



Fletcher, J., Foster, A., Goddard, P., & McSloy, A. (2016). Managing antler problems in deer. *In Practice*, 38(10), 513-519. DOI: 10.1136/inp.i5271

Peer reviewed version

License (if available):
Unspecified

Link to published version (if available):
[10.1136/inp.i5271](https://doi.org/10.1136/inp.i5271)

[Link to publication record in Explore Bristol Research](#)
PDF-document

This is the author accepted manuscript (AAM). The final published version (version of record) is available online via BMJ at <http://inpractice.bmj.com/content/38/10/513>. Please refer to any applicable terms of use of the publisher.

University of Bristol - Explore Bristol Research

General rights

This document is made available in accordance with publisher policies. Please cite only the published version using the reference above. Full terms of use are available:
<http://www.bristol.ac.uk/pure/about/ebr-terms.html>

Deer – managing problems with antlers

John Fletcher, Aiden Foster & Pete Goddard*

With Alex McSloy

* All authors contributed equally

Harthill, Reediehill Deer Farm, Auchtermuchty, Fife KY14 7HS.

John Fletcher qualified for a PhD from Cambridge on the reproductive physiology of red deer on the Isle of Rum, West Scotland in 1974. He runs his own deer farm in Auchtermuchty and writes books about deer.

School of Veterinary Sciences, University of Bristol, Langford House, Langford, BS40 5DU.

Aiden Foster graduated from the University of Bristol in 1987. He is currently senior teaching fellow in veterinary dermatology and pathology at the University of Bristol.

The James Hutton Institute, Craigiebuckler, Aberdeen, AB15 8QH.

Pete Goddard graduated from the RVC in 1979. He is the secretary/treasurer of the Veterinary Deer Society and chairs the newly formed Wild Animal Welfare Committee

Farm Animal Health and Production Group, The Royal Veterinary College, Hawkshead Lane, North Mymms, Hatfield, Hertfordshire, AL9 7TA.

Alex McSloy qualified from the University of Cambridge in 2003. She is currently a lecturer in large animal internal medicine and food animal production medicine at the Royal Veterinary College, where she also heads the clinical team at the Farm Animal Clinical Centre. Her special interests are in small ruminants, camelids and reindeer.

(Introduction/Summary paragraph) *Veterinarians may encounter deer as a result of road traffic accidents, as young deer being bottle reared, or where veterinary problems occur in farmed or park deer, or in animal collections. In all those situations antlers may prove to be the most mysterious organs and are the source of most questions received by members of the Veterinary Deer Society (VDS). The following article is based in part on material prepared for the VDS website to provide interested parties with more information about antlers. There are particular issues for practitioners when dealing with castrated deer, especially reindeer, and with orphan male calves. Hormonal intervention in deer to control the antler cycle has been used in a variety of deer species; in reindeer there are only a few formal detailed studies of the influence of hormones on the antler cycle. Consequently it is difficult to be sure of the optimum dosing regimen for managing antler problems in castrated reindeer. As a general caveat, the authors consider that reindeer are, in general, ill-suited to being enclosed in small paddocks, especially in the south of England.*

THE BACKGROUND TO ANTLERS

Antlers are one of the most amazing features of the mammal kingdom. Within mammals they are the only example of the regeneration of an entire organ and they are also the fastest growing mammalian tissue. Perhaps as a result of this they have been seized upon as symbols of growth and regeneration, and often used as medicine, in almost every culture in Europe, Asia and North America (Fletcher, 2013).

The ruminants are divided into two principal families: the horned ruminants or Bovidae including sheep, cattle, antelope, gazelle, buffalo etc., and numbering some 140 species, and the antlered ruminants or Cervidae comprising 16 genera and at least 40 species of deer. As may be imagined there are great variations amongst those 40 species including within the subject of antler growth and it is not easy to generalise across the family.

Antlers are peculiar to deer and are the most conspicuous identifying features of the Cervidae. Because deer evolved to live within northern temperate regions they are highly seasonal with their breeding cycles controlled by the seasons and mediated by day length (Lincoln, 1971a). Although some species have colonised tropical regions and have to a greater or lesser degree lost that seasonality, antlers have retained their deciduous character. In general they grow during the period of the year when animals are

sexually quiescent and mineralise and die under the influence of testosterone in preparation for the mating season. The hard antler is therefore a male secondary sexual character.

