larization data (fig. 3), the step corresponding to the DPPC transition becoming higher with time. The transfer process is however much more striking in the case of SUV (figs 2 and 3), in which a lipid transfer from DMPC to DPPC is well documented[15].

In conclusion, LUV are closer to MLV in the stability of their thermotropic properties, and closer to SUV in the absence of a well-defined pre-transition. We think that the above results could contribute to a better understanding of the general properties and stability of a very promising new type of lipid vesicle, the LUV.

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Isoenzymes of glutamate-oxalacetate transaminase in the larvae of silkworms *Bombyx mori* infected with nuclear polyhedrosis virus

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Summary. Nuclear polyhedrosis virus infection induced changes in the activity and isoenzyme pattern of glutamate-oxalacetate transaminase in the larvae of silkworm, *Bombyx mori.*

Nuclear polyhedrosis is characterized by the formation of polyhedral-shaped inclusion bodies within the nuclei of susceptible cells. Studies of insects with this type of disease have shown that large amounts of proteins are synthesized for the formation of the virus particles and inclusion bodies[2-4]. The host organism, therefore, will be affected especially in its protein and amino acid metabolism. Since transaminases are the key enzymes involved in protein and amino acid metabolism, comparative measurements of the activities of these enzymes in healthy and diseased tissues may help in understanding the physiological changes that arise from the disease. Earlier work by other investigators[5,6] indicated an increase in the glutamate-oxalacetate transaminase activity during infection. However, there is no information concerning the changes in the isoenzyme pattern of this enzyme in the infected tissues. The present work deals with a comparative study made on the activity and isoenzyme profile of glutamate-oxalacetate transaminase in healthy and diseased silkworm larvae.

Materials and methods. Healthy silkworm larvae belonging to the pure Mysore variety were obtained from the Central Sericultural Research and Training Institute (CSRTI), Mysore. The larvae were infected just after the 4th ecdysis by feeding them on mulberry leaves smeared with a nuclear polyhedrosis virus suspension. On the 5th day after inoculation, the larvae showed the typical symptoms of the disease. On the 6th day after inoculation hemolymph, fat body and intestine were collected separately from about 30 larvae, and those of control larvae fed on virus-free leaves were also obtained on the same day. The collection of hemolymph was done as described by Martignoni and Milstead[7]. Fat bodies and intestines were quickly removed and homogenized in ice-cold 0.1 M phosphate buffer pH 7.4 (1:1 w/v) in a Potter-Elvehjem tissue

Effect of nuclear-polyhedrosis on glutamate-oxalacetate transaminase activity in the larvae of *Bombyx mori**

Tissue	Group	Specific activity (μmoles/g protein/h)
Fat body	Control	72.23 ± 1.53
	Diseased	119.23 ± 1.12
Hemolymph	Control	56.73 ± 1.86
	Diseased	159.23 ± 4.55
Intestine	Control	47.73 ± 3.22
	Diseased	49.5 ± 4.42

*Values represent means ± SE for 3 batches.

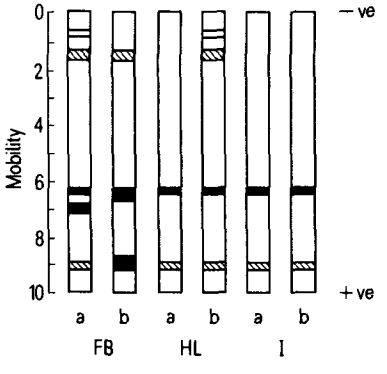

Fig. Zymogram showing the electrophoretic patterns of glutamate-oxalacetate transaminase in the fat body (FB), hemolymph (HL), and intestine (I) of healthy and infected larvae of *Bombyx mori.* a, control; b, diseased. □, Low intensity; ▨, moderate intensity; ■, high intensity.

homogenizer. The homogenates were centrifuged at 2000 rpm at 5 °C for 45 min in an MSE refrigerated centrifuge.

