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Seasonal Physiology of the Wild Raccoon Dog (*Nyctereutes procyonoides*)

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ABSTRACT—The raccoon dog (*Nyctereutes procyonoides*) is a canid omnivore with autumnal fattening and winter sleep. Farmraised raccoon dogs have elevated plasma leptin and growth hormone levels in the winter and depressed plasma cortisol and insulin concentrations during wintertime food deprivation. However, these parameters were not previously tested in the wild population. In the present study 37 wild raccoon dogs were sampled at different seasons and diverse biochemical variables were determined. The results mostly confirmed previous observations on farmraised raccoon dogs. The liver glycogen stores increased during the autumnal fattening period but were low in the winter. The liver glycogen phosphorylase activity decreased but lipase activity increased in the winter indicating the use of fat as the principal metabolic fuel. The plasma insulin concentrations were low in the winter allowing the release of fatty acids from adipose tissue. Low wintertime cortisol and thyroid hormone levels could contribute to protein sparing. Unlike on farms, wild raccoon dogs did not show seasonal fluctuations in their plasma ghrelin or growth hormone levels. The observed physiological phenomena emphasise the adaptation of the species to long periods of food scarcity in the winter.

Key words: ghrelin, leptin, *Nyctereutes procyonoides*, raccoon dog, winter sleep

INTRODUCTION

The raccoon dog (*Nyctereutes procyonoides* Gray, 1834) or *tanuki* is a nocturnal canid native to East Asia (Siivonen 1972). Between the 1930's and 1950's it was introduced to the northwestern parts of the Soviet Union. From there it dispersed to western Europe, including Finland by the mid-1970's (Kauhala 1996). The northern distribution limit of the species currently lies between 65°N and the Arctic Circle. The species has been reared commercially for the fur trade in Finland since the early 1970's with captured animals (Mäkelä 1973).

The raccoon dog is a true omnivore, and the availability of different food items affects the composition and diversity of its diet (Viro and Mikkola 1981). Wild raccoon dogs have a body mass (BM) of 3.5–10.5 kg (Kauhala 1992) and farmraised animals 5.5–14.5 kg (Asikainen *et al.,* 2002). The mean BM varies 30–40% throughout the year, being the highest in Oct-Dec and the lowest in the spring and summer (Korhonen *et al.,* 1991; Kauhala 1992).

The raccoon dog exhibits profound autumnal fattening in preparation for the winter (Kauhala 1992). It is the only canid spending 4–5 months of the midwinter in a burrow or a den in superficial hibernation-like state called winter sleep in this study. Winter sleep is different from hibernation as the body temperature remains close to normal and there can be occasional periods of arousal, food intake and defecation. This occurs in areas of snowy winters with the ambient temperature below –5 – –10°C for long periods. During winter sleep the raccoon dogs mostly fast (Nowak 1993) with fat as the principal metabolic fuel (Mustonen *et al.,* 2004). This seasonal rest is facultative and raccoon dogs are known to forage, defecate or eat snow during milder weathers (J. Asikainen, personal communication, 2003). Young individuals, who have not been able to gather sufficient fat stores, are sometimes active also during colder periods. Farmraised animals do not experience winter sleep but they also show seasonal fluctuations in their voluntary energy intake, which is highest in the autumn and declines in the winter (Nieminen *et al.,* 2002; Mustonen *et al.,* 2004). The raccoon dog is a monogamous seasonal breeder (Yamamoto 1987). Mating occurs in southern Finland in Feb or March and the gestation period is 61.0±2.0 days (Valtonen *et al.,* 1977).

