DEER GROWTH AND PRODUCTION: A REVIEW

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In a temperate environment deer have a seasonal pattern of growth in that maximum accretion of body tissue occurs in spring and summer, weight may be lost, particularly by stags, during autumn and little or no weight gain takes place over winter. This paper approaches deer growth and production from the perspective of seasonality; what is it and what can we do about it? It is written almost exclusively about red deer, primarily because the vast majority of farmed deer growth studies have been carried out on this species. The paper is in two parts, firstly a review of deer growth studies relevant to the broad theme of seasonal effects and secondly an account of recent attempts at Invermay, and elsewhere, to further understand the process of the seasonal control of growth and how it might be practically manipulated for productive advantage.

DEER GROWTH REVIEW

i. Growth and food intake cycles

The concept of a seasonal component to deer growth, even in the presence of ad libitum food, was first shown by French et al (1956) in white-tailed deer. In a series of experiments they clearly showed that young bucks grew only in summer, when food intake rose compared with winter. Studies on seasonal growth in red deer began at the Rowett Institute in Aberdeen, Scotland in the early seventies. The data indicated that male, castrated male and female red deer grew only in summer when food intake was high but not in winter despite the availability of unlimited foods; further the amplitude of seasonal change was greater for intact males (Pollock 1975, Kay 1979). In addition if the deer were placed on a manipulated photoperiod, where the frequency of daylength change was doubled, ie, a 6 month year or two 6 month cycles in one calendar year, then two cycles of food intake, growth and where appropriate antler development took place each year. Thus a growth cycle could be defined as a pattern of weight change which took place in the presence of unlimited food and could be influenced by daylength. It appeared that the long or increasing days of spring and summer stimulated appetite and growth but the short or decreasing days of autumn and winter diminished appetite and growth. The cycle of reproduction was the reciprocal of the growth cycle. Whether the growth cycle was a true cycle of growth potential or whether it was solely a consequence of the cycle in voluntary food intake was not clear. For the purpose of this review the conservative approach that there are separate but linked cycles of growth and voluntary food intake will be adopted.

In New Zealand Fennessy et al (1981) were translating their own and the Scottish findings to precise feed requirements for deer, which took into account seasonal effects. In the United States a "third" contributory cycle of metabolic rate was suggested which was thought to underlie the growth and appetite cycles. The concept was based on data which indicated that metabolic rate (oxygen consumption and heat production) was raised during summer compared with winter (Silver et al 1969). The notion equated this cycle with a kind of hibernation such that deer partly went "to sleep" to save energy during winter. However, more recent data from the same laboratory (Pekins et al 1991), which has combined new studies with a re-evaluation of old techniques, has led to the conclusion that the cycle of metabolic rate was almost solely a consequence of the food intake cycle. Thus



it is now believed that two cycles, one of growth potential and one of food intake, explain the growth seasonality we observe in deer.

ii. Physiological control of growth and food intake cycles

Almost from their discovery, attempts were made to understand the fundamental physiological mechanisms involved with the growth/food intake cycle. To begin with efforts were centred on hormones which were known to vary seasonally and fluctuations in whose levels coincided with events in the growth cycle. In view of the fact that the breeding season in males is associated with high levels of plasma testosterone (Lincoln 1971), and very low feed intake and weight loss (Kay 1979), it was thought that gonadal androgens might play a role in the growth cycle. However castrated male deer and females also have an annual growth/food intake cycle (Simpson et al 1984, Suttie and Simpson 1985), although the amplitude of the cycle is much less than for intact male deer. The currently accepted concept is that the rut, with its associated weight loss, is an addition to the annual growth cycle, not actually a part of it (Fennessy et al In Press); this addition is most likely a result of androgens, although the precise mechanism of their role is unclear. Likewise as intact male deer have higher amplitude growth/food intake cycles compared with females, gonadal steroids, particularly androgens, are thought to play a role (Suttie et al 1985); this subject is also considered in the next section.

