BEHAVIOR, BIOCHEMISTRY, AND HIBERNATION IN BLACK, GRIZZLY, AND POLAR BEARS[1]

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Abstract: Annual behavioral and biochemical patterns of black bears (*Ursus americanus*), grizzly bears (*Ursus arctos horribilis*), and polar bears (*Ursus maritimus*) were reviewed. We propose that black and grizzly bears show 4 annual physiological stages: Stage I—hibernation, in which lean body mass is preserved and body fat supplies energy; Stage II—walking hibernation, in which the biochemistry of hibernation is integrated with physical activity, but food and water intake are minimal; Stage III—normal activity, in which patterns are consistent with those of nonhibernating mammals; and Stage IV—hyperphagia, which increases fat reserves for hibernation. For polar bears, using published reports and recently collected data, we propose that all 4 stages are possible and that polar bears appear able to shift between Stages I and II in both summer and winter, which permits successful adaptation to the arctic environment.

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Field and laboratory observations of behavior of black and grizzly bears have indicated that they pass through 4 annual biochemical and physiological stages. These have been designated as Stage I—hibernation, Stage II—walking hibernation, Stage III—normal activity, and Stage IV—hyperphagia (Nelson et al. 1979). The purpose of this paper is to further define the 4 stages and to determine, using the literature and experimental data, whether the polar bear corresponds.

PHYSIOLOGICAL STAGES OF BLACK AND GRIZZLY BEARS

Stage I—Hibernation

Studies by Folk (1974) have shown that black and grizzly bears are hibernators in winter in the true sense, showing physiological patterns similar to those of deep hibernators: distinct decreases in heart rate, metabolic rate, and body temperature. However, hibernation in black and grizzly bears differs from that of deep hibernators in that bears hibernate at a near-normal body temperature, 31–35 C, and their dormancy is continuous from 3 to 7 months. Deep hibernators (bats, insectivores, and rodents) hibernate at near 0 C and undergo periodic arousals (Folk 1974).

Although expending about 4,000 kcal per day (calculations based on body fat utilization rates), the black bear does not eat, drink, urinate, or defecate. It is easily aroused into a mobile, reactive state, aware of its surroundings and able to defend itself. Female bears give birth to cubs and nurse them under these conditions (Nelson 1973).

Biochemical studies on black bears in hibernation have shown that no net formation of the common end products of protein catabolism occurs. Blood concentrations of total amino acids, total protein, urea, uric acid, and ammonia do not increase throughout winter; no evidence of intestinal storage of nitrogen has been found (Nelson et al. 1973). Lean body mass is conserved in hibernation; weight loss (between 15 and 25% of body weight) is from adipose tissue only (Nelson et al. 1975, Lundberg et al. 1976). Urine is formed daily, but that which is formed is reabsorbed into blood through the bladder wall (Nelson et al. 1975). Urea metabolism is regulated so that no net increase occurs during the state of hibernation. Prevention of uremia appears essential to maintain hibernation because injection of urea into the bloodstream of hibernating bears induces diuresis, causes intracellular dehydration, and clearly disrupts the hibernation state (Nelson et al. 1973).

Despite conservation of lean body mass and absence of net production of urea during hibernation, there is metabolic activity: transamination and catabolism of amino acids, gluconeogenesis, and formation and degradation of urea (Nelson et al. 1975, Ahlquist et al. 1976).

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In winter, urea turnover is greatly reduced because of an increase in effectiveness of protein anabolism. Amino acids enter protein anabolic pathways in preference to entering the urea cycle (Ahlquist et al. 1976, Lundberg et al. 1976).

Although some amino acids are catabolized and some urea is formed, lean body mass is preserved. This is achieved through formation of alanine from glycerol, released during lipolysis, and nitrogen released from amino acid and urea catabolism. Alanine in turn yields glucose and its metabolic byproducts and, through transamination reactions, other amino acids. The amino acids thus formed enter protein synthetic pathways more readily in winter than in summer (Ahlquist et al. 1976, Lundberg et al. 1976). The combined effect of these metabolic interactions preserves lean body mass and prevents uremia (Nelson 1980).