Leptin is a hormone secreted mainly by the white adipose tissue (WAT; Zhang *et al.,* 1994). Leptin levels of

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humans and laboratory rodents correlate positively with body adiposity (Maffei *et al.,* 1995), being suppressed by fasting and increased by refeeding (Kolaczynski *et al.,* 1996). In *ob/ob* mice, leptin decreases food intake, BM and adiposity (Pelleymounter *et al.,* 1995). In starvation, falling leptin levels disinhibit the production of hypothalamic neuropeptide Y (NPY) leading to energy preservation (Ahima *et al.,* 1996). Ghrelin is another novel signal peptide from the gastrointestinal tract and the hypothalamus (Kojima *et al.,* 1999). Its secretion is regulated by cholinergic neurons (Sugino *et al*., 2003). Ghrelin stimulates growth hormone (GH) secretion, reduces fat utilization, and increases food intake and BM. Plasma ghrelin levels are increased by fasting and reduced by refeeding and obesity (Tschöp *et al.,* 2000, 2001) and a transient ghrelin peak can be observed shortly before feeding (Sugino *et al*., 2002a, b). Ghrelin antagonizes leptin action by activating the NPY pathway (Shintani *et al.,* 2001). GH is secreted by the anterior hypophysis and promotes growth by increasing endochondral ossification (Brück 1983). In humans, hypoglycemia increases the rate of GH secretion e.g. at times of inadequate nutrition. GH is also able to increase lipolysis (Richelsen 1997).

Farmraised raccoon dogs have relatively low leptin and GH levels in the early autumn, which increase simultaneously with increasing BM and WAT mass (Nieminen *et al.,* 2001, 2002). In early Nov the animals experience a metabolic transition into winter sleep. This is characterised by rapidly fluctuating leptin and GH levels with an initial decline followed by an increase in Nov-Dec. In the winter, the plasma leptin and GH concentrations are relatively high but the ghrelin levels low. At this time of the year, these hormones probably work together to induce the use of fat as the principal metabolic fuel. Furthermore, the species has remarkable adaptations to long periods of fasting. In fact, an eight-week fast does not affect plasma leptin, ghrelin or GH levels of raccoon dogs (Nieminen *et al.,* 2002) and the animals are able to defend their muscle tissue mass without significant protein catabolism (Mustonen *et al.,* 2004).

Seasonal physiology of the raccoon dog has been studied only in farmraised animals. Yet it remains unknown how these physiological phenomena are manifested in wild raccoon dogs with smaller fluctuations in WAT mass (Mäkelä 1973; Korhonen 1988). This work is a part of a project studying seasonal metabolic and endocrinological adaptations of the species.

MATERIALS AND METHODS

The experimental animals (15 males: 10 juveniles, 5 > one year old; 22 females: 13 juveniles, 9 > one year old) were trapped or hunted with a dog between Oct 2000 and Jan 2002 according to the Finnish hunting legislation in North Carelia, Finland (62°N, 29°E). The climate of the region is boreal with the ambient temperature below freezing and permanent snow cover (40–100 cm) between November and March. The animals were housed singly in a cage and fasted for 12–24 hrs and given water *ad libitum*. They were sacrificed with an electric shock according to EU regulations (Council of the European Union 1993). All the procedures conformed to the Helsinki Convention. The age of the animals (classified as juveniles or adults) was determined according to the stage of fur development (J. Asikainen, personal communication 2002) or the annual incremental lines in the tooth sementum (Kauhala and Helle 1990). The animals were weighed and the body lengths were measured along the ventral midline from the nose to the anus (cm). From these data the body mass index [BMI = weight (kg)/length^3 (m)] reflecting body adiposity was calculated. The formula correlates (r_s = 1.000) with the obesity index empirically derived for the species (Korhonen *et al.,* 1982). Blood samples were obtained with cardiac punctures using sterile needles and syringes. They were taken into test tubes containing EDTA to prevent clotting and centrifuged at 1000 *g*. The livers and kidneys were dissected, weighed and all the samples were stored at –40°C.

The plasma glucose concentrations were determined with the liquid reagent hexokinase method, cholesterol concentrations with the enzymatic endpoint method, triglyceride concentrations with the GPO-PAP method, and creatinine concentrations with the colorimetric method of Randox Laboratories Ltd. (Swords, Ireland) with the Technicon RA-XT^{TM} analyser (Technicon Instruments Corporation, Tarrytown, NY, USA). The plasma total thyroxine (T_4), triiodothyronine (T_3), and cortisol concentrations were measured with the Spectria [^{125}I] Coated Tube Radioimmunoassay (RIA) kits (Orion Diagnostica, Espoo, Finland). The plasma insulin concentrations were determined with the Coat-A-Count Insulin kit (Diagnostic Products Corporation, Los Angeles, CA, USA). The plasma leptin concentrations were measured with the Multi-Species Leptin RIA kit (Linco Research, St. Charles, MO, USA) and ghrelin levels with the Ghrelin (Human) RIA kit (Phoenix Pharmaceuticals, Belmont, CA, USA). The plasma GH concentrations were determined using the hGH Human Growth Hormone Double Antibody kit of DPC. All the assays were previously validated for the species (Nieminen *et al.,* 2002; Asikainen *et al.,* 2003; Mustonen *et al.,* 2004).