Antlers grow by endochondral ossification from stem cells derived from the periosteum of an anatomically distinct protuberance of the frontal bone, the pedicle (Rolf and others, 2006; Mount and others, 2006; Napp and others, 2006). As antler may grow at 2 cm/day in some species it provides a valuable model for the investigation of bone formation (Bubenik, 2001).

The growing antler is cartilaginous and covered by skin with large numbers of sebaceous glands and hair. This skin is known as velvet and is heavily vascularised; the deer are described as being "in velvet". As the antlers harden in response to rising levels of testosterone the cartilage ossifies and the antler becomes effectively a dead bone attached to the pedicle. Antlers are thus very different from the horns of the Bovidae. Although any animal that grows antlers is a deer not all deer species grow antlers: the Chinese water deer (*Hydropotes inermis*), which is now well represented as a wild species, accidentally introduced to England, grows no antlers.

Apart from reindeer (*Rangifer tarandus*) and their New World wild counterpart the caribou, only male deer grow antlers. Occasionally females of other species grow antlers, including roe (*Capreolus capreolus*) and much more rarely red deer (*Cervus elaphus*). This anomaly is less rare in species in which twinning is more common leading to suggestions that antlered females may occur as a result of masculinisation by a male co-twin. It is generally believed that female reindeer grow antlers as an adaptation to their arctic habitat. Adult males and females that are not lactating cast their antlers before the winter. However, lactating females retain their antlers during the winter and are therefore able to defend snow holes and provide access to lichens and other forage for their calves when competing with other members of the herd (Henshaw 1969). The antler cycle for male and female reindeer is illustrated in Figure 1.

In other species, the fully grown and mineralised antlers are male secondary sexual characters which are hard, dead weapons and so can be broken with relative impunity in competing for females. The damage can be made good during the next antler cycle. Although the hard antlers are primarily weapons and shields they also contribute to the competitive displays between males during the mating season in the same way that body size, vocalisations and pheromones do (Bubenik and others 1982; Geist, 1991; Bubenik, 2001). Two small deer species accidentally released in England, the antlerless Chinese water deer and the muntjac (*Muntiacus reevesi*) which has only small antlers, use their canine teeth as weapons.

Red deer, fallow deer (*Dama dama*) and roe deer visibly grow their antler pedicles *in utero* in response to testosterone although by birth they may no longer be visible (Bubenik and others, 1982; Audenaerde and Simoens, 2006). It seems likely that embryonic pedicles in other deer species also differentiate in response to testosterone.

By the end of their first winter in red deer, the pedicles have made rapid growth stimulated by testosterone and from these pedicles during their first year of life red deer males develop their first antlers at a rate influenced by levels of nutrition. Having cast their first antlers, often just simple spikes, usually at about two years of age, stags grow normal adult antlers whenever testosterone levels are very low. Thus in red deer and other species which have been investigated, differentiation of the pedicle in early life is apparently stimulated by testosterone. By contrast antlers grow only when testosterone levels are at very low, baseline, levels. Therefore growth is controlled by testosterone levels which are in turn controlled by changing day length: in spring, in red deer, as a response to increasing day length, testosterone levels decline causing the antlers to fall off or 'cast'. New antlers immediately begin to grow and continue to do so very rapidly through the early summer when testosterone levels are very low indeed. With the summer solstice, luteinising hormone levels and subsequently testosterone levels begin to rise and, influenced by the declining day length, red deer stags harden or mineralise their antlers. By late summer red and fallow deer shed, or 'clean', their velvet. This covering of the live antler becomes frayed and is eventually lost, leaving the characteristic hard antler which the stag uses to exert his dominance over competing males. See Figure 2 for a graphical description of hormone changes during the red deer rut. Figure 3 illustrates antler growth.

