Journal of Andrology, Vol 19, Issue 3 266-288, Copyright © 1998 by The American Society of Andrology

JOURNAL ARTICLE

# Vitamin E deficiency causes incomplete spermatogenesis and affects the structural differentiation of epithelial cells of the epididymis in the rat

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The effects of vitamin E deficiency on the rat testis and epididymis were examined in a light- and electron-microscopic analysis. Various groups of animals were made vitamin E-deficient, beginning at postnatal day 10, via their lactating mothers, until day 21, when they were separated from their mothers. The groups were maintained thereafter on either a vitamin E-deficient or a normal diet (controls). The vitamin E-deficient animals of group A, sacrificed at day 42, revealed testes that were normal in appearance, with a full complement of germ cells when compared to their controls (group B). Group C, however, sacrificed at day 48, revealed major abnormalities in the testes, unlike both their controls (group D) and normal, untreated animals (group E). Spermatogenesis was incomplete; the most advanced cell type was predominantly step-7 spermatids. However, many of these cells, as well as earlier spermatids, appeared to undergo degeneration, evidenced by large pale areas in their nuclei, disrupted acrosomes, and a cytoplasm with uncharacteristic organelles. Multinucleated cells, characterized by their chromatoid bodies as spermatids, were often seen in the seminiferous tubule lumen. Sertoli cells were normal in appearance, except for numerous, large lipid droplets in their basal region, at stages I-VIII; in appropriate controls (group D), such droplets were absent at these stages. These lipid inclusions presumably represented the final breakdown products of the late spermatids, which were phagocytosed by Sertoli cells between days 42 and 48. However, numerous germ cells, often recognized as round spermatids, and multinucleated cells were noted in the epididymal lumen, which indicates that such cells were spared from Sertoli cell phagocytosis. These data suggest that vitamin E plays a key role in the maintenance and survival of spermatids. In the epididymis, vitamin E deficiency resulted in principal, narrow, and apical cells that showed a poorly developed secretory and endocytic apparatus at days 42 (group A) and 48 (group C), unlike those of normal, untreated animals (group E). On the other hand, clear cells of groups A and C showed a highly developed endocytic apparatus in the cauda region only, whereas in the caput and corpus regions, endocytic apparatuses were small and undifferentiated, unlike those of group E. Thus, in the epididymis, vitamin E plays a role in the structural differentiation of principal cells along the entire epididymis, whereas, in the case of clear cells, its role is region-specific. Readministration of vitamin E to the diet restored a normal appearance to both the testis and the

epididymis, which indicates that the effects on these tissues are reversible. Taken together, these data indicate that vitamin E plays important roles in maintaining the viability of the spermatid population and in allowing epithelial epididymal cells to acquire their fully differentiated structural appearance.

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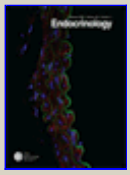

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