Zeitschrift: Schweizer Archiv für Tierheilkunde SAT : die Fachzeitschrift für Tierärztinnen und Tierärzte = Archives Suisses de Médecine Vétérinaire ASMV : la revue professionnelle des vétérinaires

Herausgeber: Gesellschaft Schweizer Tierärztinnen und Tierärzte

Band: 132 (1990)

Heft: 8

Artikel: Study on the testis selenoproteins and the effects of selenium deficiency on testicular morphology

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DOI: https://doi.org/10.5169/seals-592835

SCHWEIZ. ARCH. TIERHEILK. 132, 409–484, 1990

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STUDY ON THE TESTIS SELENOPROTEINS AND THE EFFECTS OF SELENIUM DEFICIENCY ON TESTICULAR MORPHOLOGY

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It has been known for some time that selenium (Se) is involved in male reproductive processes. Rats which had been fed a Se-deficient diet produced sperm with impaired motility and characteristic midpiece damage (1). These effects were, however, only observed in animals after long depletion periods, whereas in the first phases of insufficient Se intake the Se level in the male gonads appears to be maintained by regulatory mechanisms (2). In order to obtain more information about the role of the element in the male reproductive system, the testicular selenium metabolism and the effects of Se deficiency on testicular functions were investigated at different stages of Se depletion.

For these studies rats were fed for four generations either a Se-deficient diet with a content of 2 µg Se/kg or the same diet with 300 µg Se/kg added in the form of sodium selenite. As the males were infertile from the 2nd generation onwards, the females were mated with males which had been fed a normal commercially available rat diet.

During the first months of Se depletion the testis Se content declined only relatively slightly and accordingly no changes in the morphology of the male gonads were found. From the 2nd generation onwards, however, the testis weight (expressed as % of the body weight) decreased and in the 4th generation it was only about 40% of the adequately supplied controls. The patho-morphological examination revealed a severe bilateral atrophy of the testes with a considerable decrease in the diameter of the seminiferous tubules. In the deficient animals of the 4th generation, the mean tubule diameter was 123.5 µm compared with 258.1 µm in the controls.

The degenerative process involved the whole testes uniformly. The seminiferous tubules were almost entirely lined by Sertoli cells or Sertoli cells and a few system cells or spermatogonia which did not show mitotic activity. The basement membranes were thickened and hyaline. A few seminiferous tubules showed variable degrees of mineralization or osseous metaplasia. Peritubular connective tissue was slightly increased and showed a marked edema with very few focal infiltrates of inflammatory cells. The Leydig cells showed a distinct hyperplasia. The atrophic seminiferous tubules intermingled with a few tubules showing incomplete spermatogenic activity with differentiation proceeding only to the spermatocyte stage. Differentiated spermatozoa could not be detected either in the seminiferous tubules or in the epididymis. The infertility of the animals was verified in a fertilization test.

Spermatogenesis could, however, be restored by feeding a Se-adequate diet for 4 months. The diameter of the seminiferous tubules then increased again to a mean value of 247.7 µm. Differentiated spermatozoa developed as is to be expected in the case of undisturbed spermiogenesis, and, compared with the Se-depleted animals, the number of Leydig cells had decreased.

The findings suggest the development of a compensatory hyperplasia of Leydig cells in the Se-deprived rats to counteract testosterone deficiency. This is in accordance with the results of experiments in which LHRH or LH in the form of human chorionic gonadotropin was administrered to rats of the 1st generation which had been fed the deficient diet for 6 months. In both cases the increase in the serum testosterone level 2 hours after the stimulation was significantly lower in the Se-deficient animals than in the controls, which indicates an effect of Se-deficiency on the steroidogenesis in the Leydig cells.

In order to find out which selenoproteins are present in the male gonads and should therefore be considered in the study of the testicular functions of the element, the tissue proteins were separated by means of SDS-PAGE after in vivo long-term labeling of the animals with 75 Se-selenite. In this way 12 Se-containing proteins or protein subunits were detected, with molecular weights of 12.1, 15.6, 18.0, 19.7, 22.2, 23.7, 33.3, 55.5, 59.9, 64.9, 70.1 and 75.4 kDa. The 23.7 kDa protein is the subunit of glutathione peroxidase, the only selenoprotein so far known to have biological functions in animals. In the spermatozoa, besides a weakly labeled protein (33.3 kDa) a major selenoprotein (19.7 kDa) was found, which is most probably identical to a Se compound for which in a previous study a structural function in the membranes of the sperm mitochondria was suggested (3). With inadequate Se intake the element was preferentially incorporated into the 19.7 kDa selenoprotein in the testis and spermatozoa. This indicates that the homeostatic mechanisms for the regulation of the testis Se level (2) mainly serve to ensure the formation of this compound.

The findings of the study show that the male reproductive system is severely affected by Se deficiency and that the element is necessary for the biosynthesis of testosterone and the formation and normal development of the spermatozoa.

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