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Seasonal changes in reindeer physiology

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Abstract: The seasonal changes in the photoperiod, temperature and availability of food need to be converted to hormonal signals in order to induce adaptations in the physiology of the reindeer. The most reliable of the seasonal changes in the environment is the photoperiod, which affects the reindeer physiology through pineal gland and its hormone, melatonin. Usually there are large diurnal changes in the concentration of melatonin, but in the reindeer the daily rhythm disappears during the arctic summer to return again in the autumn. Seasonal changes in melatonin secretion are involved in the regulation of reproduction, the growth of pelage, thermogenesis, body mass and immune function. Melatonin may exert its effects through gene activation, but the mechanisms are not completely understood. Other hormones that show seasonality are thyroid hormones, insulin and leptin. Thus the observed physiological changes are a result of actions of several hormones. Appetite, energy production and thermogenesis are all vital for survival. During winter, when energy balance is negative, the reindeer uses mainly body fat for energy production. The use of fat stores is economical as the rate of lipolysis is controlled and the use of fatty acids in tissues such as muscle decreases. Only in severe starvation the rate of lipolysis increases enough to give rise to accumulation of ketone bodies. The protein mass is maintained and only in starved individuals muscle protein is used for energy production. The winter feed of the reindeer, the lichens, is poor in nitrogen and the nitrogen balance during winter is strongly negative. Reindeer responds to limited availability of nitrogen by increasing the recycling of urea into rumen. In general the adaptation of reindeer physiology enables the reindeer to survive the winter and although several aspects are known many others require further studies.

Key words: energy consumption, feed intake, leptin, lipolysis, melatonin, proteolysis, *Rangifer tarandus*, starvation.

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Introduction

Survival, growth and production of offspring are of key importance for every animal species. In arctic species, such as reindeer, these basic physiological functions are modulated by the seasonal changes in the availability and quality of food, wide changes in the temperature and photoperiod. Also in reindeer the response to these environmental cues is manifested in the seasonal changes in its physiology (Fig. 1). To be effective, the changes in physiology have to be initiated before the changes occur in the living conditions, *i.e.* the changes have to be predictive. For example the growth of winter fur coat has to begin well before the cold season. In order to predict the changes in season the reindeer has to respond to changes in their surroundings, the most important of which are temperature, availability of food and the length of the photoperiod. Among these the length of the photoperiod is the most reliable indicator of the change of the season and therefore seasonality of many physiological phenomenon is based on the length of the day.

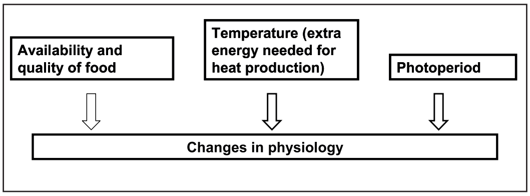

Fig. 1. Seasonal changes in photoperiod, temperature, and availability of food are the main environmental cues that through hormones induce the adaptive changes that enable the reindeer to survive the winter.

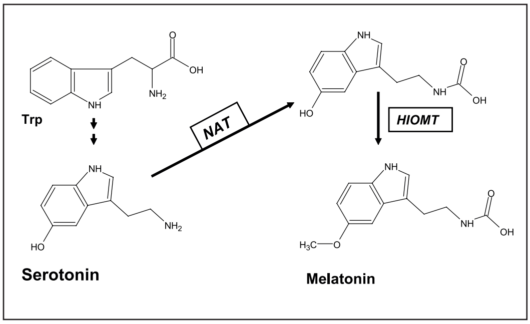

Fig. 2. Synthesis of melatonin. NAT = arylalcylamine N-acetyltransferase, HIOMT = hydroxyindole-O-methyltransferase.

Seasonal changes in melatonin and other hormones

The daylight or its absence has to be transduced to a chemical signal and this occurs in the pineal gland to which the information is carried from retina via a multisynaptic pathway. In the pineal gland the information of illumination is converted to a hormone, melatonin that in turn serves as a messenger to every organ in the body. Melatonin is synthesized during the dark period and the synthesis ceases rapidly in light (for reviews see Nelson & Demas, 1997; Malpaux *et al.*, 2001; Bartness *et al.*, 2002; Thiéry *et al.*, 2002).

