The simplest way by which the carbon atoms derived from glucose may be incorporated into amino acids and finally into the proteins occurs by reductive amination reaction which are catalysed by glutamate dehydrogenase[9]. A comparison of amination and deamination reactions catalysed by L-glutamate dehydrogenase indicates that the amination of α-keto acids is more active than the deamination reactions (table 2). Since α-keto acids are the common intermediates of glucose utilization in the filarial worms[1], it is not surprising that reductive amination of these amino acids is the basis for the appearance of radiocarbon of glucose in protein fractions like animal cells in these nematodes. Enzymes catalysing the transamination reactions between amino acids and α-keto acids are also present in *S. cervi* adult females which may also be another link between carbohydrate and amino acid metabolism. A reverse tricarboxylic acid cycle has been reported in many filarial species[10-12]. On this basis it can be proposed that the incorporation of carbon atoms of chlorella protein hydrolysate into glycogen fractions of *S. cervi* adult females is the result of deamination of amino acids of chlorella protein hydrolysate and also their entry into tricarboxylic acid cycle.

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Erythrocytie membrane changes in sheep infected with *Dictyocaulus filaria*

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Lambs infected orally with 2500 *Dictyocaulus filaria* larvae showed a significant fall in levels of erythrocyte membrane cholesterol, cholesterol: phospholipid ratio and acetylcholinesterase activity from fourth to tenth week ($P<0.05$ to $P<0.001$) post-infection. Plasma cholesterol levels in infected lambs were also significantly different from fifth week onwards from those in uninfected controls. These alterations set in one to two weeks prior to increase in osmotic fragility of erythrocytes and coincided with development of the parasite to adult stage in the infected host.

An increase in the osmotic fragility of erythrocytes has been reported in various diseases of livestock[1] and in toxic conditions[2]. Recently, increased osmotic fragility of sheep erythrocytes was reported in *Dictyocaulus filaria* infection[3]. This communication reports our preliminary findings on erythrocyte membrane changes *vis-a-vis* decreased resistance of sheep erythrocytes to osmotic lysis in *D. filaria* infection.

The study was carried out in *D. filaria* producer lambs maintained at this laboratory for vaccine production. Eight producer lambs were randomly selected and grouped into acute infection (5 lambs) and chronic infection (3 lambs) groups based on day of infection. The zero week post-infection (PI) observation for various parameters in the chronic infection group corresponds to 27th week PI. Each animal in these groups had received an infection dose of 150 *D. filaria* larvae per kg body weight. Three animals were also maintained as uninfected controls. The osmotic fragility of erythrocytes was determined as described earlier[3]. Standard methods were employed for the preparation of erythrocyte membranes[4], estimation of membrane cholesterol and phospholipids[5], and determination of acetylcholinesterase activity[6]. The study was carried out for ten weeks.

The lambs in acute stage of infection showed a decrease from fourth week onwards in membrane cholesterol (0.89 ± 0.13 to 1.31 ± 0.14 mg/ml packed cells), cholesterol to phospholipid ratio (0.61 ± 0.13 to 0.84 ± 0.12) and acetylcholinesterase activity (10.56 ± 1.75 to 16.36 ± 1.8 mol/min/RBC$\times10^{-22}$) compared to uninfected controls (cholesterol, 1.38 ± 0.09 to 1.45 ± 0.1; cholesterol to phospholipid ratio, 0.97 ± 0.09 to 1.00 ± 0.08; acetylcholinesterase activity, 18.88 ± 1.75 to 18.76 ± 1.95). However, group differences were significant from fourth or fifth week PI ($P<0.05$ to $P<0.001$.) Membrane phospholipids in the infected lambs remained unaffected during the course of infection. The changes

