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Author(s): Daniel E. Crocker, Paul M. Webb, Daniel P. Costa, and Burney J. Le Boeuf

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Protein Catabolism and Renal Function in Lactating Northern Elephant Seals

Daniel E. Crocker

Paul M. Webb

Daniel P. Costa

Burney J. Le Boeuf

Department of Biology and Institute of Marine Science,
University of California, Santa Cruz, California 95064

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ABSTRACT

Northern elephant seals, *Mirounga angustirostris*, fast completely from food and water during lactation. Previous investigations of maternal investment suggested physiological constraints on the level of energy expenditure during lactation. In this study, two components of phocid fasting physiology, protein sparing and reduced glomerular filtration rate, were examined for effects of changing body composition and lactation duration. Protein catabolism was estimated from ^{14}C -urea turnover in five mid- and five late-lactation females. Body composition was determined by using an ultrasound scanner to measure blubber depth coupled with morphometrics. Glomerular filtration rate was measured in five females at mid- and late-lactation using plasma clearance of ^3H -inulin. Protein catabolism increased significantly between measurements. The contribution of protein to metabolism varied with body composition and lactation duration. Mass-proportional glomerular filtration rate increased significantly between measurements. These data suggest that conflicting metabolic demands of lactation and fasting might constrain the duration and magnitude of maternal investment in northern elephant seals.

Introduction

Several phocid species, including northern elephant seals, *Mirounga angustirostris*, fast from food and water during the entire period of lactation, undergoing dramatic reductions in stored energy reserves (Costa et al. 1986). Measurements of maternal investment have suggested that the energy expended during

lactation is determined by the energy and protein reserves obtained during the previous 8-mo foraging trip (Crocker 1995). Analysis of lactation energetics suggests that this investment threshold might be related to the effects of body composition on the rate of lean-tissue catabolism. This model is supported by evidence of direct effects of maternal body composition on mass-controlled energy expenditure and proportional lean-tissue loss over lactation (Crocker 1995). We examined two components of phocid fasting physiology, protein sparing and reduced glomerular filtration rate (GFR), for effects of changing body composition and lactation duration.

Long-term fasting is characterized by protein sparing, reductions in metabolic rate, and reductions in water loss for urea nitrogen excretion (Goodman et al. 1980). Studies on nonlactating, fasting phocids have shown that protein stores are spared, with the bulk of energy demands being supplied by the oxidation of fatty acids (Pernia et al. 1980; Worthy and Lavigne 1987; Nordøy et al. 1990; Adams and Costa 1993). In contrast, lactation is characterized by dramatic increases in metabolism and significant transfer of nutrients and water to the mammary gland for the synthesis and secretion of milk. The general metabolism of the lactating female is reorganized in a way that ensures the appropriate nutrients are partitioned to the mammary gland (Bauman and Currie 1980). In nonfasting animals, lactation is accompanied by increased levels of food consumption and digestion (Millar 1977).

While fasting, elephant seals produce a milk with one of the highest lipid and energy contents found in nature (Riedman and Ortiz 1979). Significant increases in the rate of nutrient delivery required for milk synthesis across lactation are likely, as the energetic density of the milk increases dramatically. Comparisons of water flux data from previous milk intake studies, as well as differential pup growth rates across lactation (Kretzmann et al. 1993), suggest that milk intake rates are fairly constant. Thus, increases in the energetic density of the milk most likely represent increases in the lipid demands of milk synthesis. The relationship between lipid availability and protein use during fasting have been well documented (Lowell and Goodman 1987). Nutrient delivery to the mammary gland, together with reductions in adipose tissue stores, may have an impact on the ability to effectively spare protein while fasting. Previous milk intake studies have shown that females lose approximately 15% of their total body protein to account for the protein delivered in the milk (Crocker 1995). As this value does not include protein catabolism for maintenance metabolism, potential increases in protein loss for energy needs could move

females closer to the 30% loss of body protein considered extreme in humans (Cahill 1978).

