aplites and granitic material underwent plastic deformation during F folding and amphibolite facies metamorphism, the pegmatites acted as competent bodies undergoing rupture. Thus it is evident that the above aplites and pegmatites of the granite body are pre-S_I. The post-S_I aplites and pegmatites of the granite body are free from the effects of F folding and amphibolite facies metamorphism.

The Eastern Ghat orogenic belt rocks are believed to have been involved in two phases of metamorphism during the Eastern Ghat orogenic cycle, *viz.*, metamorphism (phase I) of the charnockites of Madras and Nilgiris and Khondalites closing at 2600 m.y. and a later metamorphism (phase II) of the belt accompanied by granitic and pegmatitic activity marking the culmination of the Eastern Ghat orogery at 1600 m.y.[2].

Evidences presented above suggest that the Kalpatta granite as a whole is either pre-tectonic or early syntectonic with respect to F folding and the accompanying retrograde amphibolite facies metamorphism (phase II). This metamorphism and deformation most probably coincide with the culmination of the Eastern Ghat orogeny closing at 1600 m.y. The late aplites and pegmatites must have intruded into the granite body after the culmination of the Eastern Ghat orogeny.

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GLYCOGENESIS IN THE LIVER OF A MURREL FISH

Literature pertaining to the localization of glycogen in the body of fishes is limited and little is known about the carbohydrate metabolism in them. The present communication records for the first time the pattern of localization and significance of glycogen in the liver cells of a murrel fish, *Ophicephalus punctatus* Bloch.

Pieces of liver from the well-fed specimens, and from those undergoing fasting, were fixed in Rossman's and Helly's fixatives at 5° C for 12 to 16 hours. Irrespective of the fixative used, the sections of liver, after conventional processing, were treated with periodic acid –Schiff (PAS) reagents, and a few sections stained with Best's Carmine technique as given by McManus and Mowry[2]. The parallel sections meant for control were incubated for ¾ of an hour in the human saliva, diluted with normal saline prior to the treatment of sections with the histochemical reagents. Some of the sections were counterstained with haematoxylin to give histological differentiation of the cells.

The histochemically prepared sections showed purple-red masses in the cytoplasm of the hepatic cells, which nearly filled the whole of the cytoplasmic space (Fig. 1). The hepatic cells

