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LETTER TO THE EDITOR

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Observations on the Lewis^a and Lewis^b activity of erythrocytes

Introduction

Information on the Lewis system was first reported in 1946 when Mourant described anti-Lewis^a antibodies which agglutinated the red blood cells of approximately 22% of Europeans [14]. Two years later Anderson discovered anti-Le^b antibodies [1] and Grubb [6] demonstrated a relationship between the Le^a and Le^b phenotypes and the secretion of ABH substances by salivary glands. It is now known that the Lewis system consists of a large family of antigens (Le^a, Le^b, Le^c, Le^d, Le^x, Le^y) controlled by the Le, Le^x, Se, H genes localized on chromosomes 11 and 19 [4, 5, 16, 18]. All contemporary reports indicate that the antigenic Le^a and Le^b activity of red blood cells results from the absorption of these antigens from the blood plasma onto the membrane surface of the erythrocytes [12, 13, 15, 17, 19, 21]. However, observation made by the author from immunochemical studies of ABH and Lewis antigens suggest another possible mechanism.

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Materials and methods

Blood samples were collected in 8 cases and typed in the ABO and Le^a and Le^b systems. Erythrocyte membranes, plasma and in 3 cases hemolysates were prepared. Glyxosphingolipids (ABH and Lewis antigens) were extracted, hydrolysed and purified using column-chromatography and thin-layer chromatography according to previously published methods [3, 7–11]. Glycosphingolipid fractions were visualized using anti-Le^a and anti-Le^b antibodies and a peroxidase with 3-amino-9-ethylcarbazol as substrate.

Results and discussion

A comparison of the chromatograms (Fig. 1A–E) obtained from the immunochemical detection of Lewis substances shows striking differences between the band intensities. The 2 lower bands of the chromatogram 1-B are much more intense than the corresponding bands in 2-B and the upper bands in the chromatograms 2-C, 2-D and 2-E are clearly more intense than the corresponding bands in 1-C, 1-D and 1-E. These differences in intensity may be explained by different absorption forces in particular glycospinogolipid Lewis chains of substances from blood plasma. Analogous findings were obtained for Le^b sub-