The synthesis of melatonin starts from the amino acid tryptophan, which is first converted to serotonin, a neurotransmitter (Fig. 2). Two enzymes are needed to convert serotonin to melatonin. The first is arylalcylamine N-acetyltransferase (NAT, EC 2.3.1.87) which catalyzes acetylation of the amino group, and the other is hydroxyindole-O-methyltransferase (EC 2.1.1.4), which adds a methyl group to the hydroxyl group. The regulation of synthesis occurs through daily changes in the activity of NAT, which appear to be endogeneous, because the fluctuation continues even if the animals are kept in constant darkness (Vanacek, 1998). Also when the continuous illumination during the summer abolishes the circadian rhythm of melatonin secretion in reindeer, the pineal gland has not lost its capacity to release of melatonin, which starts rapidly if the reindeer are exposed to darkness (Eloranta *et al.*, 1992).

Melatonin is a small lipophilic molecule, which allows it to leave the cells of the pineal gland by diffusion and also to enter the target cells by the same method. The half- life of melatonin in serum is short, about 10 min. Melatonin is removed from circulation by the liver, which converts it to sulphate or glucuronide that are biologically inactive, no more lipophilic, and can be excreted in the urine (Vanacek, 1998).

Eloranta *et al.* (1995) have studied the seasonal changes in the daily fluctuations of melatonin concentrations in reindeer. During the dark winter season, the melatonin pulse is long and its amplitude is high, while during the summer release of melatonin is negligible. The duration of the pulse allows the reindeer to distinguish seasons with short daylight from those of long daylight. But along the year, in the spring and in the autumn the photoperiod is equally long and also the amplitude and length of the melatonin pulses around the equinoxes do not differ. To be able to distinguish whether it is spring or autumn, *i.e.* whether the duration of the pulse is increasing or decreasing, the reindeer has to have a memory of previous melatonin pulses. Malpaux *et al.* (2001) have in their review listed that the three main points that are the prerequisite for the effective role of melatonin in the regulation of circadian and more importantly the circannual cycles are (i) the absence or presence of melatonin, which enables to distinguish day from night, (ii) the duration of the pulse that is needed to distiguish short days from long ones and (iii) the direction of the change that is necessary to distinguish the season. There is evidence to support the view that the direction of the change is the most important factor to synchronize the physiological functions. In sheep it has been shown that the lengthening of days is critical to synchronize the reproductive rhythm and the shortening is necessary to time the rut and also to maintain its full duration (Woodfill *et al.*, 1991).

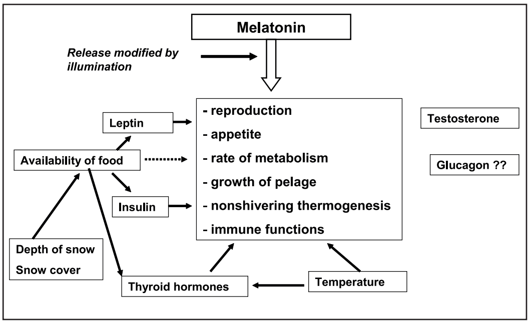

Fig. 3. Release of melatonin from the pineal gland is regulated by photoperiod, and the physiological effects of the melatonin are modified by several other hormones the concentrations of which also show seasonal variation.

The list of seasonal changes regulated or initiated by melatonin is long (Nelson & Demas, 1997; Carlberg, 2000; Malpaux *et al.*, 2001; Thiery *et al.*, 2002). Melatonin is needed to syncronize the rut so that calving occurs in the spring and enables better possibilities of survival for the offspring. Melatonin is also involved in the regulation of body mass, changes in voluntary feed intake, regulation of metabolic rate, nonshivering thermogenesis and growth of pelage. In the target tissue melatonin exerts its effect through binding to receptors which have been found both on the plasma membrane and in the nucleus (Williams *et al.*, 1996; Vanacek, 1998; Carlberg, 2000). Membrane receptors are found in the central nervous system, while in other tissues the receptors are inside the cells. Membrane receptors are found mainly in the pars tuberalis of the anterior pituitary and in many species also in the hypothalamus, although red deer and other ungulates may be devoid of the latter (Williams *et al.*, 1996). Membrane receptors of melatonin are coupled to a G-protein and binding of melatonin decreases the concentration of cAMP in the cell. The actions of melatonin are not mediated only through cAMP, because concentrations of several other signal molecules, such as those of the calcium-calmodulin signalling pathway, may be altered as well (Vanacek, 1998). The knowledge of the mechanisms of action of melatonin at the cellular level is scarce, although the reproductive and circadian actions at the level of central nervous system are assumed to be regulated through membrane receptors (Vanacek, 1998; Carlberg, 2000).

