Modification of antler growth in red deer stags by use of a synthetic progestagen

P. D. MUIR, G. K. BARRELL and A. R. SYKES

Lincoln College, Canterbury

ABSTRACT

Separate trials were conducted to investigate the effects of a synthetic progestagen—medroxyprogesterone acetate (MPA)—on red deer stags at 2 different stages of the antler growth cycle. MPA treatment of 2 stags from the time of harvest of 'A-grade' velvet stimulated the growth of additional sets of antlers in each case. This treatment also reduced blood testosterone levels and prevented the seasonal increase in testicular size. Alternatively, MPA treatment applied to 6 stags during their hard antler phase (winter) caused premature casting of the antler buttons in all animals. These findings indicate a possible role of MPA or similar compounds for the artificial manipulation of antler growth under farming conditions.

INTRODUCTION

Under normal circumstances only one set of good commercial quality velvet antlers can be harvested per year from a farmed stag. In a few cases, if the initial harvest is sufficiently early in the season, some additional velvet growth does occur although it is usually of inferior quality. Any attempt to modify the normal chain of events requires some understanding of the control mechanisms involved. However, such knowledge is currently limited.

The involvement of reproductive hormones in the antler growth and casting cycle has been suggested by studies involving castration and hormone replacement therapy. For instance, castration of stags during velvet antler growth caused the antlers to remain in the velvet state and prevented casting indefinitely (Wislocki *et al.*, 1947). Subsequent treatment with reproductively active steroids such as testosterone (Wislocki *et al.*, 1947) or oestradiol-17β (Fletcher and Short, 1974) caused completion of mineralisation with shedding of velvet: afterwards casting of the antlers occurred. Antler casting is normally associated with the period of low blood testosterone levels (Lincoln and Kay, 1979) and castration of stags during the hard antler phase caused casting a few weeks later (Goss, 1963; Lincoln, 1971; Lincoln *et al.*, 1970). Instead of castration a novel approach has been the use of anti-androgen therapy which successfully retarded the antler mineralisation process (Bubenik *et al.*, 1975). However the compound used, cyproterone acetate, had the disadvantage of high cost and deleterious effects on animal health.

Progestagens can suppress testicular function by blocking the release of pituitary luteinising hormone (Bolt, 1971). This removes the source of stimuli for testicular androgen production thereby effectively, but reversibly, 'castrating' the treated subject. Accordingly, administration of a suitable progestagen (e.g., medroxyprogesterone acetate, MPA) to deer stags should interfere with the antler growth processes and could provide a satisfactory alternative to cyproterone acetate for the manipulation of antler growth.

MATERIALS AND METHODS

Experiment 1

Three rising 3-year-old red deer stags housed indoors at Lincoln College had velvet antlers removed in the first week of December 1980. Approximately 1 week later 2 of the stags (717, 711) were each given a single intramuscular injection containing 150 mg of a depot form of MPA (Depo-Provera, Upjohn Pty. Ltd., N.Z.). This dosage was repeated at 4-weekly intervals on 2 further occasions to 717 and on 5 further occasions to 711. The third stag (752) remained as a control. These animals were fed *ad libitum* a pelleted diet containing formaldehyde-protected linseed protein concentrate.

TABLE 1 Velvet antler weight (g) in MPA-treated and control stags, Experiment 1.

Stag No.		First cut mean of both antlers	Second cut left antler only
Control	752	560	—
Treated	711	562	228
Treated	717	514	220

Experiment 2

Six mixed-age mature red deer stags which had cast hard antler buttons early in spring 1980 were allocated to 3 groups each containing 1 treated and 1 control animal. Six similar stags which had cast relatively late in 1980 were allocated to the groups on

the same basis. Group 1 treated stags were injected with MPA (as in Experiment 1) on 19 June 1981 and once again 3 weeks later. Treated stags in Groups 2 and 3 were injected with MPA, once only, on 10 July and 31 July 1981, respectively. All stags were maintained outdoors on fescue and ryegrass white clover pasture supplement with lucerne hay and pelleted concentrate (NRM deer nuts).

Blood samples were collected (weekly in Experiment 1 and 3-weekly in Experiment 2) by jugular venepuncture to provide plasma which was frozen and stored at −20°C. All plasma samples were assayed for testosterone and prolactin by radioimmunoassays as described by Barrell and Lapwood (1979). Regular measurements were taken of body weight and testicular diameter and all antler growth changes, including button casting dates (Experiment 2), were recorded. When the antlers reached commercial harvest stage they were removed; left side only in Experiment 1, both sides in Experiment 2.

RESULTS

Experiment 1

Both MPA-treated stags grew antlers which reached commercial harvest stages in 8 to 9 weeks following commencement of injections. These second antlers were about one-half the weight of those harvested immediately prior to the trial (Table 1). A short (11.5 cm) third antler growth developed from the cut stump of stag 711 after the second antler had been removed. Stripping of velvet from all remaining antlers occurred 11 and 13 weeks (717 and 711, respectively) after the final MPA injection in each case. Antler buttons were subsequently cast at approximately the same dates as in the previous year for all 3 stags.

Testicular diameters increased in the control stag, but not in the treated stags (Fig. 1).

Plasma testosterone levels rose in the control stag during the experimental period, in accordance with levels in other untreated stags housed in the same shed. The values for plasma testosterone in the MPA-treated stags were suppressed and remained low throughout the treatment period. After withdrawal of MPA plasma testosterone levels and testicular diameters of both treated stags gradually increased to control stag values (Fig. 1). Plasma prolactin levels were unaffected by the treatment.

