Effect of exogenous gonadotrophins on oestrus, the LH surge and the timing and rate of ovulation in red deer (Cervus elaphus)

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Red deer hinds (n = 38) were treated in the breeding season with five different gonadotrophin regimens to investigate the temporal relationship between oestrus, ovulation and the LH surge. All hinds were treated with progesterone-impregnated controlled internal drug release (CIDR) devices to synchronize oestrus. The five treatments were as follows: treatment 1, controls; treatments 2, 3 and 4, 1200 iu pregnant mares’ serum gonadotrophin (PMSG) was administered i.m. 72 h before CIDR device withdrawal (treatments 3 and 4 were also injected i.v. with 0.4 mg synthetic GnRH 12 or 18 h after CIDR device withdrawal, respectively); treatment 5, 200 iu PMSG was administered i.m. 72 h before CIDR device withdrawal and 0.5 iu FSH was administered in eight equal doses at intervals of 12 h starting from the time of PMSG injection. The hinds were run with crayon-harnessed stags to determine the time of oestrus onset. Blood samples were collected every 2 days for 26 days after CIDR device removal to determine concentrations of plasma progesterone and every 2 h for 72 h after CIDR device removal to determine plasma LH profiles. Laparoscopy for ovary examination was performed 6 or 12 h after oestrus onset and was repeated twice at intervals of 12 h. Final ovulation rate was determined on day 7 after CIDR device removal. All hinds received 500 µg cloprostenol i.m. on day 13. A total of 30 and 34 hinds exhibited oestrus and ovulation, respectively. Exogenous gonadotrophin administration advanced the onset of oestrus (21.1 ± 1.9 h versus 43.6 ± 2.6 h, P < 0.001) and ovulation (41.8 ± 3.1 h versus 71.3 ± 5.8 h, P < 0.001) and reduced the interval between the two events (19.1 ± 1.8 h versus 33.0 ± 0.0 h, P < 0.01). Treatment with CIDR devices alone resulted in one (n = 6) or two (n = 1) ovulation points. Exogenous gonadotrophins induced multiple ovulation points in most hinds. GnRH administration reduced the period over which multiple ovulations occurred to ≤12 h, whereas PMSG or PMSG and FSH induced ovulation over a period > 24 h. Treatment with exogenous gonadotrophins advanced the mean time to peak LH (17.1 ± 1.7 h versus 48.0 ± 3.1 h, P < 0.01) but had no effect on mean peak LH concentrations. Two hinds showed premature luteal regression. The administration of PGF₂α was effective in terminating luteal activity of multiple corpora lutea: progesterone concentration declined from 8.8 ± 1.4 to 0.6 ± 0.1 ng ml⁻¹ within 2 days of prostaglandin administration.

Introduction

The application of reproductive technology as a tool for conservation of endangered cervid species and introduction of diverse genetic material into captive populations has gained considerable popularity in the past decade. Most efforts have concentrated on propagation of male gametes by developing protocols for artificial insemination. Conception rates in the range of 50–70% are commonly achieved in red (Fennessy et al., 1990), fallow (Asher et al., 1988, 1992b; Mulley et al., 1988) and Eld’s deer (Monfort et al., 1993) following intravaginal insemination. Moreover, high fertility rates (70–80%) have been demonstrated in fallow deer (Jabbour et al., 1993) and white-tailed deer (Magyar et al., 1989) following cervical insemination.

Treatments to induce and increase ovulation rate have been applied to cervid species including red deer (Jabbour et al., 1990; Asher et al., 1992a), fallow deer (Jabbour et al., 1990), Pere David’s deer (Argo et al., 1994), white-tailed deer (Magyar et al., 1990) and chital deer (Mylera et al., 1991).
treatments comprise the administration of pregnant mares’ serum gonadotrophin (PMSG) or FSH. PMSG, a hormone with a long half-life (McIntosh et al., 1975), is usually administered in a single injection and this has resulted in more widespread application of the gonadotrophin in superovulation and embryo transfer programmes. However, as observed with domestic species, the use of superovulatory doses of PMSG alone results in ovarian overstimulation and a low recovery rate of oocytes (sheep: Jabbour and Evans, 1991a; fallow deer: Thompson and Asher, 1988). This has been attributed to a high concentration of oestrogen, which may result from a large number of follicles failing to rupture (Ryan et al., 1984; Jabbour and Evans, 1991a), a second wave of follicular growth stimulated after ovulation (Bouters et al., 1983) or an increase in oestrogen secretion by follicles following stimulation with PMSG (Jabbour and Evans, 1991a).

