1. MODELLING CELL JUNCTIONS IN THE TESTIS

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Spermatogenesis is critically dependent on the pituitary hormones FSH and LH, and on androgens produced in the testis in response to LH. However, little is known about the molecular endpoints of hormone action in the testis. Our data from rat and human models suggests that FSH and testosterone (T) regulate the formation and maintenance of cell junctions in the testis at three key sites, these being at i) the inter-Sertoli cell junctional complex, ii) between Sertoli cells and step 8 round spermatids, and iii) between Sertoli cells and step 19 elongate spermatids. These junctions are essential for maintaining the blood–testis barrier and sperm production, and are potential targets for hormonal-based contraception in men. The inter-Sertoli cell junctional complex consists of numerous junction types. We have looked at the regulation of tight junctions (TJ), adherens junctions (AJ), and the testis-specific ectoplasmic specialisation junction (ES) in vitro and in vivo, as these junctions contribute to the formation and maintenance of the blood–testis barrier. In the absence of hormones, immature AJ and TJ form, but FSH and T are respectively required for these junctions to resemble their mature in vivo phenotypes. In contrast, ES junctions do not form in the absence of hormones, but require FSH stimulation. Another site of hormone action is at the Sertoli cell–round spermatid interface. Previous data have demonstrated that this junction is regulated primarily by T in vivo. In order to identify T-regulated genes involved at junctions between these cells, we have employed laser-capture microdissection to isolate enriched populations of round spermatids and analysed these by differential display and real-time RT-PCR. A number of genes have been shown to be T-regulated, and their role(s) in junction maintenance will be discussed. The third site of action of both FSH and T is in controlling the junctions involved in the final release of mature elongate spermatids from Sertoli cells. A reduction in circulatory FSH and T levels prevents the release of sperm, which we propose is due to a change in the adhesive properties of Sertoli cell junctions at this site. This presentation will demonstrate that a variety of in vivo and in vitro models coupled with new techniques (laser-capture microdissection) and sensitive detection systems (differential display PCR, confocal microscopy) can be successfully used to delineate the hormonal regulation of cell junctions in the testis.

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