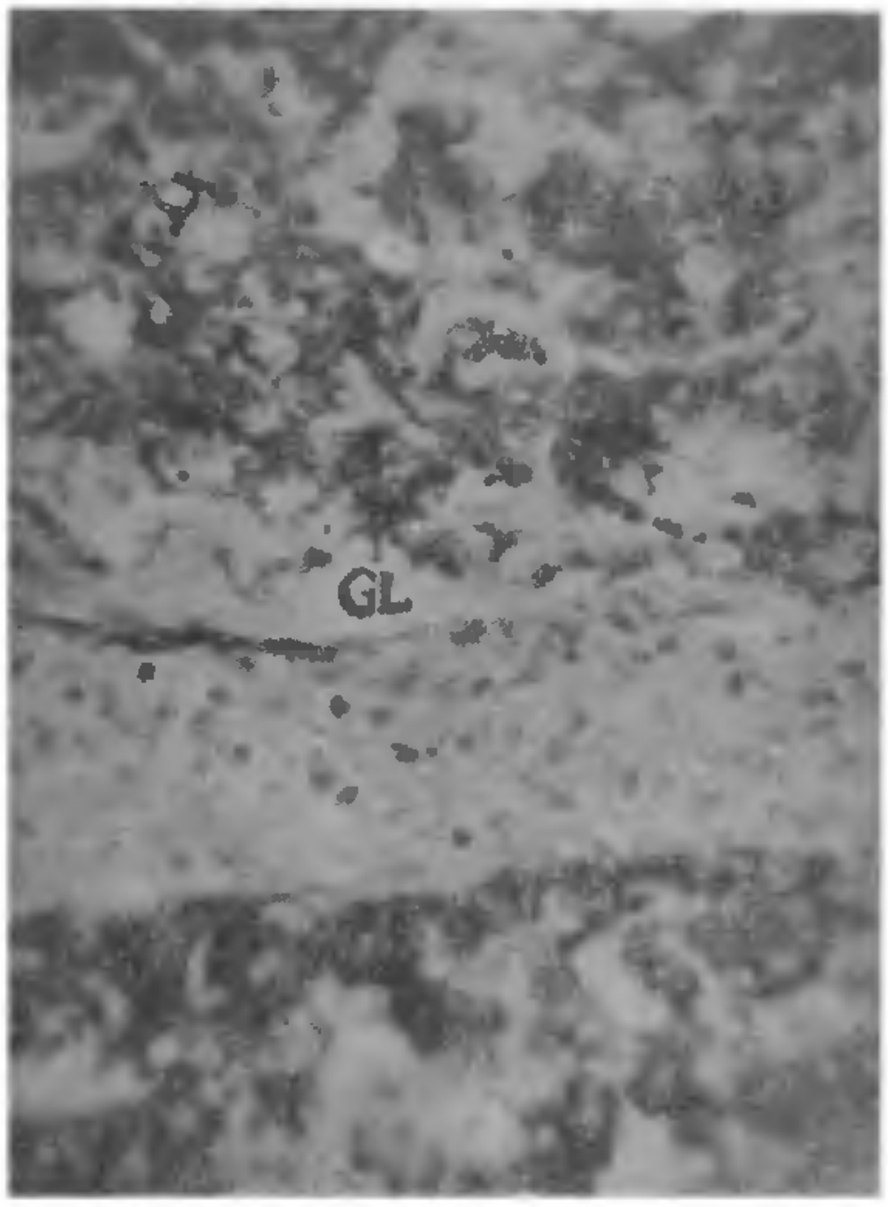

Fig. 1

appeared polygonal in shape and had generally one nucleus which occupied a central position in the cell. Some of the cells possessed two nuclei of equal size. However, it was found that their appearance had no sequence in the tissues. The purple-red stain for glycogen in the cell was found to be labile on the pretreatment of sections with the saliva. The result of the histochemical preparations with PAS always corresponded very closely with those given by the Best's carmine preparations. These reactions confirmed that the stained sites in the cells were glycogen deposits.

The pattern of localization of the glycogen deposits in the cells was characteristic. The deposits had a granulated appearance and each granule measured 0·9 to 1·2 micron in size. However, it was interesting to find that the concentration of the deposits in the different portions of the liver, and in the different feeding conditions

of the gut was not always the same. The distal and central parts of the lobes of liver had a larger concentration of glycogen in their hepatic cells than in those at the base of the liver, and the level of concentration was conspicuous in the liver of the well-fed specimens. Thus the nature of localization of the glycogenic substances in the present fish largely agreed with those of *Salmo gairdneriii*[3] and *Catla catla*[3]. However, it showed difference from the case of the mosquito fish, *Gambusia affinis*[3], for its having minute, globular and unstained lipidlike substances in between purple-red stained masses of the glycogen in the cells with the result that the aggregation of the glycogen masses was not compact in *O. punctatus*. The difference in the nature of the diet (piscivorous feeding habit of *O. punctatus*) seems to be responsible for this variation.

In an earlier investigation of this fish[4], a high level of alkaline phosphatase activity was noted in the striated border of the absorptive cells of the anterior portion (including pyloric caeca) of the intestinal tract. This provided an indirect evidence to the possibility of a vigorous phosphorylation activity in the gut-wall, and as such in the high rate of glycogenesis in the liver.

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SUSCEPTIBILITY OF AN INSECTIVOROUS BAT (*RHINOLOPHUS ROUXI*) TO EXPERIMENTAL INFECTION WITH KYASANUR FOREST DISEASE VIRUS

The involvement of chiropiera in the natural cycle of Kyasanur Forest Disease (KFD) virus was first suspected when neutralizing antibodies against KFD virus was demonstrated in two species of bats, *viz.*, a frugivorous bat, *Rousettus leschenaulti* (Pavri and Singh[1], 1965) and an insectivorous bat, *Rhinolophus rouxi* (VRC, unpublished data). Subsequently, four isolations of KFD virus were made from the spleens of naturally infected *R. rouxi* and one from their argasid tick ectoparasites (Rajagoplalan *et al.*[2], 1969). Our interest was therefore drawn to investigate viraemia in this species of bat after experimental infection with KFD virus.

While embarking on an experiment of this nature it was felt necessary to maintain these bats alive in captivity. We made several attempts in this regard. At first live and killed meal worms were left in the cages for bats to feed. Since the bats did not accept this feed, a mixture containing meal worms, ripe banana, cheese and white ants mashed into a paste was force fed to bats (Sulkin, personal communication). However, the bats did not accept this feed too. A proportion of bats survived up to 4 days under laboratory conditions. Since the bats were too small to be bled daily we decided to sacrifice them at daily intervals after experimental infection.

Twenty-five bats were inoculated subcutaneously, with 3·6 dex to 8·6 dex of KFD virus (strain isolated from *Haemaphysalis spinigera*). Two to five bats were sacrificed daily subsequent to the inoculation of KFD virus. A serial tenfold dilution of blood in rabbit serum phosphate saline (RSPS) and 10% suspension of organs such as, spleen, kidney, lung, brain and salivary glands, were inoculated intracerebrally into adult swiss mice. Confirmation of KFD virus isolated from blood and organs was made by complement fixation tests.

In the first experiment eight bats received 3·6 dex of KFD virus. The level of virus in blood ranged from traces of virus on first and second post-infection (PI) days to 3·0 to 4·0 dex/0·03 ml on third and fourth days after inoculation. KFD virus was recovered from spleen, lung and brain on 2nd PI days, and spleen lung, brain, kidney and salivary glands on third and fourth PI days. In the second experiment 17 bats received 8·6 dex of virus. The level of virus in blood ranged from 1·0 dex/0·03 ml to 3·0 dex/0·03 ml on first day to 2·7 dex/0·003 ml to more than 4·7 dex/0·03 ml on fourth PI days. Virus was isolated from spleen on first through fourth PI days.

This preliminary experiment has shown that KFD virus circulates in the blood of *R. rouxi* after experimental infection. Since the bats did not feed on the diet provided by us, they were apparently under physiological stress during the study. The dosate of virus given in one experiment was considerably high and it may be argued that viraemia recorded may not imply a replication of virus. However, in the first experiment where 3·6 dex of virus was used there was a gradual increase in the titre of virus in blood from first through fourth PI days. This would suggest that KFD virus circulates in the blood of *R. rouxi* possibly after replication.

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