In this study, we examined the relationship between protein metabolism, body composition, and lactation duration. We tested the following hypotheses: (1) that urea turnover increases significantly between mid- and late-lactation; (2) that the contribution of protein to energy metabolism increases significantly between mid- and late-lactation; and (3) that alterations in protein metabolism correlate with changes in maternal body composition. In addition, changes in renal filtration between mid- and late-lactation were examined.

Material and Methods

Study Area and Subjects

This study was conducted at Año Nuevo State Reserve, San Mateo County, California, during the 1993–1995 winter breeding seasons. Females were identified by numbered plastic flipper tags (Dalton jumbo Roto-tags, Oxon, England), which were attached to animals after weaning in previous breeding seasons. Upon arriving at the rookery, tagged females were marked with Lady Clairol hair dye (Stamford, Conn.). Females were observed daily to accurately determine parturition dates. Urea turnover was measured in five females in the middle of the lactation period (9–13 d postpartum) and five different females late-in lactation (15–18 d postpartum). In order to examine the importance of body composition and lactation duration, an experimental design with a range of postpartum injection dates was chosen. GFR was measured in five different females at both midlactation (9–10 d postpartum) and late-lactation (20–22 d postpartum) for each female. The disturbances involved in these measurements were thought to represent a high risk of mother-pup separation early in lactation. Females were allowed at least 9 d to establish a strong bond with their pups and to demonstrate typical maternal behavior.

Urea Turnover Measurements

The dilution of ^{14}C -urea over time was used to calculate-urea pool size and rate of turnover. Each female was immobilized via an intramuscular injection of approximately 1 mg kg^{-1} of a teletamine/zolazepam mixture (Telazol, Aveco, Fort Dodge, Ind.). Immobilization was maintained through intravenous injections of ketamine HCl (Aveco, Fort Dodge, Ind.). The female was administered a bolus injection of 1.0 mCi of ^{14}C -urea in a 5-mL volume into the dorsal extradural vein. Blood samples were obtained from the extradural vein every 15 min for 90–150 min to determine equilibration specific activity. Specific activity at equilibrium was reached when the specific activities of two successive samples were not significantly different. This value was not significantly different from the y -intercept calculated from the subsequent clearance curve. All females achieved

Figure 1. ^{14}C -urea turnover in 10 lactating females. Open circles are midlactation females, and closed circles are late-lactation females. Urea turnover constant (K_u) was calculated as the negative slope of a semilog plot of each clearance curve.

equilibration within the sampling period. The females were weighed, body composition was determined using an ultrasound scanner (Scanprobe, Ithaca, N.Y.) to measure blubber depth, and the truncated-cones method was used to model proportions of adipose tissue (Gales and Burton 1987). Blood samples were collected from each female at 2 and 4 d postinjection. To obtain these blood samples, a smaller dose of Telazol (0.5 mg kg^{-1}) was used for restraint, or in some cases, samples were obtained from sleeping animals without restraint.

Samples were drawn without anticoagulants and centrifuged, and the ^{14}C -specific activity of the serum was determined in triplicate. Two-hundred-microliter samples were combined with 9 mL of Betaphase scintillation cocktail (Westchem, San Diego, Calif.) and counted with a Beckman model LS 3801 scintillation counter. A curve from quenched ^{14}C standards was used to correct for any quenching due to hemolysis. This method of correcting for quench was validated by adding known quantities of isotope to unlabeled serum. Injection syringes were gravimetrically calibrated to accurately determine injection volume to within 0.001 mL . Serum urea (BUN) concentrations were determined for each sample in duplicate using a colorimetric assay (Sigma #535, St. Louis) and spectrophotometer.

