

# Selenium and Fertility in Animals and Man – A Review

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**Hansen, J. C. and Y. Deguchi: Selenium and fertility in animals and man. Acta vet. scand. 1996, 37, 19-30.** – To evaluate the information on selenium with relation to fertility in animals and man the available literature was reviewed. Selenium is incorporated in the sperm mitochondria capsule and may thus affect the behavior and function of the spermatozoon. Se seems to be essential for normal spermatozoa development in both experimental animals and in livestock and probably also in humans. Regarding selenium and female fertility only sparse information exists. In experimental animals a low selenium level affects fertility in males, but little attention has been devoted to female reproductive performance, and the data are insufficient for conclusion. In livestock numerous investigations have been performed and the effects of selenium supplementation often in combination with other antioxidants have been evaluated, but no valid conclusion can be drawn. In general adequate nutritional supply will secure optimal reproduction in both males and females, while additional supplementation seems to have a negative effect.

In humans contradictory information is found. Both low and high sperm selenium concentrations are reported to have a negative influence on the number of spermatozoa and on the motility. The optimal sperm selenium concentration waits to be defined. Some evidence indicates that a metabolic defect in a selenium incorporation into sperm cells may be associated with human infertility. No human data relating selenium to female infertility were found.

*experiment; livestock; humans.*

## Introduction

During the last decennia selenium (Se) deficiency in livestock has been recognized as a problem in various areas of the world (*Lannek & Lindberg 1975, Nebbia 1982*). As a consequence addition of selenium to foodstuffs has been widely used to provide an adequate supply.

During the 1980s Se has been recognized as being essential to humans too and several syndromes have been described in animals and in humans (*Koller & Exon 1986*) and etiologically explained as either Se deficiency or toxicity.

Furthermore a relative deficiency has been discussed in relation to cancer (*Salonen et al. 1984*), cardiovascular diseases (*Salonen 1987*), neural diseases (*Westermarck 1977*), and rheumatoid arthritis (*Tarp et al. 1989*). Se-containing preparations are commercially available and self-medication is now widespread in many countries.

Due to the toxic properties of Se, even in low doses, risks connected with use or abuse should not be underestimated and adverse effects such as acute and chronic intoxications are described

in both humans and in animals (AACT 1984, Hill *et al.* 1984, Hooper *et al.* 1985).

Se accumulates in the mammalian testis to a higher extent than it does in other tissues and it influences spermatozoal motility (Benard 1979, Hidioglou 1982, Behne *et al.* 1986). By supplementation of low doses of Se to Se deficient rats, Behne & Höfer-Bosse (1984) found indications of the existence of a mechanism in Se distribution which favors the supply of testis, while the glutathione peroxidase status has a secondary status. Thus it can be assumed that Se, besides its well-known effects on the oxidative defence system through its function in glutathione peroxidase, may play an essential role in fertility in animals and man. The purpose of this paper is to review the literature on this subject.

### Experimental animals

Se is essential for the normal development of sperm cells. Spermatozoa of rats are rich in Se (approx. 30 µg/g), of which the main part is found in a cysteine-rich polypeptide localized in the sperm mitochondrial capsules, (the "mitochondrial capsula protein" (MCP)) (Calvin *et al.* 1981, 1987, Wallace *et al.* 1984).

Se is incorporated in MCP in the early steps of the spermatogenesis under hormonal control. Behne *et al.* (1984) described the fact that hypophysectomia lead to reduced sperm Se concentration and that the concentration was normalized by administration of LH and FSH. A function of Se in the Leydig cells was indicated as plasma testosterone, after stimulation by LH-RH or LH, was lower in Se deficient rats compared to control animals, while no difference in hypophyseal LH secretion could be observed after LH-RH stimulation. Wu *et al.* (1971, 1979) found normal epididymal epithelium in Se-deficient rats, while many undeveloped and degenerated spermatozoa could be found in the

luminal epithelium. The motility of epididymal spermatozoa was found to be diminished compared to a normal function in animals with a dietary supply of 0.1 mg Se/kg, while vitamin E did not influence the motility in the Se-deficient animal.

Wu *et al.* (1971) also observed that supplementation of chromium (1 mg/kg) to Se-deficient rats stopped the spermiogenesis. This observation indicates that other essential trace elements may also influence the mammalian fertility.

A toxic effect on testicular function in rats continually supplemented with Se was described by Nebbia *et al.* (1987). 16 ppm of sodium selenite in the drinking water lead to testicular degeneration. Biochemical evidence of tissue damage (increased lactate-dehydrogenase activity) was demonstrated even at a dosage level of 4 ppm.

