Effect of deslorelin implants on follicular development, parturition and post-partum oestrus in the tammar wallaby (*Macropus eugenii*)

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**Abstract**

The effect of treatment with slow release implants containing the GnRH agonist, deslorelin, was investigated in female tammar wallabies. Pouch young were removed from 16 wallabies presumed to be carrying quiescent blastocysts. Eight received a 5 mg deslorelin implant and eight received a placebo implant. Animals were caught daily from day 25 to day 30 and their pouches inspected for newborn young and their urogenital sinus checked for a copulatory plug. Treatment with deslorelin did not affect reactivation of a dormant blastocyst and subsequent birth in 4/8 animals, but post-partum mating was inhibited in these animals. Five control and five treated animals were killed within 0–48 h post partum and their reproductive tracts analysed. At autopsy, all five control animals had large preovulatory follicles but only one deslorelin-treated animal showed signs of follicular development. These differences were also reflected in the weights of the lateral vaginae, with treated animals showing no evidence of oestrogenic stimulation. The remaining three control and three treated animals were monitored for approximately 2 years. The long-term contraceptive effects of a single 5 mg deslorelin implant lasted for just under one year. These results indicate that slow release deslorelin implants inhibit follicular development in the female tammar wallaby for extended periods of time and may have potential application in reproductive management of captive marsupials in the kangaroo family.


**Introduction**

In recent years there has been increasing attention given to the challenges of managing over-abundant marsupial populations in Australia, in particular the large macropods (kangaroos and wallabies) and the koala. Lethal control techniques, such as shooting and poisoning, are now facing increasing public opposition. Reducing population size over time by artificially reducing the fertility of individuals within the population (fertility control) has been proposed as an alternative method of population control (Bomford 1990). This has resulted in research and development of new technologies to inhibit marsupial reproduction (Nave et al. 2000, 2002).

Gonadotrophin releasing hormone (GnRH) agonist treatment has been shown to temporarily suppress ovarian cycles in dogs (Lacoste et al. 1989, Trigg et al. 2001), ewes (McNeilly & Fraser 1987), marmoset monkeys (Lunn et al. 1992), stump-tailed macaques (Fraser & Sandow 1985) and women (Fraser et al. 1990). In these species, suppression of ovarian cycles is characterised by an inhibition of follicular development, with a resultant decline in the concentrations of oestradiol and progesterone and an inhibition of oestrus. In ewes (McNeilly & Fraser 1987), heifers (Mattos et al. 2001) and women (Bider et al. 1989) ovarian follicular development is arrested at the early antral stages of development, as evidenced by the absence of large follicles in the ovaries. In addition, the luteinising hormone (LH) surge mechanism is inhibited, as is ovulation.

Treatment with long-acting implant formulations containing the GnRH agonist, deslorelin, has resulted in long-term inhibition of reproductive function in cats (Munson et al. 2001), dogs (Trigg et al. 2001), heifers (D’Occhio et al. 1996, 2000) and other wildlife species (Bertschinger et al. 2001, 2002). The longevity of these GnRH agonist formulations increases their potential application in the management of over-abundant wild and captive marsupial populations.

The tammar wallaby is well established as a model marsupial species for the study of reproduction and endocrinology (Tyndale-Biscoe & Renfree 1987, Tyndale-Biscoe &
The reproductive tract was dissected out and examined. Animals were randomly assigned to the treatment (\( n = 8 \)) or control group (\( n = 8 \)). Treated animals received a 5 mg deslorelin implant and control animals received a placebo implant. Animals were caught once every 7 days to accommodate the pouch young (PY) and/or mate were killed at the first sign of a copulatory plug. If they had not mated within 24 h of the first observation, they were presumed to be carrying a dormant blastocyst from a post-partum mating. Males were used for breeding purposes only and were not subject to any form of treatment. The Macquarie University Animal Ethics Committee approved all experimental work (approval number 99009) and the animal handling and husbandry was conducted in accordance with National Health and Medical Research Council of Australia guidelines (1990).