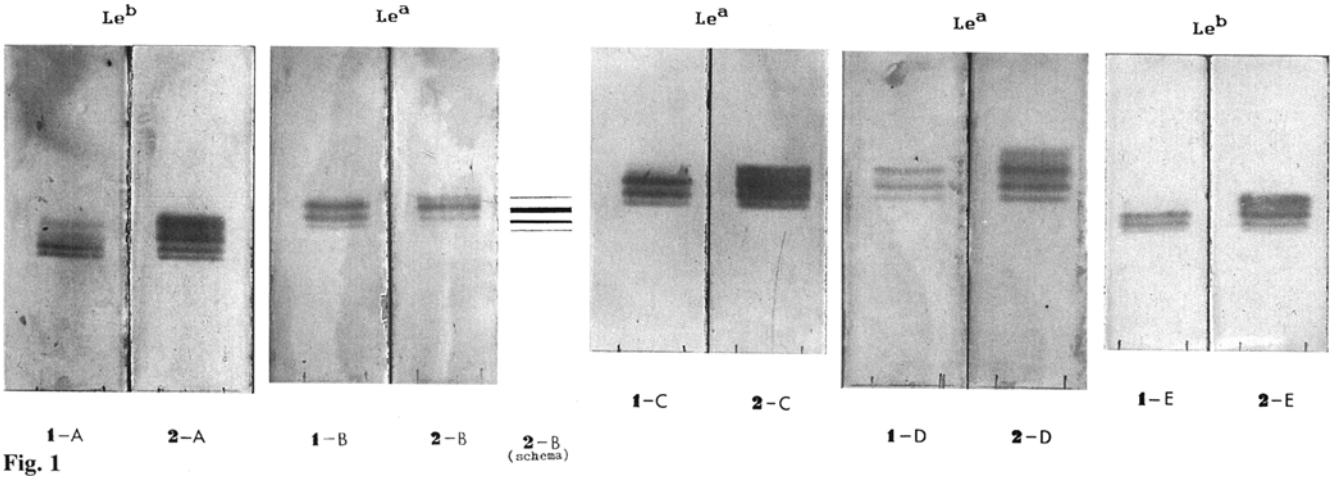

Fig. 1

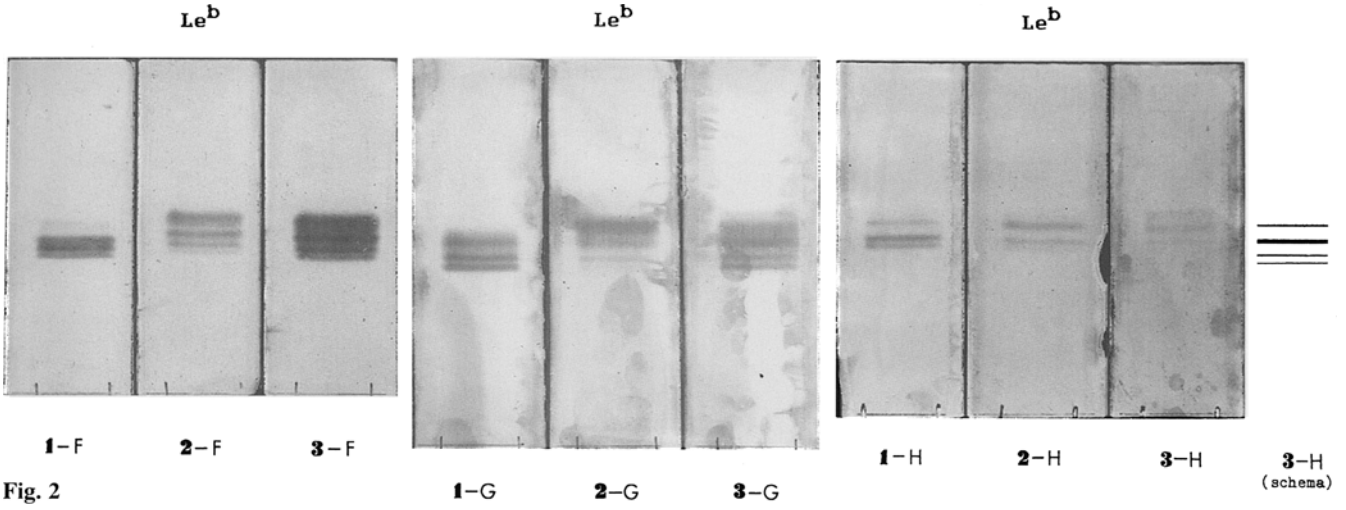

Fig. 2

stances by comparing the chromatograms 1,2,3-F, 1,2,3-G and 1,2,3-H from red cell membranes, erythrocyte contents (hemolysates) and from plasma (Fig. 2). This shows the strong expression of Le^b substances extracted from the inside of red cells (2-F, 2-G, 2-H).

These preliminary observations indicate an alternative mechanism for the expression of Le^a and Le^b substances on the erytrocyte membranes which would be a function of 2 independent mechanisms, i.e. the absorption of antigens form the blood plasma onto the erythrocyte surface and an independent production of the antigens by the red blood cells. This independent production of Lewis antigens by red blood cells could already begin in the early erythropoietic forms as suggested indirectly by the presence of small amounts of Le^a substances in the stem cells [20]. This production might then be continued in mature erythrocytes, now devoid of a nucleus, since it has been demonstrated that some delay can occur between the translation and the synthesis and storage of m-RNA in the cell cytoplasm [2]. In conclusion these preliminary observations seem to indicate an alternate and additional mechanism for the expression of the Le^a and Le^b antigens on the red cell surface. However, these are only empirical observations based on the intensity of bands obtained from plasma, hemolysates and red cell membranes and must be further investigated with regards to the specificity and identity of the fragments obtained.