The following study was designed to investigate the effect of PMSG administered alone or in combination with FSH or GnRH on the endocrine changes and the timing, duration and rate of ovulation in adult parous red deer hinds during the breeding season. The sensitivity of multiple corpora lutea to prostaglandin treatment on day 13 of the oestrous cycle was also determined by monitoring progesterone profiles.

Materials and Methods

Animals and management

A total of 38 parous red deer hinds and four mature entire stags were used between March and April 1990 in the North Island of New Zealand (37° 46’S, 175° 20’E). The animals were always held in four separate groups (A–D, n = 9–10 hinds) each with a separate mature stag, from the time of commencement of treatments. Each group had animals representative of each of the five treatment regimens, which were balanced for liveweight (overall mean ± SEM of 89.38 ± 1.4 kg). Treatment schedules were staggered 2 days between consecutive groups to restrict the number of hinds undergoing laparoscopy on any one day. The deer were contained in high-fenced paddocks (2500 m²) and grazed on ryegrass–clover pastures. Meadow hay was provided ad libitum and occasional feeding of whole kernel maize was used to habituate the deer to their handlers.

Hormone administration

Single controlled internal drug release (CIDR) devices (type-G, 0.3 g progesterone per device, Agricultural Division, CHH Plastic Products Group Ltd, Hamilton) were inserted intravaginally for a total period of 14 days from 21–27 March until 4–10 April inclusive. The devices were replaced with a new one in each animal on day 10 (31 March–6 April inclusive) to ensure that progesterone concentration remained high throughout the CIDR device insertion period. Hinds undergoing treatment 1 (n = 8) served as controls, hinds subjected to treatments 2 (n = 7), 3 (n = 8) and 4 (n = 8) received a single i.m. injection of 1200 IU PMSG (Folligon: Intervet, Lane Cove, NSW) administered 72 h before CIDR device withdrawal. Hinds undergoing treatments 3 and 4 were also injected i.v. with 0.4 mg synthetic GnRH (Gonadorelin: Fertagyl, Intervet) 12 or 18 h after CIDR device withdrawal, respectively. Hinds undergoing treatment 5 (n = 7) received an i.m. injection of 200 IU PMSG 72 h before CIDR device withdrawal and 0.5 IU ovine FSH (Ovagen: Immunochemical Products Ltd, Auckland) administered i.m. in eight equal doses (0.0625 IU), starting at the time of PMSG administration and finishing 12 h after CIDR device withdrawal. All hinds were injected i.m. with 500 µg prostaglandin F₂α analogue (Cloprostenol: Imperial Chemical Industries, Cheshire) on day 13 after CIDR device withdrawal.

Detection of oestrus

Each of the stags was fitted for 4 days with a ram mating harness (Fergus: Merck, Sharpe and Dohme NZ Ltd, Auckland) that contained a red crayon. Observations for crayon mating marks were conducted every 2 h during the 72 h after CIDR device withdrawal by close inspection of each hind. Crayons were replaced every 6–12 h.

Collection of blood samples

A total of 18 hinds (groups A and D, n = 4 for treatments 1, 2 and 3 and n = 3 for treatments 4 and 5) were mated into a covered shed, individually restrained in a pneumatically controlled cradle and blood samples taken by jugular venepuncture (in 10 ml heparinized vacutainers) every 2–3 days from the time of CIDR device withdrawal until 26 days later. Intensive blood samples were also collected every 2 h for 72 h after CIDR device withdrawal from the right external jugular vein via indwelling catheters (Intrachoc 3122; Deseret Company, Sandy, UT) that had been inserted about 2–4 h before the start of blood sampling. Blood samples were also collected every 20 min for 4 h from hinds that were treated with GnRH. Restraint was not necessary for intensive blood samplings; blood was withdrawn while groups of hinds were held in 2.4 m³ pens after repeated mustering from nearby paddocks. Blood samples were centrifuged at 1000 g for 25 min immediately after collection and the plasma aliquots stored at −20°C until assayed.

Assessment of time, duration and rate of ovulation

Hinds within each treatment were allocated randomly for initial ovarian examination by laparoscopy 6 or 12 h after the time of onset of oestrus. Laparoscopy was repeated twice at intervals of 12 h and the numbers of corpora haemorrhagica were recorded at each inspection time. The duration of ovulation was taken to be the period in which total ovulation rate increased between consecutive laparoscopies, and was estimated to be > 24 h in animals in which ovulation rate on day 7 was greater than that observed 30 or 36 h after the onset of ovulation. Hinds that did not display oestrus were inspected 72 h after CIDR device withdrawal. The final ovarian response was determined in all hinds on day 7 after CIDR device withdrawal.