Enzyme activities were determined spectrophotometrically. The liver and kidney samples were weighed and homogenized in cold citrate buffer in pH 6.5 for the glucose-6-phosphatase (G6Pase), in pH 6.1 for the glycogen phosphorylase and in cold 0.85% NaCl for the lipase measurements. The activity of G6Pase was measured using glucose-6-phosphate as substrate in the presence of EDTA after an incubation time of 30 minutes at 37.5°C (Hers and van Hoof, 1966). The glycogen phosphorylase activity was measured in the presence of glucose-1-phosphate, glycogen, sodium fluoride and AMP according to the method of Hers and van Hoof (1966). The lipase activity was measured according to the method of Seligman and Nachlas (1962) using 2-naphthyl-laurate without taurocholate as substrate. Glycogen concentrations were measured spectrophotometrically according to the method of Lo *et al.* (1970).

For statistical analyses the animals were assigned into 3 groups according to season. The aim of this classification was to describe the seasonal fluctuations of the diverse physiological and biochemical variables. The division of months into the respective seasonal groups was based on previous observations on the seasonal changes in physiological variables of the species (Nieminen *et al.,* 2002, Asikainen *et al.,* 2003, Mustonen *et al.,* 2004) and further classification was performed according to the results of this study. There was no sexual dimorphism in the measured variables, and for this reason the data for the males and the females are pooled together in the results section. The animals were also classified according to age. The only significant differences between the age groups were in the BMs, BMIs, body lengths and absolute liver weights of the animals. In other cases, the results of adults and juveniles are pooled together.

Multiple comparisons were performed with the one-way analysis of variance (ANOVA) followed by the *post hoc* Duncan's test. The normality of distribution and the homogeneity of variances

were tested with the Kolmogorov-Smirnov test and the Levene test. Comparison within groups between consecutive measurements were analysed using repeated measures ANOVA and t-test for related samples. Comparisons between the study groups were analysed with the Student's t-test and with the Mann-Whitney U test for nonparametrical data. A p-value less than 0.05 was considered to be statistically significant. The results are presented as the mean±SE.

RESULTS

The seasonal variations of the measured variables followed individual rhythms. For instance, the plasma cortisol concentrations were relatively low in the winter but quite high during the rest of the year. On the other hand, many of

Table 1. Mean BM, BMI, length, liver weight, plasma glucose, creatinine, cholesterol, triglyceride and hormone concentrations, and liver (L) and kidney (K) glycogen content and enzyme activities ± SE of wild raccoon dogs at different seasons. Late autumn = Nov (n = 9), Winter = Dec-Feb (n= 9), Spring-early autumn = April-Oct (n = 19) for BM, BMI, length, liver weight, creatinine, thyroid hormones, leptin, ghrelin, GH and insulin data; Late autumn = Oct-Nov (n = 16), Winter = Dec-April (n = 11), Spring-early autumn = May-Sep (n = 10) for other data.