Fat supplies the energy for hibernation metabolism, and metabolic water produced from it replenishes that lost through respiration (Nelson et al. 1973). Ketosis does not develop in hibernating black bears. Only slight increases were found in β-hydroxybutyrate and acetoacetate in blood, increasing from 20 ± 7 and 9 ± 2 μmol/L, respectively, before hibernation to 163 ± 70 and 81 ± 32 μmol/L during hibernation (Nelson 1980). Following 4.5 months of hibernation, the first day's output of urine by a grizzly bear contained only 5 mg of ketone bodies (Nelson et al. 1979). On the other hand, humans after 5 weeks of starvation, with free access to water, showed increases in blood of β-hydroxybutyrate and acetoacetate from 60 ± 10 and 30 ± 10 μmol/L to 5850 ± 380 and 1340 ± 140 μmol/L, respectively, and daily excreted 3 g of ketone bodies in urine (Owen et al. 1969).

The prevention of ketosis in the bear is due, in part, to glycerol metabolism. Injection of ^{14}C-labeled glycerol revealed that the labeled molecule appeared in lipid esters, including triglycerides, at an increased rate during hibernation (Ahlquist et al. 1976). Our present hypothesis is that increased triglyceride turnover in winter is sufficient to inhibit fatty acid entry into the metabolic pathways for ketone production, which effectively prevents ketosis. If ketosis developed it would predictably affect acid-base balance in an animal burning 4,000 kcal of fat daily without urinating.

Data in support of this hypothesis are found for the hoary marmot (*Marmota caligata broweri*), which gradually develops ketonemia in hibernation until arousal occurs. Ketonemia is postulated as a possible "trigger" which induces the periodic arousals in this mammal (Baumber et al. 1971). Therefore, body fat supplies the energy and water necessary for hibernation, and glycerol released from fat deposits helps prevent uremia and ketosis (Nelson 1980).

In a decade of study of hibernating bears, we have found 2 useful indicators of protein and fat metabolism which signify successful hibernation: a low ratio in the blood of urea to creatinine (25 to 35 in active bears, but 4 to 8 during hibernation) and a marked elevation of free fatty acids in serum (Nelson 1980, Nelson et al 1978*a*).

Increased levels of circulating testosterone (Nelson et al. 1978*b*) and hypothalmic hypothyroidism (Azizi et al. 1979) are also associated with hibernation. The effect of these changes is not clear at present, but they appear to be required for successful hibernation (Nelson 1980).

The importance of changes in protein and fat metabolism in producing the successful state of hibernation were highlighted by the finding that 2 black bears which died while denning in winter were uremic. Their blood urea values were 650 and 690 mg% (Nelson 1978). These animals had lost as much body weight in less than 3 weeks as hibernating bears usually lose in 3 months.

Stage II — Walking Hibernation

Although less is known about this stage than about hibernation, there is sufficient evidence to support its existence. For instance, when black and grizzly bears leave dens in springtime, they are anorectic. This phenomenon has been observed in the wild (Hock 1958) and in captive black bears taken out of their hibernaculum (Nelson 1980). Caged animals, upon emergence from their dens, do no resume normal intake of food until 10 to 14 days have elapsed, although normal rations are available during this time. After 2 weeks they eat normally.

Food intake and urine volumes were studied in a grizzly bear for 3 weeks during Stage II after it had emerged from a denning of 4.5 months (Table 1) (Nelson et al. 1979). The bear was hypophagic although it had free access to food. Water intake was low, and daily urine volume

Table 1. Comparison of urine content during Stages I, II, and III in a captive grizzly bear.

	Urine volume and content (24-hour totals)				
Stage	Volume (ml)	Nitrogen (g)	Urea Nitrogen (g)	Ammonia (mg)	Ketones (mg)
I[a]	181	1.43	0.98	72	5
II	116	0.2	0.17	41	8
	140	1.8			
III	2080	34.0	27.6	1785	33

[a] Urine collected 1 day after leaving den in spring.

varied between only 116 and 140 ml despite free access to water. Furthermore, the bear was active during the 3-week period. Nitrogen loss in urine was negligible, amounting to only 0.2 and 1.8 g per 24 hours. Daily excretion of calcium, phosphorus, and magnesium were low, varying between 2 and 8 mg per day. These data suggest that the biochemical stage of hibernation persisted in part or in full after the grizzly left the den. It began to eat and drink normally after 3 weeks.

