Table 2. F_2 segregation for fruit color and high pigmentation.

	Normal pigment			High pigment			
	Red	Yellow	Tangerine	Red	Yellow	Tangerine	Total
Webb Special × *Snowball*							
Observed	174	57		48	17		296
Expected	9	3		3	1		
$\chi^2 = 1.514$, 3 degrees of freedom, $P = 0.50–0.70$							
Webb Special × *Orange King*							
Observed	69		25	7		1	102
Expected	45		15	3		1	
$\chi^2 = 1.404$, 3 degrees of freedom, $P = 0.70–0.80$							

In addition, the fruits of the high-pigment selections exhibit a high degree of firmness. Whether this is because of a close association of independent genetic factors or because of a pleiotropic effect of one or both of the high-pigment genes has not yet been determined.

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21 February 1955.

Unusual Value for Protein-Bound Iodine in the Serum of the Opossum

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Apparently the last comparative study of serum protein-bound iodine (PBI) was made by Taurog and Chaikoff in 1946 (*1*). Since then the analytic procedure has been changed radically (*2*). Moreover, recourse to the *Handbook of Biological Data* (*3*) reveals no information concerning PBI values for the rabbit, the hamster, the sheep, or the opossum. Therefore, it was deemed important that the knowledge concerning, normal PBI values (the easiest obtainable assessment of circulating thyroid hormone) in experimental animals be extended and brought up to date. Our studies reveal that the level of PBI in the serum of the opossum is extremely low. This article is concerned with certain aspects of this result. In addition, the PBI values that we have obtained for the rabbit and the hamster supply information that was previously lacking.

Blood was obtained by cardiac puncture from three monkeys (*Cynomolgus*), four rabbits, 15 rats (Sprague-Dawley), five hamsters (*Cricetus auratus*), three opossums (*Didelphis virginiana*), three white leghorn chickens, and five guinea pigs. Jugular blood was withdrawn from three stallions directly upon slaughter at a local slaughterhouse and from a ewe housed in our animal room. In each case the respective serums were pooled and several analyses (from three to seven) were performed in duplicate upon the specimens, which were stored at 4°C between determinations. All animals were in the postprandial state when examined, and the season was summer. The method of analysis was essentially that of Barker *et al.* (*2*).

Table 1 provides the results of the analyses for PBI in the serums of the various animals used. It can be noted that the opossum serum was strikingly low in PBI. Values found for the rabbit and hamster (two specimens for which values do not appear in the literature) were in the so-called normal range. The values observed for the chicken are in good agreement with those reported by Taurog and Chaikoff (*1*). The concentration of PBI found in the rat serums are in the range noted by Taurog and Chaikoff (*1*), Halmi and Barker (*4*), and Klitgaard *et al.* (*5*). Serum PBI values for the guinea pig and the sheep are in close agreement with those disclosed by Young *et al.* (*6*) and Weeks *et al.* (*7*), respectively. Values for the horse and the monkey are in the range indicated in Albritton's *Handbook* (*3*). Thus it is clear that the technique is reliable and reproducible in our hands. Human blood samples analyzed also gave values in the range noted by Barker *et al.* (*2*) to be normal.

It is surprising to note that the opossum possesses a very low protein-bound iodine level (0.4 μg percent). In the absence of other data in the literature, it is pertinent to note that Hartman (*8*) calls attention to the fact that the body temperature of the opossum is peculiarly low—about 95° to 97°F, as established by Selenka in 1887 and more recently by Wislocki (both referred to by Hartman). Burke (*9*) also found that the rectal temperature of the opossum is about 95°F. Hartman points out that with this body temperature, the opossum alone "fails to fit into the chart of body size-temperature relationship of the animal series from the finger-sized shrew to the elephant" and that there is no explanation for this instance of nonconformity.

Immediately, therefore, one is struck by the association pointed out here between the low PBI values (reflecting a low titer of circulating thyroid hormone) and low body temperature. A comprehensive study of

Table 1. PBI values obtained from the serums of several animals.