Laparoscopy was performed under sedation after injecting 1.0–2.0 mg xylazine hydrochloride kg⁻¹ liveweight (Rompun: Bayer, Leverkusen) i.m. A presuprimal clip and scrub was carried
Table 1. Incidence of oestrus and ovulation and its duration among red deer hinds

<table>
<thead>
<tr>
<th>Treatment</th>
<th>Incidence of oestrus</th>
<th>Incidence of ovulation</th>
<th>Duration of ovulation (h)</th>
<th>Number with one ovulation</th>
<th>Number with two ovulations</th>
<th>Number with &gt; 2 ovulations</th>
</tr>
</thead>
<tbody>
<tr>
<td>CIDR device alone</td>
<td>6/8</td>
<td>7/8</td>
<td>—</td>
<td>6</td>
<td>1</td>
<td>0</td>
</tr>
<tr>
<td>CIDR + PMSG</td>
<td>6/7</td>
<td>7/7</td>
<td>&gt; 24</td>
<td>1</td>
<td>0</td>
<td>6</td>
</tr>
<tr>
<td>CIDR + PMSG + GnRH (12 h)</td>
<td>6/8</td>
<td>8/8</td>
<td>≤ 12</td>
<td>1</td>
<td>0</td>
<td>7</td>
</tr>
<tr>
<td>CIDR + PMSG + GnRH (18 h)</td>
<td>5/8</td>
<td>6/8</td>
<td>≤ 12</td>
<td>1</td>
<td>1</td>
<td>4</td>
</tr>
<tr>
<td>CIDR + PMSG + FSH</td>
<td>7/7</td>
<td>6/7</td>
<td>&gt; 24</td>
<td>0</td>
<td>0</td>
<td>6</td>
</tr>
</tbody>
</table>

CIDR: controlled internal drug release; PMSG: pregnant mares’ serum gonadotrophin.

out in the midline anterior to the udder, and local anaesthetic (Lignocaine: 2% lignocaine hydrochloride; Delta Veterinary Laboratories, NSW) was administered at the insertion points of the trocars. Sedation was reversed by injecting 0.5 mg yohimbine hydrochloride kg⁻¹ live weight (Recervyl: Aspiring Animal Services, Wanaka) i.m. External puncture wounds were treated with topical antibiotic powder (Aureomycin: Cyanamid NZ Ltd, Auckland) and 10 ml of long-acting antibiotic preparation (Propen LA: Glaxo NZ Ltd, Auckland) was administered every 48 h until cessation of laparoscopy.

Hormone assays

Blood samples collected every 2–3 days were assayed for progesterone, and intensively collected blood samples were assayed for LH. Concentrations of progesterone were measured in plasma in duplicate by direct radioimmunoassay as described for red deer by Asher (1990). All of the plasma samples from an individual hind were included within a single assay. Samples with low, medium and high concentrations of progesterone were included as control samples at frequent intervals in each assay. The interassay coefficients of variation were 24.1% for the low control (mean, 0.5 ng ml⁻¹), 12.7% for the medium control (mean, 5.4 ng ml⁻¹) and 15.2% for the high control (mean, 10.8 ng ml⁻¹) samples. The intra-assay coefficients of variation were 6.8%, 7.4% and 4.9% for the three control samples, respectively. Sensitivity of the assay, defined as the first point that was significantly different from zero, was 0.10 ng ml⁻¹.

Plasma LH concentrations were determined in duplicate by a heterologous radioimmunoassay that was described for sheep by Scaramuzzi et al. (1970) and validated for red deer plasma by Kelly et al. (1982). The ovine pituitary LH preparation used for standards and iodinated tracer was NIH-LH-S11 (National Hormone and Pituitary Programme, University of Maryland, MD). The LH antibody, which had been raised in a rabbit using NIH-LH-S11 as the antigen, was used in the assay at a final dilution of 1:200 000. Crossreactivity with other proteins has been described by Kelly et al. (1982). All the samples from a single hind were included within the same assay. Samples with low, medium and high concentrations of cervine LH were included as control samples at regular intervals in each assay. The inter-assay coefficients of variation were 23.9% for the low control (mean, 1.0 ng ml⁻¹), 4.4% for the medium control (mean, 5.4 ng ml⁻¹) and 6.9% for the high control (mean 9.3 ng ml⁻¹). The intra-assay coefficients of variation were 19.0%, 7.5% and 4.3% for the same control samples, respectively. The sensitivity of the assay was 0.03 ng NIH-LH-S11 (0.30 ng ml⁻¹).