The intracellular receptors are probably present in all cells (Carlberg, 2000). When bound to the receptor, melatonin alters the phosphorylation of a group of transcription factors RZR/ROR that are known to regulate transcription of several genes. However, the physiological role of these actions of melatonin is not yet known.

In reindeer as in other species the effects of photoperiod and season on reproduction, changes in the voluntary food intake and metabolism are probably initiated through changes in the pattern of melatonin secretion, but the profiles of many other hormones, downstream from melatonin are also altered by photoperiod and availability of food and thus the changes in these compensatory signals may be required for the actions of melatonin (Fig. 3). These signals may include hormones, such as leptin, insulin and thyroid hormones. Leptin that is synthesized in the adipose tissue, affects the same physiological phenomena as melatonin, such as appetite, thermogenesis and activity of reproduction (Chilliard *et al.*, 2001; Morgan & Mercer, 2001; Friedman, 2002; Prentice *et al.*, 2002). In sheep the photoperiod may regulate the expression of leptin gene, but it is also inhibited by food deprivation (Chilliard *et al.*, 2001). In accordance to this a recent study by Soppela *et al.* (2003) shows that also in reindeer the serum concentration of leptin decreases during winter both in *ad libitum* fed and especially in undernourished reindeer calves. This finding is in accordance with the view that leptin plays a central role in the regulation of body energy homeostasis. Also concentration of insulin that is one of the key hormones to regulate metabolism, decreases during winter (Larsen *et al.*, 1985a; Soppela *et al.*, 2003). Decreases in insulin and leptin concentrations may be interconnected, because *in* vitro data suggests that pharmacological doses of insulin may promote the expression of leptin gene (Harris, 2000).

Thyroid hormones have a central role in the regulation of basal metabolic rate and their release increases at low ambient temperatures. In reindeer controversial results have been reported on the seasonal changes in T3 and T4 concentrations with a nadir occurring either in winter or summer while others have found no seasonal variation (Ryg & Jacobsen, 1982; Nilssen *et al.*, 1985; Timisjärvi *et al.*, 1994; Bubenik *et al.*, 1998). Thus it has been concluded that changes in the concentrations of thy-

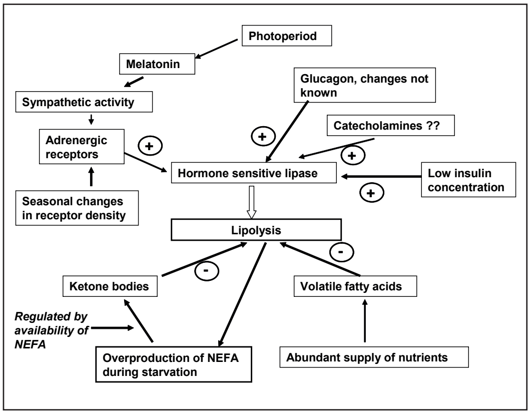

Fig. 4. Regulation of hormone sensitive lipase (HSL) and lipolysis in adipose tissue. + indicates a factor that increases the activity of HSL and – indicates inhibition.

roid hormones do not indicate variations in basal metabolic rate, but they rather fluctuate according to feeding (Ryg & Jacobsen, 1982; Ryg, 1984; Nilssen *et al.*, 1985).

Seasonal changes in energy balance

During winter energy balance of reindeer is negative. Appetite is reduced due to changes in concentrations of melatonin and leptin (Rhind *et al.*, 1998; Bartness *et al.,* 2002; Prentice *et al.*, 2002; Thiéry *et al.*, 2002), but also the availability and quality of food may contribute. The balance is also affected by the low temperatures that increase the demand of energy for heat production. To survive the reindeer is dependent on the energy stores that were deposited during the summer season, when the availability of good quality forage is abundant (Larsen *et al.*, 1985c; Suttie & Webster, 1995; Gerhart *et al.*, 1996). The strategies the reindeer uses to survive include good insulation, with the lower critical temperature as low as -30 °C (Nilssen *et al.*, 1984b), and the decrease in resting metabolic rate which is accompanied by less time spent moving (Nilssen *et al.*, 1984a, 1984b; Cuyler & Øritsland, 1993).