The rut, which in most British red deer may be considered to begin in September, effectively ends in late October although younger stags may rut later and stags may mate hinds throughout the winter. After the rut, as testosterone concentrations decline, the antlers are shed; in red and fallow deer in the early spring. Regrowth commences and the cycle is repeated. One of the first comprehensive studies of the seasonal reproductive changes in red deer stags was undertaken by Lincoln (1971a,b).

If male deer are castrated as calves before the pedicle has differentiated then no antlers can be formed. If deer are castrated when they are older and once the pedicle has become active then antlers are formed, but in the absence of testosterone the antlers remain in velvet. If the stag is castrated in the hard antler phase these antlers are cast, usually in about three weeks, new antlers grow and the antlers remain soft and live, covered in velvet for the rest of the castrate's life. If the red deer stag is castrated when he is growing his antlers then the antlers will remain in velvet and never harden. Thus in all cases, castration of the adult stag results in him remaining permanently in velvet.

Castrate red deer, being in velvet will almost always be subordinate to stags that are in hard antler during the winter and as such may be excluded from feed sources but as they do not suffer the weight losses which intact stags undergo during the rut they are not normally adversely affected and their welfare is not compromised except in the rare situation where the velvet growth becomes disorganised creating a 'perruque' or wig (Kay and Youngson, 1978). Whilst the formation of large disorganised perruque antlers (Figure 4b) is rare in castrate red deer, in roe deer castration of adult bucks invariably produces perruque antlers (Figure 4a). This perruque antler will eventually overwhelm the roe buck and kill it.

The response of fallow deer to castration is similar to that of red deer and the resultant antlers have been described as 'antleromas' (Kierdorf and others, 2004). Wild white-tailed deer (*Odocoileus virginianus*) have occasionally been reported to have antleroma type lesions and these are usually associated with alterations in circulating levels of testosterone (Munk and others, 2015).

The above is a summary of the situation in red and fallow deer and most other temperate climate deer species are approximately similar in their antler cycles and response to sex hormones although within Britain our native roe deer are seasonally different in growing their antlers during the winter. While tropical species such as muntjac are sexually active all year round.

Managing the cleaning of perruques

The veterinarian is occasionally asked to treat adult male deer that have been castrated and are consequently in a state of persistent velvet which may have developed haematoma or have hypertrophied into a perruque. As early as 1935, Georg Blauel demonstrated that oestradiol is effective in causing castrate roe bucks to clean the velvet from their perruques (Blauel, 1935). Subsequently, it was shown that oestradiol is more than ten times as effective in mineralising velvet antlers than testosterone (Goss, 1968; Bubenik, 1990b). The velvet antlers of castrate red deer have also been shown to be readily cleaned using oestradiol (Fletcher and Short, 1974). The more common approach based on the physiological control of antler growth has been to administer exogenous testosterone. However, this will make the deer rut and become aggressive – something that castration was aimed at avoiding.

For the practitioner the above may be somewhat theoretical since access to depot oestrogens or androgens that will permit levels to be maintained for a sufficient length of time may no longer be available (there are no veterinary licensed preparations and limited human hormonal products for injection are available). Published experimental results have usually entailed the use of 100 mg oestradiol-17 β implants or 1 gm implants or depot injections of testosterone (and repeated depot injections of testosterone have been tried). Implants are placed subcutaneously and removed once the velvet has been cleaned. Once the administration of exogenous hormones is stopped the antlers will be shed (although in some deer this may not be the case) and revert to new velvet growth but it will take some months for the perruque to regrow and that may be considered sufficient therapy.

Non-hormonal factors influencing antler growth

Deer often grow asymmetric antlers, usually contralaterally, as a result of lameness or amputation (Marburger and others, 1972). The mechanism for this is unknown although it has been suggested that the mechanics of limping may be a factor (Davis, 1983). Parasitism or other systemic disease may stunt antler growth. Also, it is not uncommon for individual deer to occasionally clean or cast their antlers at abnormal times for no apparent reason.

