ARTIFICIAL MANIPULATION OF ANTLER CASTING AND ANTLER GROWTH IN RED DEER

G.K. Barrell
P.D. Muir

Animal Sciences Group, Lincoln College, Canterbury.

Introduction.

Growth and regeneration of antlers in deer are unusual phenomena which probably have always attracted the attention of biologists. In spite of this the mechanisms which control these processes are still poorly understood. Our studies with red deer stags at Lincoln College have been carried out partly to provide further insight into the biology of antlers but also with the view that such studies might lead to practical techniques which could have application in the deer farming industry.

Antler casting and growth cycles.

The annual cycle of antler casting, growth and mineralisation which occurs in stags has been described by many authors, so the following is just a brief description of the events which seem to be linked to this process. In red deer (*Cervus elaphus*) and in many other deer species, antler casting seems to be a critical event as growth of new antler tissue will occur, apparently spontaneously, when the previous set of hard antlers (or their remnants) is cast off. The fact that casting of both antlers is generally synchronous suggests that the stimulus for casting is a systemically borne signal, such as a hormone circulating in the blood. In addition a connection between antler casting and gonadal function in stags has been firmly established. Removal of the testes (castration) or even suppression of their function (e.g. by immunisation of stags against gonadotrophin releasing hormone - Lincoln, Fraser & Fletcher, 1982) will cause casting of hard antlers in red deer. Both of these techniques have a number of endocrine repercussions, but the simplest explanation is that the stimulus for antler casting is the cessation of androgen secretion by the testes. However it has been difficult to provide direct proof of this because blood levels of testosterone (probably the major androgen in stags) are already very low when casting normally occurs.

Growth of new 'velvet' antlers certainly requires a virtual absence of testicular hormone secretion as a permissive factor, but Dr Jim Suttie and co-workers at Invermay Agricultural Research Centre and Auckland Medical School have recorded a positive association between antler growth and blood levels of an insulin-like growth factor (IGF1) in stags during this period (Suttie, Fennessy & Gluckman, 1983). This demonstrates a possible avenue for future manipulation of velvet antler growth but the most important factors which are currently known to determine the ultimate size of antlers are size (live weight) and age of stags (Muir, 1985).

Resurgence of testicular activity, seen as a small increase in blood testosterone levels during December (Barrell, Muir & Sykes, 1985), provides another link between the gonads and antler physiology. This seasonal rise in testosterone secretion corresponds closely with the onset of a major increase in mineralisation of velvet antlers (Muir, 1985). The association between androgen secretion and antler mineralisation had originally been established from studies based on castrated stags. Such stags can not harden their antlers until they are given exogenous steroids (Wislocki, Aub & Waldo, 1947; Lincoln, Youngson & Short, 1970; Morris & Bubenik, 1982).

Apart from the early stages of growth of 'velvet' antlers, it can be seen that regulation of two important events in the antler growth cycle, namely casting and mineralisation, is closely related to the regulation of gonadal function.

Artificial induction of casting.

A reversible suppression of testicular activity in stags can be achieved by use of the synthetic progestagen - medroxyprogesterone acetate (MPA). We have shown that a single intramuscular injection of a depot form of this steroid (0.9 mg/kg, Promone E, Upjohn N.Z.) into red deer stags when they have hard antlers will cause casting within about three weeks (Muir, Barrell & Sykes, 1982). We suspect that this effect occurs because progestagens suppress luteinizing hormone (LH) secretion from the pituitary gland, thereby depriving the testes of stimulation. Alternative possibilities for modes of action include direct effects of MPA on testosterone synthesis in the testes or interference with receptors at the site of action of testosterone. In keeping with the former mechanism proposed above we have demonstrated that MPA does suppress testosterone secretion in stags and that a programme of MPA injections given at 3-weekly intervals will delay the onset of the annual rise in blood testosterone levels associated with the rut (Fig. 1).

Two important points to note are that MPA can not induce casting of antlers which have not fully hardened and that MPA is relatively ineffective during the peak of the rut, even when antlers are hard. This latter observation is probably a reflection of the high level of pituitary activity during the rut and thus of the large doses of progestagen that are needed to overcome that level of gonadal stimulation. In connection with this point, it should be noted that the peak of the rut in young stags occurs later in autumn than for older stags. Therefore MPA will be effective for inducing premature casting in young stags only if it is given in late May or afterwards.

