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VITAMIN A DEFICIENCY AND ITS EFFECT ON THE SEXUAL ORGANS OF THE BOAR

By
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For a number of years the problems of sterility and reduced fertility have been the object of detailed investigations and studies. To some extent it has proved possible to group these afflictions according to whether they were infectious, anatomical or physiological in origin. Insufficient feeding is, without doubt, a very frequent cause of infertility as it includes lack of proteins, mineral substances and vitamins.

When it had been demonstrated that vitamin A was of decisive significance in the fertility of laboratory animals, rats and guinea pigs (*Wolbach and Howe,* 1925, 1928), the importance of the vitamin for the normal function of testis was investigated in farm animals including bulls and rams. (*Erb et al.* 1947, *Bratton et al.* 1948, *Lindley et al.* 1949). The literature on the subject however, does not seem to contain any information on the effect of vitamin A deficiency on the sexual organs of the boar.

In earlier investigations on vitamin A requirement, on its utilization and on deficiency symptoms in pigs (*Hjarde et al.* 1961), including the teratogenic effect of vitamin A deficiency (*Palludan* 1961), emphasis was placed on the effect of the lack of this vitamin on the ability to the sow to reproduce. However changes in the male sexual organs were also noted as in young boars deficient in vitamin A no spermiogenesis could be demonstrated.

The following investigations were made to further elucidate the effect of vitamin A deficiency on the sexual organs of the boar.

AUTHOR'S INVESTIGATIONS

The experiment included seven boars of Danish Land Race from two litters; numbers 80, 81, 86, 87 and 88 from one litter and numbers 89 and 90 from the other. From the age of ten to twelve weeks the boar piglets were fed with a practically vitamin A free diet (Table 1). As by far the largest part of vitamin A is

Table 1. Rations of experimental boars.

Body weight kg.	Barley kg.	Dried skim milk kg.	Daily ration Minerals, g.[1])
15—20	0.7	0.25	10
20—25	0.8	0.27	10
25—30	0.9	0.30	10
30—35	1.0	0.33	10
35—40	1.1	0.33	10
40—45	1.2	0.33	13
45—50	1.3	0.33	13
50—55	1.5	0.33	15
55—60	1.6	0.33	15
60—65	1.8	0.30	18
65—70	1.9	0.30	18
70—75	2.1	0.27	20
75—80	2.3	0.25	20
80—85	2.5	0.23	20
85—	2.2	0.23	20

[1]) 50 % dicalcium phosphate, 30 % calcium carbonate, 18 % sodium chloride, 1.9 % ferrosulphate, 0.1 % cupric sulfate. The boars were given 400 i. u. vitamin D perorally per day.

deposited in the liver, liver biopsies were made in accordance with earlier experiments during the entire experimental period in order to control the A vitamin status (Table 2).

The boars, No. 80 and 88, were each given 0.4 million international units of vitamin A at the age of 5 months and further 1.6

Table 2.

Boar No.	Age Months	I. u. vitamin A Per g. liver
81	5	42
86	5	34
87	6	22
89	3½	57
90	4½	12.5

and 0.8 million international units respectively at the age of 9 months. Boar No. 89 received 0.8 million international units at the age of 7½ months. Boar No. 86, after the first testis biopsy, which will be described later, was given 1.6 million international units of vitamin A. The other boars, 81, 87 and 90, received no vitamin A whatsoever.

Throughout the experimental period the growth rate of all the boars was the same and the characteristic symptoms of vitamin A deficiency were practically non-apparent. During the last months of the experiment the hairy coating of the boars with vitamin A deficiency became less glossy and the skin, to a greater or lesser extent, was covered with brownish, somewhat greasy crusts which could be removed without damage to the skin. These crusts were most pronounced near the eyes and at the openings of the metacarpal glands. At the age of 10 months boar No. 87 immediately prior to feeding had several general convulsive attacks, but after these had passed the pig ate one half of the ration given. These attacks recurred the same afternoon and also the following morning; between these attacks the boar had an uncertain lurching gait and was therefore killed the following morning. Boar No. 90 showed symptoms of pneumonia at the age of 7 months but was apparently cured by universal antibiotical therapy. However in the ensuing months the boar was subjected to recurrent pneumonia and in two instances to diarrhea. Between and after these attacks the boar, as compared to litter mate No. 89, developed normally but showed signs of exhaustion and lay in an apathetic state rising only to feed. Such lethargy and apparent exhaustion are often characteristic symptoms of vitamin A deficiency in pigs.

Boars 81 and 88 were killed at the same time, nearly 10 months old and 7 days later boars 89 and 90 aged 9 months were sacrified (Table 3). Boar 87, as already stated, was killed due to the increasing attacks of convulsions.

