Z Rechtsmed 86, 129–132 (1981)

Zeitschrift für
Rechtsmedizin

The Forensic Application of the Blood Group Antigens Lu^a and Lu^b

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Summary. Blood samples from 507 unrelated persons in Northrhine-Westphalia and from 254 paternity cases were tested for the Lutheran blood group antigens Lu^a and Lu^b. The gene frequencies were found to be 0.03 ($=Lu^a$) and 0.969 ($=Lu^b$).

Key words: Red blood cell antigen system Lutheran, gene frequencies, plausibility to exclude non-fathers, Lutheran system

Zusammenfassung. Anhand einer Stichprobe ($n=507$) wurden die Genfrequenzen der Antigene Lu^a und Lu^b im Raum Düsseldorf ermittelt. Die Frequenz für Lu^a beträgt 0,03; die für Lu^b 0,969. Aspekte der Lagerungsstabilität (für Identitätsgutachten), der Vaterschaftsausschlußchance sowie der Vererbung werden erörtert.

Schlüsselwörter: Lutheran-System, Populationsgenetik, Vaterschaftsausschlußchance

The Lutheran blood group system was discovered in 1945, when the first example of anti-Lu^a was identified by Callender et al. [2]. The antithetical antibody was found in 1956 by Cutbush and Chanarian [6]. The system remained simple until it was realized that several other antigens appeared to be related to the Lutheran system. Many test series with anti-Lu^a on people in different parts of the world have been reported [8, 9, 11].

This paper aims at giving a report about the frequency of the genes Lu^a and Lu^b in Northrhine-Westphalia. Furthermore, remarks on the stability of the Lu^a antigen are given as well as the plausibility to exclude non-fathers from paternity.

Material and Methods

Probands

Five hundred seven unrelated and healthy individuals living in the Düsseldorf area were investigated. Two hundred fifty-four paternity cases were analyzed.

0044-3433/81/0086/0129/$ 1.00

Sera and Controls

The following sera were used:

Anti-Lu^a (111129)	Biotest-Serum-Institut, Frankfurt/Main
Anti-Lu^a (015711A)	Behringwerke AG, Marburg
Anti-Lu^b (2538)	Fresenius, Bad Homburg
Anti-Lu^b (002239)	Merz & Dade AG, Düdingen
Anti-Lu^b (111079)	Biotest-Serum-Institut, Frankfurt/Main

All sera worked well and reliably by following the particular advices of the distributors. Lu(a+b+) test cells were obtained from Behringwerke (Sangocell I). Lu(a−b−) test cells were contributed by Sheila Cornwall (Canadian Red Cross, Toronto) and by Dr. Mary N. Crawford (Villanova), (members of SCARF exchange group).

Results

Phenotype Frequencies ($n = 507$)

	Observed	Expected
Lu(a−b+)	476 (=93.89%)	476.5
Lu(a+b+)	31 (= 6.11%)	30.0
Lu(a+b−)	0	0.5
Lu(a−b−)	0	–
Gene Frequencies		
$Lu^a = 0.630$	$Lu^b = 0.969$	

The agreement between the observed and expected phenotypes is extraordinarily close ($\chi^2 = 0.53385$).

Studies of 254 Unselected Paternity Cases

a) Exclusion from Paternity

n	Child	Mother	Lover	Exclusions in other systems
61	Lu(a−)	Lu(a−)	Lu(a−)	yes
3	Lu(a+)	Lu(a−)	Lu(a−)	yes
2	Lu(a−)	Lu(a+)	Lu(a−)	yes
11	Lu(a−)	Lu(a−)	Lu(a+)	yes
1	Lu(a−)	Lu(a+)	Lu(a+)	yes
1	Lu(a+)	Lu(a−)	Lu(a+)	yes
1	Lu(a+)	Lu(a+)	Lu(a−)	yes
0	Lu(a+)	Lu(a+)	Lu(a+)	

b) No Exclusions

n	Child	Mother	Lover
155	Lu(a−)	Lu(a−)	Lu(a−)
5	Lu(a+)	Lu(a−)	Lu(a+)
4	Lu(a−)	Lu(a+)	Lu(a−)
4	Lu(a+)	Lu(a+)	Lu(a−)
1	Lu(a+b−)	Lu(a+b+)	Lu(a+b+)
0	Lu(a−)	Lu(a+)	Lu(a+)
4	Lu(a−)	Lu(a−)	Lu(a+)
1	Lu(a+b+)	Lu(a+b+)	Lu(a+b+)

Plausibility to Exclude Non-fathers from Paternity

The plausibility to exclude any non-father from paternity is calculated by means of the formula $pq(1-pq)+2(pq)^2$. The calculation brings about a value of 2.99%.

Studies of the Stability of the Lu^a Antigen in Stored Blood Samples

After a storage time of about 6 months at 4° C, a Lu(a+) red cell was usually classified reliably. This reliability normally does not primarily depend on the age of a particular blood sample but on its grade of hemolysis. If a sample had been kept in good condition, we achieved pretty good results when introducing the Lu^a antigen in expertises on the identity of a particular sample (e.g., in cases of claimed confoundance).

Discussion

The segregation of the phenotypes in the offsprings given above supports the assumed way of autosomal codominant inheritance.

Although we did not find any Lu(a−b−) individual in our series, attention should be drawn to this phenotype! Crawford et al. [4] reported the first example of this kind and some more have been found in the meantime. Crawford et al.'s study [4] revealed that this Lu(a−b−) phenotype was controlled by just *one dominant* gene! This has been a very unusual finding, because the majority of "minus-minus" phenotypes in other systems only occur when a pair of recessive genes is inherited [10, 12].

That the Lu(a−b−) phenotype can also be due to a recessive genetic background, was suspected by Darnborough et al. [7] and later confirmed by Brown et al. [1].

Crawford et al. [5] have shown that the expression of the P_1 antigen (e.g., also Au^a and i) is altered: They noted a highly significant excess of P_1-negative individuals being Lu(a−b−) of the dominant character.

There is evidence that the dominant Lu(a−b−) state can prevent the expression of the P_1 antigen in people of this phenotype who also have inherited a P_1 gene [3].

The time and mode of action of the inhibitor locus on the Lutheran, Auberger, P, and i antigens is not known. The less appropriate notated locus *In(Lu)* [12] is known to be genetically independent of the Lutheran [13] and the P locus. Obviously, these circumstances may lead to a false exclusion from paternity (e.g., in the P system) [3].

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Received July 17, 1980