Deer of all species may fracture their antlers when they are growing. This may create substantial haematoma which will eventually mineralise. Unless the broken antler is endangering an eye no veterinary intervention is likely to be necessary. Fractures of hard antlers are extremely frequent and provided the fracture is above the coronet no action need be taken. Where the pedicle is fractured and the antler or part of the antler remains it may be necessary to tranquillise the deer and saw off the hard, insensitive antler to remove the weight. This will normally allow the pedicle to heal but may mean that

subsequent antlers do not grow in their correct position. In such cases it may be thought worthwhile to remove the pedicle. This will prevent any subsequent antler growth. The permanent prevention of antler growth in red deer by amputating the pedicle in farmed red deer calves is effective (Blaxter and others, 1988) and disbudding has been reported but is not commonly practised (Hamilton and others, 1993).

BOTTLE RAISED DEER

A frequently recurring question posed to veterinary surgeons is what action should be taken to prevent bottle raised, orphan male deer becoming dangerous as they become mature. This is a very real concern since tame male deer that have lost their fear of humans and especially if they have become imprinted on their carer, are extremely dangerous and have caused serious injuries and fatalities. By castrating the deer before the antler pedicles have become visible in the calf no antlers can be grown and the animal appears morphologically similar to the female. It is very strongly recommended that veterinary advice should always be to castrate male red, roe or fallow deer orphans as calves. Reindeer being semi-domesticated pose less risk but it would be advisable to ensure that intact male reindeer in hard antler are not used in petting collections. Intact male reindeer used in Christmas displays will not have antlers since they are shed earlier in the year. The unaccompanied public should never be allowed to enter an enclosure with tame deer of any species in hard antler.

REINDEER

In reindeer, the control of the antler cycle is different from all other deer species as alone amongst deer, females grow antlers and retain them whilst suckling to protect their young. In females, antler growth is controlled by oestrogen (Lincoln and Tyler, 1999). Intact adult male reindeer cast their antlers in early winter becoming subordinate to lactating females and to younger males which retain their antlers for longer. The castration of reindeer to prevent rutting activity is a normal practice amongst those peoples in the arctic who use reindeer for pulling sledges, for riding or even for meat. Traditionally reindeer may be castrated by biting with the teeth to crush the spermatic cord and destruction of testicular tissue by manual pressure. Reindeer calves develop pedicles very young (Blake and others, 1998) and castration, even of very young calves, is unlikely to prevent antler growth and perruque formation.

Even when more conventional castration with removal of the testicles is carried out in reindeer inhabiting arctic habitats, the antlers of castrates are normally cleaned and cast during the winter. It is thought that this is a response to cold during the winter and also to a nutritional winter deficit; castrates amongst the free ranging herd of reindeer in the Cairngorms clean and cast their antlers (*pers. comm.* Tilly Smith, The Reindeer Co., Reindeer House, Aviemore, PH22 1QU). The mechanism is thought to be through a response to adrenal steroids, probably corticosteroids (*pers. comm.* Professor Gerald Lincoln). Reindeer kept further south and used for Christmas displays in England respond differently to castration, as they do not normally clean and cast their antlers and frequently develop perruques. In the absence of available depot steroid preparations recent treatment has been by surgery.

On some holdings across England and Wales, castrated reindeer have been reported with unusual large fibropapillomatous growths. Initial investigations sought to establish an infectious viral cause but none was found (Foster and others, 2013); such lesions of the antler velvet may be similar to the perruques seen in castrates of other deer species (Figure 5). It is likely that, given that female reindeer do not develop such lesions and entire males rarely do, these are an unusual and uncommon manifestation of the complex interplay between antler development and endogenous sex hormones. These would probably be capable of being cleaned under the influence of exogenous hormones but as yet no treatments appear to have been effective and the limited number of drugs available leaves surgery as the only practicable option. In any case, some cases can have such large lesions that secondary bacterial infection and the risk of fly strike require surgical intervention (see box below). It is unclear if lesions recur every year in all cases.

A variety of terms have been used to describe abnormalities of antler growth in various species of deer where the influence of testosterone has been reduced through castration or in cryptorchidism or some form of hypogonadic state (Bubenik 1990a; such as viral infection, see Fox and others 2015). Munk and others (2015) have recommended the use of the term antleroma to describe any tumour or neoplasm composed of antler derived tissues including one or more of the velvet skin, bone, cartilage and fibrous connective tissue.