Wallace *et al.* (1983a,b, 1986) found Se to be essential for the fertility in male mice. In mice, as in rats, Se deficiency resulted in abnormal development and morphology of the sperm mitochondrial capsules, increasing in frequency through generations of Se-deficient animals. In mice (Wallace *et al.* 1983a) in contrast to rats (Wu *et al.* 1973) only a weak relation between decreased motility and morphological abnormalities has been observed. Second generation Se-deficient mice were found to have a reduced sperm count. The deficiency in mice, as in rats, is associated with the lack of the MCP-selenoprotein.

Most investigations into the relationship between selenium and fertility in experimental animals have concentrated on males. One study, however, indicated that short-term supplementation of Se by injections of sodium selenite for 2 to 3 days increased the mass of reproductive organs in female mice and increased the number of fetuses. Prolonged dosing for 20 to 130 days gave the reverse effect (Euybov *et al.* 1983).

## Livestock

### Male fertility

In boars, fed an extremely low Se diet (0.01 ppm) based upon corn and soybeans grown in the Se-poor areas of China. *Liu et al.* (1982) found a reduced number of sperms as well as a reduced number of live sperms and increased occurrence of defect heads and tails. These findings are in contrast to other studies performed by *Segerson et al.* (1981) and *Henson et al.* (1983), in which the content of Se in the basic feed was 0.025 ppm and 0.05 ppm, respectively. In none of these studies did supplementary Se have any effect on reproductive performance. Normal reproduction was observed in boars given a diet containing 0.05 mg/kg Se, while retarded sexual development was observed when increasing the content to 0.15 and 0.3 mg/kg (*Henson et al.* 1983). Post mortem examinations revealed no differences between dosage groups with regard to weight of testis, number of sperms or to sperm morphology.

*Heimann et al.* (1984) found no abnormalities in spermatozoon related to Se concentration in 12 bulls with a mean ( $\pm$  SD) blood plasma Se concentration of  $61 \mu\text{g/l} \pm 22$  and a total mean sperm Se concentration of  $461 \pm 223 \mu\text{g/l}$ .

Se seems to be essential for normal spermatozoal development in livestock, as in experimental animals. A negative effect will, however, only appear at an extremely low Se intake, and normally accepted standards seem sufficient to secure normal fertility. A moderately high supplementation may exert a negative effect. There is a lack of information for a definition of an optimal Se status with regard to male fertility.

### Female fertility

Cattle: Most investigations into Se in relation to reproduction in cattle have been devoted to studies on the effect on retained placenta. The

incidence in USA is indicated as being 10%, however, in some Se deficient areas it may be as high as 50% (*Harrison et al.* 1984). *Trinder et al.* (1969) were the first to suggest Se as an etiological factor as regards this condition, and were later supported by others (*Julien et al.* 1976, *Trinder et al.* 1973). *Harrison et al.* (1984), however, stressed that a concomitant supply of vitamin E was necessary. *Eger et al.* (1985) found that Se supply through injections of 2.3 to 23 mg Se/animal reduced the incidence of retained placenta, but also concluded that relatively low doses of 2.3 to 4.6 mg/animal were more effective than were higher doses. Also *Caufalik* (1985) reports that Se therapy (50 mg/animal) in combination with estradiol, vitamin A, D and E had a prophylactic effect. However, which of the components that was responsible for the positive effect was not revealed by the author.

*Blom et al.* (1984) found in a Danish investigation of heifers with an in general marginal Se deficient status that supplementary Se increased the incidence of retained placenta.

In the investigations where Se supplementation was found to decrease the incidence of retained placenta, the animals were in a condition of absolute or marginal Se-deficiency and probably also suffered from other kinds of nutritional unbalances. Thus *Hidiroglou et al.* (1987) found in herds with a relative high incidence of retained placenta (22.1%), and sufficient Se-supply that further supplementation did not influence the condition and concluded that retained placenta in cattle is not Se-responsive.