Animal	PBI (μg percent)
Horse	3.6 ± 0.4
Monkey	6.0 ± 0.7
Sheep	3.7 ± 0.3
Rabbit	3.3 ± 0.5
Rat	4.5 ± 0.4
Guinea pig	2.5 ± 0.5
Hamster	3.5 ± 0.4
Opossum	0.4 ± 0.2
Chicken	2.6 ± 0.3

thyroid-hypophyseal histology and physiology in the opossum appears to be well worth the undertaking, for the host of questions raised by the afore-mentioned association indicates a fundamental problem of singular importance. Is it possible that there is a low level of hypophyseal-thyroid interaction but that the tissues of this animal are adapted to small amounts of thyroxine so that their efficiency is maintained? (The opossum is sedentary but not sluggish.) Are the oxidative metabolic and enzymatic rates of the tissues of this marsupial different from those of other mammals? Experiments are being designed to elucidate these points regarding the hypothyroid animal with the hope that clues may be provided that might be important in human thyroid physiology.

References and Notes

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28 February 1955.

Synthesis of Oligosaccharides from Maltose by Rat Liver

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Enzymes that can synthesize oligosaccharides from maltose are known to be present in molds and bacteria. As far as we are aware, no such enzyme system has been shown to occur in animal tissues. Recently, however, it has been shown that an enzyme that can transfer a glucose residue from maltose to flavins (*1*) is present in rat liver. We have observed that the rat liver contains an enzyme system that can convert maltose by transglucosidation into oligosaccharides with simultaneous liberation of glucose.

The enzyme was obtained in an amorphous powder form by fractional precipitation of the liver homogenate with alcohol after the amylase had been removed by adsorption on starch. The fraction obtained between 50 and 80 percent alcohol concentration was centrifuged and was dried with cold acetone and ether. The enzyme (20 mg) was incubated at 37° ± 2°C with 1 ml of 20-percent maltose in 0.02*M* acetate buffer at *p*H 5.0. The progress of the reaction was followed by circular paper-chromatographic technique, as is described by Giri and Nigam (*2*). Figure 1 shows the formation of oligosaccharides from maltose at varying periods of incubation.

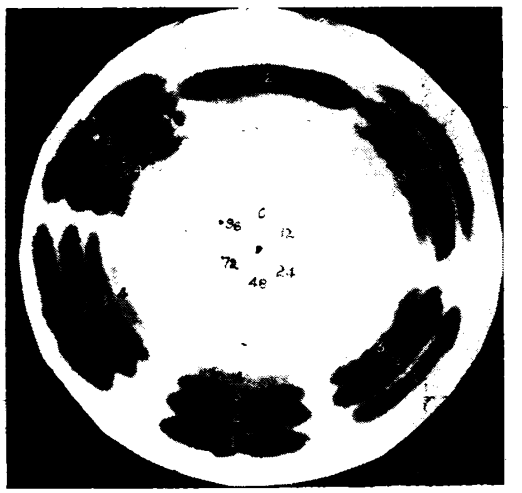

Fig. 1. Circular paper chromatogram, showing the synthesis of oligosaccharides from maltose by the enzyme prepared from rat liver. The numbers on the inner circle indicate the time of incubation in hours. Whatman No. 1 filter paper (18 cm in diameter) was used. Developing solvent: *n*-butanol, pyridine, and water (6: 4: 3); double development. The reagent used was 2-percent triphenyltetrazolium chloride in water-saturated butanol mixed with an equal volume of N alcholic potash. Band 1 indicates glucose; band 2, maltose; band 3, maltotriose; and band 4, maltotetraose.

The two oligosaccharides formed have been identified as maltotriose and maltotetraose; they were identified by our running chromatograms with known reference substances in different solvents and by determining the papergram mobilities. The oligosaccharides yield only glucose on hydrolysis, thereby showing that they contain only glucose units. The two oligosaccharides were isolated by charcoal-celite column chromatography, as is described by Whistler and Durso (*3*), and confirmed as maltotriose and maltotetraose by (i) optical rotation, (ii) partial hydrolysis with 0.1*N* sulfuric acid, (iii) preparation of acetyl derivatives, (iv) periodate and hypoiodite oxidations, (v) hydrolysis by β-amylase, and (vi) priming capacity for phosphorylase (green gram) action. Other disaccharides—lactose, sucrose, cellobiose, and isomaltose—did not show the formation of oligosaccharides. The rat brain homogenate also was found to contain the enzyme.

It is interesting to note that rat liver contains an enzyme system that brings about the synthesis of oligosaccharides containing 1 : 4 glucosidic links only, whereas the mold enzyme systems synthesize oligosaccharides having both 1 : 4– and 1 : 6– glucosidic links. It is probable that the formation of 1 : 4 and 1 : 6 links are caused by the action of two different enzymes.

A more detailed report of this investigation is in preparation.