**Materials and Methods**

**Animals**

Sixteen female tammar wallabies were synchronised by RPY on 1 March 2000 (day 0). At the time of RPY the animals were randomly assigned to the treatment (\( n = 8 \)) or the control group (\( n = 8 \)). Treated animals received a 5 mg deslorelin implant and control animals received a placebo implant. Animals were caught once every 7 days to accommodate them to catching and handling procedures. From day 25 after RPY the animals were caught daily and their pouches examined for the presence of a copulatory plug, which usually remains in position for at least 24 h post coitum (Sutherland et al. 1980). Female tammars usually mate 1–2 h post-partum (Rudd 1994), but mating is sometimes delayed until 8 h (Tyndale-Biscoe et al. 1983) or even 18 h (Harder et al. 1985) after birth when animals are frequently sampled during the birth and post-partum period. The first five animals from each group to give birth and/or mate were killed at the first sign of a copulatory plug. If they had not mated within 24 h of the first observation of a neonate (i.e. within 24–48 h of birth), they were killed at this time.

**Sample collection**

Five animals from each group were killed by an intravenous injection of pentobarbitone sodium (1 ml/2 kg; Lethabarb, Virbac Pty, Ltd, Peakhurst, NSW, Australia). The reproductive tracts were dissected out and examined.
The ovaries were checked for the presence of follicles or recent ovulations, before weighing (Sartorius electronic analytical balance, Max = 110 g, d = 0.1 mg, 1601MP8) and fixing in 10% neutral buffered formalin. The ovaries were embedded in paraffin wax, serially sectioned at 7 μm and stained with haematoxylin and eosin for subsequent histological examination and confirmation of the gross observations at the time of post-mortem. The ovaries were embedded in paraffin wax, serially sectioned at 7 μm and stained with haematoxylin and eosin for subsequent histological examination and confirmation of the gross observations at the time of post-mortem. The uteri were examined for the presence of fetuses or evidence of recent pregnancy and then weighed individually. The lateral vaginae were dissected free of adjacent connective tissue and weighed to give an index of the animal’s oestrogenic status (Short et al. 1985).

**Long-term monitoring**

The remaining three control and three treated animals were monitored once every 4–8 weeks from April 2000 until June 2002 to determine the duration of the contraceptive effects and the reversibility of treatment. All PY were removed and their age determined by measuring the head length and calculating the age from tammar wallaby PY growth tables (Poole et al. 1991). Following the resumption of reproductive activity in two of the treated animals, all of the three previously treated animals were re-treated on 15 March 2001 with a double dose of deslorelin (10 mg deslorelin).

**GnRH agonist implant**

The GnRH agonist, deslorelin (D-Trp⁶-Pro⁹-des-gly¹⁰-GnRH ethylamide), was formulated into implants that contained 5 mg deslorelin (Batch DR027A; Peptech Animal Health Pty. Ltd, North Ryde, NSW, Australia) as previously described (Trigg et al. 2001). In a real-time dissolution system the release of deslorelin was >1 μg/day for periods of approximately 1 year (Trigg et al. 2001). The in vivo release rate in tammar wallabies has not been determined. Implants were placed s.c. between the shoulder blades using a single-use commercial implanting device sterilised by e-beam radiation. The injection site was then sealed with a veterinary tissue adhesive (Vetbond, 3M Animal Care Products, St Paul, MN, USA). The dimensions of a 5 mg deslorelin implant were 2.3 mm in width and 12.5 mm in length.

**Statistical analyses**

Comparisons of the weight of reproductive tract components were analysed using two-sample t-tests. Comparisons of the number of pouch young and copulatory plugs for each group were made using the crosstabs chi-square Fisher’s Exact test feature of SPSS 1996. Data are presented as arithmetic means ± S.E.M. Results were reported as significant at P = 0.05.

**Results**

**Timing of birth**

All control and treated females were synchronised by RPY at the time of implant administration, thus demonstrating their fertility at the start of the experiment. Following deslorelin treatment there was no significant difference in the number of animals giving birth from each group (χ² = 1.067, degrees of freedom (d.f.) = 1, P > 0.05). Six (out of eight) control animals gave birth between days 25 and 27, and four (out of eight) treated animals gave birth, all on day 27 (Table 1). In one treated animal (number 691) the newborn young was found dead in the pouch.