Possible applications for this technique include advancement of casting of antler buttons so that velvet antler is ready for harvest at an earlier date. This might be useful if there is a marketing premium for early seasonal production of velvet. Alternatively it could be used to eliminate potentially late casting, such as occurs with young stags, so that the antler harvesting programme can be concentrated into a shorter span of time. By using this technique a degree of synchrony of casting can be achieved and this could be

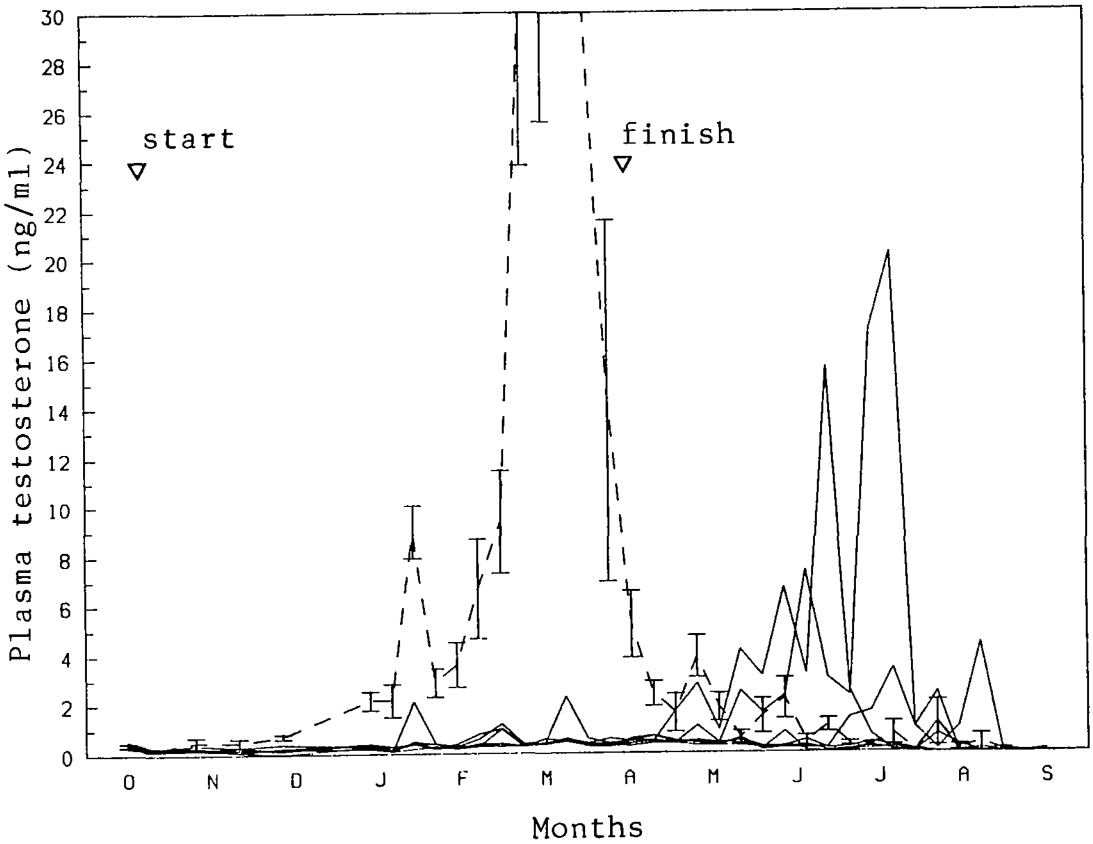

Fig 1: Plasma testosterone levels in 6 stags treated with MPA (0.9 mg/kg) every 3 weeks (between arrows). Dashed line represents mean values (±S.E.M.) from untreated stags.