The pool volume was calculated from the dilution of the injected specific activity at equilibrium. Urea pool size was calculated from the dilution volume and BUN concentration. Mean urea turnover was calculated based on the turnover constant derived from ^{14}C turnover curves and a measure of the urea pool size. This urea turnover constant (K_u) was calculated as: $K_u = -\Delta \ln(\text{SA}) \times t^{-1}$, where SA is the specific activity of the sample and t is time in days. Thus, urea turnover constant was the negative slope of a semilog plot of the clearance curve (Fig. 1). Urea pool size (N_u) in grams was calculated as: $N_u = [\text{BUN}]_{(t=0)} \times \text{CPM}_i / \text{SA}_{(t=0)}$, where CPM_i is the total in-

Figure 2. Typical clearance curve for ^3H -inulin following bolus injection. Three monoexponential curves were fit to the data using the peeling-off method. Curves 1 and 2 reflect dilution in the blood and extracellular pools respectively, and curve 3 represents renal clearance.

jected activity of ^{14}C , $\text{SA}_{(t=0)}$ is the equilibration specific activity, and $[\text{BUN}]_{(t=0)}$ is serum urea concentration at equilibrium. Assuming amino acids are the only significant source of nitrogen for urea formation, the daily rate of protein catabolism (r_p) was calculated as: $r_p = r_u \times 2.292 \text{ g protein g}^{-1} \text{ urea}$, where r_u is the mean urea turnover rate. Energy derived from protein catabolism was calculated as 17.99 kJ g^{-1} (Costa 1987). Daily female metabolism was estimated based on a regression line from mass and metabolic rate data from Costa et al. (1986), metabolic rate (MJ d^{-1}) = $0.1056(\text{mass}) + 20.0$ ($r = 0.72$, $P < 0.05$).

GFR Measurements

GFR was determined from plasma clearance rates of ^3H -inulin (Hall et al. 1977). Females were chemically immobilized as previously described. Body composition was estimated as above. For each measurement, the female was injected with 0.7–1.2 mCi of ^3H -methoxy-inulin (Dupont-New England Nuclear, Boston) in a 7–12-mL volume containing 1% inulin via the dorsal extradural vein. Blood samples (5 mL) were taken with anticoagulants at 5-min intervals for 30 min and then at 15-min intervals for at least 3 h postinjection. On some animals, concurrent blood samples were drawn from both the extradural vein and a flipper vein to ensure thorough mixing of tracer in the vascular pool. Blood samples were analyzed in triplicate as above, except that 100- μL volumes of plasma were counted in 9 mL of Ecolite scintillation cocktail (Ecolite, San Diego, Calif.). A curve calculated from quenched ^3H standards was used to correct for quench due to hemolysis.

A clearance curve for each measurement was fitted with three monoexponential curves, using the peeling-off method (Fig. 2). This method fits the monoexponential function with

the highest r^2 value to each of three sections of the curve. The three curves most likely represent dilution in the blood pool, dilution in the extracellular volume pool, and renal clearance, respectively. All renal clearance curves had an r^2 value greater than 0.99. Least squares fits for the first two monoexponential functions had r^2 values ranging from 0.86 to 0.99, with the lower value representing the second curve from a female exhibiting some apneustic behavior during the measurement. Seals exhibit some peripheral vasoconstriction during apnea, which may cause slight variations in dilution kinetics.

The area under each curve was calculated using standard numerical integration and summed to obtain a total area under the curve (A); GFR was then calculated as: $\text{GFR} = \text{CPM}_{\text{inj}}/A$, where CPM_{inj} is the total radioactivity injected. This method of quantifying GFR has been validated against true inulin clearance measurements by means of urine collection (Hall et al. 1977). BUN and creatinine concentrations were determined for each sample in duplicate using a colorimetric assay (Sigma #535 and #555) and spectrophotometer. A BUN : creatinine ratio was calculated for each female at each measurement.