<table>
<thead>
<tr>
<th>Structures present in ovary</th>
<th>Birth</th>
<th>Copulatory plug</th>
<th>Post-mortem</th>
</tr>
</thead>
<tbody>
<tr>
<td>Control</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>692</td>
<td>Day 27</td>
<td>Day 27</td>
<td>Day 27</td>
</tr>
<tr>
<td>1BE0558T</td>
<td>Day 27</td>
<td>Day 27</td>
<td>Day 27</td>
</tr>
<tr>
<td>1E710E8T</td>
<td>Day 27</td>
<td>Day 27</td>
<td>Day 27</td>
</tr>
<tr>
<td>537</td>
<td>Day 27</td>
<td>Day 28</td>
<td>Day 28</td>
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<tr>
<td>548</td>
<td>Day 26</td>
<td>Day 26</td>
<td>Day 26</td>
</tr>
<tr>
<td>686</td>
<td>Day 27</td>
<td>No</td>
<td>NA</td>
</tr>
<tr>
<td>1E71BB2T</td>
<td>No</td>
<td>Day 30</td>
<td>NA</td>
</tr>
<tr>
<td>543</td>
<td>No</td>
<td>No</td>
<td>NA</td>
</tr>
<tr>
<td>Treated</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>683</td>
<td>Day 27</td>
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<td>Day 28</td>
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<td>532</td>
<td>No</td>
<td>Day 26</td>
<td>Day 26</td>
</tr>
<tr>
<td>689</td>
<td>No</td>
<td>No</td>
<td>NA</td>
</tr>
<tr>
<td>20166E3T</td>
<td>No</td>
<td>No</td>
<td>NA</td>
</tr>
<tr>
<td>542</td>
<td>No</td>
<td>No</td>
<td>NA</td>
</tr>
</tbody>
</table>

NA, not applicable; * young found dead in pouch.

Table 1 Incidence of birth and mating in control and deslorelin-treated female tammar wallabies between days 25 and 30 after removal of pouch young, and structures present in the ovaries at post-mortem.
Copulatory plugs were observed in a significantly greater number of control than treated females between day 25 and day 30 (control, 6/8; treated, 1/8; $\chi^2 = 6.349$, d.f. = 1, $P < 0.05$). One treated female mated on day 26, but no PY was observed in the pouch. Examination of the reproductive tract of this female suggested that she was recently gravid, as one uterus was significantly larger than the other. Of the six control animals that gave birth, copulatory plugs were observed in all but one animal. This animal was not killed and following RPY a second young was born 27 days later suggesting that a post-partum mating had occurred, but this was not evident from physical examination.

**Effects on the reproductive tract**

At the time of post-mortem there were marked gross morphological differences between the reproductive tracts of control and deslorelin-treated animals (Fig. 1a and b). This was also reflected in the weights of the various components of the reproductive tract. The vaginal complex (median and lateral vaginas) of control animals was significantly heavier than treated animals (control, 23.10 ± 3.27 g; treated, 7.28 ± 2.70 g; $P < 0.01$). There was a tendency for both uteri to be heavier in control than treated animals, but this difference was not significant (parturient uterus: control, 1.78 ± 0.41 g; treated, 1.04 ± 0.11 g; non-parturient uterus: control 1.04 ± 0.40 g; treated 0.35 ± 0.07 g; $P > 0.05$).

In all five control animals there was a regressing CL from the recent pregnancy in one ovary (Fig. 2a) and a large pre-ovulatory follicle (diameter $> 3.5$ mm) in the contralateral ovary (Fig. 2b). In contrast, deslorelin-treated animals showed a normal regressing CL in one ovary (Fig. 2c) and early antral stage follicles (Fig. 2d) in the second ovary. A large pre-ovulatory follicle was only observed in one deslorelin-treated animal (Fig. 2e and f), and this was the same animal that mated within the 24h preceding post-mortem on day 26. In the remaining four treated animals, there was no evidence of preovulatory follicles or recent ovulations in either ovary.