In view of the fact that the plasma concentrations of the pituitary hormone prolactin vary seasonally, highest levels being detected in summer and lowest levels in winter (Mirarchi et al 1978, Brown et al 1979) thus roughly coinciding with the cycle of voluntary food intake (Suttie and Kay 1985), this hormone has been studied to determine whether it has any causal link with food intake. Curlewis et al (1988) treated adult red deer hinds with a long acting formulation of a prolactin release antagonist, bromocriptine, at two dose levels, from February-November (Northern Hemisphere). They found no effect of the low dose bromocriptine of 5 mg/week, on either prolactin or food intake but 12.5 mg/week lowered the amplitude, but did not suppress the food intake and prolactin cycles. The authors concluded however, that it was not possible to definitively state that the reduction in amplitude of the food intake cycle was due to the reduction in prolactin. Rather they considered that the changes in prolactin might only parallel neuroendocrine activity which influenced seasonal functions. In a further study by the same group, Milne et al (1990) treated red deer hinds with domperidone, a prolactin releasing agent, or bromocriptine in Although bromocriptine treatment lowered prolactin and food intake, domperidone had no effect on food intake despite the fact that it raised prolactin levels. In a study in reindeer Ryg and Jacobsen (1982) administered bovine prolactin during winter to young male reindeer. Somewhat enigmatically they reported a significant increase in liveweight without a concomitant increase in food intake. The results of the Invermay prolactin administration trial are in the next section. Although there is abundant evidence of an association between prolactin and food intake, cause-effect relationships have proved elusive.

In the last six years at Invermay we have been studying growth hormone and insulin-like growth factor 1 and their roles in the annual cycle of growth and food intake. Although preliminary studies of growth hormone in red deer did not reveal a seasonal cycle (Brown et al 1979) studies by Bubenik et al (1975) in white-tailed deer indicated that plasma levels of growth hormone were elevated in spring. At Invermay we have extended these findings

to include a measurement of the pulsatile release characteristics of growth hormone (Suttie et al 1989). Growth hormone in deer is released in episodes or pulses, but the size and the frequency of these pulses varies during the year. In early spring the pulses are of high amplitude and occur with a frequency of 4 per day. This results in a seasonal peak in mean daily plasma growth hormone level. At other times of the year the frequency of growth hormone pulses is less and the amplitude of the pulses is much less. We believe that the pulse amplitude is the major determinant of the seasonal (spring) peak in growth hormone level. Growth hormone is known to cause the release of insulin-like growth factor 1 (IGF1) from the liver. We have found that there is a seasonal peak of IGF1 in deer about one month after the growth hormone peak and, further, this peak of IGF1 occurs at the same time as peak velvet antler growth and body growth. Thus we conclude that the growth rhythm is partly due to a seasonal change in plasma IGF1. Recent research (Suttie et al 1991) has shown that if deer are exposed to a manipulated photoperiod of 16 hours light followed by 8 hours dark (16L:8D) over winter then IGF1 levels are raised dramatically in contrast to deer kept on 8L:16D. This means that the mechanism involved with the transduction of photoperiodic information to body growth is via changes in IGF1. As melatonin treatment can also influence IGF1 (Suttie et al In Prep), we have strong evidence relating photoperiod to growth via melatonin and growth factors. At this time we do not know the mechanisms whereby the melatonin information alters IGF1 secretion but presumably these are through classical neuroendocrine pathways.

Thus we are building up a picture of the physiological control mechanisms regulating seasonal growth in deer but many gaps remain, particularly in the seasonal control of appetite.

An important point to raise at this stage is the fact that deer digest feed better in summer compared with winter (Milne et al 1978) Recently Domingue et al (1991) have shown that concomitant with an increase in voluntary food intake during summer, and an increase in rumen pool size, digestibility of cellulose and hemicellulose increases by 5-7%. The physiological (endocrine) mechanisms are not clear. Clearly the increased digestibility must contribute to a relatively higher nutrient availability per unit of food eaten in summer compared with winter.

Although we know very little about the annual cycle of nutrient partitioning in deer one very clear anatomical consequence of this is apparent; seasonal fat deposition and mobilisation. All deer, but entire stags, in particular, lay down enormous quantities of fat in summer and mobilise it in autumn and winter. This topic has been extensively reviewed elsewhere (Drew 1985). It is fair to say we understand very little of the hormonal control mechanisms which underlie this cycle of tissue deposition. Recent research at Invermay points to seasonal differences in insulin kinetics in the glucose tolerance test such that glucose is cleared faster in spring. This may mean that the balance between growth hormone and insulin changes seasonally in deer. However the neuroendocrine control of this is a further unknown.