One of the most pronounced changes is the marked decrease in the voluntary feed intake during winter, a finding that is apparent also when the reindeer are fed high quality feed all year round (McEwan & Whitehead, 1970; Ryg & Jacobsen, 1982; Nilssen *et al.,* 1984b; Larsen *et al.*, 1985c; Mesteig *et al.*, 2000). Although it is known that hormones such as melatonin and leptin affect appetite, correlations between feed intake and hormone concentrations are generally low (Rhind *et al.*, 2000). This has been taken as an indication that single samples taken at interval of several days cannot adequately describe the relationship (Rhind *et al.*, 2000). Likewise the question whether the decrease in appetite precedes that in metabolism or vice versa is still an open. There is evidence to suggest that the adaptations of voluntary feed intake are initiated by the changes in the secretion of melatonin, with many other hormones downstream from melatonin probably contributing as well (Rhind *et al.*, 1998), but also data to suggest that metabolic changes precede those of appetite has been reported (Thiéry *et al.*, 2002).

Because the availability of food cannot be predicted in the early winter the survival strategy has to be based on economic and controlled use of energy stores. About 85% of energy is stored in the body fat deposits under the skin, around internal organs, such as heart and kidneys, and also in the bone marrow. The other major form of energy is body protein, which accounts for almost 15% of energy in the body. Carbohydrate, mainly glycogen in the liver and muscles, although important during acute stress situations, does not play any major role in the survival.

Circannual changes in lipid metabolism

The use of lipids during winter has been demonstrated in several studies. Usually the stores are at maximum in October and reach a nadir in April – June (Larsen & Blix, 1985; Gerhart *et al.*, 1996). The amount of lipid stored or the decrease in fat stores is determined by the balance between the rates of lipolysis and lipogenesis. Studies on Norwegian reindeer have shown that adaptive changes occur in both of these (Larsen *et al.*, 1985c). In adipose tissue lipolysis is catalyzed by hormone sensitive lipase (HST), the activity of which is under hormonal control by insulin, catecholamines and glucagon (Fig. 4). Insulin is a very potent inhibitor of HSL, while catecholamines and glucagon activate it. As stated above, the concentration of insulin is low during winter (Larsen *et al.*, 1985a; Soppela *et al.*, 2003), a condition that favours lipolysis. Of the other hormones that regulate lipolysis, adrenaline and noradrenaline show no significant seasonal changes (Larsen *et al.*, 1985a), and nothing is known about the annual changes in the concentrations of

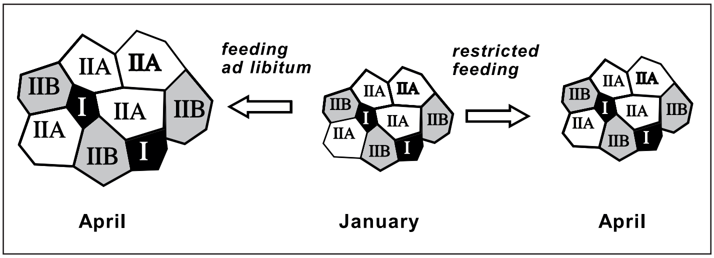

Fig. 5. Changes in cross sectional area in the middle gluteal muscle in reindeer calves. One group of calves was fed a pelleted feed ad libitum and the other group was fed lichens at 60% level of *ad libitum* intake.

glucagon. Thus it has been suggested that serum concentrations of catecholamines do not explain the elevated rate of lipolysis, although the stress caused by sampling may partially mask the changes in their concentrations. The activation of lipolysis may also be regulated via sympathetic activity, possibly by melatonin (Bartness *et al.*, 2002), and the subsequent release of noradrenalin from neurons. Because the observed rate of any physiological event regulated by several hormones is a sum effect of all hormones in question, it can be suggested that the low concentration of insulin and concentrations of catabolic hormones together with the neuronal activation result in increased rate of lipolysis. In reindeer lipolysis is increased in a controlled manner. This is indicated by the stable serum concentrations of nonesterified fatty acids over the main part of the winter (Larsen *et al.*, 1985a; Soveri *et al.*, 1992; Bubenik *et al.*, 1998; Soppela *et al.*, 2000) with increases in ketone bodies that would be an indication of massive increase in lipolysis occurring only in early spring (Soveri *et al.*, 1992; Soppela *et al.*, 2000). The increase in ketone body concentrations is apparent also in reindeer that are fed *ad libitum* high quality feed throughout the winter (Pösö *et al.*, 1994; Soppela *et al.*, 2000).

