## 68. SOURCE OF INHIBIN IN THE TAMMAR WALLABY, MACROPUS EUGENII

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The relationship between the gonadal peptide inhibin and other hormones is well understood in both sexes of eutherian mammals. Inhibin has been barely investigated in marsupials: the inhibin α-subunit gene has been characterised and its expression studied in possums [1], and the first account of circulating inhibin in male wallabies is reported in these Proceedings [2]. This study reports the results of an experiment examining the source of inhibin in tissues and fluids from adult female wallabies at post mortem. Tissues were snap frozen, homogenised in buffer to produce an extract assayed for total inhibin [3] ± 6% PEG and antisera to determine antibody specific binding. Inhibin is present in a wide range of tissues. Poor inhibin binding was observed in neural tissues, except the pituitary. Most body tissues, e.g. muscle, spleen, heart, bladder, were all negative for inhibin. High inhibin levels were seen in adrenal tissue sampled during gestation and lactation but whether this was from adrenal medulla or cortex was not determined. Median vagina, cervix, endometrium and myometrium had no inhibin. Oviductal tissue was positive, but levels were not as high as ovarian tissue. Ovarian tissue was divided into component parts (corpus lutea and follicles of different ages, interstitial and ovarian tissue excluding the CL or large follicle) and the inhibin levels were ranked: gestating ovary > CL  $\ge$  follicle >follicle drained of fluid > old CL > CL in lactation > corpus albicans > active CL. Follicular fluid (wFF) is the highest source of inhibin. Both allantoic and yolk-sac fluid contained inhibin, presumably from a fetal source. Tissues in the female wallaby produced inhibin, predominantly from ovarian tissue, with the levels varying as the pregnant cycle progresses. These results are consistent with the tissue distribution of inhibin in the possum [1]; however, the form inhibin takes in different tissues requires

(1) Vanmontfort, D., et al. (1998) J. Mol. Endocrinol. 21: 141–152; (2) Borchers, C.E., et al. (2003) Reprod. Fertil. Dev. Suppl. 15: Abs. 67; (3) Robertson, D.M., et al. (1988) Mol. Cell. Endocrinol. 58: 1–8.