Statistical analyses

Data are presented as mean ± SEM within each treatment and were analysed by x² tests and analysis of variance. Mean LH profiles were obtained by normalizing the data about the time of the LH surge peak.

Results

Oestrus and ovulation

Of the 38 hinds in the trial, a total of 30 and 34 exhibited oestrus and ovulated, respectively (Table 1). There was no difference between treatments on the incidence of oestrus or ovulation. However, the administration of exogenous gonadotrophins compared with CIDR treatment alone advanced the time to onset of oestrus (21.1 ± 1.9 h versus 43.6 ± 2.6 h, P < 0.001) and ovulation (41.8 ± 3.1 h versus 71.3 ± 5.8 h, P < 0.001) and reduced the interval between the two events (19.1 ± 1.8 h versus 33.0 ± 0.0 h, P < 0.01, Table 2).

Treatment with CIDR devices alone resulted in one (n = 6) or two (n = 1) ovulation points. Treatment with exogenous gonadotrophins increased the total follicular response and numbers of corpora lutea (P < 0.01, Table 3). The majority of the hinds treated with PMSG with or without FSH had multiple corpora lutea (Table 1), with final ovulation rates ranging from 1 to 23, 1 to 17, 1 to 12 and 0 to 19 for hinds treated with PMSG, PMSG and GnRH at 12 h, PMSG and GnRH at 18 h, and PMSG and FSH, respectively. The administration of GnRH reduced the duration of multiple ovulation to ≤ 12 h, whereas hinds treated with PMSG or PMSG and FSH ovulated over a period > 24 h.

Endocrine response

Plasma LH profiles normalized around the LH peak for the various treatments are presented in Fig. 1. (One hind treated with CIDR devices alone that showed no increase in LH concentration and failed to exhibit oestrous behaviour or to ovulate was excluded from the analyses.) Treatment with exogenous gonadotrophins advanced the mean time to LH
peak (17.1 ± 1.7 h versus 48.0 ± 3.1 h, \( P < 0.01 \)), but had no effect on the mean peak LH concentrations. The overall mean peak LH concentration was 40.1 ± 4.6 ng ml\(^{-1}\). There was no apparent effect of GnRH administration on the mean time to LH peak. However, all hinds treated with GnRH 12 or 18 h after CIDR device withdrawal had LH peaks 14 or 20 h after CIDR device withdrawal, respectively (2 h after injection of GnRH). In contrast, the range in the timing of the LH peak for hinds treated with CIDR devices, PMSG or PMSG and FSH was 42–52 h, 10–12 h or 12–36 h after CIDR device withdrawal, respectively. The occurrence of oestrus varied in relation to the preovulatory LH surge, ranging from 38 h before to 24 h after the LH peak. The timing of ovulation ranged from 2 to 42 h after the LH peak.

The mean concentration of progesterone at the time of CIDR device withdrawal was 2.5 ± 0.2 ng ml\(^{-1}\). Luteal development was evident in all hinds after increasing the plasma concentration of progesterone starting on day 4 after CIDR device withdrawal. Peak concentration of progesterone for mono-ovulating hinds was 2.9 ± 1.3 ng ml\(^{-1}\). However, in hinds treated with exogenous gonadotrophins, there was a positive increase in progesterone concentrations with increasing numbers of corpora lutea (\( P < 0.05 \)). The overall peak progesterone concentration for superovulated hinds (treatments 2–5 inclusive) was 11.4 ± 2.1 ng ml\(^{-1}\). Two hinds treated with PMSG or PMSG and GnRh after 12 h showed premature luteal regression (following detection of three corpora lutea and multiple large follicles in each hind) as revealed by a decline in progesterone secretion after day 7 following removal of CIDR devices. Treatment with prostaglandin on day 13 of the oestrous cycle resulted in reduced plasma progesterone concentrations in hinds from all treatment groups (Fig. 2). Progesterone concentrations declined from a peak of 8.8 ± 1.4 to 0.6 ± 0.1 ng ml\(^{-1}\) within 2 days of prostaglandin administration.

