

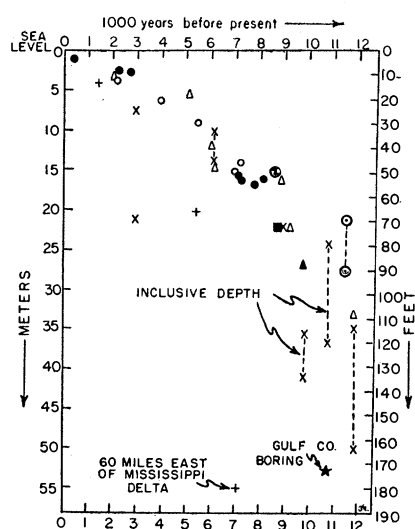


Fig. 1. Radiocarbon ages relative to depths below present sea level. Samples of wood and shells are believed to have been deposited essentially at sea level. No correction has been made for compaction or subsidence from other causes. ■, wood (Suess, U.S. Geological Survey, Australia, 9); ○, shells, and ●, peat (deVries and Barendsen, Holland); △, shells, and ▲, wood (Magnolia Petroleum Laboratory, Texas Gulf); +, shells, and ×, wood (Kulp, Lamont Geological Observatory, Mississippi Delta); ⊙, wood (Kulp, Bermuda); ★, shells (Humble Oil Company, Texas Shelf).

the various localities. This may indicate that the observations reflect the actual eustatic change that accompanied the melting of the last ice sheets rather than the effects of local tectonic movements or compaction. It is obvious, however, that many more measurements from a variety of locations must be made before it will be possible to distinguish conclusively between eustatism, tectonic movements, and compaction.

A possible temporary halt in the rise of the sea level some 7000 or 8000 years ago seems to be indicated by samples from both North Holland and the Gulf Coast. Such a halt may conceivably correspond to the Cochrane halt in the retreat of the ice (5).

The picture becomes confused prior to the last 10,000 years, which may be due to imperfection of the data, or, possibly, to an oscillation of the sea stand connected with the Mankato readvance of the ice sheets around the Great Lakes (6). Evidence for or against changes in the rate of eustatic rise in correlation with oscillations of the ice sheets might prove helpful in determining whether or not glacial readvances were synchronous on different continents and in the two hemispheres.

Perhaps the most interesting question connected with these observations is that

of the supposed postglacial higher sea stand during the "climatic optimum" (7). Neither the samples from the Gulf Coast nor those from North Holland give any indication of a postglacial sea level higher than that of the present. As yet no evidence from carbon-14 measurements has been produced that would prove such a high sea level, and a sample (W-185) of shells from one of the low terraces from Western Australia (+ 10 to 12 ft), which was expected to date this sea stand, gave an age of more than 30,000 years.

Any assistance in securing samples for radiocarbon determinations that would date a sea level as accurately and as conclusively as possible and hence give a bearing on the afore-mentioned problems would be greatly appreciated.

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7. Plant fossils indicate that during a short period about 5000 years ago the climate may have been warmer than it is now. See R. F. Flint, *Glacial Geology and the Pleistocene Epoch* (Wiley, New York, 1947), pp. 187-191.
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21 December 1955

Lactose in the Sapotaceae

Lactose has been reported (1) to occur in the tropical fruit *Achras sapota*, but the supporting evidence is minimal. In order to confirm this report, a preliminary investigation (2) has been made in this laboratory on the fruits of the aforementioned species as well as on the related species *Mimusops roxburghiana* and *Ponteria campechiana* (3).

Samples of 30 to 40 g (wet weight) of the fresh fruit were extracted with 50-percent ethanol until extraction of sugar was complete. The total amount of sugar present was determined as glucose by the anthrone method (4). The bulk of the sugar present (glucose) was removed by fermentation with washed *Saccharomyces cerevisiae*. Extracts of all three fruits, when examined chromatographically (butanol, pyridine, and water, 6 to 4 to 3), showed evidence of a compound

Table 1. Lactose content of fruit of *Achras sapota*, *Ponteria campechiana*, and *Mimusops roxburghiana*.