At the time when boars 81 and 88 were killed, 10 months old, a testis biopsy was performed on their litter mate, boar 86. The operation was made under universal anesthesia using a liver harpoon with a diameter opening of 3.5 mm.; about 2 g. testis tissue was removed from the right testicle. The following day the boar received an intramuscular injection of 1.6 mill. international units of vitamin A in an aqueous dispersion. Two months later a testis biopsy was performed on the left testicle under local

Table 3. Data of boars.

Boar No.	Body weight kg.	Age months	I.u. vit. A per g. liver	Sexual organs g						
				Testes			epididymis	vesiculæ seminales	gll. bulboure-thrales	corpus prostatae
				right	left	total				
89 C	114	9	208	240	245	485	110	193	98	6.0
90	114	9	< 0.4	73	76	149	77	43	67	7.2
88 C	130	10	525	285	276	561	138	83	50	7.0
81	130	10	< 0.5	140	157	297	126	55	57	9.0
87	140	10	< 0.3	136	141	277	148	150	107	10.2
80 C	173	13	370	389	394	783	173	156	66	8.0
86	174	13	440	331	273	604	115	115	46	9.5

C: control boars.

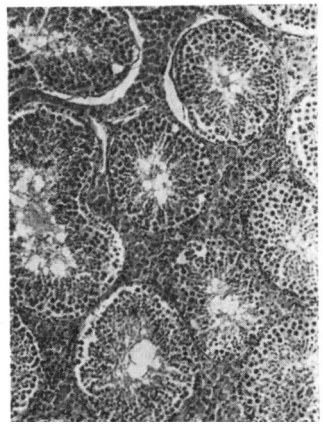
anesthesia, and one month after this operation boar 86 and the control boar 80, both 13 months old, were killed.

From the autopsy boar 90 was found to have a chronic, purulent bronchopneumonia and boar 87, chronic fibrous pleuritis.

In Table 3 are given the ages of the boars when killed, their body weight, vitamin A content in the liver and the weight of the testicles and of the secondary sexual glands, except the disseminated prostate. As may be seen from this table, the weight of the testes of boars 81 and 87, deficient in vitamin A was one half, and of boar 90, less than one third of the weight of the testes of the control boars. In this connection it is also worth noting that boar 86, which, according to the liver analysis of litter mates, boars 81 and 87, might be expected to have been vitamin A deficient at the age of 10 months, but which had received this vitamin 3 months before slaughtering, had a testes weight not essentially lower than the control boar, No. 80.

As seen from the table, the weight of the secondary sexual organs varied greatly. Moreover there seems to be no characteristic relation between the A vitamin status of the animal and the size of these glands. However it should be kept in mind that the weight f. inst. of the seminal vesicle varies greatly, depending on the fullness of that organ. These figures should therefore be accepted with some reservation.

For microscopic investigations, samples were taken from all



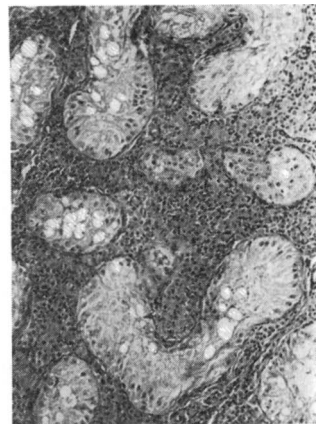
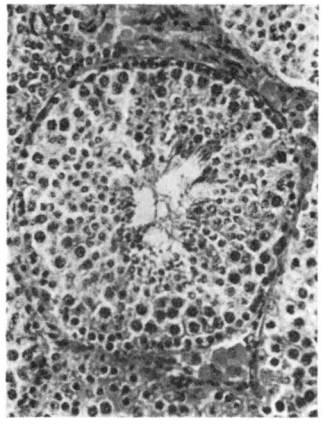


Fig. 1. Boar 89. Testis. Normal spermiogenesis and interstitial tissue. Hematoxylin-Eosin. (magn. appr. 70 ×).

Fig. 2. Boar 90. Testis. Vitamin A deficient. No spermiogenesis. Small tubules and a "compensatory" hypertrophy of Leydig cells are seen. Hematoxylin-Eosin. (magn. appr. 70 ×).



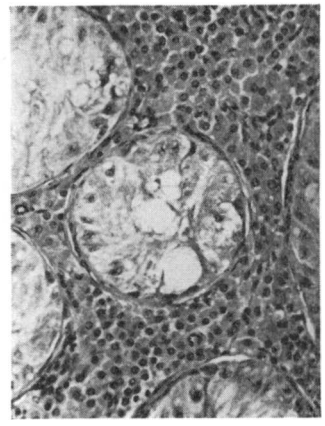


Fig. 3. Boar 89. Tubule from testis shown in Fig. 1. Hematoxylin-Eosin. (magn. appr. 170 ×).

