Reports

Age of Bed V, Olduvai Gorge, Tanzania

Abstract. Various finds of hominid remains in the Olduvai Gorge, Tanzania, have focused interest upon the age of the deposits in the sequence of six beds. After bed I was dated by the potassium-argon decay method, an absolute date of 10,400 years has now been obtained with the radiocarbon method from a sample of mammalian bones for bed V.

Previous age determination by the K^{40} -Ar⁴⁰ decay method (1) and subsequent fission-track dating (2) have yielded similar dates for the deposits of bed I of the Olduvai Gorge in northern Tanzania. These techniques were used on geological deposits of various types of tuff associated with the fossil remains in this bed (Table 1). However, C¹⁴ dating can be applied directly to the bone samples collected from bed V.

Locally, in a few places, this bed contains mammalian bones and also rare mollusks of a species of *Limno*colaria (3) found today in arid regions of Tanzania. The sample dated consists of bones from the fawn-colored aeolian deposit of bed V on the northern side of Olduvai Gorge where it is cut obliquely by the fifth fault. At that particular site area, bed V is about 14 feet (4.60 m) thick, but a large part of the top portion of the aeolian deposit and the overlying caliche have been eroded.

It is this feature which made the collection of samples possible. Bed V is a geologically very young deposit and underlies the main body of caliche which separates it from bed VI, which consists of black volcanic ash. The C¹⁴ date establishes the upper limit in age for the later upper Capsian and also the date of the dry period succeeding the last phase of the Gamblian pluvial (4). Bed V overlies with angular unconformity the older beds I to IV in many places and occasionally even the basaltic lava underlying bed I. It filled the mature valley created after faulting and cutting during the

1 NOVEMBER 1968

Upper Pleistocene and rests upon a late upper Kenya Capsian industry.

The sample of fractured mammalian bones obtained from bed V was slightly covered by caliche. The specimens were not appreciably fossilized, and their hollow parts were completely filled with sand. Detailed faunal classification of the sample was not available.

Approximately 2 pounds (900 g) of bone were first washed in distilled water and subsequently analyzed by a recently developed method of collagen isolation (5). The bones are placed in 1.0N HCl at room temperature; the mineral matter is dissolved and the collagen is insoluble. Since the mineral matter did not entirely dissolve in the bed V sample, further treatment with more concentrated HCl completed the process. The insoluble mineral matter was then filtered off with the collagen remaining on a glass-paper filter. The isolated collagen was left overnight in 1.0N NaOH at room temperature to remove alkali-soluble contaminants, such as humic acids. After filtration on a Büchner funnel and washing with distilled water, the sample was dried in an oven.

After combustion, the radioactivity of the sample was counted as UCLA-1321 for 4215 minutes as CO_2 gas at 1 atm in a proportional counter (7.5 liters). The error listed is based on a one-sigma statistical counting error. The date was then calculated on the basis of a 5730 \pm 40-year half-life, giving an increase of approximately 3.0 percent in age over calculations based on the older value of the half-life of 5568 \pm 30 years. The sample showed an age of 10,400 \pm 600 years.

Examination of the dates in Table 1 shows that, in addition to the basal lava, only the ages of two beds are actually known. It should be noted that a prior estimation of the age of bed II by the K^{40} -Ar⁴⁰ method at 490,000 years may have to be revised (6).

The dating of bed I by K^{40} -Ar⁴⁰ and fission-track dating made possible the establishment of a plausible age for the Upper Villafranchian. The UCLA-1321 radiocarbon sample now gives an age for the termination of the Upper Pleistocene and beginning of the Holocene at Olduvai that is in good

Table 1. Dating of deposits from several beds of the Olduvai Gorge in northern Tanzania (3).