What controls lipolysis during winter? The regulation of lipolysis and lipogenesis are regulated in such a manner that they are not active simultaneously and thus during winter the rate of lipogenesis is low, while during summer, when the availability of high quality feed is abundant, lipogenesis is the predominant pathway (Larsen *et al.*, 1985c). The data on isolated adipocytes suggest that in winter the enzymes of the lipogenic pathway are downregulated (Larsen *et al.*, 1985c). On the other hand the activity of hormone sensitive lipase, the key enzyme in lipolysis, is higher in winter in comparison to values measured in summer (Larsen *et al.*, 1985b). However, in isolated adipocytes the stimulation of lipolysis by adrenalin is less in winter than in summer, which suggests that lipolytic capacity is regulated also at receptor/post-receptor level (Larsen *et al.*, 1985c). In addition volatile fatty acids and ketone bodies have been found to regulate lipolysis in isolated adipocytes (Larsen *et al.*, 1983). In summer, when the feed intake and the production of volatile fatty acids in the rumen are high and there is no need for mobilization of lipids, volatile fatty may control lipolysis. If the rate of lipolysis during winter increases above the level needed for energy, the oversupply of free fatty acids will increase the production of ketone bodies that in turn slow down lipolysis (Fig. 4). Furthermore reduced activity of 3-hydroxyacyl-CoA dehydrogenase in muscles during winter suggests that their capacity to use of fatty acids may be decreased (Kiessling *et al.*, 1986; Pösö *et al.*, 1996).

Seasonal changes in protein metabolism

In comparison to our knowledge on lipid metabolism very few studies have been performed on annual changes in protein metabolism. Protein as an energy source differs from carbohydrates and lipids, because there is no special storage protein, such as triglycerides for lipids or glycogen for carbohydrates. Thus when protein is broken down at a rate that is higher than the synthesis of proteins there will be loss of structural elements or enzymes that are needed to catalyze the metabolism. Thus only during extreme starvation does protein degradation occur to any greater extent.

A special feature of protein metabolism in reindeer is that lichen, which is the main winter feed, is low in nitrogen (Nieminen, 1980). This together with reduced feed intake results in strongly negative nitrogen balance during winter (McEwan & Whitehead, 1970; Gerhart *et al.*, 1996). This is demanding for the reindeer, especially to pregnant females that have to use their own tissues to produce amino acids for the growing fetus and later in spring for milk proteins. Reindeer are extremely well adapted to large variations in the nitrogen content and may have a greater potential to recycle urea than domestic ruminants (Wales *et al.*, 1972). Thus a decrease in urea concentration is frequently seen in winter (Soveri *et al.*, 1992; Pösö *et al.*, 1994), a finding that has been taken as an indication that the recycling of urea to rumen is more effective during the winter. Increases in urea concentrations during winter may indicate severe starvation and greatly increased utilization of protein as an energy source (Valtonen, 1979).

The body protein mass is greatest in October and lowest in late spring (Gerhart *et al.*, 1996) and the loss of protein over winter may be up to 29%. It has been suggested that Svalbard reindeer that live in extremely harsh conditions, may use IIB fibres in their muscles as a protein source during winter (Kiessling & Kiessling, 1984). This was indicated by the decrease in the cross sectional area of these fibres. To study whether moderate undernutrition also causes reduction in the IIB fibre area Finnish reindeer calves were fed lichens for 6 weeks at a level that was 60% of their *ad libitum* intake of lichens (Pösö *et al.*, 2001). Other calves fed *ad libitum* with commercial feed served as controls. Although the calves in the lichen group lost about 8.8 kg of weight, the cross sectional area of all muscle fibre types remained unchanged during the starvation period (Fig. 5). In the control group there was an increase in the fibre area indicating that the rate of protein synthesis was greater than that of protein degradation. Interestingly the activity of cathepsin B, one of the proteolytic enzymes in the lysosomes, measured as an indicator of lysosomal protein degradation, was also decreased indicating that a similar control of protein catabolism prevails as seen in the controlled lipolysis over the period of low availability of food (Pösö *et al.*, 2001).

