14. EFFECTS OF A HIGH MATERNAL PHYTOESTROGEN DIET ON THE REPRODUCTIVE DEVELOPMENT OF MALE OFFSPRING

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Environmental oestrogens have been implicated in the reported decline in human sperm counts [1,2]. Administration of oestrogen during the neonatal period has been shown to reduce Sertoli cell numbers [3]. This project investigated the effects of a high maternal phytoestrogen (PO) diet during pregnancy and lactation on the reproductive tract of their male offspring. Three treatment groups were used: LP, male animals born to mothers that had received low PO diet (112 μ g/g isoflavanoid) and weaned on to a low PO diet; HP, male animals whose mothers were transferred to a high PO diet (465 $\mu g/g$ isoflavanoid) at the time of mating and weaned on to a high PO diet; HLP, male animals whose mothers were transferred to a high PO diet at the time of mating and then weaned onto a low PO diet. Groups of male rats (n = 8) were killed at 18 days and 16 weeks (adult) postpartum. One testis from each animal was fixed in Bouin's fluid and the other frozen. Blood was collected for hormone assays. Sertoli cell and germ cell numbers were counted using the optical disector method [4]. At 18 days postpartum, the rats exposed to the high PO diet had significantly fewer Sertoli cells (P<0.001), and higher plasma FSH concentrations (P < 0.005) than the LP rats. Sertoli cell (P < 0.01) and total germ cell (P < 0.01) numbers were significantly reduced in the HP and HLP adult rats. Returning the rats to a low PO diet (HLP) at weaning (21 days postpartum) did not increase Sertoli or germ cell number. No changes in plasma FSH. LH or testosterone or testicular levels of testosterone or dihydrotestosterone were seen between the adult rats. Within the testis each Sertoli cell can only support the development of a finite number of germ cells. These data suggest that a high PO diet during pregnancy and lactation decrease Sertoli cell number and the potential sperm production of male offspring.

(1) Carlsen E et al. (1992) BMJ **305**, 609–613. (2) Sharpe RM & Skakkebaek NE (1993) Lancet **341**, 1392–1395. (3) Atannossova N et al. (1999) Endocrinol. **140**, 5364–5373. (4) Wreford NG (1995) Micros. Res. Tech. **32**, 423–436.