**Discussion**

The average intervals between withdrawal of progesterone implants and oestrus or ovulation are similar to those reported previously for red deer (Asher et al., 1992a) and fallow deer (Jabbour et al., 1993). Treatment with CIDR devices alone was effective in inducing oestrus and ovulation in most of the hinds. This result is in contrast to a previous report in which oestrus and ovulation failure was observed in most (75%) of the hinds treated with CIDR devices alone (Asher et al., 1992a). This finding suggests subtle seasonal effects on oestrous synchrony in red deer hinds. The present study was conducted approximately 2 weeks later than that reported by Asher et al. (1992a). The administration of exogenous gonadotrophins significantly advanced the time to onset of oestrus and ovulation following withdrawal of CIDR devices (Thompson et al., 1990). This finding is similar to reports in superovulated sheep following oestrus synchronization with CIDR devices. PMSG enhances the recruitment of follicles and steroidogenesis (Jabbour and Evans, 1991a) by acting directly on the ovary. This presumably results in an earlier attainment than usual of threshold concen-

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**Table 2.** Mean (± SEM) times to onset of oestrus and ovulation in red deer from removal of CIDR devices and the mean interval between the two events

<table>
<thead>
<tr>
<th>Treatment</th>
<th>Time of onset of oestrus (h)</th>
<th>Time to first ovulation (h)</th>
<th>Interval from oestrus to ovulation (h)</th>
</tr>
</thead>
<tbody>
<tr>
<td>CIDR device alone</td>
<td>43.6 ± 2.6</td>
<td>71.3 ± 5.8</td>
<td>33.0 ± 0.0</td>
</tr>
<tr>
<td>CIDR + PMSG</td>
<td>16.6 ± 2.7</td>
<td>31.0 ± 5.7</td>
<td>14.0 ± 4.6</td>
</tr>
<tr>
<td>CIDR + PMSG + GnRH (12 h)</td>
<td>23.0 ± 4.7</td>
<td>53.0 ± 5.6</td>
<td>24.0 ± 2.7</td>
</tr>
<tr>
<td>CIDR + PMSG + GnRH (18 h)</td>
<td>21.6 ± 3.7</td>
<td>34.5 ± 3.8</td>
<td>15.0 ± 1.7</td>
</tr>
<tr>
<td>CIDR + PMSG + FSH</td>
<td>22.9 ± 4.2</td>
<td>46.3 ± 4.1</td>
<td>22.0 ± 2.9</td>
</tr>
</tbody>
</table>

CIDR: controlled internal drug release; PMSG: pregnant mares’ serum gonadotrophin.

**Table 3.** The ovarian response of red deer hinds following stimulation with CIDR devices and exogenous gonadotrophins

<table>
<thead>
<tr>
<th>Treatment</th>
<th>Total number of follicles</th>
<th>Number of large unruptured follicles</th>
<th>Number of corpora lutea</th>
</tr>
</thead>
<tbody>
<tr>
<td>CIDR device alone</td>
<td>1.4 ± 0.3</td>
<td>0.4 ± 0.3</td>
<td>1.0 ± 0.2</td>
</tr>
<tr>
<td>CIDR + PMSG</td>
<td>10.7 ± 4.1</td>
<td>2.3 ± 0.5</td>
<td>8.4 ± 2.7</td>
</tr>
<tr>
<td>CIDR + PMSG + GnRH (12 h)</td>
<td>9.1 ± 2.2</td>
<td>1.6 ± 0.3</td>
<td>7.5 ± 2.1</td>
</tr>
<tr>
<td>CIDR + PMSG + GnRH (18 h)</td>
<td>6.5 ± 1.3</td>
<td>2.4 ± 0.7</td>
<td>4.3 ± 1.5</td>
</tr>
<tr>
<td>CIDR + PMSG + FSH</td>
<td>12.7 ± 1.7</td>
<td>4.0 ± 1.1</td>
<td>8.7 ± 2.3</td>
</tr>
</tbody>
</table>

Values are means ± SEM.

CIDR: controlled internal drug release; PMSG: pregnant mares’ serum gonadotrophin.
Fig. 1. Profiles of mean (± SEM) plasma LH concentration normalized around LH peak in red deer hinds following treatment with (a) CIDR devices alone (n = 3), or with (b) 1200 IU pregnant mares’ serum gonadotrophin (PMSG) (n = 4); 1200 IU PMSG and GnRH administered (c) 12 h (n = 4) or (d) 18 h after CIDR device withdrawal (n = 3); (e) 200 IU PMSG and 0.5 IU ovine FSH (n = 3).