In summary, the seasonal changes in the growth, energy metabolism and reproduction of reindeer show that the reindeer are well adapted to the arctic climate. Our understanding on the physiology of these changes is increasing, but still very little is known about the molecular mechanisms. The role of photoperiod and thus melatonin as the major regulator is accepted, but the role of other hormones, such as leptin, insulin, glucagons and thyroid hormones, is less clear and even less is known about the interactions of the hormonal effects.

References

Bartness, T. J., Demas, G. E. & Song, C. K. 2002. Seasonal changes in adiposity: the roles of the photoperiod, melatonin and other hormones, and sympathetic nervous system. – *Exp. Biol. Med.* 227: 363-376.

Bubenik, G. A., Schams, D., White, R. G., Rowell, J., Blake, J. & Bartos, L. 1998. Seasonal levels of metabolic hormones and substrates in male and female reindeer (*Rangifer tarandus*). – *Comp. Biochem. Physiol. C* 120: 307-315.

Carlberg, C. 2000. Gene regulation by melatonin. – *Ann. NY Acad. Sci.* 917: 387-396.

Chilliard, Y., Bonnet, M., Delavaoud, C., Faulconnier, Y., Leroux, C., Djiane, J. & Bocquier, F. 2001. Leptin in ruminants. Gene expression in adipose tissue and mammary gland, and regulation of plasma concentration. – *Dom. Anim. Endocrinol.* 21: 271-295.

Cuyler, L.C. & Øritsland, N. A. 1993. Metabolic strategies for winter survival by Svalbard reindeer. – *Can. J. Zool.* 71: 1787-1792.

Eloranta, E., Timisjärvi, J., Nieminen, M., Leppäluoto, J. & Vakkuri, O. 1995. Seasonal onset and disappearance of diurnal rhytmicity in melatonin secretion in female reindeer. – *Am. J. Zool.* 35: 203-214.

Eloranta, E., Timisjärvi, J., Nieminen, M., Ojutkangas, V., Leppäluoto, J. & Vakkuri, O. 1992. Seasonal and daily patterns in melatonin secretion in female reindeer and their calves. – *Endocrinol.* 130: 1645-1652.

Friedman, J. M. 2002. The function of leptin in nutrition, weight, and physiology. – *Nutr. Rev.* 60: S1-S14.

Gerhart, K. L., White, R. G., Cameron, R. D. & Russell, D. E. 1996. Body composition and nutrient reserves of arctic caribou. – *Can. J. Zool.* 74: 136-146.

Harris, R. B. S. 2000. Leptin – much more than a satiety signal. – *Annu. Rev. Nutr.* 20: 45-75.

Kiessling, K.-H. & Kiessling, A. 1984. Fibre composition and enzyme activities in five different muscles from the Svalbard reindeer. – *Comp. Biochem. Physiol.* 77A: 75-78.

Kiessling, K.-H., Kiessling, A., Nilssen, K. & Andersson, I.-L. 1986. Histochemical and enzymatic differences in skeletal muscle from Svalbard reindeer during the summer and winter. – *Rangifer* 6: 2-7.

Larsen, T. S. & Blix, A. S. 1985. Seasonal changes in total body water; body composition and water turnover in reindeer. – *Rangifer* 5: 2-9.

Larsen, T. S., Lagercrantz, H., Riemersma, R. A. & Blix, A. S. 1985a. Seasonal changes in blood lipids, adrenaline, noradrenaline, glucose and insulin in Norwegian reindeer. – *Acta Physiol. Scand.* 124: 53-59.

Larsen, T. S., Nilsson, N. Ö. & Belfrage, P. 1985b. Seasonal changes in hormone-sensitive lipase activity in adipose tissue from Norwegian and Svalbard reindeer. – *Acta Physiol. Scand.* 125: 735-738.

Larsen, T. S., Nilsson, N. Ö. & Blix, A. S. 1983. Effects of volatile fatty acids and ketone bodies on lipolysis in isolated adipocytes from Norwegian reindeer (*Rangifer tarandus). – Acta Physiol. Scand.* 117: 451-455.

Larsen, T. S., Nilsson, N. Ö. & Blix, A. S. 1985c. Seasonal changes in lipogenesis and lipolysis in isolated adipocytes from Svalbard and Norwegian reindeer. – *Acta Physiol. Scand.* 123: 97-104.

