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Rickets in Polar Bear Cubs: Is There a Lesson for Human Infants?

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Some polar bear cubs raised in American zoos have developed metabolic bone disease, which has not been seen in wild bears [1–3]. Although the reasons that these cubs develop classic radiological features of rickets and sustain antebrachial fractures are complex, an obvious question is whether a fuller understanding of the process can provide insight into the nutrition of human infants, especially preterm infants. In unraveling the pathophysiology of metabolic bone disease in captive polar bears, a familiar theme emerges with a new twist: human milk is for humans and polar bear milk is for polar bears.

A pregnant sow in the wild consumes a diet of ringed seals, certain whales and some fish before denning and delivering her cubs. She fasts during the entire denning period, utilizing fat stores for nutrition and milk production [4]. The cubs weigh 600–700 g at birth and nurse for several months before emerging from the den [5, 6]. Growth of the cub in the den is remarkable, not unlike the growth of human pre-term infants. By age 12 weeks, cubs weigh 8,000–9,200 g [6]. The cubs emerge from the den at the end of the winter and are soon swimming, but they remain with their mothers and continue to nurse. Polar bear milk is extremely high in fat [5], especially saturated fat, and reflects the marine diet of the mother in the summer and early fall prior to birthing. The levels of calcium and phosphorus are high (23 and 15.4% of ash weight of milk, respectively) [5]. It contains approximately 675 IU/l of vitamin D and high levels of vitamins A and E. It also contains the amino acid taurine at levels >20-fold higher than in cow milk-based formula.

The polar bear has become the poster child of the green revolution as the shrinking polar ice caps attest to global warming. Virtually every wildlife rescue and environmentally conscious organization employs pictures, cards, cartoons and stuffed toys featuring this comely bear. Our immediate instinct is to try to save polar bears, especially orphaned cubs. Rescued cubs whose mothers have died, or captive cubs that may become victims of infanticide and cannibalization, are typically hand-reared with a formula developed for other animals, in particular canine pups [2, 4]. It is in captive cubs fed canine formula that rickets have been reported [1, 3].

Several factors contribute to metabolic bone disease in cubs. Their black skin and hollow, tube-like hairs make photocutaneous synthesis of vitamin D unlikely. In fact, studies of skin samples reveal extremely low levels of 7-dehydrocholesterol [7], the precursor of vitamin D. Fat-soluble vitamin absorption by the gut requires bile salt action to form lipid micelles. Polar bear bile acids, including the unique ursodeoxycholic acid, can only be conjugated with taurine [8]. The fat content of cow milk-based infant formula is far lower than polar bear milk [1–3]. Total lipids in bear milk range from 27 to 35%, whereas cow milk usually contains less than 10% lipids. Vitamin D is higher in bear milk than in supplemented cow milk (675 vs. 400 IU/l) [1]. Accordingly, hand-rearing with a standard formula provides a diet that is often quite different from that which is found in nature. Of particular note, the vitamin D in cow milk-based formula cannot be adequately absorbed if the diet is devoid of sufficient taurine to form bile salts. Hand-reared cubs were supplemented with higher levels of lipids, but in the absence of effective bile salts this resulted in steatorrhea [3]. Based upon new knowledge of the composition of polar bear milk, efforts are underway to improve the formula fed to captive polar bear cubs.

The main features of metabolic bone disease are single or multiple fractures following trivial falls (20 fractures in 14 captive bears), and radiologic evidence of rickets, such as widening and fraying of the metaphysis [3, 9, 10]. Serum calcium and phosphorus concentrations are variable, but alkaline phosphatase activity is extremely high (>3,000 IU/l) [3]. Serum 25(OH) vitamin D values are <7.5 ng/ml.

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With very high doses of vitamin D supplementation (10,000 IU/day), rickets is reversed, serum calcium, phosphorus and alkaline phosphatase values normalize, and fractures heal [3].

The lessons for pre-term human infant nutrition are several. The growth rate of polar bear cubs must be tremendous for them to reach an adequate size in time to emerge from the den. The very low birth weight pre-term infant, particularly one whose weight is similar to that of a 600 g cub, lacks the nutritional advantages of placental nutrient transfer. Similar to the less-than-optimal formula-fed captive cubs, the milk designed by nature for full-term infants is not optimal for pre-term infants. Growing cubs seem to require a predictable dietary source of vitamin D; the concentration in polar bear milk is, on average, 8- to 15-fold higher than in human milk and 1.5-fold higher than supplemented cow milk. With abundant vitamin D, cubs can be raised in the dark.

Human infants can utilize both glycine and taurine to conjugate bile acids, hence their milk supply does not have to be so high in taurine content [11]. On the other hand, the polar bear cub can tolerate a high lipid diet without steatorrhea or atherosclerosis provided there is adequate taurine available to conjugate bile acids and permit lipid and fat-soluble vitamin absorption. In low birth weight infants, dietary taurine supplementation of formula or provision of human milk alters duodenal bile acid concentrations and conjugation patterns. Glycoconjugates predominate in non-taurine supplemented formula-fed infants [11].

In essence, an accident of nature, the occurrence and pathogenesis of rickets in polar bear cubs reared in American zoos, can remind us of the nutritional needs and potential inadequacies in the diet of human low birth weight infants.

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References

1 Chesney RW, Hedberg GE, Rogers QR, Dierenfeld ES, Hollis BE, Derocher A: Does taurine deficiency cause metabolic bone disease and rickets in polar bear cubs raised in captivity? Adv Exp Med Biol 2009;643:325–331.

2 Hedberg GE, Dunker F, Chesney RW: Pathogenesis of metabolic bone disease in captive polar bears *(Ursus maritimus)*. Polar Bears International 2007 (http://www.polarbears-international.org/pbhc/hedberg.htm).

3 Kenny DE, Irlbeck NA, Eller JL: Rickets in two hand-reared polar bear *(Ursus maritimus)* cubs. J Zoo Wildl Med 1999;30:132–140.

4 Kenny DE, Bickel C: Growth and development of polar bear cubs at Denver Zoological Gardens. Int Zoo Yb 2005;39.

5 Cook HW, Lentfer JW, Pearson AM, Baker BE: Polar bear milk. IV. Gross composition, fatty acid, and mineral constitution. Can J Zool 1970;48:217–219.

6 Derocher AE, Nelson RA, Stirling I, Ramsay MA: Effects of fasting and feeding on serum urea and serum creatinine levels in polar bears. Marine Mammal Sci 1990;6:196–203.

7 Kenny DE, Irlbeck NA, Chen TC, Lu Z, Holick MF: Determination of vitamins D, A, and E in sera and vitamin D in milk from captive and free-ranging polar bears *(Ursus maritimus)* and 7-dehydrocholesterol levels in skin from captive polar bears. Zoo Biol 1998;17:285–293.

8 Hofmann AF: The continuing importance of bile acids in liver and intestinal disease. Arch Intern Med 1999;159:2647–2658.

9 Johnston G, Cutchins J: Andy Bear – A Polar Cub Grows Up at the Zoo. New York, Morrow, 1985.

10 Lin RC, Engeli E, Prowten AW, Erb HN, Ducharme NG, Goodrich LR: Antebrachial fractures in four captive polar bears *(Ursus maritimus)*. Vet Surg 2005;34:358–365.

11 Jarvenpaa AL, Rassin DK, Kuitunen P, Gaull GE, Raiha NC: Feeding the low-birth-weight infant. III. Diet influences bile acid metabolism. Pediatrics 1983;72:677–683.