With contribution from Alex McSloy – Management of antler velvet lesions (antleromas) in reindeer

Sedation, analgesia and careful attention to haemostasis should be used when removing all or part of the antler with such lesions. It is important to accurately weigh reindeer before administering drugs and not to guess their body weight. Care must be exercised in the handling of such deer – some are halter trained others are challenging to handle.

Sedation: A combination of intramuscular xylazine and ketamine (0.3 mg/kg and 0.5-1 mg/kg respectively) should make the deer recumbent. In quiet deer, accustomed to being handled, it may be possible to use intravenous medication such as 0.075 mg/kg xylazine with 0.1 mg/kg diazepam IV then IV ketamine as needed.

In a hospital situation it is possible to protect the airway from regurgitation with endotracheal intubation and mixed air delivery throughout the procedure and isoflurane if needed. However, such equipment will not be practical when using propane disbudding irons to cauterise blood vessels. Where inhalation anaesthesia is not employed then there may be a need for top up with ketamine at 1 mg/kg IV. Sedation can be reversed with intramuscular administration of atipamazole.

Analgesia: Intravenous meloxicam at 0.5 mg/kg and a ring block with procaine.

Haemostasis: A tourniquet can be applied to the base of each antler at the start. For significant haemorrhage haemostats are used to handle blood vessels, twist and leave on, as in cattle. For small bleeds, a propane disbudding iron can be used.

Antibiosis: Intramuscular administration of penicillin / streptomycin combination for three days. Wounds can be dressed with oxytetracycline based spray and the owner should be advised about fly control if the time of year and conditions dictate.

At the time of writing, the authors are not aware of any reliable data on non-surgical treatment options that result in antler control in castrated reindeer, though exogenous testosterone, oestrogen or corticosteroids are likely to cause antler hardening. The normal signal for antler shedding in species other than reindeer, as noted earlier, is a decline in the concentration of circulating testosterone (female reindeer antlers are controlled by oestrogen of course) which "permits" new antlers to start growing. Oestrogens may have a minor secondary role to play in the antler cycle of male reindeer (Bubenik and others, 1997). So, giving testosterone or oestrogen could help in castrates which fail to harden their antlers. However, in reindeer, antler cycle control is more complex than in red deer, involving oestrogen and prolactin, and there is much debate about what endogenous concentrations of testosterone are required to regulate these processes. Furthermore, as noted above, there is evidence that castrated prepubertal calves will still go on to grow normal antlers and shed them as part of the cycle – albeit with some time delays for shedding in some cases (Lincoln & Tyler, 1992). Similarly in older castrates the cleaning of the velvet may be protracted and antler shedding delayed compared with entire males (Lincoln & Tyler, 1994).

Any exogenous treatment would need to exceed a threshold dose for a sufficient period and given that a normal hormonal environment will not have preceded this, adequate resolution of such problems is recognized as a significant challenge. (Sometimes, in the long term, sepsis and fly worry become serious secondary problems if the new growth continually fails to harden.) In practice, despite giving testosterone injections, some people have reported failure of attempted treatment. It is likely that a significant threshold concentration of circulating testosterone for a sufficient period (perhaps 2-3 weeks) to enable mineralization to occur was not achieved. As an example, we have a report of the off-label use of two doses of 2.5 ml IM of a 50 mg/ml injection (i.e. 125 mg) of testosterone, roughly 3 weeks apart (since such solutions are likely to have an active period of up to 28 days) but this was ineffective and it is possible that the dose was too low for a reindeer bull which is likely to weigh about 150 kg (weight can vary hugely). More encouragingly, there is a report of using 250 mg of testosterone IM as a one-off treatment which eventually proved effective but it is likely that repeated (and perhaps slightly higher) doses would have given a better result. Thus one option for successful management of antler problems with castrated reindeer is to use around 10 mL of a 50 mg/mL sterile, non-aqueous solution of

testosterone, possibly with a second dose three weeks later if there is no initial effect. Alternatively, silastic tubing implants containing oestradiol or testosterone could be administered in June and removed in October although the optimal dose of either hormone for this mode of treatment is unknown (and subject to the availability of products). All of the experimental work in red deer has been conducted using subcutaneous implants of crystalline testosterone – normally 1 gm implants estimated to release 2.5 mg testosterone per day, or silastic implants. Of course, giving large doses of testosterone to castrated males may induce aggressive behaviour in the recipient – something castration was aimed at avoiding. Consequently the use of 100 mg oestradiol-17 β implants releasing 0.25 mg/day may be preferable. (Both of these hormones took 3 – 4 weeks to initiate antler cleaning in red deer.)