	Late autumn	Winter	Spring-early autumn
BM (kg)	6.54 ± 0.40^{b}	5.26 ± 0.28^{ab}	4.36 ± 0.96^{a}
BMI (kg/m^3)	27.3 ± 1.1^{b}	22.4 ± 1.2^{a}	23.5 ± 2.5^{ab}
Length (cm)	61.81 ± 0.77	61.73 ± 0.47	55.89 ± 2.27
Liver weight (kg)	0.29 ± 0.01^{b}	0.22 ± 0.01^{a}	0.19 ± 0.02^{a}
Liver weight/BM ‰	44 ± 3	42 ± 2	50 ± 4
Glucose (mmol/l)	7.13 ± 0.48	8.11 ± 0.75	7.87 ± 1.15
Creatinine (μmol/l)	106.33 ± 14.99^{b}	96.71 ± 7.68^{b}	70.06 ± 2.57^{a}
Cholesterol (mmol/l)	5.11 ± 0.42	4.46 ± 0.67	4.51 ± 0.28
Triglycerides (mmol/l)	1.41 ± 0.23	1.22 ± 0.26	0.99 ± 0.18
T_4 (nmol/l)	24.11 ± 1.81^{ab}	18.68 ± 1.73^{a}	26.35 ± 1.94^{b}
T_3 (nmol/l)	0.87 ± 0.08^{b}	0.51 ± 0.05^{a}	0.79 ± 0.10^{b}
T_3/T_4 %	3.71 ± 0.31	3.02 ± 0.53	3.16 ± 0.31
Leptin (ng/ml)	3.00 ± 0.86	1.24 ± 0.18	2.12 ± 0.14
Ghrelin (ng/ml)	2.33 ± 0.13	2.21 ± 0.37	2.18 ± 0.08
Ghrelin/leptin	1.01 ± 0.14^{a}	1.82 ± 0.35^{b}	1.11 ± 0.08^{a}
Growth hormone (ng/ml)	1.14 ± 0.21	1.18 ± 0.13	0.87 ± 0.12
Insulin (μIU/ml)	22.94 ± 9.65^{b}	3.51 ± 1.06^{a}	8.75 ± 2.36^{a}
L Glycogen (mg/g)	15.63 ± 3.31^{b}	7.31 ± 1.51^{a}	5.03 ± 0.95^{a}
K Glycogen (mg/g)	0.57 ± 0.04	0.46 ± 0.08	0.55 ± 0.03
L G6Pase (μg P/mg/h)	29.53 ± 1.83^{a}	32.42 ± 2.91^{a}	42.11 ± 2.14^{b}
K G6Pase (μg P/mg/h)	18.90 ± 1.57^{b}	12.43 ± 1.27^{a}	21.51 ± 1.40^{b}
L Phosphorylase (μg P/mg/h)	41.62 ± 1.79^{b}	28.27 ± 1.35^{a}	41.84 ± 1.95^{b}
K Phosphorylase (μg P/mg/h)	4.77 ± 0.30	4.30 ± 0.27	4.44 ± 0.20

P: one-way ANOVA. Means with different superscripts differ at $p < 0.05$.

Table 2. BM, BMI, body length and absolute liver weight of juvenile and adult wild raccoon dogs. Data are mean ± SE. Spring-early autumn = April-Oct, late autumn = Nov, winter = Dec-Feb.

		n	Weight (kg)	Length (cm)	BMI (kg/m^3)	Liver weight (g)
Spring-early autumn	Juveniles	7	2.77 ± 0.15*	51.5 ± 0.7*	20.5 ± 0.6	149 ± 9*
	Adults	3	8.07 ± 1.97	64.7 ± 1.2	29.5 ± 6.8	274 ± 18
Late autumn	Juveniles	9	5.69 ± 0.42*	60.7 ± 0.9	25.2 ± 1.2*	264 ± 17*
	Adults	7	7.64 ± 0.52	63.3 ± 1.2	30.0 ± 1.3	328 ± 16
Winter	Juveniles	8	5.07 ± 0.28	61.4 ± 0.5	21.9 ± 1.2	216 ± 11
	Adults	3	5.80 ± 0.75	62.7 ± 0.9	23.8 ± 3.5	221 ± 30

* = Significant difference between the juvenile and adult animals, t-test, $p < 0.05$.