NOVEL STUDY APPROACHES AND ATTEMPTS AT GROWTH PROMOTION IN DEER

Introduction

Many early attempts to increase growth in deer by steroid use, inwintering and supplementary feeding have been dealt with in previous reviews to the NZVA Deer Branch (Suttie 1985, 87). Since then Mulley (1990) has confirmed that Ralgro Zeranol® is effective as a growth promotant in fallow deer and Freudenberger *et al* (1991) have shown that immunisation against LHRH may have beneficial growth effects in stags. Duckworth and Barrell (1990) have found that immunisation against melatonin has a growth promoting effect, perhaps by allowing a free-running of seasonal cycles although Ataja (1990) could not repeat the weight gain advantage. However innovative and integrated attempts at growth promotion in deer within the levels of acceptability to producers, exporters and markets have been lacking. The next 3 sections describe novel approaches to growth study in deer and attempts at growth promotion by altering the growth cycle and by modifying the existing cycle.

NOVEL APPROACHES

IGF1 in relation to carcass size and body composition

As mentioned in the review IGF1 correlates with body growth. However, it is not known whether there is a relationship among plasma IGF1, carcass parameters and body composition. In addition, previous work had been carried out in pens with animals fed concentrate rations and it was felt that the conclusions should be validated for animals at pasture. Twenty eight 10-month-old stags were kept at pasture from September-February and bled and weighed fortnightly. Plasma was analysed for IGF1. All stags were slaughtered in February (aged 15 months); organ weights were recorded and the carcasses analysed chemically for water, fat and ash. Primary data for IGF1 and live weight shown in Figure 1 indicate that there was a seasonal increase and decrease in plasma IGF1 confirming what we had previously shown in penned deer. The stags grew rapidly in spring and early summer but growth rate slowed in mid-summer before increasing again in late summer.

Table 1 shows that IGF 1 correlated significantly and positively with total live weight gain, carcass length and weight, femur weight, organ and muscle weights and fat-free carcass components (except ash). This means that IGF1 appears strongly related to lean growth, but not fat. The relationship between overall liveweight gain and IGF1 is significant, but if this relationship is divided into two periods, before and after the summer solstice, the relationships vary, Table 2. We speculate that the reason for the variation is that prior to the summer solstice the stag lays down primarily lean tissue but after, ie, during autumn, primarily fat is deposited. IGF1 correlates with lean, not fat, therefore the correlation between liveweight gain and IGF1 breaks down over this period. In the biometric analysis attempts were made to determine whether the relationships with IGF1 were influenced by IGF1 alone or were related via body size. This analysis failed to distinguish body size effects for all measured parameters except pancreas weight. This means that IGF1 has an independent effect on pancreas size but influences other parameters via its effect on overall body size. The relevance of this for pancreas function is not known, but the role of IGF1 on body size control is further emphasised. Whether exogenous IGF1 would act as a growth promotant in deer is a moot point.

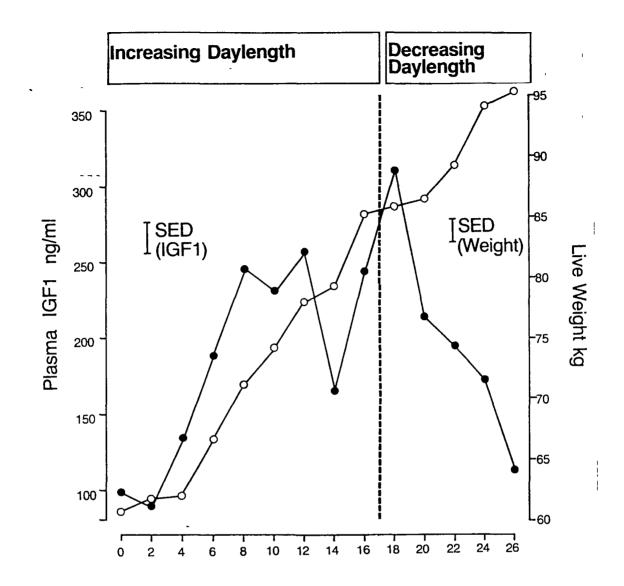


Figure 1. Mean liveweight (O) and plasma IGF1 (•) of 28 stags for 26 weeks. SED = standard error of the difference.