Female reindeer antler cycles have been controlled experimentally by implanting oestradiol into reindeer in velvet to clean the antlers and removing the implants four months later to induce antler casting (Lincoln and Tyler, 1999). Oestradiol is less likely than testosterone to stimulate aggression.

ADVISORY NOTE

Serious consideration should be given to the advisability of keeping reindeer in mild temperate regions not only because of the possible problems associated with antler hypertrophy in castrates but also because these are sub- arctic species prone to a number of diseases which they may not encounter in their native regions.

Any hormonal treatments must be accompanied by the usual off-label use warning according to the prescribing cascade principles (and, if products do not have an authorisation for use in any food-producing animals, the consequent prohibition of treated animals from entering the human food chain) with the informed consent of the owner and noting that at such a dose there may be unwanted side effects which might include, at least, hair loss at the injection site. Injecting too large a volume of an oily solution at one site may be a cause for concern. This dose may also cause the recipient to come back into the rut and potentially lead to aggressive behaviour. Given the lack of evidence for optimal dosing, in some cases it may be very difficult to cause the shedding of persistently velveted antlers in castrated reindeer bulls using any hormonal therapy.

It should be noted that the removal of growing (velvet) antlers is illegal in the UK but velvet antlers can be removed (following suitable analgesia/anaesthesia) by a veterinarian on veterinary / welfare grounds.

While we believe that the advice given here offers our best understanding, colleagues following these guidelines should alert owners to the potential risks and uncertainties involved and do so at their own risk. The Veterinary Deer Society would be interested to hear from colleagues who have been involved in dealing with antlers of red deer and reindeer in these situations so that a collective view of effective practices can be built up.

Further information about some of the legal requirements that pertain to reindeer can be found in the APHA document: <http://ahvla.defra.gov.uk/documents/surveillance/diseases/reindeer.pdf>

For example, the movements of reindeer should be recorded under the AMLS and by the owner under the requirements of The Movement of Animals (Records) Order 1960. The transport of reindeer can raise some complexities given that regular appearances at a Christmas event may require repeated movements between the venue and temporary accommodation before returning to the home location.

Acknowledgements

Tracy Dewey for help with Figure 1; Nick Crabb for preparing Figure 2; James Barnett & APHA for the images of reindeer fibropapillomatous lesions; Gemma Thorpe and Pavel Scherer for images. Tilly Smith for her information about the Cairngorm Mountain reindeer herd.

References

AUDENAERDE, P.M.F. & SIMOENS, P.J.M. (2006) Fetal differentiation of the antler developing area in red deer (*C. elaphus*). In: Bartos, L., Dusek, A., Kotrba, R., Bartosova-Vichova, J. editors. Advances in Deer Biology. Prague; 2006. p.164 (Abstract).

BLAKE, J.E., ROWELL, J.E. & SUTTIE, J.M. (1998) Characteristics of first-antler growth in reindeer and their association with seasonal fluctuations in steroid and insulin-like growth factor 1 levels. *Canadian Journal of Zoology* **76**, 2096-2102.

BLAXTER, Sir K., KAY, R.N.B., SHARMAN, G.A.M., CUNNINGHAM, J.M.M., EADIE, J. & HAMILTON, W.J. (1988) Farming the Red Deer – the final report of an investigation by the Rowett Research Institute and

the Hill Farming Research Organisation published by Department of Agriculture and Fisheries for Scotland p.125.

BLAUDEL, G. (1935) Beobachtungen über die Entstehung der Perücke beim Rehbock [Observations on the origin of the Roe deer wig]. *Endokrinologie* **15**, 321-329.