Fig. 4. Boar 90. Tubule from testis shown in Fig. 2. In the small tubuli only Sertoli cells are present. Hematoxylin-Eosin. (magn. appr. 170 ×).

the organs and tissues. However the following description will only include the histological findings in the sexual organs. Sections were prepared of the tissues fixed in formalin and the following stains were made on the histological cuttings: v. Gieson-Hansen, Hematoxylin-eosin, Sudan III, Nile blue, PAS, the Feulgen reaction and the so-called long Ziehl-Neelsen.

Microscopic investigations of testis from the control boars showed that these were normally developed with active, spermatogenetic epithelium (Figs. 1 and 3). In the vitamin A deficient boars the histological picture of testis varied somewhat in spite of certain common characteristics, so that the most pronounced divergences from normal were found in boars 87 and 90, with slightly less pronounced divergences in boar 81. The number of tubuli as compared with the control animals was unchanged but the size of all tubules was considerably below normal (Figs. 2 and 4). The tubules of boar 90 contained almost without exception cells which must be recognized as the sustentacular cells of Sertoli (Fig. 4). The basally placed nucleus was triangular to oval in shape with a pronounced nucleolus and the cells often formed a syncytium. The cytoplasma contained granules and many larger and smaller vacuoles were visible in the paraffin wax sections. In the majority of tubules including boar 87, only Sertoli cells were found. However in a few tubules it was possible to demonstrate poorly differentiated spermatogonia and spermatocytes. In no cases could spermia be demonstrated in the lumen. In boar 81 the histological picture was even more heterogenous, as some tubuli contained only sustentacular cells, while in other tubuli it was possible to demonstrate the various stages of spermatogenesis as well as a content of spermia in the lumen. However there were always fewer germ cells than normal and they largely consisted of spermatogonia and the first stages of spermatocytes (Fig. 8). Besides spermia in the lumen there were numerous desquamated cells representing various stages of spermatogenesis and also quite divergent cell types in the form of giant cells (Fig. 9).

The interstitial tissue in testis likewise showed characteristic changes. In the vitamin A deficient boars proliferation of the connective tissue was apparent both in the septula of the testis and around the separate tubules. The number of interstitial cells, the Leydig cells, seemed to be increased in all the vitamin A deficient boars. However a count showed that this proliferation

was only apparent because of the diminished size of the tubules and that actually the total number of Leydig cells was of the same order in both the vitamin A deficient and in the control boars.

As a rule the interstitial cells are polygonal and sharply defined with a spherical to ovoid nucleus often placed excentrically. This nucleus contains one, sometimes two nucleoli as well as fine chromatin granules generally placed near the nuclear membrane. The cytoplasma, which is strongly eosinophilic contains many granules. In boar 90 the Leydig cells were generally below normal in size and varied in shape from polygonal to rounded, together with more oblong forms. The size of the nuclei varied and the chromatin showed a tendency to concentrate in larger granules. In this boar it was characteristic that the cytoplasma of the interstitial cells was only slightly eosinophilic.

The changes just noted were apparent also in boar 81, but to a lesser degree. However in the third boar deficient in vitamin A, boar 87, the Leydig cells were very large with pronounced eosinophilic cytoplasma.

As already stated, a testis biopsy was made in boar 86 immediately prior to receiving vitamin A. At that time the boar was 10 months old. By histological examination the relatively small tubuli were found to contain many Sertoli cells and very few spermatogonia (Fig. 5). In the interstitial tissue there were numerous Leydig cells with slightly eosinophilic cytoplasma. When the boar was 12 months old *i. e.* 2 months after receiving vitamin A, a testis biopsy was again performed (Fig. 6). The single tubuli were now considerably larger in diameter, but the histological picture of the spermatogenetic tissue varied greatly. In some tubules, except for Sertoli cells only a few spermatogonia

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Fig. 5. Boar 86. Testis. Vitamin A deficient. The tubule shows advanced injury only containing Sertoli cells. Hematoxylin-Eosin. (magn. appr. 225 ×).

Fig. 6. Boar 86. Testis 2 months after vitamin A therapy. The tubule is of larger size with several layers of germ cells and cellular debris. Hematoxylin-Eosin. (magn. appr. 225 ×).

Fig. 7. Boar 86. Testis 3 months after vitamin A therapy shows nearly normal histological structure. A few vacuoles are to be seen between the cells of the germinal epithelium. Hematoxylin-Eosin. (magn. appr. 225 ×).