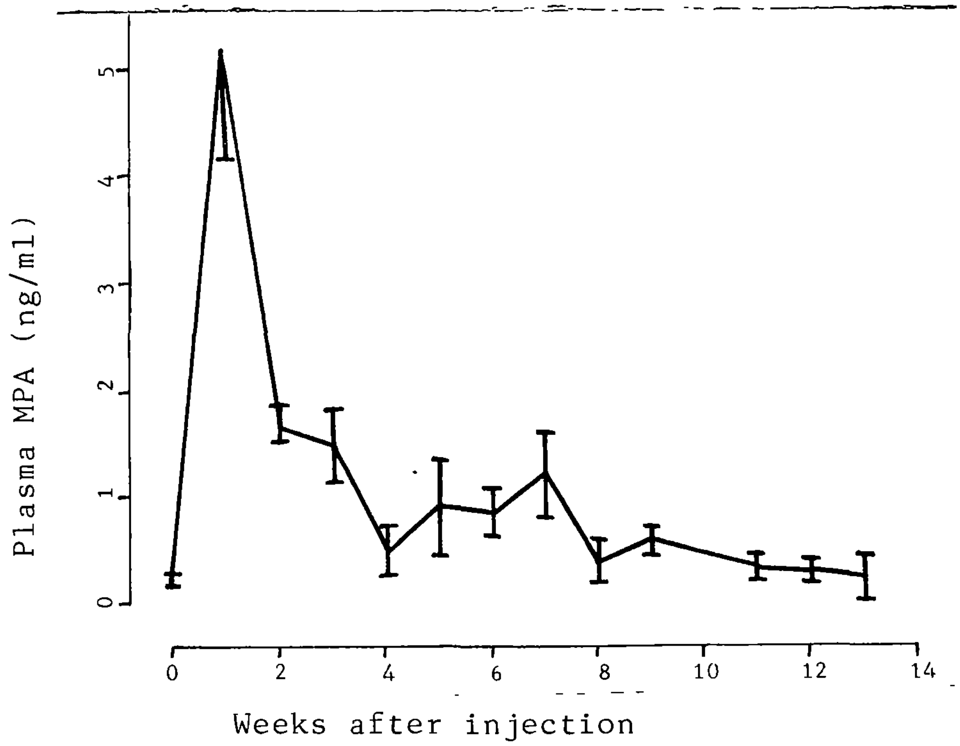

Fig 2: Mean plasma MPA levels (±S.E.M.) in 10 stags after a single injection (0.9 mg/kg, i.m.) of MPA.

exploited to reduce velvetting costs. In addition, induction of antler growth could provide a tool which would enable stags to be sent away for slaughter at different times of the year, yet still have produced a crop of velvet prior to their despatch. We have used MPA successfullly after velvetting to enable the growth of second and third crops of regrowth velvet which, even though smaller than the initial crop, were graded as marketable.

There still remains one large snag. MPA, or any progestagen, is not licensed for use in stags. Therefore it is important to note that in spite of the potential for pharmacological manipulation of velvet growth, progestagens and similar compounds can not be used commercially in stags at present. Also there is little likelihood of this procedure being approved in the near future.

If the current restriction on the use of hormones in stags is eventually removed there is still the possibility of drug residues remaining in body tissues. In the case of MPA administration as a means of inducing casting of antlers, we have made some effort to determine the disappearance of this drug following intramuscular injection of the depot preparation. We developed a chemical assay which utilised an antibody (kindly provided by Dr K.T. Kirton, Upjohn, U.S.A.) which had been raised in rabbits as a specific binding agent for MPA. This assay (radioimmunoassay) enabled us to measure MPA levels in the blood plasma of stags down to as low as 0.1 ng/ml. As can be seen in Figure 2, blood plasma levels reached the background value (about 0.5 ng/ml) within 10 to 12 weeks after an intramuscular injection of 150 mg (approximately 0.9 mg/kg). Assuming that levels in blood are representative of those in antler and muscle tissue, it can be concluded from these studies that MPA is not present in appreciable amounts after 11 weeks following injection. Velvet antlers would not be harvested for sale within this time scale if MPA was employed to induce casting, so residues of this hormone would not be expected to occur in commercially harvested antlers. Likewise, the meat from stags which are slaughtered <u>after</u> velvetting at the normal harvest stage would not be expected to contain any MPA residues.

<u>Suppression of the rut.</u>

One possible avenue for MPA treatment of stags arises from its ability to suppress testosterone secretion during the rut. This can be achieved if MPA treatment commences prior to the rut. In stags which are not being held for breeding purposes this could be utilised to overcome behavioural problems and reduce the severity of rut-induced weight losses. We have obtained some data which show that this latter object is feasible. In this study stags were injected with MPA (0.9 mg/kg) every 21 days from the date of velvet removal until mid winter. Weight losses over the rut in the treated group were only one third of those in the control group (Table I). In a subsequent study where each treated stag received a single MPA injection (0.9 mg/kg) prior to the rut, results were not so clear cut (Table II). There is considerable variation in the date of onset of the rut, therefore it is probable that in

TABLE I - Body weight losses of stags during the rut and the effect of repeated progestagen treatment (0.9 mg/kg MPA, given i.m. every 3 weeks).

