Lactose Intolerance in Captive Nocturnal Prosimians
(*Perodicticus potto*): A Twenty-one Year Record

U. M. COWGILL and S. J. STATES

*University of Pittsburgh*

**ABSTRACT.** A colony of pottos was captured in December 1959 and brought to the United States. At that time part of their diet consisted of a high protein porridge made with whole milk. In addition, their drinking water contained an antibiotic to protect them from possible infection resulting from the change in habitat. No intestinal incontinence resulted from this treatment. Antibiotic addition to the diet ceased in 1961. In 1971 the remaining pair of animals developed a calcium deficiency. This was alleviated by adding 5 g calcium lactate to the drinking water and serving whole milk every other day. In 1977 the male developed acute lactose intolerance. His feces became bacteriologically sterile. His mate, who died on January 9, 1979, produced feces containing enteric organisms throughout her life. A large number of dairy products were served the animals in the attempt to alleviate this problem. Acidophilus milk or tinned 2% milkfat containing milk with 100% of the lactose hydrolyzed, produced no intestinal incontinence in the animal. It is suggested that sterile stools resulted from a fungal infection that killed the *Escherichia coli*. In addition it is proposed that non-pathogenic bacteria brought about an alteration of the brush border of the columnar epithelial cells of the villi which synthesize the enzyme lactase, such that lactase production was seriously reduced.

**INTRODUCTION**

Lactase is synthesized in the brush border of the columnar epithelial cells of the villi. The two sugars, glucose and galactose, are absorbed by the intestine, ultimately passing into the blood stream. When the enzyme lactase is unavailable or is present in insufficient quantity to reduce the lactose into the two monosaccharides, the undigested lactose is carried to the colon where it undergoes fermentation by intestinal bacteria. This process eventually produces carbon dioxide, lactic acid and other short-chain acids. The ultimate result is intestinal incontinence. A review of this whole process has been published by KRETCHMER (1971a, b).

A number of hypotheses have been put forward to explain the origin of lactose intolerance. Some have suggested that lactase synthesis may be an adaptation resulting from the consumption of dairy products (CUATRECASAS, LOCKWOOD & CALDWELL, 1965; WEN et al., 1973). Other studies have been unable to confirm this suggestion (LANG-WIRZ, 1977; KLOTZ, 1964;HUANG & BAYLESS, 1968). A genetic interpretation has most recently been advanced. A detailed review is presented by McCracken (1971) and Simoons (1980). There is much discussion on the etiology of lactose intolerance but there is relatively little information on the development of lactase insufficiency in adulthood after many years of milk consumption. This type of problem is clearly not genetic.

The purpose of this paper is (1) to describe the case of one male *Perodicticus potto ibeanus* THOMAS, 1910 who developed a severe lactose intolerance problem after 18 years of milk consumption; (2) to describe the resolution of this problem; and (3) to offer an hypothesis as to the origin of this metabolic anomaly.
MATERIALS AND METHODS

The animals were fed only milk for one day. The feces from both animals were collected immediately after production.

Lactose intolerance may be ascertained from the results of three different types of examinations. Lactose tolerance is indicated by a neutral pH. The female potto's fecal material always exhibited an hydrogen ion concentration between 7 and 8. That of the male's fecal material was 5.4. This acidity results from the ultimate bacterial breakdown of lactose within the colon into lactic acid and other short chained fatty acids. A “urine dipstick” may be purchased in any pharmacy. This dipstick is inserted into the fecal specimen. After removal, followed by a minute of rest, the paper is quickly rinsed with doubly distilled water and the color of the dipstick examined.

A second indicator of lactose intolerance is the presence of glucose in the feces. Lactose is hydrolyzed by the enzyme lactase into glucose and galactose. This process normally occurs in the jejunum and the proximal ileum. The enzyme is synthesized in the brush border of the epithelial cells of the villi. The products of this hydrolysis are absorbed by the intestine, passing eventually into the blood stream. Lactose intolerance may be indicated by the presence of glucose in the stool since, though it may have been hydrolyzed in the colon, it was not absorbed and hence may be detected in the fecal material. A glucose “dipstick” inserted into a fecal specimen providing a reading of one-plus or more is an indicator of lactose intolerance. The female produced fecal material that gave no indication of detectable glucose by this method. The microbiologically sterile fecal material of the male produced a reading of three-plus on the glucose-indicating dipstick, confirming the suggestion that he was suffering from lactose intolerance.

The third type of study involves taking a blood sample after sufficient milk consumption and measuring the glucose content. A normal animal will show a rise in blood glucose after sufficient milk consumption. This observation stems from the reduction of lactose into glucose and galactose and their ultimate absorption into the blood stream, thereby causing a rise in blood glucose. Fasting blood glucose for the female P. potto who was lactose tolerant, was 100 mg dl⁻¹. On consumption of 50 g of milk this figure would rise to 122 mg dl⁻¹. This clearly indicated that the lactose was being hydrolyzed and the resulting glucose was being absorbed into the blood stream. Fasting blood glucose for the lactose intolerant male was 70 mg dl⁻¹ (COWGILL & ZEMAN, 1980b). After consumption of 50 g of milk this figure was unchanged. This indicated that glucose was not being absorbed into the blood stream, and therefore, that lactose was not being hydrolyzed in the villi of the jejunum.

All milk utilized in these experiments was homogenized, pasteurized milk supplemented with vitamins A and D such that consumption of 227 g would supply 10 and 25 %, respectively, of the human daily requirement. Two per cent milk refers to milk containing only 2 % milkfat. The types of milk used in this study are enumerated in Table 1.

Lactase enzyme (LactAid) was employed to hydrolyze the lactose. Seventy per cent reduction is achieved by adding 8 drops of the enzyme to 1 l of milk, while 100 % reduction is achieved with 15 drops. Subsequent to enzyme addition, the milk is magnetically stirred for 0.5 hr and refrigerated for 24 hr before use.

The lactose content of milk is about 5 %. Complete hydrolysis produces 2.5 % each of glucose and galactose. Acidophilus milk containing no lactose and 2 % milkfat was also employed in this study.