Plant	Dry wt. (% of wet wt.)	Total sugar (% of dry wt.)	Lactose (% of total sugar)
<i>A. sapota</i>	25	74	0.023
<i>P. campechiana</i>	33	69	0.043
<i>M. roxburghiana</i>	41	38	0.028

having the same R_f as a lactose standard. In order to remove interfering compounds, the extracts were adsorbed on charcoal-celite columns (5), which were then washed with water to remove salts and monosaccharides. Lactose and small amounts of higher sugars were eluted with 50-percent ethanol.

In order to confirm the presence of lactose qualitatively and to effect a quantitative estimation, we employed a purified preparation of *Escherichia coli* beta-galactosidase (6). This enzyme was devoid of alpha-glycosidase activity. The glucose that was split from the lactose by enzyme action was estimated spectrophotometrically with a Beckmann model DU instrument. Into a silica cuvette was pipetted 0.8 ml of buffer [0.1M tris(hydroxymethyl)aminomethane at pH 7.2 and $10^{-3}M$ MgCl₂], 30 μlit of Zwischenferment (5 mg/ml), 20 μlit of TPN (8 mg/ml), 10 μlit of ATP (50 mg/ml), and 50 to 100 μlit of the sample. At this stage, any trace of glucose in the sample was detected by the increase in optical density at 340 mμ. The beta-galactosidase was then added (20 μlit) by micropipette, and the changes in optical density were followed until the rate became negligible. In this procedure, 36 μg of lactose caused a rise in density of 0.560 in 15 minutes. The theoretical value of 0.620 could be obtained by using more enzyme and a longer observation time. Only a sugar containing glucose bound in a beta-glycosidic linkage will be assayed by this procedure.

It is evident (Table 1) that lactose is present only in small quantity but is readily determinable. About 10 to 20 mg would be present in a whole ripe fruit. It is our intention to examine fruits of other members of the Sapotaceae and to inquire into the mode of lactose formation in them.

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References and Notes

1. R. Ulrich, *La Vie des Fruits* (Masson, Paris, 1952).
2. This work was performed under contract No. AT(45-1)-507 between the U.S. Atomic Energy Commission and the University of Oregon.
3. These fruits were obtained through the good offices of the Agricultural Experiment Station,

- Paramaribo, Surinam, and the Sub-Tropical Experiment Station, Homestead, Fla.
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Effect of Growth Hormone and Acromegaly on Plasma Phospholipid

We have used the technique of labeling with radiophosphorus to study the effect of administered anterior pituitary growth hormone and of acromegaly on plasma phospholipid. In both cases there is a lowering of the rate of plasma phospholipid formation, if "formation" is defined as the sum of metabolic processes and membrane transfers involved in the entry of newly-formed phospholipid into the blood.

Although it appears that plasma phospholipids are made and destroyed in the liver, these processes are independent of severe liver injury. Experiments in man, by Cornatzer and Cayer (1), have demonstrated a constant rate of phospholipid formation in any individual in spite of time and change in liver function. Among individuals, however, they found a wide normal variation. Zilversmit's (2) experiments with adrenalectomized dogs have focused attention on endocrine control of the liver-plasma phospholipid system.

In our experiments, 100 to 400 μ c of labeled inorganic phosphate were given intravenously, and plasma samples were drawn at 15, 24, 40, and 60 hours. The plasma was extracted with trichloroacetic acid (TCA), and the relative specific activity of phosphorus in both the TCA-soluble fraction and phospholipid fraction was determined. These values were plotted against time; both the curves obtained and the average value of stable phospholipid phosphorus were considered. When the effect of growth hormone was studied, each patient acted as his own control, with pretreatment and treatment curves being determined at an interval of 1 week. Studies were also made on a series of acromegalic patients to show the effect of excess endogenous growth hormone.