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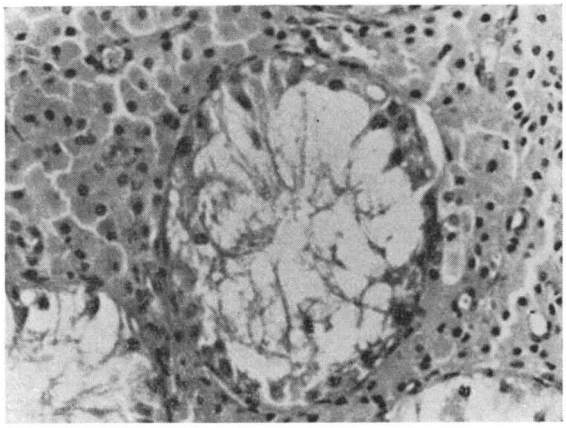

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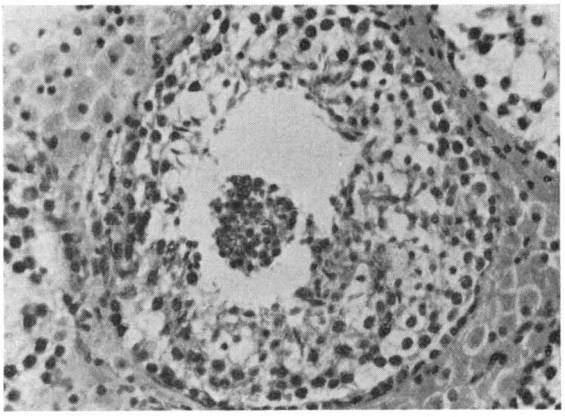

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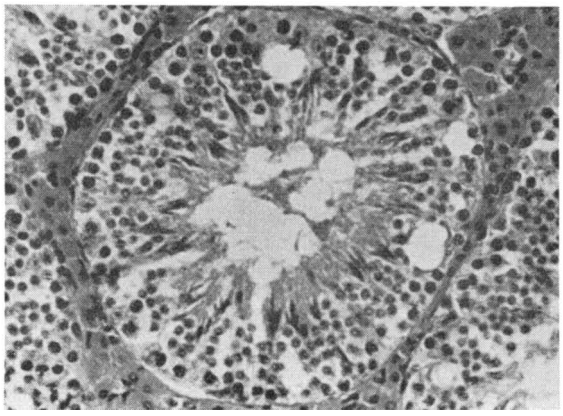

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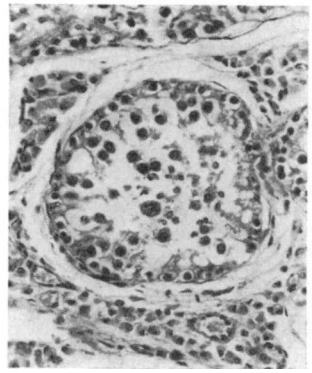

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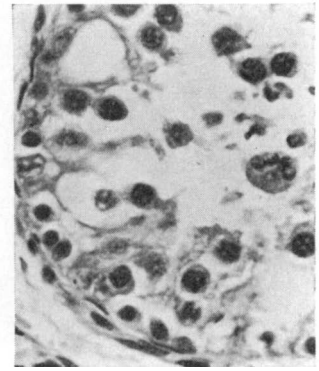

F i g. 8. Boar 81. Testis. Vitamin A deficient. A few layers of undifferentiated germ cells are present. Hematoxylin-Eosin. (magn. appr. 170 ×).

F i g. 9. Boar 81. Part of the same tubule as in fig. 8 showing the sloughing of germ cells together with a multinuclear cell. Hematoxylin-Eosin. (magn. appr. 420 ×).

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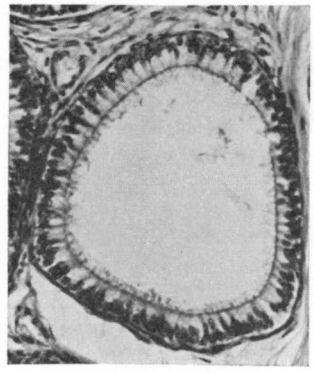

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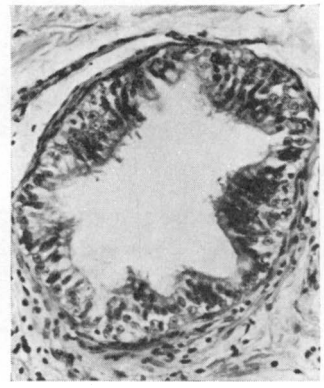

F i g. 1 0. Boar 80. Normal efferent ductule of testis. Hematoxylin-Eosin. (magn. appr. 170 ×).