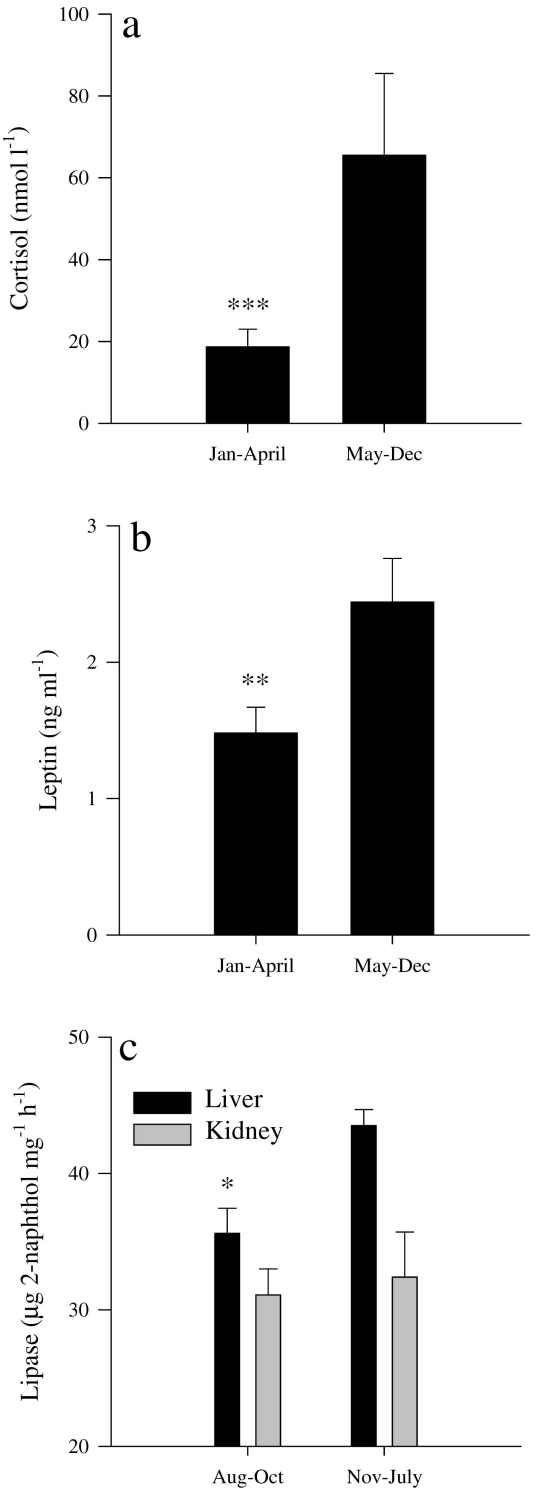

Fig. 1. Plasma cortisol concentrations (a; n = 10 Jan-April, n = 25 May-Dec), leptin concentrations (b; n = 9 Jan-April, n = 24 May-Dec) and liver (c; n = 16 Aug-Oct, n = 20 Nov-July) and kidney (c; n = 14 Aug-Oct, n = 9 Nov-July) lipase activities of the wild raccoon dogs at different seasons (mean±SE). *** = Significant difference between seasons (t-test, $p < 0.001$), ** = significant difference between seasons ($p < 0.02$, Mann-Whitney U test), * = significant difference between seasons (t-test, $p < 0.02$).

the enzymatic variables, such as the liver G6Pase activities clearly showed another type of seasonal variation with three distinct periods. Due to these different seasonal rhythms of the variables the seasons were classified as follows: Late autumn = Nov, winter = Dec-Feb, and spring-early autumn = April-Oct for BM, BMI, length, liver weights, creatinine, thyroid hormones, ghrelin, GH and insulin data; late autumn = Oct-Nov, winter = Dec-April, and spring-early autumn = May-Sep for the other variables. The cortisol and leptin data were divided between Jan-April and the rest of the year and the liver and kidney lipase data between Aug-Oct and the rest of the year. Spring-early autumn represents the active foraging period including most of the autumnal fat storage. Late autumn is the period of the final fat storage and the metabolic transition into winter catabolism and winter is the period of winter sleep.

The BMs of the raccoon dogs were relatively low in the spring-early autumn, increased in the late autumn and started to decline in the winter (ANOVA, $p < 0.05$; Table 1). The same was observed in the BMIs. The absolute liver weight also followed the same pattern, but the relative liver weights showed no seasonal changes. The juvenile and adult raccoon dogs differed from each other only in the BM and BMI values, body lengths and in the absolute liver weights (t-test, $p < 0.05$; Table 2).