BUBENIK, G.A., BUBENIK, A.B., STEVENS, E.D. & BINNINGTON, A.G. (1982) The effect of neurogenic stimulation on the development and growth of bony tissues. *Journal of Experimental Zoology* **219**, 205-216

BUBENIK, A.B. (1990a) Evolution of horns, pronghorns, and antlers. In 'Horns, pronghorns and antlers' Bubenik, G.A. and Bubenik, A.B. eds. Springer Verlag, New York, NY, USA. pp 79-81.

BUBENIK, G.A. (1990b) Neuroendocrine regulation of the antler cycle. In 'Horns, pronghorns and antlers' Bubenik, G.A. and Bubenik, A.B. eds. Springer Verlag, New York, NY, USA. pp 265-297

BUBENIK, G.A., SCHAMS, D., WHITE, R.J., ROWELL, J., BLAKE, J. & BARTOS, L. (1997) Seasonal levels of reproductive hormones and their relationship to the antler cycle of male and female reindeer (*Rangifer tarandus*). *Comparative Biochemistry and Physiology B Biochemistry and Molecular Biology* **116**, 269-277.

BUBENIK, G.A. (2001) Deer antler- a wonder of nature: structure and function of antlers, regulation of their development, and their potential in medicine, pp3-15 Proceedings of the Antler Science and Product Technology Symposium, held in Banff April 2000 Sim, J.S., Sunwoo, H.H., Hudson, R.J. & Jeon B.T. eds.

DAVIS, T.A. (1983) Antler asymmetry caused by limb amputation and geophysical forces. In: Brown, R.D. (editor), Antler development in *Cervidae*. Caesar Kleberg Wildlife Research Institute, Kingsville, TX, USA. pp. 223-230.

FLETCHER, J. (2013) Deer. Reaktion Books: London.

FLETCHER, T.J. & SHORT R.V. (1974) Restoration of libido in castrated red deer stag (*Cervus elaphus*) with oestradiol-17 β . *Nature* **248**, 616-618.

FOSTER, A.P., BARLOW, A.M., NASIR, L., WILSON, C.D., EVEREST, D.J., FINNEGAN, K. ERDELYI, C.J. & SCHOCK, A. (2013) Fibromatous lesions of antler velvet and haired skin in reindeer (*Rangifer tarandus*). *Veterinary Record* **172**, 452.

FOX, K.A., DIAMOND, B., SUN, F., CLAVIJO, A., SNEED, L., KITCHEN, D.N. & WOLFE, L.L. (2015) Testicular lesions and antler abnormalities in Colorado, USA mule deer (*Odocoileus hemionus*): a possible role for epizootic hemorrhagic disease virus. *Journal of Wildlife Diseases* **51**, 166-176.

GEIST, V. (1991) Bones of contention revisited: did antlers enlarge with sexual selection as a consequence of neonatal security strategies? *Applied Animal Behaviour Science* **29**, 453-469.

GOSS, R.J. (1968) Inhibition of growth and shedding of antlers by sex hormones. *Nature* **220** (5162), 83-85

HAMILTON, W.J., KYLE, D.J. & ROBSON, M.G. (1993) Disbudding of red deer stag calves to prevent antler growth. *Veterinary Record* **132**, 62-63.

HENSHAW, J. (1969) Antlers the bones of contention. *Nature* **224** (5223), 1036-1037.

KAY, R.N.B. & YOUNGSON, R.W. (1978) Perruque red deer stag. *Deer* **4**, 329.

KIERDORF, U., KIERDORF, H., SCHULTZ, M. & ROLF, H.J. (2004) Histological structure of antlers in castrated male fallow deer (*Dama dama*). *Anatomical Record Part A* **281**, 1352-1362.

LINCOLN, G.A. (1971a) The seasonal reproductive changes in the red deer stag (*Cervus elaphus*). *Journal of Zoology* **163**, 105-123.

LINCOLN, G.A. (1971b) Puberty in a seasonally breeding male, the red deer stag (*Cervus elaphus*). *Journal of Reproduction and Fertility* **25**, 41-54.