The ovaries of control animals tended to be heavier than the ovaries of treated animals. For the ovary with the regressing CL, this difference was not significant (control, 201 ± 37 mg; treated, 142 ± 17 mg; $P > 0.05$). For the contralateral ovary (the ovary associated with the non-parturient uterus) there was a significant difference when animal 532 was excluded on the basis that it did not respond to the treatment in the same fashion as the remainder of the population and was therefore an outlier. The ovary with the large preovulatory follicle in control animals was on average double the weight of treated animals without large follicles (control, 229 ± 41 mg; treated, 111 ± 22 mg; $P < 0.05$).

**Long-term reproductive success**

After the period of daily capture, the remaining six females were monitored at less regular intervals. One treated animal (number 689) gave birth early in May 2000 (Table 2), but did not give birth again before administration of a second deslorelin implant. The remaining two treated females gave birth early the following breeding season, 340 and 359 days after the implant was administered (Table 2). After treatment with a further 10 mg deslorelin, none of the treated animals gave birth for the

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**Figure 1** Ventral view of the reproductive tracts of (a) control animal no. 537 and (b) deslorelin-treated animal no. 691 within 0–48 h of birth and/or post-partum mating. In both animals the parturient uterus is on the right hand side of the photograph. Scale bar = 10 mm. Ov, ovary; Ut, uterus; LV, lateral vagina; MV, median vagina; Bl, bladder; UGS, urogenital sinus.
remainder of the 2001 breeding season or during the 2002 breeding season, a period of 464 days. Two of the control animals bred within 60 days of administration of a placebo implant (Table 2). The remaining animal died within 3 months of placebo implant administration and did not breed during this time.

**Discussion**

Treatment with the GnRH agonist, deslorelin, at the time of RPY did not appear to inhibit the reactivation of a quiescent blastocyst and subsequent birth, but successfully inhibited follicular development and post-partum oestrus in 4/5 animals. Moreover, these contraceptive effects can
be maintained for periods of approximately 1 year. This demonstrates the potential of deslorelin implants for controlling reproduction in tammar wallabies.

The inhibition of follicular development and oestrus during deslorelin treatment in the present study was consistent with findings from other species. Short-term treatment of heifers with deslorelin resulted in a decline in the numbers of class 2 (6 to 9 mm) and class 3 (>9 mm) follicles, although the number of class 1 (<6 mm) follicles was not affected (Mattos et al. 2001). Similarly, ewes treated with the GnRH agonist, buserelin, had no follicles >2.5 mm in diameter (McNeilly & Fraser 1987). In both species, inhibition of follicular development was accompanied by a decline in peripheral plasma follicle-stimulating hormone (FSH) concentrations and an absence of pulsatile release of LH, suggesting that gonadotrophin support is not required for the maintenance of smaller follicles, but is required for the development of larger follicles (McNeilly & Fraser 1987, Gong et al. 1996). Although the concentrations of FSH and LH were not measured in the present study, the absence of follicular development suggests that there was inadequate gonadotrophic support for normal follicular development. A similar attenuation in the growth of follicles was reported in female tammars following passive immunisation against GnRH (Short et al. 1985) and hypophysectomy (Hearn 1974, Panyaniti et al. 1985).

Studies in heifers have demonstrated that suppression of follicular growth was caused by decreased gonadotrophic support rather than a direct action of GnRH agonist on the ovaries (D’Occhio et al. 2000). In addition, no binding sites for GnRH or GnRH agonists have been identified in bovine ovarian tissues (Gong et al. 1996). The GnRH receptor transcript has recently been detected in ovarian tissue of tammar wallabies (Cheung & Hearn 2002). Therefore, it is possible that deslorelin is having a direct effect at the level of the ovaries as well as at the level of the pituitary, as occurs during GnRH agonist treatment in the rat (Janssens et al. 2000). It has been hypothesised that GnRH agonists may directly affect ovarian steroidogenesis in the rat via ovarian GnRH receptors which are part of a local autoregulatory system (Roth et al. 2001). The role of ovarian GnRH receptors in the tammar wallaby deserves further investigation.