Stage III—Normal Activity

Studies done on bears during this phase, postulated to last from May to September, indicate that captive black bears are active and eat 5,000 to 8,000 kcal per day. They cannot duplicate hibernation responses in summer when starved outside under ambient temperature or housed in hibernation-like conditions (in the dark and in the cold). Like other starving warm-blooded animals including humans, nonhibernating bears continuously utilized muscle as a source of energy and became dehydrated and uremic when denied access to water (Nelson et al. 1975).

During the period of normal activity when bears were eating and drinking ad libitum, daily urine volume and nitrogen losses were in the range of 2 L and 16 g in black bears (Nelson et al. 1973) and 2 L and 34 g in a grizzly bear (Table 1).

Stage IV—Hyperphagia

Black bears demonstrate hyperphagia in the fall, increasing their daily intake from 8,000 to 15,000 to 20,000 kcal. Grizzly bears observed in the wild feed 20 hours per day in the late fall to prepare for hibernation and consume upwards of 20,000 kcal per day (Nelson 1980).

The prodigious food intake during this phase is confirmed by polydypsia, polyuria, and large nitrogen losses in urine. Two black bears studied during this phase showed daily urine volumes between 8 and 16 L and nitrogen losses between 104 and 69 g, respectively (Nelson et al. 1975).

PHYSIOLOGICAL STAGES OF POLAR BEARS

Background

In polar bears, the sequence of changes from one stage to another is far from clear. In fact, there appears to be no orderly sequence, although all 4 stages can be identified. For instance, at Churchill, Manitoba, adult male and female polar bears, yearlings, and cubs have been observed in dens during August. They have also been observed during August walking along the water, swimming, and sitting by Hudson Bay. No evidence was found that vegetation had been eaten. About 100 caribou and thousands of snow geese were in the same area as summer denning bears. There was no evidence that polar bears fed on them. Only occasional scats were seen.

It was decided to study this population of polar bears to determine whether any showed biochemical evidence of hibernation as judged against data obtained from black bears. Blood parameters and glycerol metabolism were examined by methods similar to those used for black bears (Nelson 1980).

Methods

Four adult males, 3 adult females and 5 cub polar bears who were not denning and 1 adult male who was, were immobilized with phencyclidine hydrochloride and sparine (Nelson et al. 1973). Blood samples were obtained from the femoral vein and analyzed as previously described for urea, glucose, cholesterol, triglycerides, phospholipids, free fatty acids, total protein, amino acids, creatinine, corticosteriods, glucagon, and insulin (Nelson et al 1973, Palumbo et al. 1980). Glycerol metabolism was followed by injecting 250 μCi of ^{14}C-labeled glycerol and determining its fate in various intermediates for a 4-hour period. Substances assayed were labeled glycerol, glucose, lactate, plasma proteins, amino acids, and urea. Methods

Table 2. Comparison of blood values (mean ± standard error) found in wild and captive polar bears in summer and in black bears in summer and winter.

	Polar bears					Black bears[a]	
	Wild, active			Wild, denning	Captive	Hibernation, Stage I	Active, Stage III
Compound(s)	Adult males (N=4)	Adult females (N=3)	Cubs and yearlings (N=5)	Adult male (N=1)	Adult females (N=3)		
Urea mg%	16±1	7.1±0.7	8.2±1.26	17.2	24.1±4.7	35	20
Creatinine mg%	2.4±0.2	2.6±0.3	1.9±0.4	2.2	1.2±0.2	1.1±0.1	3.2±0.5
Urea/creatinine	6.7±0.9	2.7±0.1	4.5±0.5	7.8	20.3±3.4	35	6.5
Glucose mg%	112±21	158±17	141±22	146	147±21	98	92
Cholesterol mg%	370±24	257±42	353±27	163	271±15	390	520
Triglycerides mg%	249±19	352±25	342±14	385	204±31	203	310
Phospholipids mg%	546±33	802±96	775±66	620	169±55	465	610
Free fatty Acids (μEq/l)	1458±246	733±396	948±349	3300	335±275	330±75	1350±110[b]
Total proteins gm%	7.6±1	7.9±0.6	7.3±0.5	9.1	7.9±0.7	7.6±0.1	8.0±0.3
Corticosteroids μg%	29±1					7.8±2.5	1.8±0.2[c]
Glucagon (pg/ml)	100±9					175±48	78±13[c]
Insulin (μU/ml)	41±3					17±3	14±6[c]

