55. PENILE DEVELOPMENT IN TAMMAR WALLABIES: A CONTINUING ENIGMA

Michael W. Leihy, Geoffrey Shaw, Marilyn B. Renfree and Jean D. Wilson
Department of Zoology, University of Melbourne, Victoria 3010

Androgens from the developing testes induce differentiation of the phallus in all mammals. In keeping with this concept, administration of androgen to (1) or transplantation of testes into (2) female tammar wallaby pouch young (PY) causes development of a male phallus. Phallic development in tammar males begins relatively late (3) and at a time (after day 60 of pouch life) when there is no sexual dimorphism in levels of plasma androgens (4). To address this dichotomy, we performed two studies. To determine if the late onset of phallic development is due to inactivation of testosterone (by conversion to androstenedione) at earlier stages, we compared the effects of methyltestosterone enanthate (MTE) (which cannot be oxidized to methylandrostenedione) and testosterone enanthate (TE) in female PY beginning at day 20. MTE did not accelerate phallic development whereas TE accelerated phallic growth, suggesting that the delay in male phallic development is due to limiting levels of endogenous androgens. Secondly, to address the fact that levels of androgens (testosterone (T), dihydrotestosterone (D), and androstanediol (A)) are not different in male and female PY between days 60 and 150, we considered the possibility that these hormones are released in a pulsatile fashion missed on routine sampling. Administration of a GnRH analogue to day-104 PY caused only a 55% increase in plasma T with no change in D or A, and we found no evidence of diurnal or pulsatile secretion (levels of T, D, and A averaged 58, 17, and 8 ng/dL) in day 90-115 male PY bled hourly around the clock. In summary, the mechanism by which androgens virilize the phallus is unclear. Possible explanations include the presence of some unidentified androgen in plasma (such as a conjugate), or action of unbound testosterone (circumventing the absence of a high affinity transport protein in this species) that is difficult to measure with available techniques.

(1) Leihy *et al*. (2003) *Endocrinology* **143**: 2643–51. (2) Tyndale-Biscoe and Hinds (1989) *Reprod. Fertil. Dev.* **1**: 243–54. (3) Butler *et al*. (1999) *Anat. Embryol*. **199**: 451–457. (4) Wilson et al. (1999) *Biol. Reprod*. **61**: 471–475.

10.1071/SRB03Ab55