LINCOLN, G.A. & TYLER, N.J.C. (1992) Antler growth in male and female reindeer calves occurs in the absence of the gonads. In: The Biology of Deer, RD Brown editor; Springer New York. 493-498. http://dx.doi.org/10.1007/978-1-4612-2782-3_118

LINCOLN, G.A. & TYLER, N.J.C. (1994) Role of gonadal hormones in the regulation of the seasonal antler cycle in female reindeer, *Rangifer tarandus*. *Journal of Reproduction and Fertility* **101**, 129-138.

LINCOLN, G.A. & TYLER, N.J.C. (1999) Role of oestradiol in the regulation of the seasonal antler cycle in female reindeer, *Rangifer tarandus*. *Journal of Reproduction and Fertility* **115**, 167-174.

MARBURGER, R.G., ROBINSON, R.M., THOMAS, J.W., ANDREGG, M.J. & CLARK, K.A. (1972) Antler malformation produced by leg injury in white-tailed deer. *Journal of Wildlife Diseases* **8**, 311-314.

MOUNT, J.G., MUZYLA, M., ALLEN, S., OKUSHIMA, S., ALTHNAIAN, T., MCGONNELL, I.M. & PRICE, J.S. (2006) Antlers may regenerate from persistent neural crest like stem cells. In: Bartos, L., Dusek, A., Kotrba, R. & Bartosova-Vichova, J. editors. *Advances in Deer Biology*. Prague; 2006. p.161 (Abstract).

MUNK, B.A., GARRISON, E., CLEMONS, B. & KEEL, M.K. (2015) Antleroma in a free-ranging White-tailed deer (*Odocoileus virginianus*). *Veterinary Pathology* **52**, 213-216.

NAPP, J., WIESE, K.G., KIERDORF, U., KIERDORF, H., SEYMOUR, N., SCHLIEPHAKE, H. & ROLF, H.J. (2006) Stem cells isolated from the regenerating antler express key markers of the osteogenic lineage. In: Bartos, L., Dusek, A., Kotrba, R. & Bartosová-Vichová, J. editors. *Advances in Deer Biology: Deer in a changing world*. Prague: Research Institute of Animal Production. p162 (Abstract).

ROLF, H.J., KIERDORF, U., KIERDORF, H., SEYMOUR, N., NAPP, J., SCHLIEPHAKE, H. & WIESEL, K.G. (2006) Visualization and characterization of stem cells from the regenerating deer antler. In: Bartos, L., Dusek, A., Kotrba, R., Bartosová-Vichová, J., editors. *Advances in Deer Biology: Deer in a changing world*. Prague: Research Institute of Animal Production. p160 (Abstract).

Information about the Veterinary Deer Society may be found at: <http://www.vetdeersociety.com/>

Figure 1

This figure represents the seasonal pattern of antler growth in male and female reindeer in relation to their reproductive cycle.

Reproduced with permission - LINCOLN, G.A. & TYLER, N.J. (1994) Role of gonadal hormones in the regulation of the seasonal antler cycle in female reindeer, *Rangifer tarandus*. *Journal of Reproduction and Fertility* 101, 129-138. Bioscientifica. ISSN: 0022-4251.

Figure 2

The seasonal cycle of testosterone levels and antler growth in red deer. The cycle has been studied in depth and is better understood in red deer than in other species; it is thought that the role of testosterone in controlling antler cycles is universal across deer species with the exception of female reindeer in which oestrogen is the controlling hormone. The graph is based on studies performed in red deer (Lincoln 1971a,b).

Figure 3

The growth cycle of deer antlers (Powerpoint file)

Figure 4a

An example of Perruque in a castrated roe deer (Reproduced with permission from Pavel Scherer's book "Roe deer and their antlers (A stalker's lifetime experience)" English edition.

Figure 4b

Perruque in a red deer castrate which is an uncommon condition in this species (reproduced with permission of Gemma Thorpe)

Figure 5

Images of hyperplastic fibropapillomatous changes to velvet skin in castrated reindeer. Courtesy James Barnett and supplied to APHA