Prolactin administration during winter

In the review above it was stated that prolactin had been implied as a growth/food intake regulator but little cohesive evidence was available to confirm any such effect. We decided to treat stags with prolactin during their first winter of life to determine whether there was an effect on growth and food intake. Twelve 6-month old red deer stags were randomly allocated to either treatment with daily subcutaneous injections of 350 I.U. bovine prolactin made up in bicarbonate buffered saline (n=6) or daily subcutaneous injections of buffer alone. Treatment began in June and concluded in September, this period being the normal duration of the seasonal nadir in the prolactin annual cycle in deer. The animals were penned individually and fed a concentrate diet to appetite. The coat was observed as it was known from studies in carnivores and rodents that prolactin treatment influences moult and regrowth pattern. Food intake and live weight were measured until November to determine any residual effects of treatment with prolactin.

TABLE 1. Mean values (\pm SEM) for total live weight gain and body composition data at slaughter and correlation coefficients for the relationship with mean IGF1 for n=28 stags. Percentage carcass components are of hot carcass weight. n.s, no significance, *P<0.05, **P<0.01, ***P<0.001.

<u>Parameter</u>		Mean ± sem	Correlation coefficient	
Total Live Weight G	Total Live Weight Gain (kg)		0.592***	
Carcass components				
Hot Carcass	Length (cm)	111.0 ± 0.9	0.546**	
2200 0000	Weight (kg)	55.8 ± 1.3	0.521**	
Femur	Length (cm)	28.8 ± 0.2	0.363 n.s.	
	Weight (g)	463.0 ± 10.1	0.466*	
Organs	Liver (g)	1524.0 ± 27.3	0.419*	
018	Pancreas (g)	87.9 ± 3.4	0.490*	
	Spleen (g)	240.0 ± 8.8	0.148 n.s.	
	Kidneys (g)	223.0 ± 6.1	0.438*	
	Testes (g)	80.0 ± 2.9	0.407*	
M. semimembranosus (g)		1141.0 ± 64.2	0.464*	
Kidney fat (g)		138.0 ± 12.1	0.279 n.s.	
Weight of fat (kg)		5.1 ± 0.1	0.236 n.s.	
Water (kg)	~6/	35.9 ± 0.4	0.549**	
Ash (kg)		3.3 ± 0.1	0.254 n.s.	
Protein (kg)		11.5 ± 0.2	0.464*	
Fat-free (kg)		50.7 ± 0.6	0.528**	
Percentage of	fat	9.0 ± 0.4	0.059 n.s.	
10.001111100 01	water	64.6 ± 0.3	0.130 n.s.	
	ash	5.7 ± 0.1	-0.342 n.s.	
	protein	20.7 ± 0.2	-0.105 n.s.	
	fat-free	91.0 ± 0.5	-0.059 n.s.	

Prolactin treatment significantly increased food intake and liveweight gain in winter, Table 3. During spring although food intake was similar, live weightgain was slightly higher in the group previously treated with prolactin. This is important as one might have expected compensatory weight gain to take place in the control group - but this did not occur. (This is a relevant recurrent theme in the next sections). Although the prolactin treated group had slightly bigger antler, this effect was not significant. Plasma prolactin levels were significantly raised by the once daily injection of prolactin by September, and even in November levels of prolactin were slightly (not significantly) higher in the group previously treated with prolactin. The prolactin treated stags moulted their winter coat and grew a summer coat in mid winter; no such effect was seen in the buffer control group who moulted in spring at the normal time.

TABLE 2. Correlation between total plasma IGF1 for each stag over each two week period and antler growth rate and liveweight gain.

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Antler growth rate	0.338 ***
Liveweight gain (overall)	0.117 *
Liveweight gain (before summer solstice)	0.418 ***
Liveweight gain (after summer solstice)	-0.273 **

TABLE 3. Influence of prolactin treatment on food intake, growth, antler size and plasma prolactin concentrations. SED is the standard error of the difference.

		Prolactin <u>Treated</u>	Control	SED
Food intake	Winter	1.42	1.23	0.04
kg/day	Spring	1.85	1.84	0.08
Live weight gain g/day	Winter	215	173	16
	Spring	282	258	29
Antler length	September	5.6	4.1	0.8
	November	33.6	27.8	5.2
Plasma prolactin ng/ml	June September November	0 3.1 62.7	0.7 15.2 49.4	0.2 9.0 10.9

Prolactin treatment during winter elevated food intake, liveweight and caused a moult to take place. This finding concurs with data from Curlewis *et al* (1988) that blocking prolactin secretion in summer reduces food intake, lowers growth and delays the moult. Thus prolactin must be included in the list of growth regulators for deer. At this time its precise role, the mechanism of action and the interactions with other known regulators such as IGF1 are not known. The fact that advantages in winter liveweight gain were maintained in spring is a useful pointer for manipulative work.