Glutamate-oxalacetate transaminase activity was determined in the tissue homogenates and hemolymph by the method of Tonhazy (cf. Bergmeyer[8]). Protein concentration was estimated by the method of Lowry et al.[9]. Isoenzymes of glutamate-oxalacetate transaminase were studied by polyacrylamide disc gel electrophoresis[10] followed by enzyme-specific staining of the gels. About 200 µg of protein from each sample was layered on the gel (5%) and electrophoresis was carried out applying a current of 3 mA/gel at 5 °C. Tris-glycine buffer pH 8.5 was used. The gels were then incubated in 30 ml of 0.05 M Tris-HCl buffer, pH 7.4, containing 50 mM DL-aspartic acid, 20 mM 2-oxoglutarate, 0.05 ml glutamate dehydrogenase (obtained from Sigma Chemical Company, USA.), 20 mg of NAD^+ 12 mg nitro blue tetrazolium (NBT), 2 mg of phenazine methosulfate (PMS).

Results and discussion. The results of the determinations of the glutamate-oxalacetate transaminase activity are given in the table. In fat body and hemolymph of the infected larvae there was a significant increase in the activity of the enzyme whereas the gut tissues did not show any changes. As reported by other investigators[5,11], the viral infection also induces an elevation in the concentration of some of the amino acids, especially glutamic acid, aspartic acid and alanine, which are readily transaminated and used in the virus multiplication process. Increased transaminase activity in the infected fat body indicates that it is the main seat of synthesis of this particular virus. In the case of hemolymph, the increase in the enzyme activity may be due to the release of the enzymes from the disintegrating fat bodies during infection. This fact is also evident from the electrophoretic patterns of the isoenzymes, shown in the figure. The isoenzyme pattern in the intestinal tissues shows no difference, whereas there is a distinct change in the fat body and hemolymph of healthy and infected larvae. The slow migrating bands found in the enzyme from fat body appear to be specific to fat body tissue, since they are not found in either hemolymph or intestine. But these bands appeared in the hemolymph of diseased larvae, indicating that they are liberated from the fat body undergoing destruction caused by the virus. Further, one of the fast migrating bands found in the fat body of healthy larvae is not found in those of infected larvae. This may be due to the destruction of this enzyme along with other tissue proteins. The remaining bands in the fat bodies of infected larvae are intensified, indicating that these isoenzymes are responsible for the increased activity during infection. Thus the changes in the activity and isoenzyme profile of transaminases in the normal and infected tissues can be considered as indicators of the biochemical changes produced by polyhedrosis virus in the insect hosts.

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A lectin in the pollen of marihuana, *Cannabis sativa* L.

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Summary. A lectin was found in the pollen of *Cannabis sativa* L. which reacted with papain treated erythrocytes. The lectin has an affinity for glucose containing receptor sites.

Lectins or agglutinins found in plants have been used to study the structure and topography of cellular membranes[1,2]. Several lectins have been reported which react weakly or not at all with unaltered red cells but react strongly with protease-treated red cells[3]. In addition, lectins have been found throughout some plant species in the seeds and other parts of the growing plant[4].

Cannabis sativa L. is generally regarded as a highly variable species[5] of considerable forensic interest. A lectin has been reported in the seeds of *Cannabis*[6] and in an effort to confirm this result the pollen was examined as a new and unreported source of lectins. The pollen and seeds from locally grown *Cannabis sativa* L. were collected, crushed and extracted into 0.1 m phosphate buffered saline at pH 7.5. The crude extract was tested against untreated human red cells of various blood group specificities without result. When tested against papain treated red cells, the lectin from the pollen showed a low avidity but a titre of $^1/_{128}$ without regard to a recognizable blood group. The reaction with neuraminidase-treated red cells was very weak as was the reaction with ficin treated cells. The activity of the lectin was also apparently directed toward a site different from that of the T, Tn and Tk cryptantigens. The extracts of marihuana seeds behaved similarly although preparations were of a much lower titre.

Inhibition studies with simple sugars showed the lectin to react primarily with glucose and several of its derivatives: glucosamine, N-acetyl D-glucosamine and methyl D-glucopyranoside. The lectin also reacted with galactose to a lesser extent. Concentration by ultrafiltration showed the molecular weight of the lectin to be in excess of 15,000.

The receptor site for the lectin appears to be away from the periphery of the red cell shielded by the protein which is