Different precipitin reactions of the *Abrus precatorius* lectin

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Summary. Different precipitin reactions of the *Abrus precatorius* plant lectin with various galactan-polysaccharides are described and compared with a number of other anti-galactose lectins from different sources.

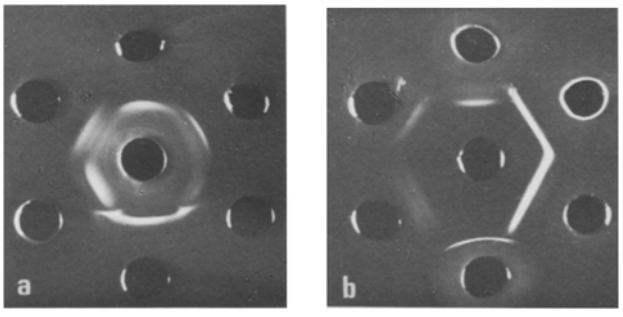
An interesting precipitin reaction of the *Abrus precatorius* lectin (India) has been described earlier in this Journal by Bird[1]. He found that *Pneumococcus* type XIV polysaccharide, as well as human ABH blood group specific glycoproteins, especially their *Pneumococcus* type XIV cross-reactive precursor substances, gave strong precipitin reactions with an extract from the seeds of *Abrus precatorius*. From these results, it could be concluded that a common N-acetyl-lactosamine structure was responsible for this phenomenon.

Because of the great importance of this precursor substance as a tumor characteristic or tumor associated antigen[2], and in order to check whether the *Abrus* lectin can be used as a specific marker, we re-investigated the precipitin reactions of the *Abrus* lectin, using a material of highly purified *Abrus* precipitin, which had been obtained by affinity chromatography on Con A Sepharose, a method which has been developed by us in modifying previous isolation procedures by other authors[3-5].

The results of our experiments are summarized in the figure: Whereas the precipitin reactions of the *Abrus* lectin with different glycosubstances are shown in the figure, a, in the figure, b the comparison of the *Abrus* lectin with other lectins from different sources and with a similar specificity is demonstrated. It can be deduced from the figure, a that the *Abrus* lectin does not only precipitate with *Pneumococcus* type XIV polysaccharide and human blood group substances, but also reacts as an anti-galactan and as a marker for some neuraminidasetreated (serum)glycoproteins[6].

On the other hand, when compared with certain other anti-galactan or anti-β-galactosyl lectins (figure, b), no close relationship to these heterophile reagents from plant and invertebrate *(Tridacna clams)* is visible, when testing against a purified galactan standard substance.

Our results confirm the anti-β-galactosyl specificity of the *Abrus* lectin, but demonstrate also that the various cross-reactions of the precipitin (and agglutinin), because of its detection of β-galactosyl groups in different linkages (1-4, 1-6), limits its use as a specific marker and do not facilitate the characterization of the corresponding cell surface receptor, which triggers also the mitogenic stimulation of lymphocytes by this lectin.



Different precipitin reactions of the *Abrus precatorius* lectin. (Numbers should be read clockwise from 12 o'clock (= 1) on.)
a Precipitation of the *Abrus* lectin with different glycosubstances. 1 = *Helix pomatia* (snail) galactan, 2 = *Achatina fulica* (snail) galactan, 3 = *Lymnaea stagnalis* (snail) galactan: polysaccharides consisting mainly of D-galactose in β-glycosidic linkage, 4 = Serum glycoprotein (human haptoglobin), 5 = *Pneumococcus* type XIV polysaccharide, 6 = Pneumogalactan from bovine lung. Center: *Abrus* lectin.
b Precipitation of purified *Achatina fulica* galactan by different lectins. (Center: *Achatina fulica* galactan.) 1 = *Abrus precatorius*, 2 = *Ononis spinosa*, 3 = *Glycine soja*, 4 = *Tridacna maxima*, 5 = *Tridacna gigas*, 6 = *Ricinus communis*.

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Human adenosine deaminase and chromosome 20[1]

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Summary. In patients exhibiting in marrow cells deletion of the long arm of chromosome 20, the specific activities of adenosine deaminase in cells of the peripheral blood are normal. This suggests that the gene for adenosine deaminase is not localized to the distal segment of the long arm of this chromosome.