Figure 1A shows composite curves from six endocrinologically normal patients, each of whom had two series of determinations with no intervening

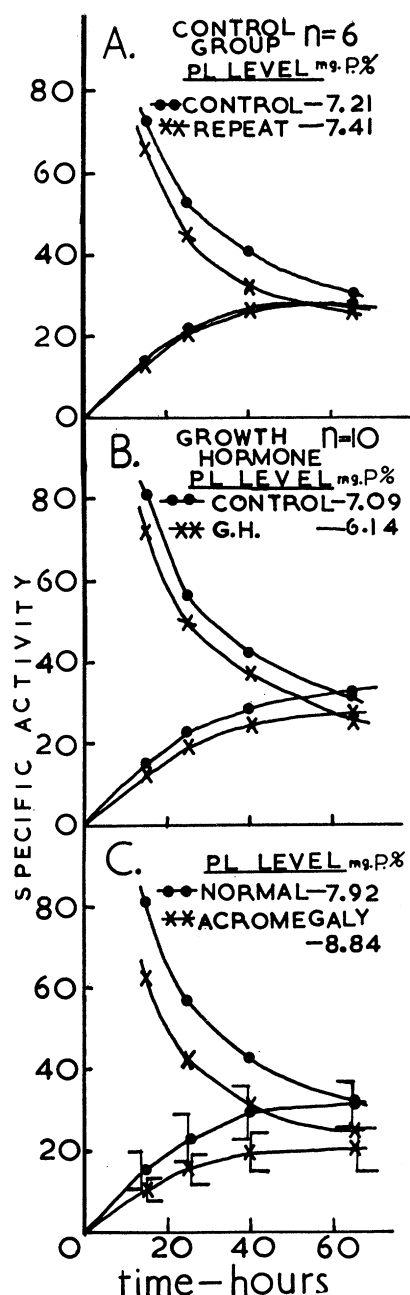


Fig. 1. The specific activity is expressed as a ratio of the percentage of the dose in counts per minute $\times 10^{-4}$ divided by milligrams of phosphorus.

treatment. The rising curves show specific activity of the phospholipid fraction; the falling curves show that of the TCA-soluble fraction. In each case the two phospholipid curves were identical. This confirms the constancy of the curve and the validity of repeating the test after such a short interval. The figure also shows that the TCA-soluble and

phospholipid curves intersect at their peaks, giving a Zilversmit product-precursor relationship. There is no significant change in the total phospholipid-phosphorus values.

The effect of the intramuscular administration of growth hormone is shown in Fig. 1B. Ten patients received 100 mg daily in four divided doses. The treatment curve is flatter than the control curve, and the plasma-phospholipid phosphorus is significantly decreased. These changes, both of curve and level, were constant in direction in each patient studied and indicate a decrease in the rate of plasma-phospholipid formation. This decrease was the only metabolic effect of growth hormone observed. There was no clinical change, nitrogen retention, or alteration in glucose tolerance.

The study of the effect of increased secretion of growth hormone was made in a series of 16 patients with typical acromegaly. The results are shown in Fig. 1C, in which a composite curve for 20 control patients is compared with a composite curve for 16 acromegalics. The crossbars represent the standard deviation for each point in time. The phospholipid curves of the acromegalics are lower, and the specific activity curve of the TCA-soluble fraction does not intersect the peak of the phospholipid curve as it does in normals. This was true in each individual case. The *F* test of variance showed that the difference between the groups is highly significant. The total phospholipid-phosphorus values were not abnormal.

The agreement between the results obtained with administered growth hormone and those found in acromegaly suggests that growth hormone decreases the rate of formation of plasma phospholipid. Since the total value does not fall in acromegaly, the destruction of plasma phospholipid must also be slow, indicating a slow phospholipid turnover in this condition. This is the only constant metabolic effect of growth hormone thus far demonstrated in man.

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12 December 1955

The person who won't take advice isn't necessarily any more stubborn than the one who is offering it.—ANON.