Stag	Pre-rut body weight (kg)	Post-rut body weight (kg)	Body weight loss (kg)
Control Stags			
627	228.0	156.0	72.0
600	193.5	169.5	24.0
87	203.5	173.5	30.0
60	175.5	139.5	36.0
622	168.5	144.0	24.5
761	135.5	122.5	13.0
Mean	184.1	150.8	33.3
S.E.M.	±13.0	±7.9	±8.4
MPA Treated Stags			
451	183.0	172.0	11.0
887	205.0	193.0	12.0
405	173.0	165.5	7.5
899	177.5	158.5	19.0
99	168.5	155.0	13.5
717	128.0	125.0	3.0
Mean	172.5	161.5	11.0
S.E.M.	±10.3	±9.1	±2.2

TABLE II - Body weight losses of stags during the rut and the effect of a single i.m. injection of a progestagen (0.9 mg/kg MPA) given prior to the rut.

Stag	Pre-rut body weight (kg)	Post-rut body weight (kg)	Body weight loss (kg)
Control Stags			
231	196.5	176.0	20.5
10	200.0	158.5	41.5
99	185.0	153.0	32.0
289	164.0	153.5	10.5
899	178.0	152.5	25.5
Mean	184.7	158.7	26.0
S.E.M.	±6.5	±4.5	±5.2
MPA Treated Stags			
887	215.5	196.0	19.5
451	192.5	175.5	17.0
60	172.5	152.0	20.5
761	160.5	142.0	18.5
711	159.0	142.0	17.0
Mean	180.0	161.5	18.5
S.E.M.	±10.7	±10.6	±0.7

some of the treated stags MPA may have been largely eliminated from the body before the rut had commenced (e.g. refer to Fig. 2). As a result the effects of MPA on body weight loss would have been less marked.

The untoward effects of rutting on behaviour and live weight and their consequences for management of stags, especially during winter months, warrant further studies in this area of rut suppression.

Growing additional sets of antlers.

In their natural state, stags are limited to one set of antlers per year because of the annual daylight cycle. Additional sets of antlers can be grown and cast by artificially providing the annual lighting cycles within a compressed time scale. Three sets per year have been obtained in this manner from red deer stags (Suttie, Corson & Fennessy, 1984) and up to four from sika stags (Goss & Rosen, 1973).

We have utilised the ability of MPA treatment to induce early casting of antlers as a means for growing additional antlers. Since new velvet antlers will grow when casting has occurred, there is no difficulty in producing a set of A-grade velvet antlers by July in our red deer stags. However the stumps which are left behind after the velvet has been harvested do not calcify because testosterone secretion during winter is not high enough to induce a high degree of mineralisation. As a result the stumps will not be cast off in spring, since only fully calcified, hardened antler material can cast. So, a stag which has been treated in this manner will grow just one set of antlers in that year, albeit a few months earlier than usual.

The answer to this problem is to provide an artificial supply of testosterone, or any compound which will induce calcification, after the velvet has been harvested. This will harden the stumps (buttons) and, provided the drug is then removed, casting will occur in spring followed by growth of a second crop of velvet antlers. We have achieved this by administering subcutaneously tablets containing about 100 mg of crystalline oestradiol-17B to induce calcification (Barrell & Muir, 1984). Oestradiol has the advantage over testosterone in that it seems to be more potent, on a weight basis, for producing this effect (Goss, 1968; Fletcher & Short, 1974; Fletcher, 1978). Oestradiol is a naturally occurring hormone and thus its use in animals is likely to be less objectionable than for a synthetic hormone like MPA. Nevertheless, it also can not be used in stags because it is not yet licensed for these purposes.

Quite apart from the velvet industry is the possibility of using this approach to provide stags with trophy antlers outside the normal season. Use of MPA in April to induce casting, followed by oestradiol when the antlers have attained full size, will mean that stags treated in this manner could then carry their fully developed, hard antlers over the next twelve months. We have utilised a similar procedure to obtain two fully grown, fully hardened sets of antlers within one year on a single stag. Alternatively, timely administration of testosterone or an oestrogen could be used to delay casting and thus extend the period when stags are in hard antler.

Suppression of antler growth.

