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Galactose Metabolism in the Sea Lion.* (31554)

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Galactosemia is characterized by the lack of galactose-1-P uridyl transferase (UDP glucose: α-D-galactose-1-phosphate uridyl transferase 2.7.7.12), an important enzyme of galactose metabolism. When infants afflicted with this inborn error of metabolism are fed galactose, they develop cataracts, cirrhosis, malnutrition, and mental retardation. The milk of the sea lion is devoid of carbohydrate and hence, galactose(1). Sunshine and Kretchmer have shown that neither of the disaccharides lactose or sucrose are absorbed by the sea lion intestine, but that glucose is absorbed and apparently metabolized(2). It seemed possible that this animal had lost its capacity to metabolize galactose and might serve as an admirable model for study of the pathogenesis of galactosemia in man.

Materials and methods. Two California sea lions (*Zalophus californianus*) were investigated. One weighed 12 kg and was approximately 3 months of age; the other weighed 32 kg and was approximately 16 months old. Galactose-1-phosphate uridyl transferase was assayed by our modification of the UDPG consumption assay(3). Galactokinase (ATP: D-galactose-1-phosphotransferase 2.7.1.6) activity was estimated by measuring the phosphorylation of galactose-1-C14 by a minor modification of the system described by Ng *et al*(4). Blood galactose levels were determined by the method of Roth *et al*(5).

Results. The galactokinase and galactose-1-phosphate uridyl transferase activities of the red cells of the infant and sub-adult sea lions are presented in Table I and are compared with values as obtained on normal human subjects. Liver tissue from the infant sea lion was also examined for galactokinase and galactose-1-phosphate uridyl transferase activity. High levels of activity of both enzymes were found, but the procedures used were not adequate for quantitation of enzyme activity in this tissue.

The sub-adult sea lion (32 kg) was given 0.91 g of galactose/kg body weight and serial estimations of blood galactose levels were made on blood obtained by flipper puncture. The results of these studies are shown in Fig.

TABLE I. Galactokinase and P-Gal Uridyl Transferase Activity in Human and Sea Lion Blood Samples.

Specimen		Galactokinase activity, μMole Gal-1-P formed/hr/g Hb	P-gal uridyl transferase activity, μMole UDPG consumed/hr/g Hb
Sea lion	Infant	1.46	11.11
	Sub-adult	1.53	8.62
Human	Infant	2.40*	24.0 (approx.)
	Adult	.91*	24.0

* Average value from Ng, Donnell and Bergren (4).

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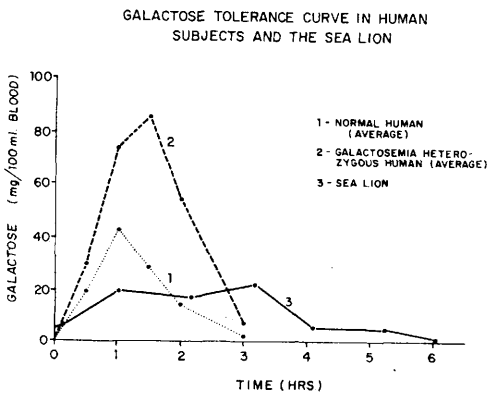

FIG. 1. Galactose tolerance curves in human subjects and a sea lion. Galactose levels after administration of .91 g of galactose/kg body weight are compared with results reported on human subjects receiving approximately twice this dose of galactose.

1 and are compared with average normal galactose tolerance tests and tests on heterozygotes carried out under similar conditions in human subjects by Donnell *et al* (6). The capacity of the sea lion to metabolize galactose is clearly demonstrated by these studies, the curve returning to baseline levels after 4 hours. The somewhat prolonged elevations of blood galactose levels may be due to the long intestinal tract of this species, resulting in long-continued absorption of the monosaccharide.

These studies indicate that although, unlike most other mammals, the infant sea lion is not presented with a large quantity of dietary galactose, the enzymes of galactose metabolism remain intact. Unlike the human, in which galactokinase activities are several-fold higher in infants than in adults(4), sub-adult sea lion galactokinase activity was approximately the same as that found in the infant sea lion. This suggests that the persistence of the enzymes of galactose metabolism in the sea lion may be a necessity because of the relatively high galactose content of mucopolysaccharides of various aquatic organisms (7,8). These findings indicate that the sea lion is not a suitable species to serve as a model for the study of the pathophysiology of galactosemia.

Summary. Studies on the enzymes, galactokinase and galactose-1-phosphate uridyl transferase have shown that these enzymes are present in the blood and liver of sea lions. A galactose tolerance test on a sea lion has revealed that these animals are able to metabolize galactose.

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Aortic Ruptures in Turkeys Induced by Diethylstilbestrol and Dienestrol Diacetate.* (31555)

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The administration of diethylstilbestrol (DES) to turkeys by either the parenteral (1,2) or oral(3) routes causes mortality from aortic ruptures. The possibility that other synthetic estrogens might induce aortic rhexis in turkeys has not been investigated. This paper describes experiments which were conducted to determine if dienestrol diacetate (DD) was comparable to DES in ability to cause such lesions.

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Materials and methods. Broad-Breasted