In Norway *Ropstad et al.* (1987) investigated 17 herds with normal reproductivity performance and 47 herds with abnormal. A considerable part of the herds had a marginal Se deficiency defined as blood Se concentrations below 50  $\mu\text{g/l}$ . In herds with a high Se status increased time from calving to last insemination compared to herds with low Se status was found. No

differences according to general health conditions were observed between low and high Se status herds. Corresponding negative findings were observed by *Blom et al.* (1984) in a study of Se supplementation to 17 Danish herds. *Spears et al.* (1986) found that a combination therapy of Se and vitamin E to beef cattle in a condition of marginal Se deficiency did not influence conception rate or interval between calvings. It did, however, reduce mortality in the offspring. Also other studies have shown negative results of a combined Se vitamin E therapy on reproductive performance in cattle with sufficient Se supply (*Hidiroglou et al.* 1987, *Koppel et al.* 1984, *Stowe et al.* 1988). *Segerson & Libby* (1982) observed in 41 charolais cows with a marginal Se deficiency status (mean blood serum Se concentration 23 µg/l) that a combination of injections of 40 mg Se and 544 IU α-tocopherol every 14 days did not influence fertility, but that the number of sperms per ovum was correlated to the Se status of the cows. The authors suggested that Se facilitates the intrauterine sperm transport.

A positive effect of Se on neonatal mortality in calves and on general reproductive performance was reported by *Sanders* (1984) based on an investigation of two low Se status herds (herd I: 150 cows with blood Se concentrations from 23 to 44 µg/l; herd II: 70 cows with a blood serum Se between 30 and 54 µg/l). They recommended oral supplementation as more effective than parenteral administration, a view also pointed out by *Hansen & Kristensen* (1979).

*McClure et al.* (1986) observed an increased conception rate after oral supplementation of Se to cows with a mean whole blood glutathione peroxidase activity below 70 units/g hemoglobin; also *Tasker et al.* (1987) have reported increased conception rate after injections of Se. In a number of publications from East European countries it is reported that a mixed anti-

oxidative therapy exerts a positive effect on cattle reproduction. Unfortunately most of these publications do not indicate Se status prior to treatment and it cannot be ascertained whether the positive finding is due to the effect of Se or to other antioxidants. The multifactorial cause-effect relationship was indicated by *Grace* (1988), who showed that high intake of molybdenum can reduce fertility in cows.

Regarding cows the literature indicates that only in cases of evident Se deficiency, probably in connection with other nutritional deficiencies and poor hygiene, Se supplementation in combination with vitamin E is of importance for the reproductive ability. In well-fed animals signs of Se deficiency are very rare and furthermore moderate supplementation may give rise to negative effects. It is thus recommended that Se supplementation is only used on with a well documented deficiency (*Blom et al.* 1984). In cattle a blood serum Se concentration below 50 µg/l has been regarded as an indication of deficiency.

Pigs: *Chavez & Patton* (1986) investigated the effect of administration of 3 mg Se and 408 I.U. d-α-tocopherol to sows 3 times during pregnancy as a supplement to a feed containing 0.1 mg/kg of Se. No effect was observed in blood concentrations of neither Se, vitamin E, nor the glutathione peroxidase activity. In spite of no observed biochemical effects treatment improved litter size and birth weight. Neonatal mortality too was reduced in the treated group compared to controls. On the basis of these findings *Chavez & Patton* (1986) recommend supplementation to sows of both Se and vitamin E beyond the recommended nutrition standards of 0.1 mg/kg of Se and 15 IU/kg of vitamin E. The documentation for this recommendation seems, however, to be weak and insufficient. *Jensen et al.* (1984) found that single injections of 0.05 mg/kg of Se, as sodium selenite to 350 sows and gilts did not influence litter size and

neonatal mortality nor could any influence on the estrus cycle be demonstrated.

A report on positive effects of Se supplementation is given by *Momcilo et al.* (1987), who found increased litter size and slaughter weight in sows followed through 2 generations and supplied with 0.1 ppm Se in the feed compared to sows on a 0.03 ppm diet. In a Polish study (*Wandurski* 1988) better effect of vitamin E therapy (single injection of 0.6 g vitamin E) on fertility was found compared to a combination therapy of 0.376 g vitamin E and 5.5 mg sodium selenite per animal. This study had no control animals and the basic Se status was not indicated.

It may be concluded that it is not a documented fact that supplementation with Se alone or in combination with other antioxidants has any beneficial effect on reproduction in sows when recognized dietary standards are fulfilled.

**Sheep:** Intervention trials have shown that orally administered Se is effective in reducing incidence of infertility in ewes and in increasing number of born lambs (*Hartley et al.* 1961, *Andrews et al.* 1968). *Segerson & Ganapathy* (1979) found that Se supplementation increased the uterine contraction thereby increasing the chance of fertilization. *Hartley* (1963) regarded Se deficiency as a cause of early embryo mortality (3-4 weeks of pregnancy).