GROWTH PROMOTION BY ALTERING SEASONAL CYCLES

This section considers two attempts at growth promotion by altering the timing of seasonal events. One could draw an analogy with attempts to advance the breeding season to suit management options; here we are trying to advance the growing season.

Photoperiodic induction of growth during winter

In the review above it was stated that we knew that changes in daylength, in some way, controlled the seasonal cycles of growth and food intake. Long days are associated with growth (and reproductive quiescence) short days with weight loss or limited growth (and in the case of adult animals, reproduction). What if we exposed deer to the long days of summer during winter, would we get growth rates associated with summer? We have carried out experiments in the context of our winter-housing programme (see below). Thirty recently weaned stag calves were randomly allocated to one of three treatment groups in April as follows. One group (n=10) was exposed to a light regime, indoors, which followed exactly the changes which were taking place outdoors (sine control). A second group (n=10) was exposed to a light regime indoors such that on the 11th of April the daylength was adjusted to that of the winter solstice and then increased progressively to simulate the natural changes in daylength in spring (sine advance). A third group (n=10) was exposed on the 11th of April to a photoperiod of 16L:8D indoors, that is the number of hours of light at the summer solstice at 45° South. On September 6 they were released outdoors. The indoor pens were carefully light proofed so that the animals were oblivious to outdoor daylength changes. The lights which provided 500 lux one meter above the floor, were switched on and off automatically each day by a series of timers. The groups of stags were fed to appetite and weighed weekly. Five stags in each group were blood sampled, at intervals, and the resultant plasma analysed for IGF1.

Both light advance treatments increased liveweight gain during winter (Table 4). In the case of the 16L:8D group, mean liveweight gain over winter was higher than achieved by the control group outside in spring. During spring the animals which gained most in winter gained least. The significant increases in growth rate were accompanied by significant increase in plasma IGF1, particularly so in the 16L:8D group.

TABLE 4. Influence of daylength on liveweight gain and IGF1 secretion.

		Sine control	Sine advance	16L:8D	SED
Liveweight gain g/day	Winter	153	206	274	8.5
	Spring	252	242	136	9.0
Week of treatment (winter	.)				
IGF1 ng/ml	4	172	233	170	25
	6	213	220	259	23
	9	233	222	481	40
	14	262	310	647	40
	18	425	515	861	82

This study shows that the growth cycle can be advanced by increasing the number of hours of daylight during winter, and points to an increase in IGF1 as part of the mechanism for this effect. In that deer are housed commonly during winter, this method provides a simple, non-invasive manipulative tool for growth promotion. Further research to refine the system is underway.

Melatonin manipulation of growth

In as much as photoperiod regulates growth, the hormone which transduces the photoperiodic effects to the neuroendocrine system, melatonin, might be considered an effective cycle alteration treatment. This has been clearly shown for reproduction (Webster et al 1991). However in that study weight gain pattern was also manipulated. Treatment of mature stags with Regulin® (18 mg melatonin/implant) implants in October and November increases summer growth rates and leads to an advancement of the attainment Whether this technique has any applicability in a venison of peak animal weight. production system with young stags or indeed whether the technique will succeed in increasing growth in this age cohort is under investigation. One important feature which distinguishes younger from older animals in terms of seasonal growth is that young animals are still growing a frame; in contrast older animals are gaining and losing weight but frame size must remain constant. Also we are not sure at present whether younger animals process photoperiodic information or respond to that information in quite the same way as older animals. Much of our current photoperiodic research at Invermay is designed to find the physiological mechanisms which underlie photoperiodically controlled changes in food intake and growth rate.