References

1. Andersen PH (1948) The blood group L system. A new blood group L_2. A case of epistasy within the blood groups. APMIS 25:728–731
2. Barouki R (1990) Expression des genes au cours de la spermatogenese. Médecine/sciences 6:785–790
3. Brockhaus M (1985) Detection of glycolipid with monoclonal antibodies. Immunol Methods III:133–145
4. Ceppellini R, Siniscalco M (1955) Una nuova ipotesi genetica per il sistema Lewis secretore e suoi reflessi nei riguardi di alcune evidence di linkage con altri loci. Rev Inst Sieroter Ital 30: 431–445
5. Couillin Ph, Mollicone R, Griscard MC, Gibuad A, Ravise N, Feingold J, Oriol R (1991) Chromosome 11q localisation of one of the three expected genes for the human α–3–fucosyltransferases, by somatic hybridization. Cytogenet Cell Genet 56:108–11
6. Grubb R (1948) Correlation between Lewis blood group and secretor character in man. Nature 162:933
7. Hakomori S (1978) Isolation of blood group ABH-active glycolipids from human erythrocyte membranes. Methods in Enzymology vol L Complex Carbohydrates part C:207–211
8. Hansson GC, Karlsson G, Larson BE, Samuelsson BE, Thurin J, Bjursten LM (1985) Detection of blood group type glycosphingolipid antigens on thin-layer plates using polyclonal antisera. J Immunol Methods 83:37–42
9. Hauser R, Rand S, Lenz J, Brinkmann B (1989) Nachweis von Glykosphingolipiden der ABH- und Le-Antigene auf Dünnschichtplatten mittels der PAP-Methode. Z Rechtsmed 103: 47–56
10. Karlsson KA (1987) Preparation of total nonacid glycolipids for overlay analysis of receptors for bacteria and viruses and for other studies. Methods in Enzymology 138:212–220
11. Koscielak J, Miller-Podraza H, Zdebska E (1978) Isolation of poly(glycosyl)ceramides with A, B, H and I blood-group activities. Methods in Enzymology vol L Complex Carbohydrates part C:211–216
12. Lindströme K, Shanwell A, Jacobsson A, Lönnqvist B, Samuelsson BE (1992) Alteration of glycosphigolipid-based blood group antigen expression on eryhtrocytes and in plasma studied on consecutive samples after a blood group 0 to A bone marrow transplantation. Glycobiology 2:529–534
13. Marcus DM, Cass LE (1969) Glycosphingolipids with the Lewis blood group activity: uptake by human erythrocytes. Science 164:553–555
14. Mourant AE (1946) A new human blood group antigen of frequent occurrence. Nature 158:237–238
15. Needs ME, McCarthy DM, Barrett J (1987) ABH and Lewis antigen and antibody expression after bone marrow transplantation. Acta Haemat 78:13–16
16. Nishihara S, Nakazato M, Kudo T, Kimure H, Ando T, Narimatsu H (1993) Human α–1, 3–fucosyltransferase (FucT-VI) gene is located at only 13 Kb 3′ to the lewis type fucosyltransferase (FucT-III) gene on chromosome 19. Biochem Biophys Res Commun 190:42–46
17. Oriol R, Le Pendu J, Sparkes RS, Sparkes MC, Crist M, Gale RP, Terasaki PI, Bernoco M (1981) Insights into the expression of ABH and Lewis antigens through human bone marrow transplantation. Am J Genet 33:551–560
18. Oriol R, Le Pendu J, Mollicone R (1986) Genetics of AB0, H, Lewis, X and related antigens. Vox Sang 51:161–171

19. Renton PH, Hancock JA (1962) Uptake of A and B antigens by transfused group 0 erythrocytes. Vox Sang 7:33–38
20. Schenkel-Brünner H, Hanfland P (1981) Immunochemistry of the Lewis blood-group system III. Studies on the molecular basis of the Le[x] property. Vox Sang 40:358–366
21. Sneath JS, Sneath PA (1955) Transformation of the Lewis groups of human red cells. Nature 176:172