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.

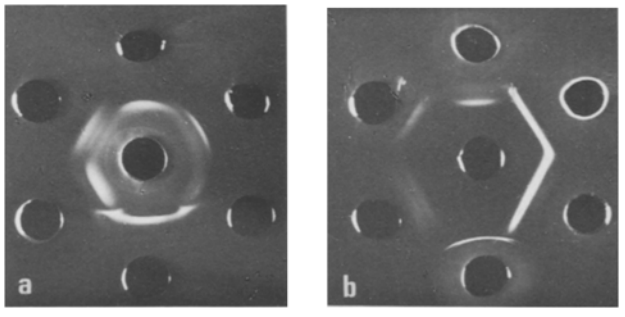
An interesting precipitin reaction of the *Abrus precatorius* lectin (India) has been described earlier in this Journal by Bird[1]. He found that *Pneumococcus* type XIV polysaccharide, as well as human ABH blood group specific glycoproteins, especially their *Pneumococcus* type XIV cross-reactive precursor 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