[a] Unless otherwise indicated, all black bear data from Nelson et al. (1973).
[b] Data from Ahlquist et al. (1976).
[c] Data from Palumbo et al. (1983).

were as described by Lundberg et al. (1976) and Ahlquist et al. (1976). When possible, data were compared with data from black bears and from 2 captive polar bears at Churchill, Manitoba, and 1 at Point Barrow, Alaska, who were fed ad libitum and given free access to water. The captive bears were immobilized as described above for data aquisition.

Results

There was little or no difference in data obtained from the wild Churchill polar bears and hibernating black bears (Table 2). Data from captive polar bears, on the other hand, resembled data from black bears in Stage III.

The most striking findings for the wild polar bears were the low urea/creatinine ratios and the elevated levels of lipids, especially free fatty acids (Table 2). These data indicated a biochemical state of successful hibernation (Nelson 1980).

Captive polar bears had urea/creatinine ratios and blood lipid levels resembling those of Stage III black bears and nonhibernating mammals, such as human beings. (Differences between wild and captive polar bears noted in this study confirm data published by Lee et al. (1977), who found that wild polar bears have persistently lower levels of urea and higher levels of cholesterol than a captive bear. They did not determine creatinine.)

The results of the glycerol experiments in wild polar bears were similar to those in hibernating black bears (Fig. 1). Activity appeared in glucose, lactate, and serum proteins. However,

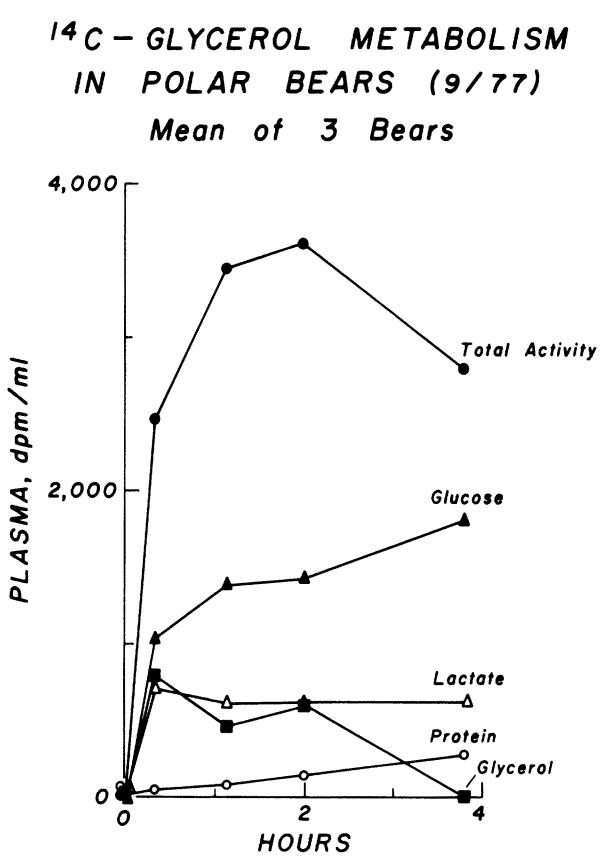

Fig. 1. Appearance of labeled glucose, lactate, and protein and disappearance of labeled glycerol after injection of ^{14}C-glycerol into 3 wild polar bears in summer.

Table 3. Comparison of plasma amino acids (in μmol%; mean ± standard error) in 3 wild adult polar bears and in 3 hibernating black bears.