The plasma glucose, total cholesterol and triglyceride concentrations remained stable at all seasons, but the plasma creatinine concentrations increased in the late autumn and remained at a relatively high level in the winter (ANOVA, $p < 0.05$; Table 1). The plasma T_4 and T_3 concentrations were lower in the winter compared to the spring-early autumn (ANOVA, $p < 0.05$), but no seasonal changes were detected in the T_3T_4 ratios. The plasma insulin concentrations were the highest in the late autumn (ANOVA, $p < 0.05$). There was no clear seasonal pattern in the plasma ghrelin or GH concentrations. The plasma cortisol and leptin concentrations were lower between Jan-April than during the rest of the year (t-test, $p < 0.001$; Fig. 1a and Mann-Whitney U test, $p < 0.02$; Fig. 1b).

The liver glycogen content was the highest in the late autumn but the kidney glycogen levels did not fluctuate (ANOVA, $p < 0.05$; Table 1). The liver G6Pase activity was the highest in the spring-early autumn and declined in the late autumn, while the kidney G6Pase and the liver glycogen phosphorylase activities decreased in the winter (ANOVA, $p < 0.05$). The kidney phosphorylase activities remained at the same level throughout the year. The liver lipase activity was lower during Aug-Oct compared to the rest of the year (t-test, $p < 0.02$; Fig. 1c) but in the kidney lipase activity there were no seasonal changes.

DISCUSSION

The raccoon dog is an interesting model to study seasonal obesity, fasting and winter sleep. It is of medium size, common in nature and easily reared in fur farms. The sea-

sonal physiological changes observed previously in farmraised animals (Nieminen *et al.,* 2001, 2002; Mustonen *et al.,* 2001, 2004; Asikainen *et al.,* 2002, 2003) could for the most part be detected in their wild counterparts, too. A major problem in the present study was the lack of repeated measurements of the different biochemical values of individual animals. Yet to obtain this the wild raccoon dogs would have to be restrained and kept in a facility, which would have made them essentially farmraised individuals.

In the autumn the raccoon dog very effectively stores energy reserves as subcutaneous fat (Korhonen 1988). This could be clearly seen in the higher BMs and BMIs of the animals in the late autumn. This phase was also characterised by the high liver weights, liver glycogen stores and glycogen phosphorylase activities. The high plasma insulin concentrations observed in the late autumn could enhance the storage of energy as fat and glycogen (Brück 1983). G6Pase liberating glucose into the circulation during e.g. gluconeogenesis (Harris 1986) is not as important during this time of year. In fact, the high plasma insulin levels could have contributed to this by downregulating gluconeogenesis (Kaloyianni and Freedland 1990) leading to a relatively low G6Pase activity.

During Aug-Oct, the liver lipase activity was lower than in the winter also fitting to these observations on the autumnal physiology of the wild raccoon dog. As fat is being deposited in subcutaneous adipose tissue (Korhonen 1987), it is prudent to keep its hepatic degradation and thus the lipase activity low. Unlike in farmraised raccoon dogs (Mustonen *et al.,* 2004), the plasma lipid levels of the wild animals did not show any clear seasonal variations. The relatively small number of wild animals available to this study could have masked some of the effects observed on fur farms. However, both the farmraised (Mustonen *et al.,* 2004) and wild raccoon dogs can maintain their plasma glucose levels stable despite of great fluctuations in energy intake and adipose tissue mass. It could be of importance for the species to keep the circulating glucose concentrations on a level that is high enough to enable physical activity, such as foraging or avoiding predators even in relatively unfavourable conditions.

In Finland the raccoon dog spends the coldest part of the year in superficial winter sleep like bears (*Ursus* spp.) and badgers (*Meles meles*; Siivonen 1972). The liver glycogen stores of wild raccoon dogs decreased as the animals entered winter metabolism. At the same time, the glycogen phosphorylase activity declined to a lower level. This points to the significance of adipose tissue as the main source of metabolic energy in the winter. The importance of fat is further emphasised by the higher liver lipase activities in Nov-July compared to Aug-Oct (Fig. 1c).