*Vipond* (1984) reported that dosing to ewes of 5 mg Se together with other minerals and vitamins 2 weeks before mating had no effect on reproduction independent of the pre-dosing Se status evaluated by erythrocyte GSH-Px activity. Also *Kott et al.* (1983) found no effect of injection of 4 mg of Se monthly during pregnancy with regard to number of lambs born. However, a combined Se vitamin E therapy increased the survival of lambs significantly. *Euybov et al.* (1983) reported that optimal dosing of sodium selenite to ewes (without actually defining what optimal means) increased the mass and volume

of reproductive organs, increased fertility and number of live born lambs and birth weight. *Patkowska-Sokola* (1984) also reported positive effect on fertility, number of lambs born, and reduced mortality of lambs when the ewes were treated with injections of 5 mg Se as sodium selenite 4 weeks before mating and 8 mg 4 weeks before parturition. These animals had been grazing on sulfate-rich soils.

On the basis of the existing literature the effects of Se supplementation seem to be more effective in ewes than in dairy cows; this may be due to the fact that either the Se requirements in dairy cows have become more accurately defined or that cattle when compared to sheep are less sensitive to Se deficiency (*Grace* 1988). Otherwise in ewes as in cows the effect of Se on reproductive performance seems to be non-specific and probably related to the nutritional status. It seems advisable to establish the selenium status of the animals before a supplementation programme is implemented (*Van Ryssen et al.* 1992).

**Horses:** *Monroe et al.* (1988) did not find any effect of injections of 2.5 mg Se/45 kg to 8 pregnant mares compared to 8 control mares with regard to conception rate, gestational length, hormonal status, number of liveborn foals, stillborn foals, aborted foals, number of agalactia in mares, or retained placentas. Neither could *Taylor et al.* (1985) find any improvement in reproduction of pony mares as a result of Se supplementation.

**Poultry:** Reduction in egg production and increased embryonal mortality related to Se deficiencies has been observed in White Leghorn hens fed a 0.03 mg/kg Se diet without supplement of vitamin E (*Cantor & Scott* 1974). Both effects were restored to normal by adding sodium selenite in a concentration of 0.1 mg/kg. *Latshaw et al.* (1977) and *Combs & Scott* (1979) showed that a minimum of 0.05 mg/kg feed is required to maintain normal egg produc-

tion in hens, while Se concentrations below 0.1 mg/kg lead to reduced activity of GSH-Px and reduced hatching of eggs.

*Petersen & Jensen* (1984) investigated the effect on hens of a Se supplementation of 0.1 mg/kg to a basal diet containing 0.1 to 0.2 mg/kg. No effects were observed on number and weight of eggs, on fertility of eggs or percent of eggs hatched. Furthermore, no influence on mortality of hens and chickens was observed. The Se concentrations in eggs was increased from 0.13 to 0.19 mg/kg (yolk + white). A negative effect of Se supplementation in turkeys was observed by *Høj* (1983), who found reduced fertility in animals fed diets containing 0.32 and 0.47 mg/kg compared to a control group on a diet with Se concentration of 0.2 mg/kg. No reports have shown that Se deficiency influences male reproduction in poultry (*Combs & Combs* 1986).

In poultry Se is essential to maintain normal egg production and high hatchability of eggs. A dietary concentration of 0.1 mg/kg seems necessary to hens and 0.2 mg/kg for turkeys. A concentration of 0.3 mg/kg or more may have negative effects.

**Fur animals:** Injections of 0.3 mg Se to bitches of the blue fox, according to *Jørgensen et al* (1987), have been shown to be without effect on fertility, number of liveborn whelps, whelp mortality, and on the incidence of endometritis. No influence on GSH-Px activity was found, which could indicate that the animals, prior to therapy, were in Se balance and thus excreted the single parenteral dose quickly without any biochemical effects. *Kuliev* (1984) found that intravenous injection of 0.1 mg Se/kg as sodium selenite to nutria every month from birth increased the weight of ovaria, the number of follicles and corpora lutea. Unfortunately this publication provides no information on basic Se status in the animals.