F i g. 1 1. Boar 90. Efferent ductule of testis showing proliferation and metaplasia of epithelium. Hematoxylin-Eosin. (magn. appr. 170 ×).

were to be seen, but in most of the tubules there were several germ cells and in a few tubules all the generations of spermatogenesis. The number of cell layers and the density of the cells were much below normal. In the lumen there were only a few sperms, but numerous desquamated cells. As a result of the growth of the tubules, the interstitial layer formed a smaller portion of testis than earlier and the Leydig cells had a strongly eosinophilic stained granulated cytoplasma.

When boars 80 and 86 were killed at the age of 13 months, some of the tubuli in boar 86 showed a nearly normal histological picture of the germinal epithelium (Fig. 7). However in some parts of the testis vacuoles of the cytoplasma were visible and the spermatogenetic epithelium was not as well developed in respect to number and density of cells as in the control boar, no. 80.

Sudan III staining was performed on frozen sections of the testicles to demonstrate possible lipoids in the Leydig and Sertoli cells. The result was that neither in the control boars nor in the vitamin A deficient boars could any content of lipoids in the Leydig cells be demonstrated. However the Sertoli cells contained many droplets of fat, mainly located in the basal part of the cells and so distributed that the lipoid content seemed to be largest in the vitamin A deficient boars. This is probably because the germinal epithelium was poorly developed in these boars and the lipoid content of the Sertoli cells dominated the histological picture.

It is well known that in connection with atrophy of testis, ceroid pigment is often observed in the Leydig cells. Therefore a long Ziehl-Neelsen stain was made to demonstrate the pigment. However in none of the boars was there any positive reaction to that staining method.

The epithelium in rete testis, normally low columnar, showed in the vitamin A deficient boars a tendency to proliferation with cells containing very large nuclei, but poor in chromatin.

In boar 90 very characteristic changes were observed in the epithelium of the efferent ductules. The epithelium consists normally of ciliated cells alternating with groups of secreting cells (Fig. 10), but in boar 90 there was a multilayered proliferating epithelium with numerous vacuoles between the cells (Fig. 11). Some eosinophilic lenkocytes could be demonstrated in lumen.

The histological investigation of epididymis comprised of sec-

tions from the head, the body and the tail. In all portions the duct was somewhat dilated in boars 81 and 90 and the epithelium considerably lower than in the control boars, while in the third vitamin A deficient boar, 87, numerous vacuoles were found in the epithelium. In all the boars, however, the cells seemed to have normal secretory function. In the lumen, both in control boars and in boar 81, sperms were found in all the sections and in boar 81 some desquamated cells were demonstrated. In the two other vitamin A deficient boars no sperms could be found in the head or in the body of epididymis, but a few sperms and desquamated cells were observed in the tail.

By microscopical investigation of the body of the prostate gland of all the boars, the glandular tissue was found normal with active secretory epithelium. However, in all vitamin A deficient boars numerous prostatic concretions were found. These rounded, concentrically built, small bodies of various sizes, stained strongly blue black in hematoxylin preparations, and were to be found either intraepithelially or free in the cavities — often together with desquamated glandular cells.

Neither in the disseminated portion of prostata or in the Cowpers glands was any deviation from normal observed in the vitamin A deficient boars and the epithelium by the histological staining methods used was found to have normal secretory activity. In the seminal vesicles in boar 90 the glandular epithelium had large nuclei which stained very weakly by the Feulgen reaction. In boar 81 the epithelium of the seminal vesicles showed in some places a tendency to proliferation under formation of a multilayered epithelium.

DISCUSSION

As early as 1925, *Wolbach* and *Howe* published a detailed account of the pathological effects of vitamin A deficiency in rats and a few years later (1928) in guinea pigs. These investigators discovered that testis, after foregoing oedema, showed pronounced atrophy with the result that the tubules only contained cells derived from the sustentacular cells. The cells of the efferent ductules of the testis increased in number and there were often large vacuoles between the cells. In the epididymis the cells became slightly atrophic, but retained their normal characteristics, whereas the secondary sex glands showed the sequence of atrophy, keratinization and fibrosis. Very compre-

hensive investigations of rats have been made by *Mason* (1930, 1933), who not only described changes in testis due to vitamin A deficiency, but also subsequent histological regeneration processes after later application of that vitamin. This investigator pointed out that testicular injury was often apparent before the external manifestations of vitamin A deficiency appeared and that spermatogenesis frequently persisted in spite of considerable depletion of the germinal epithelium. The regeneration after vitamin A deficiency injuries to rats required from five to thirteen weeks, which suggests a more serious disturbance in the germinal epithelium than after inanition. Mason demonstrated atrophy of the secondary sexual glands in vitamin A deficient rats but he assumed that this reduction was due to decreased feed intake of the rats and the resulting lack of energy, protein, etc.

