and Shrivastava (1980) and Koley and Shrivastava

Table 4: Parental mating combination LH⁻ x LH⁻ and LH types in offspring:

Mating	*Number of families*	*Number of offspring*	*Offspring phenotype*
A/LH⁻ x A/LH⁻	02	03	A/LH⁻ = 2; O/LH⁻ =1
A/LH⁻ x B/LH⁻	10	21	A/LH⁻ = 5; B/LH⁻ = 6 AB/LH⁻ = 4; O/LH⁻ = 6
A/LH⁻ x AB/LH⁻	02	05	A/LH⁻ = 4; B/LH⁻= 1
B/LH⁻ x B/LH⁻	08	18	B/LH⁻ = 14; O/LH⁻ = 4
B/LH⁻ x AB/LH⁻	06	14	B/LH⁻ = 10; AB/LH⁻=3 A/LH⁻ = 1
A/LH⁻ x B/LH⁻	01	04	A/LH⁻ = 3; B/LH⁻=1
Total	29	65	

(1992) regarding the genetics of the LH-specificity and this trait may be applied usefully in paternity determination and many other important field in immunogenetics.

KEY WORDS Inheritance. LH-specificity. Polymorphism.

ABSTRACT LH-specificity refers to weak or strong agglutination of erythrocytes obtained with the lectin from *Erythrina lithosperma*. Since the trait has shown promise of being a useful serological tool, an attempt was made to explore the genetics of this red cell membrane specificity. For this 199 families representing 398 parents and 430 offspring, from Madhya Pradesh, Central India were tested with the anti-LH lectin. Results suggested an autosomal dominant mode of inheritance for this trait. From pedigree analysis it was established that the genes responsible for the weak LH phenotype are dominant over its allele responsible for strong LH phenotype.

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