For those who do not wish to be bothered with antler growth at all, for example some farmers with fallow deer, polled stags must be a desirable proposition. Geoff Asher at Ruakura Agricultural Research Centre has been highly successful in polling fallow bucks (pers. comm.). His technique involves disbudding (removal of all primordial pedicle tissue) the bucks at 5 months of age. This eliminates antlers, and as a consequence, also reduces fighting between bucks in yards.

There has been little call for similar studies in red deer. Owners of red deer stags generally are appreciative of antler growth but there are many who find regrowth after velvet harvest a nuisance. Regrowth velvet is seldom of sufficiently marketable quality to justify the cost of harvest, yet it can develop into undesirable antler spikes which invariably have to be removed anyway. Consequently there is considerable interest in suppressing velvet regrowth and we have already carried out preliminary studies using oestrogens or testosterone to achieve this. Although results are encouraging, we have not gathered sufficient data yet to justify any conclusions about the most suitable procedure to use.

Conclusion.

Currently there is a surge of research activity being carried out in N.Z. and overseas into the biology of antler growth in deer. Our contribution from Lincoln College has helped to demonstrate that the antler growth cycle in red deer stags can be manipulated by using endogenous hormones, but it must be appreciated that many of the results so far are preliminary. The various procedures need to be repeated a number of times and their consequences studied over a few years before their use can be advocated. In addition the commercial use of hormones and their agonists awaits further drug licensing.

We are confident that farmers and veterinarians will continue to be able to exercise an increasing degree of control over the growth of antlers. This will depend on maintenance of the research effort which is being applied to this field.

References.

Barrell, G.K. & Muir, P.D. (1984). Experimental manipulation of antler growth in red deer stags by treatment with exogenous steroids. Proc. Endocr. Soc. Aust. 27: S14.

Barrell, G.K., Muir, P.D. & Sykes, A.R. (1985). Seasonal profiles of plasma testosterone, prolactin and growth hormone levels recorded from some red deer stags in New Zealand. In: Biology of deer production. Eds. P.F. Fennessy & K.R. Drew. Royal Society of N.Z., Bulletin 22: Wellington. (In press).

Fletcher, T.J. (1978). The induction of male sexual behavior in red deer (*Cervus elaphus*) by the administration of testosterone to hinds and estradiol-17B to stags. Horm.

Behav. 11: 74-88.
Fletcher, T.J. & Short, R.V. (1974). Restoration of libido in castrated red deer stag (Cervus elaphus) with oestradiol-17B. Nature 248: 616-618.
Goss, R.J. (1968). Inhibition of growth and shedding of antlers by sex hormones. Nature 220: 83-85.
Goss, R.J. & Rosen, J.K. (1973). The effect of latitude and photperiod on the growth of antlers. J. Reprod. Fert. Suppl.19: 111-118.
Lincoln, G.A., Fraser, H.M. & Fletcher, T.J. (1982). Antler growth in male red deer (Cervus elaphus) after active immunization against LH-RH. J. Reprod. Fert. 66: 703-708.
Lincoln, G.A., Youngson, R.W. & Short, R.V. (1970). The social and sexual behaviour of the red deer stag. J. Reprod. Fert. Suppl. 11: 71-103.
Morris, J.M. & Bubenik, G.A. (1982). The effect of androgens on the development of antler bone. In: Antler development in cervidae. Ed. R.D. Brown. Caesar Kleberg Wildlife Research Institute: Kingsville, Texas. pp.123-141.
Muir, P.D. (1985). Studies on the growth and compositional development of antlers in red deer (Cervus elaphus). Ph.D. Thesis: Lincoln College.
Muir, P. D., Barrell, G.K. & Sykes, A.R. (1982). Modification of antler growth in red deer stags by use of a synthetic progestagen. Proc. N.Z. Soc. Anim. Prod. 42: 145-147.
Suttie, J.M., Corson, I.D. & Fennessy, P.F. (1984). Voluntary intake, testis development and antler growth patterns of male red deer under a manipulated photoperiod. Proc. N.Z. Soc. Anim. Prod. 44: 167-170.
Suttie, J.M., Fennessy, P.F. & Gluckman, P.D. (1983). Insulin like growth factor 1: antler stimulating hormone? Proc. Endocr. Soc. Aust. 26: S28.
Wislocki, G.B., Aub, J.C. & Waldo, C.M. (1947) The effects of gonadectomy and the administration of testosterone propionate on the growth of antlers in male and female deer. Endocrinology 40: 202-224.