In the years which have passed since the first investigations on the effect of vitamin A deficiency on the male sexual organs of rats and guinea pigs were performed, numerous experiments have been made, not only with laboratory animals but also with farm animals f. inst. bulls and rams. Extensive investigations on cattle have been made by *Bratton et al.* (1948). In these, by feeding bulls with a vitamin A deficient ration, an increase of abnormal spermatozoa in semen was demonstrated and by histological investigation of testis, degeneration of the germinal epithelium was observed. *Madsen et al.* (1948) found that semen production in bulls, fed a diet deficient in vitamin A, continued even after clinical symptoms of A avitaminosis were very pronounced. The semen showed a marked increase in the percentage of abnormal spermatozoa and of cellular debris with progressive decline in mobility. In vitamin A deficient rams, *Lindley et al.* (1949) found that the testicles were decidedly smaller than those in the control rams, the semen produced was of low quality and histologically degeneration of the germinal epithelium was demonstrated.

As stated, the effect of vitamin A deficiency on the sexual organs of the boar has apparently not previously been made the object of special investigations, but under other pathological conditions, similar histological changes have been noted in the appearance of testis. In cryptorchid boars *Teilum* (1950) thus observed suppressed spermiogenesis, an excessive lipoid content in the Sertoli cells, whilst the numerous Leydig cells contained no lipoids

by Sudan staining. This investigator found by histological examination in several animal species (dog and cat for instance) a considerable content of lipoids in the Leydig cells, but, in agreement with the findings in the experimental boars described here, no lipoids could be demonstrated histologically in the interstitial cells of young normal boars.

The effect of vitamin A deficiency on the testes of the boar is not only a quantitative change under guise of a reduction in size of these organs, but to a far greater extent a qualitative alteration with decreased or totally discontinued spermatogenesis. In this connection it should be noted that the histological findings varied somewhat in the vitamin A deficient litter mates, 81 and 87. Presumably, however, a certain physiological variation will always be present, even in litter mates.

Two months after vitamin A application to the vitamin A deficient boar, 86, the appearance of the spermatogenetic epithelium had totally changed. From consisting almost exclusively of Sertoli cells, some tubules at any rate now included all stages of spermatogenesis and when the boar was killed a month later the microscopic findings approached normal even more closely. It should however be stated that the processes of regeneration after such a pronounced A avitaminosis, apparently proceeds rather slowly. This must be expected from the histological appearance of testis under the deficiency as, of the germinal epithelium, only a very few spermatogonia were to be found.

As already stated, in boars 87 and 90, ten and nine months old respectively, no spermatogenesis was observed. However in the tail of epididymis of both boars, sperms and desquamated cells were demonstrated. Therefore in these boars it could not be a case of retarded development of spermatogenetic tissue but an atrophy of the formerly active germinal epithelium.

Again, as already stated, the size of the secondary sexual glands varied somewhat, but aside from incipient changes in the epithelium of the seminal vesicle these organs were found to have an apparently normal secretory function. The degree to which the rather heterogenous histological picture of the Leydig cells is expressive of the endocrine function of these cells, is not easy to determine. However there seems to be a certain relationship between the appearance of the cells, f. inst. the content of eosinophilic granules in the cytoplasma and the relative size of the secondary sexual glands.

The concretions in prostata already described, are often present in older boars (*Ellenberger,* 1911). However in this experiment, as they only occurred in the vitamin A deficient boars and in consideration of the youth of the boars, it may be assumed that the formation of the concretions is due to vitamin A deficiency, possibly in connection with incipient focal metaplasia and degeneration of the epithelium of the prostate gland.

In the epithelium of the efferent ducts of testis in boar 90 some proliferation and metaplasia was observed which to a certain degree resembled the change in the epithelium in the salpinx in vitamin A deficient gilts described earlier (*Hjarde et al.* 1961).

Some investigators (*Wolbach and Howe,* 1928) have called attention to the fact that in vitamin A deficient guinea pigs there were pathological changes in semen and in testis before the animals showed clinical symptoms of vitamin A deficiency. On the other hand it is stated (*Bratton et al.* 1948, *Lindley et al.* 1949) that production of semen continued in bulls and rams even after clinical symptoms of vitamin A deficiency were apparent. As already stated, the vitamin A deficient boars, 81 and 86, appeared to be clinically normal, but boar 87 and to a lesser degree boar 90, showed characteristic symptoms of A avitaminosis. This condition, that boars in spite of pronounced degenerative changes in testis and discontinued spermatogenesis, do not necessarily show typical symptoms of vitamin A deficiency, may be of decisive importance under practical conditions, where by clinical examination alone, it is impossible to demonstrate the cause of deficient semen production.

The important question is to elucidate in which way the vitamin deficiency causes the testicular changes here described. There are two main possibilities; it can be an effect of vitamin A deficiency on the secretory function of endocrine organs or it may be a more direct effect of the deficiency on the spermatogenetic epithelium.