The enzyme adenosine deaminase (ADA) catalyses the irreversible deamination of adenosine to inosine. In human erythrocytes, this activity occurs as a monomeric protein which exhibits electrophoretic polymorphism depending upon an autosomal gene with 2 codominant alleles, ADA_1 and ADA_2[2,3]. Tissues, other than red blood cells, contain in addition to this polymorphic protein, one or more additional forms[4,5]. The findings on the nature of isoenzymes in tissues of patients with severe combined immunodeficiency and ADA deficiency[6,7], together with the demonstration of

Specific activity of adenosine deaminase in patient with 20q- polycythaemia rubra vera

Patient	Sex	Haematocrit %	Red cell mass ml/kg	Percentage of marrow cells 20q	Adenosine deaminase (nmoles/h mg protein) Erythrocyte	Leukocyte	Lymphocyte
1	M	77	59	100	60	2,060	4,450
2	F	50	ND	100	53	913	1,670
3	M	49.5	44	60	97	1,390	2,180
4	F	71	37	0	105	1,950	3,650
Normal	F 37-47 M 42-52		F 22-29 M 25-32		63 ± 24	750 ± 280	2,205 ± 1,170

NP = not performed.

the intercovertibility between the molecular forms of ADA[6,8], suggest that the heterogeneity of ADA in human tissue is the result of modification of a single gene locus product[6,9]. Cell hybridization studies assign this locus to chromosome 20[10]. Because a deletion of the long arm of this chromosome, karyotypically designated as 20q-, occurs in marrow haemopoetic precursors of subgroups of patients with polycythaemia rubra vera and refractory sideroblastic anaemia[11], we thought it would be of interest to determine the activity of ADA and its molecular forms in cells of the peripheral blood of these patients.

Methods. Routine haematological procedures were performed by standard techniques. Bone marrow chromosome prepartions were made by a modification[12] of the direct method of Kiossoglou et al.[13]. The Giemsa banding technique used was according to Summer et al.[14].

Erythrocytes, leukocytes and lymphocytes from heparinized peripheral blood were separated and processed as described previously[5,15]. Adenosine deaminase activity was assayed by the conversion of radiolabelled adenosine to inosine and hypoxanthine[6], and this activity in leukocyte and lymphocyte extracts were chromatographed on Biogel A-0.5 M[5].

Results and discussion. Results are shown in the table. In marrow precursors of 3 of 4 patients with polycythaemia rubra vera studied deletions varying in size from $\frac{1}{3}$ to $\frac{2}{3}$ of the long arm of chromosome 20 were found. 2 patients (No. 1 and 2) had the partially deleted chromosome (20q-) in 100% of cells analysed whilst patient No. 3 showed a mosaic cell pattern, i.e. 60% with the 20q- chromosome and 40% with no abnormalitiy. Patient No. 4 had a completely normal karyotype in all cells examined.

The specific activities of ADA in cells of peripheral blood of all 4 patients were within the normal range and gel filtration of leukocyte and lymphocyte extracts demonstrated the presence of both the 296,000 and 34,000 molecular forms of ADA[5].

In the light of assignment of the locus for the catalytic unit of ADA to chromosome 20[10] the present data are of interest. If the ADA gene was located on the deleted portion of chromosome 20, in the absence of translocation, the ADA activity in peripheral blood erythrocytes would either be absent or markedly reduced. That this is not the case suggest that the locus for human ADA is either on the short arm or close to the centromere on the long arm of chromosome 20.

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Destruction of the platelet aggregating activity of ristocetin A[1]

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Summary. Hydrolysis of ristocetin A in 0.1 N HCl at 37 °C for 2 h resulted in the loss of its ability to induce platelet aggregation in platelet-rich plasma derived from guinea-pigs and humans. However its antibiotic activity against *Staph. aureus* was not lost.

The glycopeptide antibiotic[2], ristocetin A, underwent extensive testing which demonstrated its efficacy against gram positive organisms[3-7]. Its use as an antibiotic was later discontinued partially as a result of its ability to induce thrombocytopenia and thrombophlebitis[5,8,9]. Howard and Firkin[10] found that platelets in plasmas derived from patients with von Willebrand's disease would not aggregate in its presence. It was later demonstrated that the defect lay in the factor VIII molecules present in plasmas from von Willebrand's patients[11].

While studying the mechanisms of ristocetin induced platelet aggregation, we found that mild acid hydrolysis of