Amino acid	Hibernating black bear[a]	Wild polar bears
Taurine		15.1±1.5
Aspartic acid	1.1±0.1	0.7±0.1
Threonine	19.6±1.0	13.2±3.3
Serine	15.4±2.0	13.8±1.1
Asparagine and glutamine		113±14
Proline	16.5±3.0	14.6±2.8
Glutamic acid	15.1±4.0	11.0±0.3
Citrulline		5.4±1.0
Glycine	49.1±10.0	37.0±0.2
Alanine	40.9±6.0	27.4±8.2
l-amino-N-butyric acid		4.5±0.1
Valine	26.1±3.0	19.2±1.6
Cystathionine		0.7±0.1
Methionine	3.9±0.3	2.3±0.4
Isoleucine	10.3±3.0	6.1±1.8
Leucine	18.4±2.0	12.1±1.9
Tyrosine	6.4±2.0	4.2±0.6
Phenylalanine	10.9±2.0	7.0±0.5
Cystine	7.2±1.0	
Histidine	8.0±1.0	
Lysine	28.9±9.0	
Arginine	17.7±2.0	

[a] Values from Nelson et al. (1973).

there was a quantitative difference in that, in polar bears, activity was slow to appear in serum proteins and tended to remain in glucose longer. However, no labeled urea was found in plasma of polar bears injected with labeled glycerol. This is similar to findings in hibernating black bears in which no labeled urea is detected after glycerol injections. When similar experiments were done in Stage II black bears, labeled urea was always found in plasma (Ahlquist et al. 1976).

Blood amino acid concentrations in wild polar bears were similar to those in black bears in Stage I or Stage III except for a lower concentration of alanine (Nelson et al. 1973) (Table 3). The more sluggish conversion of glycerol into plasma proteins by wild polar bears than by hibernating black bears may be responsible. Insulin, glucagon, and corticosteroids were present in plasma of the wild polar bears.

DISCUSSION

The summer studies indicated that the wild polar bears found at Churchill, Manitoba, were in Stage II. That is, a biochemical state of hibernation was found, yet the bears were active while eating little. Stage I was ruled out because of the presence of physical activity in all bears except 1 and the presence of some scats. Stages III and IV were ruled out because no signs of normal food intake or hyperphagia were observed.

In support of classifying the summer polar bear in Stage II, Craighead has field notes and photographic evidence obtained in summer from South Twin Island, Hudson Bay, showing that wild polar bears eat little, are lethargic, and have increased body fat reserves. Summer observations in the Churchill, Manitoba, area by Best (1976) further indicated that polar bears spent little time feeding (3%) and that the energy content of the food was low. Knudsen (1973) states, "My observation that the bears spent little time feeding while on the island during summer is further supported by the fat condition of the animals upon their arrival. Their general condition suggested that they could survive a summer by utilizing their fat reserves."

The wild polar bears' adaptation to the severe cold of winter must be so efficient that in summer it has to compensate. Stage II would allow denning at will and not require much food or water intake.

In winter, biochemical studies of wild polar bears are sparse and do not allow for interpretation of stages. However, field observations suggest that wild polar bears spend some time in both Stages I and II. For instance, wild pregnant females, females with young cubs, barren females, and adult and immature male polar bears have all been observed to den in winter. Pregnant females spent the longest time in dens; adult male bears spent the least, denning for up to 7–8 weeks (Perry 1966:78–79, Stroganov 1969). In our opinion, based on studies of black and grizzly bear denning (Nelson et al. 1979, Nelson 1980), a polar bear spending 8 weeks in a den would require the biochemistry of hibernation (Stage I) for survival.

However, wild polar bears can also be quite active in winter. We propose that when active they are in Stage II. This contention is based on observations that polar bears eat intermittently and that they restrict their diet primarily to fat. Physical activity rules out Stage I and irregular feeding rules out Stages III and IV. The restriction of food intake to fat further supports the concept that Stage II is present. The biochemical reactions of hibernation would not be disrupted by eating fat, but would be if protein were eaten (Nelson 1980).

The intermittent consumption of food and its restriction to primarily blubber has been observed by several investigators. Perry (1966) and Stroganov (1969) noted that wild polar bears fed primarily on the blubber of seals although consuming some hide as well. Stirling (1974) estimated that bears kill a seal about once every 5 days. Stirling and McEwan (1975) state that "after a polar bear kills a seal it feeds predominately on the blubber and often abandons the meat. Blubber is the preferred part of the seal. Exceptions occur such as when a small seal is killed by a particularly hungry bear or by a female bear with 1 or 2 cubs. Then a large part, or all of the carcass is consumed." Stirling (1974) noted, "The skin and fat were eaten first, sometimes in a very exacting manner. For example, one bear about 0.5 km distance was observed carefully using its incisors like delicate clippers to remove only the fat from the carcass leaving the meat."

