Journal of Andrology, Vol 11, Issue 4 344-352, Copyright © 1990 by The American Society of Andrology

JOURNAL ARTICLE

Does ethane 1,2-dimethanesulphonate (EDS) have a direct cytotoxic effect on the seminiferous epithelium of the rat testis?

R. L. Sprando, R. Santulli, C. A. Awoniyi, L. L. Ewing and B. R. Zirkin
Department of Population Dynamics, Johns Hopkins University, Baltimore, Maryland 21205.

The authors examined the possibility that ethane 1,2-dimethanesulphonate (EDS) has a cytotoxic effect on spermatogenesis that is not secondary to androgen withdrawal resulting from the well known cytotoxic effect of EDS on Leydig cells. Adult male rats were implanted with polydimethylsiloxane (PDS) capsules containing testosterone (T) and estradiol (E), and were simultaneously injected with EDS. The PDS-TE implants, by inhibiting luteinizing hormone (LH) production, prevented Leydig cells from repopulating the testis and clamped testosterone within the seminiferous tubules at increasing concentrations relative to implant size. In rats that received EDS alone, the number of advanced spermatids per testis was significantly reduced by 2 weeks, but within 8 weeks returned to the numbers maintained in vehicle-injected control rats or in vehicle-injected rats that received testosterone- and estradiol-filled capsules of 24 cm and 0.1 cm, respectively (PDS-24TE). Surprisingly, in rats that received an EDS injection plus PDS-24TE implants, the number of advanced spermatids per testis was significantly reduced at 8 weeks and severe seminiferous tubule atrophy occurred despite the fact that the testosterone concentration was sufficient to quantitatively maintain spermatogenesis in vehicle-injected rats. In rats injected with EDS and implanted with 24 cm testosterone but not estradiol-filled capsules (PDS-24T), the advanced spermatid number per testis was significantly higher than that in the EDS plus PDS-24TE rats, but significantly lower than that in control rats. These results suggest that EDS may have a cytotoxic effect on the seminiferous epithelium that is independent of the elimination of Leydig cells, and the EDS and estradiol act synergistically to exert a profound toxic effect on spermatogenesis.

This article has been cited by other articles:

Biol Reprod, March 1, 2004; 70(3): 600 - 607.
[Abstract] [Full Text] [PDF]

BIOLOGY *of* REPRODUCTION HOME

G. Rothschild, C. M. Sottas, H. Kissel, V. Agosti, K. Manova, M. P. Hardy, and P. Besmer
A Role for Kit Receptor Signaling in Leydig Cell Steroidogenesis
Biol Reprod, September 1, 2003; 69(3): 925 - 932.
[Abstract] [Full Text] [PDF]