In the farmraised raccoon dog weight-regulatory hormones – ghrelin and GH – interact in order to maintain lipolysis during the winter months (Nieminen *et al.,* 2002). In the wild raccoon dogs, however, no clear seasonal changes could be observed in the circulating concentrations of these hormones. Of course, the farmraised raccoon dog accumulates more subcutaneous fat due to very favourable feeding conditions (Asikainen *et al.,* 2003) and the lack of winter sleep (Korhonen 1987). It is possible that the smaller amount of fat gathered in the wild (Mäkelä 1973; Korhonen 1988) could dampen these changes even in the presence of other seasonal factors, such as the photoperiod and its effector hormone melatonin. However, the observed pattern of relatively low plasma leptin concentrations during Jan-April and the higher levels during the other months could be related to the function of leptin as a signal of nutritional status (Maffei *et al.,* 1995). The lower wintertime leptin levels could thus reflect the diminishing amount of adipose tissue during the winter sleep and its immediate recovery period. As the replenishment of fat stores started in the spring and continued in the summer and autumn the leptin levels increased to a higher level than in the winter.

In the winter the plasma insulin concentrations of wild raccoon dogs declined to about 15% of the values observed in the autumn. These findings are analogous with fasting dogs (*Canis familiaris*; de Bruijne *et al.,* 1981) and farmraised raccoon dogs (Mustonen *et al.,* 2004). It is known that insulin inhibits the hydrolysis of triglycerides and the release of fatty acids from WAT (Cahill 1976). The low insulin levels encountered in the wintering raccoon dogs of this study are probably required for sufficient triglyceride mobilization during the winter. Low insulin can also induce the sparing of carbohydrates by decreasing glucose uptake to muscle and fat, enable the decrease in metabolic rate (Rothwell *et al.,* 1983) and stimulate gluconeogenesis and ketogenesis (Kaloyianni and Freedland, 1990) required for successful winter life.

The plasma T_4 and T_3 concentrations of the wild raccoon dogs decreased in the winter. The same phenomenon has been previously observed in farmraised raccoon dogs (Korhonen 1987; Nieminen *et al.,* 2001). Decreased thyroid hormone levels downregulate metabolic rate and can thus contribute to energy saving (Brück 1983). Thyroid hormone levels also decrease in the actively wintering mink during the cold season (*Mustela vison*; Boissin-Agasse *et al.,* 1981). The same has also been documented in the wintering beaver (*Castor canadensis*; Aleksiuk and Cowan 1969) with reduced thyroid activity, normal body temperature, low food intake and lethargy, phenomena that characterise the winter sleep of the raccoon dog. In this respect, the wild raccoon dog is very similar to other species experiencing food scarcity as a part of their natural seasonal rhythms.

The lowered plasma cortisol concentrations during Jan-Apr also fit to this pattern of energy saving (Fig. 1a). Fasting decreases the plasma cortisol levels of farmraised raccoon dogs in the winter (Mustonen *et al.,* 2004). As the raccoon dogs are able to defend against protein catabolism, it is possible that this efficient protein sparing would require low circulating cortisol and thyroid hormone concentrations (Goldberg *et al.,* 1980, Mustonen *et al.,* 2004) observed in this study, too. The observed wintertime increase in the plasma creatinine levels has also been previously docu-

mented in denning bears (Nelson *et al.,* 1973) and in food-deprived farmraised raccoon dogs (Mustonen *et al.,* 2004).

During the spring and summer raccoon dogs replenish their energy reserves. The adipose tissue mass is at its lowest seasonal level and the animals have to forage actively to meet their metabolic demands. The relatively low fat stores and increased locomotion set demands for intermediary metabolism to maintain blood glucose levels stable. The glycogen stores were still low, but the G6Pase activity increased in spring-early autumn liberating more glucose into the circulation when foraging activity returned. At the same time, the glycogen phosphorylase activity returned to the autumnal level indicating increased carbohydrate turnover.

The results of this study indicate that the wild raccoon dog has adapted to marked seasonal changes in its energy reserves. As a consequence the species has been able to colonize new areas effectively. The physiological systems of the species optimize the use of relative autumnal abundance with effective energy storage and the mobilization of fuel from adipose tissue in the winter. In the wild raccoon dog, these phenomena are not reflected in the concentrations of the weight-regulatory hormones as clearly as in the farmraised animals.

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