FSH REGULATES SERTOLI CELL AND SPERMATOGONIAL POPULATIONS IN THE ADULT DJUNGRARIAN HAMSTER

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The hormones that regulate spermatogonial (Sg) development are ill defined; in part owing to lack of appropriate Sg-enriched experimental models. The photo-inhibited hamster model provides a rich source of Sg, thus making it an ideal model to study their control. This study aimed to assess the effects of FSH and testosterone on the re-initiation of Sertoli cell and Sg development in the gonadotrophin-deplete Djungarian hamster, as induced by photo-inhibition. Long day (LD) photoperiod (16L:8D) adult hamsters were exposed to a short day (SD) photoperiod (8L:16D) for 11 weeks to suppress gonadotrophins, resulting in a Sg only testis. Animals then received FSH alone or in combination with either testosterone or the anti-androgen, flutamide, for 7 days. Another group received testosterone alone. Bouin’s fixed testes embedded in resin were used for the determination of Sertoli and early germ cell number using the optical disector stereological technique. The number of Sertoli cells, type A Sg, type B Sg/preleptotene spermatocytes (S’cytes) and leptotene/zygotene S’cytes were suppressed in SD controls, to 66%, 34%, 19% and 10% (all \(P<0.01\)) of LD control values, respectively. Later germ cell types were not observed. FSH treatment, in the absence/presence of testosterone increased Sertoli cell number (\(P<0.01\)) to normal LD values. Similarly, FSH treatment in the absence/presence of testosterone increased type A Sg, type B Sg/preleptotene S’cytes and leptotene/zygotene S’cytes to ~85%, 69% and 80% (all \(P<0.001\)) of LD controls, respectively. Testosterone alone did not affect Sertoli and germ cell numbers and remained at SD controls values. These data demonstrate that the re-initiation of Sg is dependent on FSH, with testosterone playing no role. Surprisingly the adult Sertoli cell population in this model is hormonally dependent. This naturally occurring model now provides an extraordinary opportunity to understand the mechanism (apoptotic and or proliferative) by which FSH regulates Sertoli and germ cell development.

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