MODIFICATIONS OF EXISTING CYCLES

Winter Housing

Fennessy et al (1981) indicated that deer kept indoors during winter had lower energy requirements than those outdoors. Thus for any given food intake, growth rate was increased in animals indoors because more of the nutrients were available for tissue accretion. This factor, in addition to advantages of feed utilisation, easy care, increased stock numbers and minimising of pasture damage during winter has lead to the growth in popularity of inwintering particularly of weaner deer in Otago/Southland. At Invermay we began inwintering research in the winter of 1987. This was essentially a pilot trial, but replicated studies took place in the winter of 1988 and 1989. All animals were fed lucerne hay and a barley based concentrate ration. Group food intake was recorded daily and all animals were weighed weekly. In both years the trial design was similar. Duplicate groups of weaner stag calves (n=10 per group) were kept indoors in pens and outdoors on gravel feeding pads on either a high or a low plane of nutrition during winter from May to September. Liveweight gain was recorded after the stags had been released to pasture in September until November (1988) or April (1989). In both years live weight gain for the high plane groups was similar, indoors and outdoors, but food intake required to achieve these growth rates was slightly less indoors compared with outdoors (Table 5). Growth rates of the low plane group were very low in all groups, the food intake of the indoor groups was much less than those outdoors. This tends to confirm earlier data of energy savings by housing. During spring all groups grew well; although those fed the low diet during the winter grew significantly better during spring. However, despite the "compensatory growth" in spring, the animals which suffered nutritional restriction during winter did not fully catch up on those animals fed well. Thus although deer voluntarily reduce food intake in winter, they are capable of growth if fed well and the growth advantages of this treatment in winter remain in spring and summer.

TABLE 5. Winter and spring live weight gain (LWG) (g/day) and winter voluntary food intake (VFI) (kg/day) in 1988 and 1989.

		Indoors		<u>Outdoors</u>		<u>SED</u>
		<u>High</u>	Low	<u>High</u>	Low	
1988						
LWG	Winter	.151	-0.012	.157	0.043	
2,,0	Spring	.288	.348	.293	.352	
VFI	Winter	1.61	0.84	1.71	1.17	0.044
<u>1989</u>						
LWG	Winter	.206	.035	.194	.058	
LWO	Spring	.301	.339	.342	.349	
VFI	Winter	1.52	0.80	1.58	0.96	0.038

TABLE 6. Winter weight gain, casting date and velvet antler weight in 2- and 3-year old stags fed diets containing 5% or 20% extruded cottonseed meal in winter. The velvet antler weight was adjusted to compensate for the fact that 3-year old stags had their velvet antler removed at a later stage of development compared with the 2-year old animals.

	2-year old		3-year old	
	<u>5%</u>	<u>20 %</u>	<u>5%</u> <u>20%</u>	
Winter weight gain kg	13.3	13.8	15.8 10.0	
Mean casting date	14 Oct	16 Oct	19 Sept 16 Sept	
Antler weight kg	1.04	1.06	1.68 1.76	

Decisions to house deer in winter must be made on the basis of many factors, but clearly energy savings can be made. It is also clear from this study that if deer can be fed well during the winter then they will be bigger in the spring/summer or alternatively will reach acceptable slaughter weight earlier.

Protected protein

Protected protein means a protein source which is either naturally resistant or is treated so as to make it resistant to degradation in the rumen by micro-organisms. The protein which escapes digestion in the rumen flows into the small intestine where it is digested and absorbed. Normally if a high level of very digestible protein (>16% Digestible Crude Protein DCP) is fed then rumen micro-organisms destroy the increment above 16% and the net result is that the ruminant has available to it an outflow from the abomasum of about 16%. Protecting the protein results in a higher protein availability for growth. The

technique is widely used in the wool growing industry. We decided to carry out a trial to compare 2 diets, one containing a high proportion of a protected protein source (extruded cottonseed meal, Harroways, Green Island, Dunedin, New Zealand), the other a low proportion of the protected protein. The diets were fed to rising 2- and 3-year old stags during winter, from May to late August. The high diet contained 20% w/w extruded cottonseed meal with barley, bran, pollard and a mineral vitamin pre-mix. The low diet contained 5% w/w extruded cottonseed meal with the same ingredients as above but with peameal to bring the % DCP to the same as the 20% diet. These diets were fed at the rate of 1.5 kg/head/day with ad libitum access to meadow hay. The animals were weighed before and after the trial, antler casting date was recorded, as was velvet antler yield. There were 25 stags in each group, balanced for the 2 age groups. Data for the 2 age groups and the different diets were analysed separately. There were no significant differences in winter live weight gain, casting date or velvet antler weight due to treatment, although the 3-year old stags cast their antlers earlier and had higher velvet antler yields (Table 6).