Results

Urea Turnover Measurements

Urea turnover constant, urea turnover rate, protein catabolism rate, and estimated contribution of protein toward metabolism are shown in Table 1. Urea turnover increased significantly from mid- to late-lactation. Urea turnover constant was significantly greater for day 15–18 postpartum ($\bar{X} = 0.726 \pm 0.044$) than for day 9–13 postpartum ($\bar{X} = 0.550 \pm 0.100$; $t = 3.35$, $P < 0.01$). The contribution of body protein to estimated metabolism was significantly higher in late-lactation females than in midlactation females ($t = 4.12$, $P < 0.01$), supplying $11.2\% \pm 0.8\%$ of energy during late-lactation and $7.9\% \pm 1.6\%$ of energy at midlactation. Urea turnover constant varied significantly with adipose tissue proportions ($r^2 = 0.90$, $P < 0.01$; Fig. 3) and days postpartum ($y = 0.28 + 0.27x$; $r^2 = 0.66$, $P < 0.01$). The proportion of estimated energy metabolism from protein varied significantly with adipose tissue proportions ($r^2 = 0.73$, $P < 0.05$; Fig. 4A) and days postpartum ($r^2 = 0.74$, $P < 0.01$; Fig. 4B). BUN measurements were not significantly correlated with turnover constant ($r = 0.08$, $P > 0.05$) and were not significantly different between mid- and late-lactation samples ($t = 0.08$, $P > 0.05$).

GFR Measurements

Specific activities of blood samples taken simultaneously from flipper veins and the extradural vein differed by less than 3%, indicating thorough mixing in the vascular pool. Absolute and mass-proportional GFR increased between measurements. Data obtained from all measurements are shown in Table 2.

Table 1: Urea turnover constant, BUN, urea turnover rate, rate of protein catabolism, estimated daily metabolism, and the contribution of body protein to estimated metabolism for midlactation and late-lactation females

	Urea Turnover Constant	Adipose Tissue (%)	BUN (mg dL ⁻¹)	Urea Turnover Rate (g d ⁻¹)	Rate of Protein Catabolism (g d ⁻¹)	Daily Metabolism (MJ)	Contribution of Body Protein to Estimated Metabolism (%)
Midlactation females:							
1	.43	32.8	26.0	69.4	202.6	54.4	6.7
2	.47	32.5	22.1	72.1	210.6	58.3	6.5
3	.53	30.8	22.0	74.7	218.1	56.7	6.9
4	.65	28.3	26.0	84.0	245.3	48.5	9.1
5	.67	29.2	25.0	119.6	349.2	62.2	10.1
Mean	.55 ^a	30.7 ^b	24.2	84.0 ^c	245.2 ^d	56.0	7.9 ^e
SD	.10	1.8	1.8	18.5	54.0	4.5	1.5
Late-lactation females:							
1	.68	28.4	24.8	152.4	444.9	69.6	11.5
2	.70	27.4	24.3	105.4	307.9	54.3	10.2
3	.70	27.6	22.5	131.6	384.0	59.6	11.6
4	.75	25.6	21.4	124.7	364.1	61.8	10.6
5	.80	27.4	24.0	117.3	342.5	50.1	12.3
Mean	.73 ^a	27.3 ^b	22.3	126.3 ^c	368.7 ^d	59.1	11.2 ^e
SD	.04	.9	1.3	15.7	45.7	6.7	.7

Note. The urea turnover constant (K_u) was calculated as: $-\Delta \ln(SA)t^{-1}$, where SA is the specific activity of the plasma, and t is time. Urea turnover rate (r_u) was calculated as: $K_u \times N_u$, where N_u is urea pool size. Rate of protein catabolism (r_p) was calculated as: $r_u \times 2.292$ g protein g⁻¹ urea. Daily metabolism (MR) was estimated as: 0.1056 (mass) + 20.0 MJ. Contribution of body protein to estimated metabolism was calculated as: $100 \times (r_p \times 0.01799 \text{ MJ g}^{-1})/\text{MR}$. Means with the same superscript letters for mid- and late-lactation females are significantly different ($P < 0.01$).