In deslorelin-treated tammars, oestradiol concentrations would presumably have remained at basal levels, as is the case in heifers (Mattos et al. 2001) and mice (Bokser et al. 1989), as the preovulatory follicle is known to be the source of the high oestradiol concentrations coincident with behavioural oestrus following the pregnant and oestrous cycle (Harder et al. 1985). The inhibition of oestrus and the absence of an oestrogenic response in the vaginae further support this hypothesis.

Deslorelin treatment did not affect normal gestation or parturition in 60% (3/5) of animals. This is in accordance with the results of Short et al. (1985) who showed that passive immunisation against GnRH does not inhibit gestation or progesterone secretion after RPY. After hypophysectomy, pregnancies proceed to term but parturition does not occur (Hearn 1974), thereby demonstrating that gonadotrophin secretion is not required for successful luteal reactivation or gestation.

In the control group, parturition successfully occurred and live young were observed in the pouch of all five of the animals in which reproductive tracts were analysed. In the treated group, live young were observed in only three animals. Whether the loss of two out of five young in treated animals compared with none out of five in control animals is a direct result of deslorelin treatment requires further investigation. The three treated animals in which live young were observed successfully suckled these young for periods of between 24 and 48 h suggesting that the early phase of lactation was not affected.

The maintenance of follicular development and subsequent occurrence of oestrus in one treated animal demonstrates that there are individual differences in the response to deslorelin treatment, and highlights the possibility that an unknown proportion of the population may not be responsive to the contraceptive effects of deslorelin treatment. The occurrence of oestrus during deslorelin

### Table 2 Duration of contraception in long-term deslorelin-treated female tammar wallabies, compared with the reproductive success in control female tammars.

<table>
<thead>
<tr>
<th>Animal no.</th>
<th>Treatment</th>
<th>Treatment date</th>
<th>Date of birth</th>
<th>Days post-treatment</th>
</tr>
</thead>
<tbody>
<tr>
<td>689</td>
<td>Deslorelin (5 mg)</td>
<td>01-Mar-00</td>
<td>01-Mar-00</td>
<td>61</td>
</tr>
<tr>
<td></td>
<td>Deslorelin (10 mg)</td>
<td>15-Mar-01</td>
<td>—</td>
<td>&gt;464*</td>
</tr>
<tr>
<td>20166E3T</td>
<td>Deslorelin (5 mg)</td>
<td>01-Mar-00</td>
<td>04-Feb-01</td>
<td>340</td>
</tr>
<tr>
<td></td>
<td>Deslorelin (10 mg)</td>
<td>15-Mar-01</td>
<td>23-Feb-01</td>
<td>&gt;464*</td>
</tr>
<tr>
<td>542</td>
<td>Deslorelin (5 mg)</td>
<td>01-Mar-00</td>
<td>—</td>
<td>359</td>
</tr>
<tr>
<td></td>
<td>Deslorelin (10 mg)</td>
<td>15-Mar-01</td>
<td>23-Feb-01</td>
<td>&gt;464*</td>
</tr>
<tr>
<td>1E71BB2T</td>
<td>Placebo</td>
<td>01-Mar-00</td>
<td>28-Apr-00</td>
<td>58</td>
</tr>
<tr>
<td></td>
<td>Placebo</td>
<td>15-Mar-01</td>
<td>02-May-01</td>
<td>48</td>
</tr>
<tr>
<td>686</td>
<td>Placebo</td>
<td>01-Mar-00</td>
<td>28-Mar-00</td>
<td>27</td>
</tr>
<tr>
<td></td>
<td>Placebo</td>
<td>15-Mar-01</td>
<td>11-May-01</td>
<td>57</td>
</tr>
<tr>
<td>543</td>
<td>Placebo</td>
<td>01-Mar-00</td>
<td>—</td>
<td>b</td>
</tr>
<tr>
<td></td>
<td>Placebo</td>
<td>15-Mar-01</td>
<td>—</td>
<td>c</td>
</tr>
</tbody>
</table>