A negative result? Yes, in the sense that there were no significant differences due to diet but all stags gained about 13 kg - at a time of the year when they might have been expected to remain at the same weight or even lose weight. It seems that even the low level of protection gave a growth response. A trial will shortly be in progress to further compare protected protein; this time a very low protein control group will be used.

High winter protein

Although DCP levels greater than 16% result in rumen degradation of much of the protein, we carried out a small trial to compare growth rate of weaner stags fed 16% DCP with stags fed 23%. From June to September stags aged 7 months (n=6 per group) were fed to appetite diets composed of soybean meal, barley, bran and pollard with the soybean component varied to achieve the different values for DCP. From September all animals were fed the 16% DCP diet for a further 3 months. All animals were weighed weekly and food intake was recorded. There were no significant differences in food intake either during or after the period of increased protein feeding (Table 7). Although there were no significant differences in live weight gain, there were strong trends that the animals fed the higher protein grew better during winter and this effect was carried over to spring.

TABLE 7. Effect of winter protein level on food intake and live weight gain during winter and spring.

		23% DCP	16% DCP	<u>SED</u>
Food intake				
kg/day	Winter	1.38	1.23	0.04
	Spring	1.93	1.84	0.07
Live weight gain				
g/day	Winter	203	173	21
	Spring	313	282	24
	Overall	255	210	22

Caution should be expressed over these results as the differences are non-significant. However, the effects are large enough to necessitate further scrutiny with larger groups. The trend is consistent with the concept that winter gains are held during spring, therefore meaning that extra effort on winter feeding reaps benefits later.

Steroids

In previous reports to this Association we have shown that increasing steroid levels during spring when these are naturally low results in a significant growth promotion. In this study we compared the growth promotion activity of testosterone with oestradiol. In September, 30 rising 3-year old stags were allocated to either control, testosterone or oestradiol treatment, (n=10 per group). The testosterone treatment was 60 cm of silastic tubing and the oestradiol treatment 10 cm of silastic tubing packed with either crystalline testosterone or oestradiol (Sigma Chemical Co, St Louis, MO) respectively. The implants were positioned subcutaneously in the groin region under Rompun anaesthesia with local analgesia. The stags were weighed at monthly intervals for 90 days, until December. The growth rates were significantly higher for the steroid treated groups (Table 8).

TABLE 8. Effect of steroid treatment during spring on liveweight gain in rising 3-year old stags.

	<u>Control</u>	Testosterone	Oestradiol	SED
Liveweight gain (g/day)	171	202	221	41

This result confirms earlier data that steroid supplementation is a growth promotant. The desirability of this result is lessened in the light of marketing and economic pressures which dictate that venison should remain "clean and green". It is however valid to point out that the existing growth cycle can be manipulated in this way.

Other non-Invermay concepts

At Invermay we have fed high protein diet to deer. In Denmark high energy diets, up to 13 MJME/kg DM have been successfully fed (Frank Vigh Larsen, Personal communication). On these diets Danish red deer have approached a growth rate of 400 g/day in spring. Whether this is due to a genetic difference in growth rate is not known.

Clearly considerable research is being undertaken on novel pasture species to increase growth in deer, particularly during winter and lactation. Results are presented elsewhere in these Proceedings (Wilson *et al*). This is outside the scope of this review.

Likewise using novel deer species such as Pere David's or Rusa or their hybrids with red deer might result in significant growth promotion either by altering feed requirements or the seasonal cycles of demands for feed. No detailed studies on this aspect are to hand.

CONCLUSIONS

This review has covered what a growth cycle is and why we have confidence in the concept of growth and intake cycles. Much is poorly understood of the physiological

control of these cycles, particularly appetite, nutrient utilisation for growth, nutrient partitioning and the neuroendocrine control mechanisms and their interactions which underlie these mechanisms. Many of these factors have widespread relevance to animal and human biology, not just seasonality, and not just deer!

However, from our basic research programmes we have identified two areas of non-manipulative natural growth promotion, namely photoperiodic shifting of the cycles during winter indoors and the concept of "targeted" increased winter nutrition, from which unexpected carry-over benefits are reaped.

We know little; at least we are beginning to understand how little we know.

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