| Bed | Geological sequence | Absolute age (yr) | Dating method | Major find |
|---------------|--------------------------------|-------------------------------------|----------------------|--|
| VI | Holocene | | | |
| Caliche | Holocene | | | |
| V | End of Upper Pleistocene | 10,400 ± 600 | Radiocarbon | Homo sapiens sapiens "Oldoway Man" |
| Va | Upper Pleistocene | | | |
| IV | Upper to Middle Pleistocene | | | Homo sapiens neanderthalenis "Acheulian Man" |
| ш | Middle Pleistocene | | | |
| II | End Villafranchian | | | Australopithecus Homo habilis Pithecanthropus "Chellean Man" (Homo leakeyi) |
| I | Villafranchian | $2.03 \pm 0.28 	imes 10^6$ | Fission-track (2) | Australopithecus Zinjanthropus boisei |
| Lava Tuffs | Pliocene | $1.75 	imes 10^6$ $4 	imes 10^6$ | K-Ar (3) K-Ar (3) | Homo habilis (Pre-Zinjanthropus) |

agreement with dates for that event from other continents. It also establishes the Holocene age of bed VI overlying the caliche above bed V (7).

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Ouabain Hypoglycemia: Insulin Mediation

Abstract. The fall in blood sugar occurring during infusion of ouabain (1 microgram per kilogram per minute for a 60-minute period) in dogs is accompanied by an increased uptake of glucose and potassium by the liver. Concurrently, plasma insulin in the portal blood increases significantly. This increase appears to be a result of increased insulin secretion caused by ouabain.

The hypoglycemic effect of ouabain in the dog has been described (1). This hypoglycemia was reportedly more marked when ouabain was combined with insulin than when either drug was administered separately (2), and it was not present in pancreatectomized animals (3). The purpose of our experiments was to determine whether there is an interaction of ouabain with the insulin in the blood, or whether ouabain



Fig. 1. Differences in glucose and potassium concentration between plasma of portal and hepatic vein blood in six dogs after intravenous administration of ouabain (1 μ g kg⁻¹ min⁻¹ for 60 minutes).

causes an increase in plasma insulin levels.

Laparotomies were performed on 12 mongrel dogs, anesthetized and prepared as described (4). A polyvinyl catheter was then placed into the portal vein through a branch of gastrosplenic vein without disturbing the stem of the portal vein. Another catheter was inserted under fluoroscopy through the right jugular vein into a hepatic vein. Catheters were also placed in a femoral vein and artery. After a control period of 30 minutes, ouabain $(1\mu g \text{ kg}^{-1} \text{ min}^{-1})$ was administered intravenously (femoral vein) for 60 minutes. The experiment was continued for 60 minutes after the end of ouabain administration. Blood samples were taken at regular intervals from portal vein, hepatic vein, and femoral artery for measurements of the concentrations of glucose, potassium, and insulin. The insulin was quantitated by radioimmunoassay (5) and the result is expressed in terms of a porcine standard (6) by the use of a guinea pig antiserum which reacted essentially identically with porcine and canine insulin.

After ouabain administration, the glucose in arterial blood decreased from 103 to 80 mg per 100 ml (-23)percent, P < .05). The portohepatic difference (+6 mg per 100 ml) in plasma glucose during the control period indicates a net output, as expected in fasting animals. After ouabain administration, this trend was reversed. The portohepatic difference, amounting to 13.6 mg per 100 ml (P < .05), indicates a net uptake of glucose by the liver (Fig. 1). Changes in the concentra-

tion of potassium in the plasma paralleled the changes in glucose concentration: the portohepatic difference increased significantly in comparison with control (-0.4 meq/liter, P < .05). Other results indicating the role of insulin in the mediation of the hypoglycemia caused by ouabain have been reported (3).

The amount of insulin in the plasma of the portal blood during ouabain infusion increased significantly (by 125 percent, P < .05) in comparison with the concentration in plasma of portal blood of the control group (Fig. 2). An hour after the end of the ouabain infusion, plasma insulin of portal blood tended to return to control values.

In the plasma of arterial blood of controls, the initial concentrations of insulin were lower than those found in the plasma of portal blood, as expected from the inactivation of insulin by the liver (7). The increase of plasma insulin in peripheral blood after ouabain administration, although consistent, was lower (increase of 77 percent as compared to 125 percent) than that observed in portal blood. These observations indicate that the increased amount of insulin in the plasma during ouabain administration is not due to a change in





SCIENCE, VOL. 162