In hormone production, interest is primarily concentrated on the gonadotropic hormones of the pituitary gland, yet the possibility that a change in function of the thyroid gland may be manifested cannot be rejected. Various opinions have been expressed as to the importance of the anterior pituitary. *Mayer and Goddard* (1951) found that injection of gonadotropic hormones in vitamin A deficient rats stimulated the development of the secondary sexual organs. They concluded therefore that the cause of atrophy

of the secondary sexual organs was a failing function of the testis due to reduced secretion of the gonadotropic hormones. However *Sutton and Brief* (1939) reported that gonadotropin secretion was increased by vitamin A deficiency which was presumably due to influence of abnormally functioning sexual glands. In experiments with vitamin A deficient rams already described, *Lindley et al.* (1949) demonstrated that injection of testosterone propionate, or pregnant mare serum, exerted no beneficial effects on semen production or quality. This also seems to indicate that neither the gonadotropic hormones or testosterone were of any great importance for the change in the sexual glands under vitamin A deficiency. These investigators found cysts in the pituitary body, but in the boars, here described, no such characteristic histological changes could be demonstrated.

As was stated in an earlier report (*Hjarde et al.* 1961), confirmed as characteristic by continued experiments, gilts and sows often had rather long heat periods and a small return rate. This does not indicate any reduced secretion of the gonadotropic hormones in these animals.

Using a method introduced by *Sørensen* (1958), it was possible to measure the thyroxin secretion by means of I^{131} in the above mentioned boars (*Palludan and Sørensen,* 1962). It appeared that the thyroxine secretion of the vitamin A deficient boars was one half to one third of the thyroxine secretion of the control boars. It therefore seemed reasonable to investigate the extent to which this condition affected testis function. *Berliner and Warbritton* (1937) found that thyroidectomy in rams resulted in an increase in the number of abnormal sperms and a decrease in the amount of semen, whereas a subsequent dosage with thyroxine restored the normal production of semen. However on the basis of the above and other similar investigations, there is little reason to believe that the effect of vitamin A deficiency on spermatogenesis is exclusively, or even mainly, due to a reduction of the hormone production of the thyroid gland.

Several experiments have substantiated the observation that the pathological changes due to vitamin A deficiency are found in many epithelial structures and are most pronounced during the growth and differentiation of the tissues as is seen in fetal life by many malformations, while in growing and in adult animals among other disturbances, keratinization of the epitelia occurs, since some tissues are more vulnerable than others.

Furthermore there are certain variations from one animal species to another and even individual differences within the same species. For instance it is characteristic of many animal species that vitamin A deficiency has proved injurious to the vaginal mucous membrane and to the spermatogenetic epithelium of testis before other symptoms of deficiency are manifested. This may be due to the more organized and specialized nature of these epithelial tissues.

By means of tissue cultures *Fell and Mellanby* (1952) demonstrated that an addition of an excess of vitamin A has a similar, but a more rapid effect on the cultivated tissue than the effect from feeding an animal on a diet with a high vitamin A content. This, and many similar experiments indicate that the vitamin affects the various tissues directly. Taking all these observations and results into consideration it seems correct to assume that vitamin A will also directly affect the epithelium in the tubules of testis, either the spermatogenetic epithelium or the Sertoli cells. These cells comprise of the nutritive element and it is therefore possible that a change in the metabolism of the Sertoli cells could be the actual cause of discontinued spermiogenesis.

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SUMMARY

In continuation of a series of experiments on the importance of vitamin A deficiency for pigs an investigation was made to elucidate the effect of A avitaminosis on the sexual organs of the boar. The experiment included seven boars of Danish Land Race from two different litters. Three of the boars were used as control animals, three were fed a vitamin A deficient diet during the entire experimental period and a fourth was given vitamin A after having been depleted. Some of the vitamin A deficient boars showed no clinical symptoms of A avitaminosis, while one of the boars was killed due to repeated attacks of convulsions.

Examination of the sexual organs showed that the testicle weight in the A avitaminotic boars was one half to one third of normal.

By histological investigation, atrophy of the tubules was found in testis of the A avitaminotic boars as well as discontinued spermatogenesis, a "compensatory" increase of Leydig cells and in one of the boars incipient metaplasia of the epithelium in the efferent ducts.

Pathological changes in the secondary glands were not very pronounced. In the body of prostate numerous concretions were found in the deficient boars and the epithelium of the seminal vesicles showed incipient degenerative changes.

A testis biopsy was performed on an A avitaminotic ten months old boar and by histological examination characteristic atrophy of testis was found. The boar was given vitamin A and two months later a testis biopsy was again taken. A conspicuous proliferation of the germinal epithelium was apparent with formation of a few sperms. When the boar was killed a month later, the epithelium of the tubules was found to be nearly normal.