Malpaux, B., Migaud, M., Tricoire, H. & Chemineau, P. 2001. Biology of mammalian photoperiodism and the critical role of the pineal gland and melatonin. – *J. Biol. Rhytms* 16: 336-347.

McEwan, E. H. & Whitehead, P. E. 1970. Seasonal changes in the energy and nitrogen intake in reindeer and caribou. – *Can. J. Zool.* 48: 905-913.

Mesteig, K., Tyler, N. J. C. & Blix, A. S. 2000. Seasonal changes in heart rate and food intake in reindeer *(Rangifer tarandus tarandus*). – *Acta Physiol. Scand.* 170: 145-151.

Morgan, P. J. & Mercer, J. G. 2001. The regulation of body weight: lesson from the seasonal animal. – *Proc. Nutr. Soc.* 60: 127-134.

Nelson, R. J. & Demas, G. E. 1997. Role of melatonin in mediating seasonal energetic and immunologic adaptations. – *Brain Res. Bull.* 44: 423-430.

Nieminen, M. & Heiskari, U. 1989. Diets of freely grazing and captive reindeer during summer and winter. – *Rangifer* 9: 17-34.

Nilssen, K. J., Bye, K., Sundsfjord, J. A. & Blix, A. S. 1985. Seasonal changes in T_3, FT_4, and cortisol in free-ranging Svalbard reindeer (*Rangifer tarandus platyrhynchus*). – *Gen. Comp. Endocrinol.* 59: 210-213.

Nilssen, K. J., Johnsen, H. K., Rognmo, A. & Blix, A. S. 1984a. Heart rate and energy expenditure in resting and running Svalbard and Norwegian reindeer. – *Am. J. Physiol.* 246: R963-R967.

Nilssen, K. J., Sundsfjord, J. A. & Blix, A. S. 1984b. Regulation of metabolic rate in Svalbard and Norwegian reindeer. – *Am. J. Physiol.* 247: R837-R841.

Prentice, A. M., Moore, S. E., Collinson, A. C. & O'Connell, M. A. 2002. Leptin and undernutrition. – *Nutr. Rev.* 60: S56-S67.

Pösö, A. R., Heiskari, U., Lindström, M., Nieminen, M. & Soveri, T. 2001. Muscle fibre growth in undernourished reindeer calves (*Rangifer tarandus tarandus* L.) during winter. – *Comp. Biochem. Physiol. A* 129: 495-500.

Pösö, A. R., Nieminen, M., Raulio, J., Räsänen, L. A. & Soveri, T. 1996. Skeletal muscle characteristics of racing reindeer (*Rangifer tarandus*). – *Comp. Biochem. Physiol.* 114A: 277-281.

Pösö, A. R., Nieminen, M., Sankari, S. & Soveri, T. 1994. Exercise-induced changes in blood composition of racing reindeer (*Rangifer tarandus tarandus* L.). – *Am. J. Physiol.* 267: R1209-R1216.

Rhind, S. M., McMillen, S. R., Duff, E., Hirst, D. & Wright, S. 1998. Seasonality of meal patterns and hormonal correlates in red deer. – *Physiol. Behaviour* 65:295-302.

Rhind, S. M., McMillen, S. R., Duff, E., Kyle, C. E. & Wright, S. 2000. Effect of long-term feed restriction on seasonal endocrine changes in Soya sheep. – *Physiol. Behaviour* 71: 343-351.

Ryg, M. 1984. Seasonal changes in the relationship between food intake and serum triiodothyronine in reindeer. – *Comp. Biochem. Physiol.* 78A: 427-429.

Ryg, M. & Jacobsen, E. 1982. Seasonal changes in growth rate, feed intake, growth hormone, and thyroid hormones in young male reindeer (*Rangifer tarandus tarandus*). – *Can. J. Zool.* 60: 15-23.

Soppela, P., Heiskari, U., Nieminen, M., Salminen, I. & Sankari, S. 2000. The effects of prolonged undernutrition on serum lipids and fatty acid composition of reindeer calves during winter and spring. – *Acta Physiol. Scand.* 168: 337-350.

Soppela, P., Saarela, S. & Nieminen, M. 2003. The effects of wintertime undernutrition on plasma leptin and insulin levels in juvenile reindeer. – *Rangifer* Report 7: 56.

Soveri, T., Sankari, S. & Nieminen, M. 1992. Blood chemistry of reindeer calves during the winter season. – *Comp. Biochem. Physiol.* 102A: 191-196.

