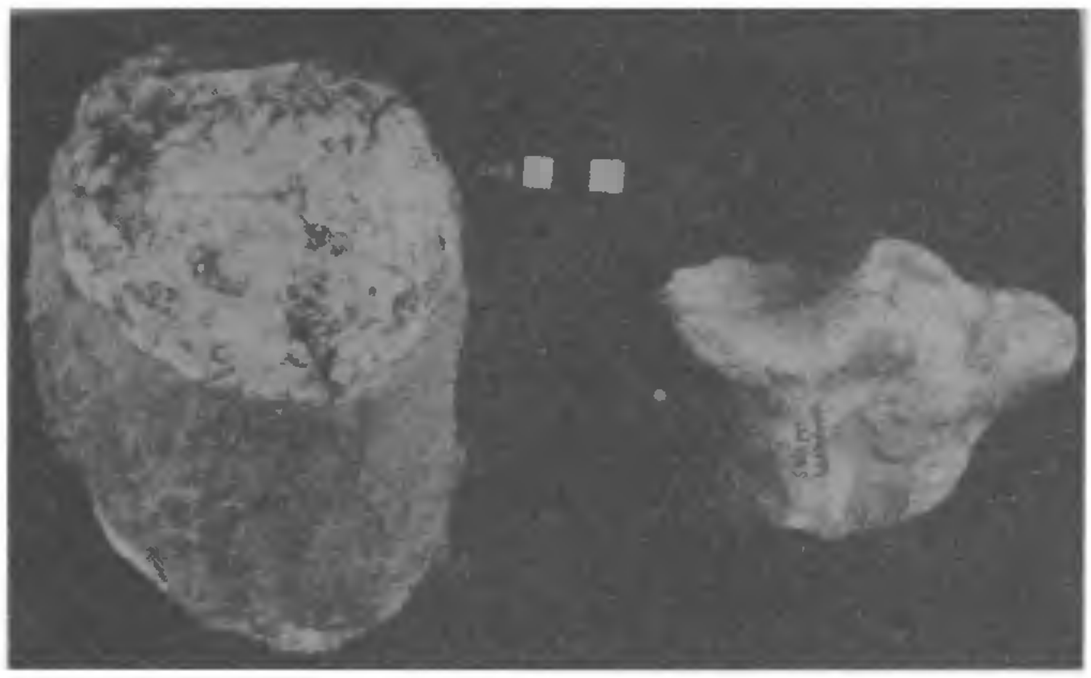

**Figure 3.** Fossilized tusk of *Elephas* sp (left) and Scapula of *Hexaprotodon* sp (right) from Yedurwadi.

**Figure 4.** Acheulian artefacts from Yedurwadi.

and four flakes (two side- and two end-flakes). The discoidal and amorphous cores have small and big flake scars with deep to shallow negative bulb of percussion. The flakes are thick with secondary flake scars on the dorsal side. As a whole the assemblage represents Acheulian, perhaps late Acheulian.

Geomorphologically, the older deposits occur in the form of an inlier, surrounded from three sides by mid- to late-Holocene sediments in a localized depression of a valley pediment, drained by the link-channel of river Krishna. On the basis of the fossil-fauna from the site and radiometric dates of the older deposits from other parts of Western Upland Maharashtra[4], the fossiliferous and implementiferous sediments of Yedurwadi can be tentatively assigned a late Pleistocene age.

The finding of hippo from Yedurwadi, is a noteworthy addition to the faunal list of Western Deccan Volcanic Province, as it extends the geographical extent of the animal further south of river Ghod.

The discovery of the Acheulian artefacts from the Deccan Trap region of Upper Krishna valley proves the potentiality of the area. Nonetheless, the specific typological and chronological interpretations must await the discovery of primary sites and datable assemblage from this part of the Krishna valley.

2 April 1986; Revised 16 June 1986

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## INCIDENCE OF G-6-PD DEFICIENCY IN THE VARIOUS ETHNIC GROUPS OF JAMMU REGION (JAMMU AND KASHMIR STATE)

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GLUCOSE 6-phosphate dehydrogenase (G-6-PD) is an important enzyme found in the human red cells, granulocytes, lymphocytes, platelets, liver cells, etc. It catalyzes the direct oxidation in the pentose phosphate pathway, converting glucose-6-phosphate to 6-phosphogluconate and maintaining glutathione in the reduced state (GSH). The latter is vital for protecting haemoglobin, red-cell membrane and enzymes of the red-cells from oxidative damage[1]. The deficiency of this enzyme in red blood cells is

known to be of greater clinical significance than in other cells. G-6-PD deficiency subjects are not only susceptible to primaquine and other 8-aminoquinoline antimalarial drugs but also to a number of other drugs, fava beans and certain stresses including infections and diabetic acidosis[2]. G-6-PD deficiency varies greatly in its prevalence throughout the world. The present study reports the occurrence of this disorder in the population of Jammu region of Jammu and Kashmir State.

The study involved about 2,000 apparently healthy subjects belonging to various ethnic groups of the region under investigation. The blood samples were analyzed by Methaemoglobin reductase test[3] and further confirmed by fluorescent spot test[4] and dichlorophenol indophenol colour change[5] methods.

Of the 2,000 subjects investigated 168 were found to be deficient in G-6-PD enzyme. This included 151 males and 17 females. Hindus exhibited the maximum frequency of deficient subjects (9.2%) followed by Muslims, Sikhs and Christians (table 1).

The present study revealed a higher percentage of the G-6-PD deficient gene (about 8.4%). In the literature the prevalence of this deficiency is reported to range from 0.6 to 19% in different parts of the country. A study at Chandigarh shows that the deficiency in the Punjab[6] is about 6.9%. The overall incidence of the deficiency is reported to be higher among South Indians than in North Indians[7]. However, the maximum incidence of this deficiency in the world is reported in the populations around the Mediterranean[2] Sea (Italy 33%, Greece 32%). It is likely that this "Mediterranean" G-6-PD deficiency gene had its origin in the north and then spread throughout the adjoining regions. This hypothesis finds support even in India as most of the foreign invasions on India were from the Northern regions.

**Table 1** *Distribution of G-6-PD deficiency in various ethnic groups of Jammu region*

| Ethnic group | No. of samples investigated | No. of Individuals with G-6-PD enzyme deficiency | | | Deficiency (%) |
|---|---|---|---|---|---|
| | | Male | Female | Total | |
| Hindus | 1,250 | 105 | 10 | 115 | 9.20 |
| Muslims | 342 | 22 | 4 | 26 | 7.60 |
| Sikhs | 322 | 20 | 2 | 22 | 6.83 |
| Christians | 86 | 4 | 1 | 5 | 6.87 |
| Total | 2,000 | 151 | 17 | 168 | 8.40 |

The above data confirms the earlier reports[8,9] that this trait of deficiency of the enzyme is greater in males than in females except for a single report[10] to the contrary. This is obvious as the gene for this enzyme is present on the X-chromosome.

The probable cause for the maintenance of the higher frequency of the G-6-PD deficient gene appears to be malaria. It appears that the deleterious effects of both thalassaemia and G-6-PD deficiency are balanced in the population by a conferred protection against malaria. The hypothesis is supported by population data by various earlier workers[11–13]. However, Friedman[14] is of the opinion that some characteristics of the variant red-cell act directly to inhibit the invasion or intracellular development of the malarial parasite.

9 December 1985; Revised 24 July 1986


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