*Duration of contraception to date at the end of the 2002 breeding section; †, no births detected following treatment; ‡, animal died on 28 May 2001.
treatment has also been noted in individual heifers. Oestrus in these cows indicated that ovarian follicular growth and oestrogen production continued during deslorelin treatment. The higher levels of oestrogen were able to stimulate oestrus but were presumably insufficient to stimulate a preovulatory LH surge and ovulation, because there was no increase in plasma progesterone concentrations after oestrus (D’Occhio et al. 1996). It has been demonstrated that treated heifers will not respond to GnRH stimulus with an LH surge, which could explain this presumed lack of ovulation (Bergfeld et al. 1996). The current understanding is that cows treated with GnRH agonist can show some growth of ovarian follicles, but ovulation does not occur, either because follicle maturation is not complete or the gonadotroph cells in the pituitary become insensitive to natural GnRH (D’Occhio et al. 1996). In the present study, the ‘non-responder’ was killed before the time of ovulation so we do not know whether ovulation would have occurred normally. It is possible that although there was obviously sufficient gonadotrophic support for follicular development, the pituitary may have been desensitised to the extent that this animal would have failed to generate a preovulatory LH surge, resulting in a failure to ovulate. In a study by Lunn et al. (1992), one buserelin-treated marmoset monkey (out of six) appeared to continue to have ovarian cycles based on plasma oestradiol and progesterone concentrations. However, this animal failed to respond to a GnRH challenge 3 weeks after buserelin implant insertion, suggesting that the pituitary was desensitised.

The long-term effects of deslorelin treatment were monitored in the three remaining treated animals. One treated female (689) gave birth within 2 months of deslorelin administration, suggesting that follicular development and ovulation had not been inhibited in this animal. There are two possible explanations for the continued reproductive activity in this animal. The dose administered (5 mg) may have been insufficient to desensitise the pituitary and significantly reduce gonadotrophin secretion within one oestrous/pregnant cycle, or this individual may be resistant to the contraceptive effects of deslorelin treatment. After removal of the pouch young there was no evidence of reproductive activity in this female for 10 months. In addition, after administration of a higher dose the animal did not breed for a period of 16 months (experiment continuing at time of writing). This suggests that this animal is not resistant to the contraceptive effects of deslorelin, but there may have been insufficient time or dose for down-regulation to occur. Ineffective pituitary suppression has been suggested as a potential reason for higher conception rates in heifers treated with low doses of deslorelin (D’Occhio et al. 2000). The remaining two treated animals first bred approximately one year after treatment with 5 mg deslorelin. After administration of 10 mg deslorelin, none of the treated animals bred during the following 16 months. The use of higher doses of deslorelin has been associated with longer contraceptive duration in heifers (D’Occhio et al. 2000) and male dogs (Trigg et al. 2001). The results from this small tammar trial suggest that a similar dose–response relationship may exist for the tammar. However, these results should be viewed cautiously as the first and second dosages were from different batches of deslorelin implants and the long-term effects of previous treatment on the pituitary in tammar wallabies are unknown.

In summary, deslorelin implants were successful at inhibiting follicular development and mating in the tammar wallaby. The long-term contraceptive effects lasted for approximately one year. This demonstrates the potential of these implants to reversibly inhibit reproduction in macropodid marsupials. The successful reactivation of quiescent blastocysts and the probable maintenance of lactation following their birth may limit the usefulness of this contraceptive in the majority of macropodid species which mate post-partum. However, post-partum mating is not a feature of the eastern grey kangaroo, M. giganteus, or the koala, Phascolarctos cinereus, which are two of the species presenting widespread management challenges. Therefore, deslorelin may be an efficacious fertility control option for these species and further trials are warranted. The relationship between dose and duration of action should be investigated in an attempt to gain longer contraceptive duration. Trials are also required in a larger group of animals to gain a more accurate estimation of the contraceptive success rate in the tammar wallaby, and the basis for any non-response.

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