Absolute GFR ranged from 167.8 to 420.3 mL min⁻¹. Mean GFR as a proportion of body mass was 0.49 ± 0.09 mL min⁻¹ kg⁻¹ for midlactation females and 1.03 ± 0.12 mL min⁻¹ kg⁻¹ for late-lactation females. This difference was significant

Figure 3. Changes in urea turnover constant (K_u) with decreasing adipose tissue proportions. Open circles are midlactation females (9–13 d postpartum), and filled circles are late-lactation females (15–18 d postpartum). The equation for the regression line is: $y = 0.05x - 2.06$ ($r^2 = 0.90$, $P < 0.01$).

($t = 8.07$, $P < 0.01$). On average, mass-specific GFR increased $114\% \pm 41\%$ between measurements. These values were compared to predicted mammalian GFR based on allometry as $5.35 \times (\text{body mass})^{0.72}$ mL min⁻¹ (Edwards 1975). Midlactation females exhibited $50\% \pm 7.9\%$ of predicted GFR, while late-lactation GFR was $97.3\% \pm 12.4\%$ of this value. GFR as a proportion of lean mass showed similar trends (0.74 ± 0.15 mL min⁻¹ kg⁻¹ at midlactation, 1.43 ± 0.18 mL min⁻¹ kg⁻¹ at late lactation; $t = 7.21$, $P < 0.01$). Creatinine concentrations declined significantly from 1.69 ± 0.19 to 1.11 ± 0.16 mg dL⁻¹ ($t = 9.41$, $df = 4$, $P < 0.01$). The BUN : creatinine ratio in plasma was significantly correlated with mass-specific GFR ($r = 0.88$, $P < 0.05$).

Discussion

Our data support the hypothesis that female elephant seals have a reduced ability to spare protein as lactation progresses. Urea turnover and the contribution of protein to energy metabolism increased significantly between mid- and late lactation. These changes varied directly with changes in maternal body composition. This suggests a dynamic relationship between the energetic demands of lactation and adipose tissue

Figure 4. Changes in the contribution of protein to energy metabolism with decreasing adipose tissue proportions (A) and increasing lactation duration (B). Open circles are midlactation females (9–13 d postpartum), and filled circles are late-lactation females (15–18 d postpartum). The equations for the regression lines are: A, $y = -0.79x + 32.36$ ($r^2 = 0.73$, $P < 0.01$); and B, $y = 0.50x + 2.81$ ($r^2 = 0.74$, $P < 0.01$).

proportions that determines the extent of protein catabolism. This represents a potentially important constraint on the duration and magnitude of maternal investment in northern elephant seals. These data explain the observed relationship between maternal body composition and proportional lean-tissue loss over lactation and give an empirical basis to the concept of a threshold level of proximate reproductive expenditure in northern elephant seals.

These findings are consistent with previous observations that rates of protein catabolism may be determined by the relative sizes of fat and protein pools and that at least some of the protein sparing associated with fasting is due to reductions in metabolic rate (Henry et al. 1988). Continued partitioning of nutrient reserves toward production of an increasingly lipid-rich milk might result in catabolism of functionally important lean tissue. In absence of the protein lost for milk synthesis, these changes might still have a significant impact on fasting duration. When added to the protein losses required for milk

production, these changes impose significant limitations on the female's ability to maintain lactation.

Increases in the rate of urea production during lactation are apparently rapidly matched by increases in urea excretion, resulting in little change in BUN concentrations. The efficiency of urea excretion drops with increasing urinary concentration and decreasing urine flow rate, because increased transit time in the distal nephron favors passive urea absorption (Bankir et al. 1993). While investigations in fasting, weaned elephant seal pups demonstrate decreases in GFR coupled with increases in urinary urea concentration (Pernia et al. 1989; Adams and Costa 1993), diminished urine volumes are associated with decreases in amino acid oxidation and improved tubular reabsorption.