However, when the wild polar bear does eat in winter, it consumes a huge meal. This could be argued as representing hyperphagia. However, hyperphagia connotes eating any and all food and eating almost all of the time. The wild polar bear does not show this behavior in winter but appears to regulate precisely its food intake by intermittent eating. Certainly, when active, it requires more energy than when denning. In winter, polar bears swim in the sea water for days and perform long arctic movements. Their energy cost for walking is 2-fold higher than normally predicted (Oritsland et al. 1976). Obviously the arctic activity of the polar bear in winter extracts a high energy demand and this is met by eating seal blubber.

It is tempting to speculate that fat cell size of the polar bear is involved in regulating its winter eating. For instance, when the fat cell shrinks to a critical size because of loss of fat, a signal may be released which stimulates fat consumption until the cell returns to a certain maximum size. Body fat could be effectively replenished by such a mechanism. However, there are no data to confirm this hypothesis.

Stage II in winter would reduce the need for water. Surely, water requirements for polar bears must represent a special problem when the only available source is cold snow and ice from which salt has been leached. If wild polar bears were similar to captive bears (who eat protein), about 8 L of urine would be produced daily (Nelson et al. 1979). Taking 8 L of water in the form of cold snow or ice would impose an undue energy stress on the wild polar bear just to warm it to body temperature.

In all probability, the wild polar bear need not take in much, if any, water in winter simply because it eats mostly blubber. The catabolic products of fat combustion are only carbon dioxide and water. Carbon dioxide can be easily excreted through respiration. The metabolic water produced could maintain normal body hydration, as it does in denning black bears (Nelson et al. 1973). If polar bears ate much protein, water requirements would be markedly increased because its end products of catabolism (urea, ammonia, uric acid, and creatinine) must be excreted in urine.

Obviously protein is ingested because it is in hide and blubber. However, if the quantity is low, as observed for winter polar bears, urinary excretion of its end products should not impose a great stress on body water reserves. Polar bears eating 45,000 kcal of fat will receive 5 L of water from its metabolism which can be used for urine formation (Nelson 1973). Furthermore, in winter, the polar bear may not need much dietary protein if its metabolism is similar to that in summer, which is like that of hibernating black bears. These latter animals use only body fat to supply energy for metabolic processes; no lean body mass is utilized (Nelson et al. 1973, 1975, Lundberg et al. 1976). No other animal has been shown to have this ability to completely protect muscle mass under starvation conditions at near normal body temperature. It would be of great advantage to the polar bear to have a similar type of adaptation when it roams the polar ice. Certainly, what is known about wild polar bears in summer now suggests that they can utilize a type of metabolic adjustment which allows them to roam freely but not have to feed and drink water daily.

Wild polar bears also require adequate vitamin, mineral, and trace metal intake for survival. However, depot fat, in this case the blubber of seals, is thought also to be the chief storage depot for these substances. Blubber, along with the hide of the seal, could easily supply all essential nutrients demanded by winter activity. When the wild polar bear hibernates, if it is similar to the

black bear, no nutrient intake is required and excellent health is maintained.

Nutrient requirements for nondenning, lactating, and growing bears are different. These animals need protein for milk production and growth and have been observed consuming protein in winter. However, this may not affect their water balance to any great degree since the protein most likely is being used for growth requirements rather than being catabolized (Munro 1970).

Few data exist about a Stage IV (hyperphagia) in polar bears. However, our summer observations of wild polar bears have indicated increased body fat reserves, suggesting that hyperphagia may occur or that fat cells are filled prior to entering the summer period.

CONCLUSIONS

Thus, the observations of polar bear behavior and biochemistry support a hypothesis that a continuous capacity to hibernate, winter or summer, may exist. Polar bears appear to have incorporated a feeding behavior into their adaptation so that by eating primarily fat, the least physiologic stress is produced on their biochemical adaptation. Obviously, more data are required to test this hypothesis, such as information on drinking, urination and defecation patterns, along with some basic blood and metabolic studies of polar bears in winter.

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