Experiment 2

MPA-treated stags in Group 1 cast their hard antler buttons 3 to 4 weeks after their first injection and in Groups 2 and 3, 2 to 3 weeks after the single injection (Fig. 2). All treated stags cast buttons considerably earlier than in the previous year and were well in advance of untreated stags, most of which cast within

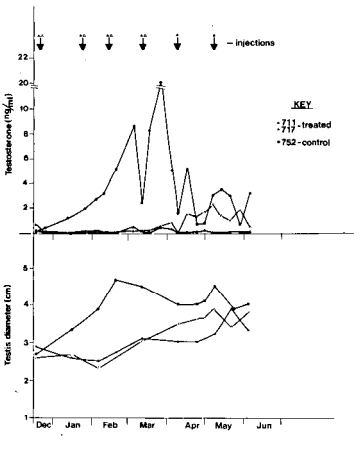

FIG. 1 Testosterone levels and testis size—Experiment 1

a week of their 1980 casting dates. Stags which cast buttons late in 1980 appeared to respond to treatment as rapidly as those which cast early. Antler growth following casting in all stags was normal in terms of the patterns of growth achieved in the previous year. As a result the dates of velvet harvest from the treated stags was much earlier than in the control animals.

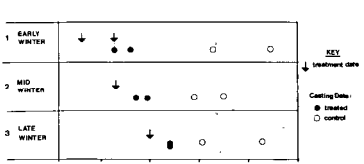

FIG. 2 Antler casting dates for treated and control stags—Experiment 2.

During the course of this experiment plasma testosterone and prolactin levels were low in all stags and testicular sizes decreased. No effect of treatment on these parameters could be detected.

DISCUSSION

Experiment 1 was a pilot trial, but the results obtained demonstrated clearly that MPA suppressed testicular steroidogenesis and stimulated secondary antler growth at a time when such regrowth would not have been expected to occur. Also the antler hardening following withdrawal of MPA and their subsequent casting near the previous year's dates indicated that the effects of the drug were not permanent. This anti-androgenic effect of MPA in stags is in accord with such effects reported from other studies with the same compound in male rats (Barbieri and Ryan, 1980), dogs (Wright *et al.*, 1979) and humans (Rivarola *et al.*, 1968). Together with the finding of Bubenik *et al.* (1975) that the anti-androgen cyproterone acetate retarded *mineralisation of antlers in white-tailed deer*, these results support the view that the antler growth cycle is mediated in part by testicular function. Likewise the premature induction of button casting, unequivocably due to MPA (Experiment 2), was similar to the effects of castration during the hard antler phase (Goss, 1963; Lincoln, 1971; Lincoln *et al.*, 1970) and would indicate that antler casting was a consequence of a sudden reduction in testicular activity. Nevertheless some caution should be applied to this interpretation since the data from our untreated stags suggested that testicular activity (estimated on the basis of plasma testosterone levels) was already minimal many weeks prior to the spring casting period. Our understanding of the precise biological mechanisms involved in antler casting remains incomplete.

In spite of gaps in our understanding of antler growth it seems feasible to consider applying the findings reported here directly to stag management systems. However, before MPA or any other synthetic progestagen could be used to advance or synchronise the date of velvet harvest on commercial farms, a careful check on the possibility of drug residues in the velvet product, or even in the carcass, should be carried out. This is a current undertaking in our laboratory.

There is a lack of information on the effect of MPA treatment on long-term stag fertility. However, evidence suggests that progestagen treatment does not affect the subsequent fertility of humans (Brotherton, 1972) and rats (David *et al.*, 1963). Despite these findings it is not yet recommended that MPA treatment be given to any stag which could be used for breeding purposes.

ACKNOWLEDGEMENTS

To A. S. Familton and M. Keeley for assistance with deer handling, Miss J. Wilkinson for performing hormone assays and to Upjohn Ltd, for donation of MPA.

REFERENCES

Barbieri, R. L.; Ryan, K. J., 1980. *Acta endocr., Copenh., 94:* 491.

Barrell, G. K.; Lapwood, K. R., 1979. *J. Endocr., 80:* 397.

Bolt, D. J., 1971. *J. Reprod. Fert., 24:* 435.

Brotherton, J., 1972. *Biblphy Reprod., 20:* 913.

Bubenik, G. A.; Bubenik, A. B.; Brown, G. M.; Wilson, G. A., 1975. *J. Exp. Zool., 194:* 394.

David, A.; Edwards, K.; Fellowes, K. N. and Plummer, J. M., 1963. *J. Reprod. Fert., 5:* 331.

Fletcher, T. J.; Short, R. V., 1974. *Nature, No. 5449, 248:* 616.

Goss, R. J., 1963. *Mechanisms of hard tissue destruction.* Pub. No. 75, American Association for the Advancement of Science, Washington, USA, p 339.

Lincoln, G. A., 1971. *J. Zool., Lond., 163:* 195.

Lincoln, G. A.; Kay, R. N. B., 1979. *J. Reprod. Fert., 54:* 209.

Lincoln, G. A.; Youngson, R. W.; Short, R. V., 1970. *J. Reprod. Fert., Suppl. 11:* 71.

Rivarola, M. A.; Camacho, A. M.; Migeon, C. J., 1968. *J. clin. Endocr., 28:* 679.

Wislocki, G. B.; Aub, J. C.; Waldo, C. M., 1947. *Endocrinology, 40:* 202.

Wright P. J.; Stelmasiak, T.; Black, D.; Sykes, D., 1979. *Aust. vet. J. 55:* 437.