The cause of the characteristic changes in testis is discussed and the conclusion drawn that vitamin A, in all probability, directly affects the spermatogenetic epithelium.

ZUSAMMENFASSUNG

A-Vitaminmangel und dessen Effekt auf die Geschlechtsorgane des Ebers.

In der Fortsetzung einer Reihe von Versuchen über die Bedeutung des A-Vitaminmangels für Schweine wurde eine Untersuchung mit dem Ziel ausgeführt, den Einfluss dieser Avitaminose auf die Geschlechtsorgane des Ebers zu beleuchten.

Der Versuch umfasste sieben Eber der dänischen Landrasse aus zwei Würfen. Drei von diesen Ebern erhielten während der ganzen Versuchsperiode A-Vitamin-freies Futter, dem vierten Eber wurde nach Erschöpfung seines Organismus an A-Vitamin dieses Vitamin verabreicht, während drei Eber als Kontrolltiere in diesem Versuch dienten.

Keiner der Eber mit A-Vitaminmangel zeigte charakteristische Symptome hierfür, dagegen musste einer von diesen Ebern wegen wiederholter Krampfanfälle getötet werden.

Die Untersuchung der Geschlechtsorgane ergab, dass das Gewicht der Hoden bei den A-avitaminotischen Ebern die Hälfte bis zu einem Drittel des normalen Gewichts betrug.

In den Hoden wurden bei diesen Ebern histologisch Tubuliatrophie, aufgehörte Spermiogenese und anscheinend Vermehrung der Leydig-Zellen sowie bei dem einen der Eber beginnende Metaplasie des Epithels in den Ductuli efferentes nachgewiesen.

Pathologische Veränderungen in den sekundären Geschlechtsdrüsen traten nur geringgradig hervor. In der Prostata der Eber mit A-Vitaminmangel fanden sich zahlreiche Konkremente, und das Samenblasenepithel zeigte beginnende degenerative Veränderungen.

Eine Testisbiopsie wurde bei einem der Eber mit A-Vitaminmangel ausgeführt, als derselbe 10 Monate alt war, und histologisch war die charakteristische Atrophie der Hoden vorhanden. Daraufhin wurde dem Eber A-Vitamin verabfolgt, und die zwei Monate später wiederholte Testisbiopsie ergab Proliferation des Keimepithels mit Bildung einer geringen Anzahl von Spermien. Bei der einen Monat später erfolgten Tötung erwies sich das Tubuliepithel als fast normal.

Die Ursache der charakteristischen Veränderungen in den Hoden wird diskutiert und die Schlussfolgerung gezogen, dass das A-Vitamin wahrscheinlich seine Wirkung direkt auf das spermiogenetische Epithel ausübt.

RESUMÉ

A-vitaminmangelens indflydelse på ornens kønsorganer.

I fortsættelse af en række forsøg over A-vitaminmangelens betydning for svin er der udført en undersøgelse med henblik på at belyse denne avitaminoses indflydelse på ornens kønsorganer.

Forsøget omfattede syv orner af Dansk Landrace fra to kuld. Tre af ornerne fodredes A-vitaminfrit i hele forsøgsperioden, en fjerde fik efter at være udtømt for A-vitamin dette vitamin tilført, medens tre af ornerne indgik i forsøget som kontroldyr.

Nogle af de A-vitaminmanglende orner viste ingen karakteristiske symptomer på A-vitaminmangel, medens en af ornerne måtte aflives på grund af gentagne krampeanfald.

Ved undersøgelse af kønsorganerne fandtes, at testikelvægten hos de A-avitaminotiske orner androg halvdelen til en trediedel af det normale.

I testis påvistes hos disse orner histologisk tubuliatrofi, ophørt spermiogenese og tilsyneladende forøgelse af Leydig-cellerne, samt hos en af ornerne begyndende metaplasi af epitelet i ductuli efferentes.

Patologiske forandringer i de sekundære kønskirtler var lidet fremtrædende. I prostata fandtes hos de A-vitaminmanglende orner talrige konkrementer, og sædblæreepitelet viste begyndende degenerative forandringer.

En testisbiopsi blev udført på en A-vitaminmanglende orne, da den var 10 måneder gammel, og histologisk fandtes den karakteristiske atrofi af testis. Derefter fik ornen tilført A-vitamin, og 2 måneder senere udtoges atter en testisbiopsi, hvor der påvistes proliferation af kimepitelet med dannelse af et fåtal af spermier. Ved aflivningen en måned senere fandtes et næsten normalt tubuliepitel.

Årsagen til de karakteristiske forandringer i testis diskuteres, og det konkluderes, at A-vitaminet sandsynligvis udøver sin virkning direkte på det spermiogenetiske epitel.

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