The use of exogenous gonadotrophins for superovulation has been attempted in a number of cervid species (e.g. fallow deer: Jabbour et al., 1990; red deer: Jabbour et al., 1990; Asher et al., 1992a; Argo et al., 1994; Père David’s deer: Argo et al., 1994). However, this has always resulted in disappointing recovery and fertilization rates of oocytes (Jabbour et al., 1990; Argo et al., 1994). This has been attributed to recruitment of multiple follicles over an extended period after withdrawal of CIDR devices. In this study, this contention is further supported by the observation that, in hinds treated with PMSG alone, the number of ovulations on day 7 was higher than after the last laparoscopy performed during the 72 h after CIDR device removal, which may result in excessive secretion of oestrogen. Argo et al. (1994) demonstrated in red deer and Père David’s deer that follicular development and consequently oestrogen secretion commenced before the termination of oestrous synchronization treatment. This may ultimately accelerate the transport of gametes through the reproductive tract. Embryos or ova arriving prematurely in the uterus will degenerate and be expelled promptly via the cervix (Whyman and Moor, 1980).

Alternatively, the extended duration of ovulation may contribute to the reduced fertilization rates observed in superovulated fallow deer (HN Jabbour, unpublished); red deer and Père David’s deer (Argo et al., 1994). This may necessitate a double insemination regime to ensure that viable spermatozoa are present when oocytes are shed from a second follicular wave. This hypothesis is supported by other studies in which
embryos were recovered at disparate stages of development following superovulation of red and Peré David’s deer (Argo et al., 1994). This problem may be overcome by the administration of GnRH 12 h after CIDR device withdrawal, which results in more synchronous timing of ovulation in a cohort of hinds and may also reduce the secretion of oestradiol, as has been demonstrated in sheep (Jabbour and Evans, 1991b).

The administration of GnRH had no effect on the peak LH concentration, suggesting that in intact hinds the positive feedback response of oestrogen on the hypothalamus–pituitary axis is an all-or-none response. Such a result is similar to reports that increasing doses of PMSG failed to increase LH secretion in sheep (Evans and Robinson, 1980). However, it contradicts reports that in ovariectomized fallow deer (Jabbour et al., 1992) an increase in the magnitude of the LH surge occurs with increasing doses of oestradiol benzoate administered by injection. In the study described here, treatment with GnRH resulted in more synchronous timing of the LH surge, which ultimately reduced the spread in the timing of ovulation. The timing of ovulation in relation to the LH peak was not constant in the different treatment groups and in one hind ovulation was observed as early as 2 h after the LH peak. This finding suggests that ovulation may result from a direct action of PMSG on the ovary (Cameron and Batt, 1991), and is supported by observations in fallow deer in which ovulation and premature luteinization of follicles in some females was induced in the complete absence of an LH surge (Jabbour et al., 1993).

The relationship between the number of corpora lutea and plasma progesterone concentrations during the luteal phase is in agreement with the findings of Kelly et al. (1982) and Asher et al. (1992a). This provides further evidence that luteal tissue is the principal source of progesterone during the oestrous cycle of red deer (Adam et al., 1985). The administration of prostaglandin on day 13 of the oestrous cycle was effective in reducing plasma progesterone concentration within 2 days. This result suggests effective demise of the corpora lutea in superovulated hinds. Return to oestrus was not monitored in this study. However, Asher et al. (1992a) reported that return to oestrus occurred over 7 days after prostaglandin administration to a group of superovulated hinds.

The reason for the occurrence of premature luteal regression is not clear. It may be related to excessive follicular stimulation or high circulating concentrations of oestrogen during the early luteal phase (Armstrong et al., 1982). In sheep, the incidence of premature luteal regression was associated with secondary peaks of oestrogen secretion after the LH surge (Jabbour and Evans, 1991a). The secondary oestrogen peaks may have induced a premature luteolytic process; whether this is a direct or indirect action is unclear. Wilbank (1966) and Schoonmaker et al. (1982) reported early regression of the corpus luteum in Hereford heifers and Rhesus monkeys, respectively, following exogenous injection of oestrogen shortly after ovulation. Schoonmaker et al. (1982) speculated that in the Rhesus monkey oestrogen may act on the hypothalamo–hypophyseal axis to inhibit LH secretion, thus denying the corpus luteum necessary luteotrophic support. However, it is possible that excessive amounts of oestrogen stimulate the secretion of luteolytic prostaglandin. Battye et al. (1988) reported that the administration of flunixin meglumine, an inhibitor of prostaglandin synthetase, prevented premature luteal regression in PMSG-treated goats.

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