Suttie, J. M. & Webster, J. R. 1995. Extreme seasonal growth in arctic deer: comparisons and control mechanisms. – *Amer. Zool.* 35: 215-221.

Thiéry, J. C., Chemineau, P., Hernandez, X., Migaud, M & Malpaux, B. 2002. Neuroendocrine interactions and seasonality. – *Domest. Anim. Endocrinol.* 23: 87-100.

Timisjärvi, J., Ojutkangas, V., Eloranta, E., Nieminen, M., Leppäluoto, J., Liimatainen, S. & Vuolteenaho, O. 1994. Annual variations in serum thyriod-stimulating hormone and thyroid hormones and in their responses to thyrotrophin-releasing hormone in the reindeer. – *J. Endocrinol.* 141: 527-533.

Valtonen, M. 1979. Renal responses of reindeer to high and low protein diet and sodium supplement. – *J. Sci. Agr. Soc. Finland* 51: 387-419.

Vanacek, J. 1998. Cellular mechanisms of melatonin action. – *Physiol. Rev.* 78: 687-721.

Wales, R. A., Milligan, L. P. & McEwan, E. H. 1972. Urea recycling in caribou, cattle and sheep. – *Proc. 1st Int. Reindeer/Caribou Symp. Fairbanks, Alaska* 297-307.

Williams, L. M., Hannah, L. T., Kyle, C.E. & Adam, C. L. 1996. Central melatonin receptors in red deer (*Cervus elaphus*). – *Gen. Comp. Endocrinol.* 104: 1-6.

Woodfill, C. J. I., Robinson, J. E., Malpaux, B. & Karsch, F. J. 1991. Synchronization of the circannual rhythm of the ewe by discrete photoperiodic signals. – *Biol. Reprod.* 45: 110-121.

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Abstract in Finnish / Tiivistelmä:

Valaistus, lämpötila ja ravinnon saatavuus vaihtelevat vuodenajan mukaan. Jotta nämä muutokset voisivat saada aikaan adaptiivisia muutoksia porossa, ne täytyy muutta hormonisignaaleiksi. Luotettavin näistä edellä mainituista ympäristön vuodenaikaismuutoksista on valo, joka vaikuttaa poron elintoimintoihin käpylisäkkeen ja sen erittämän hormonin, melatoniinin, välityksellä. Melatoniinin plasmapitoisuuksissa on havaittavissa selkeä vuorokausirytmi, joka porolla häviää kesällä ja alkaa uudestaan syksyllä. Melatoniini-hormonin vuodenaikaisvaihtelut ovat mukana säätelemässä lisääntymistä, talvikarvan kasvua, lämmöntuottoa, elopainoa ja immuunitoimintoja. Melatoniini vaikuttaa geeniaktivaation kautta mekanismeilla, joita ei vielä tarkkaan tunneta. Muita hormoneja, joiden erityksessä on havaittu vuodenaikaisvaihtelua, ovat kilpirauhashormonit, insuliini ja leptiini. Havaitut muutokset ovat ilmeisesti usean hormonin yhteisvaikutuksen aiheuttamia. Ruokahalu sekä energian- että lämmöntuotto ovat keskeisiä hengissä säilymisen kannalta. Talvella poron energiatase on negatiivinen ja se käyttää lähinnä varastoimiaan rasvoja energian tuottoon. Rasvojen käyttö on ekonomista, sillä rasvojen hajoaminen, lipolyysi, on säädeltyä ja rasvahappojen käyttö lihaksissa vähenee talvella. Vasta vakavasti nälkiintyneissä poroissa lipolyysi aktivoituu siten, että myös ketoaineita alkaa kertyä vereen. Valkuaisaineiden määrä vähenee vähemmän kuin rasvojen ja ainoastaan nälkiintyneet porot käyttävät lihasten valkuaisaineita energiantuottoon. Poron talviravinnossa, jäkälässä, on vain vähän typpeä, joten talvisin typpitasapaino on voimakkaasti negatiivinen. Poro reagoi tähän vähäiseen typpimäärään lisäämällä urean kierrätystä pötsiin. Kokonaisuudessaan poron elintoimintojen sopeutuminen auttaa poroa selviytymään talven yli. Vaikka adaptaatiosta on joiltakin osin kertynyt runsaasti tietoa, on siinä myös paljon selvitettävää.