in erythrocyte membrane constituents had set in one to two weeks prior to the significant decrease ($P<0.01$ to $P<0.001$) in the resistance of sheep erythrocytes to osmotic lysis (0.77 ± 0.02 to 0.82 ± 0.02 initiation; 0.54 ± 0.05 to 0.65 ± 0.02 completion). In chronically infected lambs, the various erythrocyte membrane constituents were restored to near-normal (cholesterol, 1.28 ± 0.12 to 1.32 ± 0.11; cholesterol to phospholipid ratio, 0.93 ± 0.09 to 1.00 ± 0.08; acetylcholinesterase activity, 17.90 ± 1.45 to 18.15 ± 1.25). The erythrocytes of these animals continued to remain osmotically more fragile (0.70 ± 0.02 to 0.74 ± 0.02 initiation; 0.50 ± 0.02 to 0.56 ± 0.03 completion) than those of uninfected controls (0.67 ± 0.01 to 0.70 ± 0.03 initiation; 0.46 ± 0.01 to 0.48 ± 0.01 completion). However, the differences were not significant.

It is interesting to observe that the increase in erythrocyte fragility is preceded by a decrease in various membrane constituents during acute course of infection. Further, the alterations in these constituents coincide with the development of the nematode parasite to adult stage and its establishment in the air passages of the host, causing progressively increased interference with free flow of air and gaseous exchange in the lungs. In acute infection, the parasite is known to cause hypoxia and hypoxaemia[7], whose degree is associated with the extent of increase in osmotic fragility of the erythrocytes in this host–parasite system[8]. This is further supported by the present observation of restoration of osmotic fragility to near normal level in chronically ill animals.

Altered susceptibility of erythrocytes to osmatic lysis has been reported in disease and toxic conditions[3], besides structural defects and damage to erythrocyte membranes[9]. The decrease in membrane cholesterol, cholesterol to phospholipid ratio and acetylcholinesterase activity, seem to be the direct effects of hypoxic injury suffered by the sheep erythrocytes during acute infection. This is corroborated by the restoration to near-normal of these membrane constituents and erythrocytic fragility in chronic stage of infection (27th week PI onwards), when the infected host is relieved to a large extent from the hypoxic effects of the disease through various host compensatory mechanisms, besides worm expulsion from the lungs by acquired immune mechanism.

This happens to be the first study of erythrocyte membranes in helminth infections of man or animals.

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Some observations on goats following administration of *Leptospira interrogans* serovar *hardjo*

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***Leptospira interrogans* serovar *hardjo* cells were inoculated in two goats and bacteriological, serological and biochemical observations were made at various intervals. The goats exhibited a biphasic fever curve, the increase in temperature (104–105°F) being the first observed at 9th or 10th day post-inoculation (DPI) lasting up to 20th or 21st DPI. The second curve was observed between 22nd or 25th DPI and 32nd DPI. No other clinical sign was observed in any goat. During pyrexic phase leptospires were visible microscopically in the blood. However, attemps to culture the organisms, either from the blood or urine or tissue collected during necropsy failed. Antibodies against the *hardjo* cells were detected in goats during the initial pyrexic curve employing microscopic agglutination test (MAT) (at 14th DPI) and indirect haemagglutination test (IHA) (at 7th DPI). MAT titres in the goats remained more or less unchanged whereas the IHA titres declined. Biochemical studies carried out using sera of infected goats indicated an increase in liver enzymes namely alanine aminotransferase and aspartate aminotransferase and elevated bilirubin and cholesterol levels, suggesting liver damage. Albumin concentration in goats decreased with increase in globulin concentration.**

LEPTOSPIRA INTERROGANS serovar *hardjo* is an important pathogen of almost all the farm animals. The characteristic clinical features which develop in animals following the infection with this organism are abortion, still birth, infertility and mastitis 1,2. In goats, however, the only clinical sign which has been reported to develop following the administration of *hardjo* strains is a rise in rectal temperature[1]. No other information on infection in goats is available.

In the present study efforts were made to study biochemical changes in goats inoculated with *L. interrogans* serovar *hardjo*. Bacteriological and serological observa-