Some investigators have suggested that GFR might increase in parallel with urine concentrating ability and ensure a normal rate of urea excretion in the face of a high, concentration-dependent urea resorption, with only small changes in plasma urea levels (Bankir et al. 1993). Fractional resorption of urea can be estimated using the following equation: % urea resorbed = $100 - [100 \times \text{urea excretion rate} / (\text{GFR} \times \text{BUN})]$. If we calculate the resorbed fraction using data from the present study, estimated fractional resorption increases from 51% to 62% from mid- to late lactation. In other words, the efficiency of urea excretion decreases from 49% to 38% of filtered urea. The well-established decline in the efficiency of urea excretion with increasing urine osmolality suggests that increases in rates of urea excretion in combination with stable or reduced urine flow would require an increase in GFR or an elevation in plasma urea levels. In the case of high urine concentrating activity, enhanced GFR reduces the increase in plasma urea levels required to increase rates of urea excretion (Bankir et al. 1993). Thus, increases in GFR over lactation might be an adaptation to decreased efficiency of urea excretion and greater urinary urea concentration necessitated by increased urea excretion rates and water stress late in the fast.

Renal blood flow and GFR are regulated in response to changes in arterial pressure and tubular flow rates determined by the solute concentrations of the tubular fluid. Significant reductions in the levels of serum proteins have been observed during lactation in northern elephant seals (J. Williams, unpublished data). In addition to supporting the idea of increased levels of protein use, this potentially affects the colloidal properties of the plasma. The movement of the fluid from the glomerular capillary into Bowman's capsule is opposed by the oncotic pressure of the plasma proteins. The reductions in plasma protein concentrations could potentially increase the flow rate of tubular fluid and increase GFR. It has been suggested that a vasopressin-induced, intrarenal urea recycling mechanism could indirectly depress tubuloglomerular feedback while improving urinary concentration, resulting in increased GFR (Bankir et al. 1993).

The observed increase in the BUN : creatinine ratio is oppo-

Table 2: GFR, mass, mass-specific GFR, and GFR as a proportion of lean-tissue mass for midlactation and late-lactation females

	GFR (mL min ⁻¹)	Mass (kg)	GFR/Mass (mL min ⁻¹ kg ⁻¹)	GFR/Lean Mass (mL min ⁻¹ kg ⁻¹)
Midlactation females:				
1	167.8	420	.40	.58
2	234.9	553	.43	.63
3	168.2	276	.61	.92
4	191.1	438	.44	.65
5	281.5	472	.60	.92
Mean	208.7	432	.49	.74
SD	43.8	90	.09	.15
Late-lactation females:				
1	317.4	313	1.01	1.40
2	381.5	448	.85	1.16
3	195.6	200	.98	1.36
4	381.2	323	1.18	1.64
5	420.3	370	1.14	1.61
Mean	339.2	331	1.03	1.43
SD	79.0	81	.12	.18

Note. All means are significantly different ($P < 0.01$) between mid- and late-lactation females.

site that reported in fasting elephant seal pups (Adams and Costa 1993), polar bears (Ramsay et al. 1991), gray wolves (Delguidice et al. 1987), prairie dogs (Harlow and Buskirk 1991), and martens (Harlow and Buskirk 1991). The increases in BUN : creatinine ratios in the present study appear to result from reductions in serum creatinine concentrations. The production of nonmetabolizable creatinine from the irreversible hydrolysis of creatine phosphate, primarily in skeletal muscle, should qualitatively reflect the level of muscle degradation. As BUN concentrations did not reflect the increases in urea production, we would interpret the increases in BUN : creatinine ratios as representing increased creatinine clearance due to increases in GFR. BUN : creatinine ratios (range: 12.6–25.0) were significantly higher than the value of 10, suggested by Nelson et al. (1984) to indicate attainment of efficient protein sparing in fasting bears.

The data presented here support the concept of homeorhesis during lactation in northern elephant seals, where milk composition is maintained at the expense of other biological processes. Increasing rates of protein catabolism across lactation and their relationship to maternal body composition suggest potential physiological constraints on the magnitude and duration of maternal investment during lactation in northern elephant seals. Changes in protein catabolism and kidney function during lactation support the idea that maternal metabolism and physiology are being modified in ways different from typical fasting physiology to meet the energy and nutrient demands of lactation. These data provide continued support for the

concept of a physiologically determined threshold level of reproductive expenditure in this species.

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