## Humans

For approximately 30 years it has been known that Se is essential to the spermiogenesis in experimental animals. In spite of this knowledge the interest in Se in relation to human fertility appeared only recently. *Bleau et al.* (1979) reported a mean normal Se sperm concentration of 190  $\mu\text{g/l}$  and found high concentrations related to low sperm motility and reduced number of live spermatozoa. The authors suggested that Se might be involved in certain cases of human infertility. In a later investigation (*Bleau et al.* 1984) of 125 men from childless couples a mean seminal Se concentration of  $71.3 \pm 29.7 \mu\text{g/l}$  (range 7–230  $\mu\text{g/l}$ ) was found. More than 85% of the total semen Se was found in the seminal plasma. A significant positive correlation between spermcount and sperm Se concentration was found. Motility was found to be highest when the seminal concentration was between 50 and 69  $\mu\text{g/l}$ . When concentrations were below or above this interval motility was decreased and the incidence of asthenospermia high. On the basis of these results *Bleau et al.* (1984) suggest a seminal Se concentration between 40 and 70  $\mu\text{g/l}$  to be optimal. When seminal concentrations were above 80  $\mu\text{g/l}$  they observed a high abortion rate and signs of ovarian dysfunction in women, based upon the assumption that husband and wife normally have the same intake of Se from diet and environment.

In a methodological study of trace element determination in seminal plasma and spermatozoa *Pleban & Mei* (1983) found in men admitted to a fertility clinic seminal Se plasma concentrations from 21 to 191  $\mu\text{g/l}$  and from 0.51 to 4.63  $\mu\text{g/l}$  in spermatozoa. *Saaranen et al.* (1986) found in a similar group of 70 Finnish men, a mean concentration in seminal fluid of  $33.4 \pm 14.1 \mu\text{g/l}$ . This was less than half the level detected in blood serum, which was  $78.2 \pm 9.9 \mu\text{g/l}$ . *Sustomaa et al.* (1987) have re-

ported a spermatozoal concentration of  $1.70 \pm 0.1 \mu\text{g/g}$  in a pooled sample. These results are in agreement with the findings of *Bleau et al.* (1984), but are considerably lower than those originally reported by *Bleau et al.* (1979), where the mean sperm concentration was reported to be  $190 \mu\text{g/l}$ . In the later findings they reported a seminal concentration of  $80 \mu\text{g/l}$  or more having a negative effect. The early report certainly indicates too high a concentration level, which might be a result of a methodological error, however, the possibility of differences in the intake of Se between the populations cannot be excluded.

*Takasaki et al.* (1987a,b) determined Se concentrations in seminal fluid, seminal plasma and spermatozoa in 32 healthy fertile men and in 73 infertile (62 had oligozoospermia and or asthenospermia, 11 had azoospermia). No differences among the groups were seen as regards total sperm and seminal plasma Se concentrations. In seminal fluid the mean concentration was  $80.1 \pm 21.6 \mu\text{g/l}$  in the fertile group v.s.  $77.1 \pm 25.2 \mu\text{g/l}$  in the infertile group, and the seminal plasma concentrations were  $70.4 \pm 18.6$  and  $69.5 \pm 21.9 \mu\text{g/l}$  respectively. In the spermatozoa the mean concentration in the fertile group was significantly lower compared to the infertile group ( $12.7 \pm 70 \mu\text{g}/10^8$  cells versus  $29.7 \pm 23.4 \mu\text{g}/10^8$  cells). In contrast to this *Vanha-Perttula et al.* (1988) did not find any differences between Se concentrations in spermatozoa from fertile ( $1.8 \pm 0.8 \mu\text{g Se/g d.w.}$ ) and from infertile men ( $1.3 \pm 0.6 \mu\text{g Se/g d.w.}$ ). *Takasaki et al.* (1987a,b) reported a positive significant correlation between seminal plasma concentration and spermatozoal density while a negative correlation was observed between spermatozoal Se concentration and density. Also the percentage of mobile spermatozoa was negatively correlated to the seminal plasma concentration. The authors suggested a connection between a high spermatozoal Se concentra-

tion and male infertility and suggested that Se exerts a negative effect on sperm motility and on spermiogenesis.

The results by *Takasaki et al.* (1987a,b) do not agree with the assumption made by *Bleau et al.* (1984) that a sperm concentration between 40 to  $80 \mu\text{g/l}$  is optimal as both most fertile and infertile men in the Japanese study were found to be within this interval. On the other hand the possibility that a low Se intake per se might influence fertility negatively cannot be excluded, thus *MacPherson et al.* (1994) found that a supplement of  $100 \mu\text{g Se}$  to young infertile men with documented low dietary selenium intake increased sperm mobility significantly compared to a placebo group.

*Roy et al.* (1990) found no significant correlation between Se level in the seminal plasma and sperm count or motility. However, in view of the known poor correlation of these semen parameters with the incidence of pregnancy the authors state that assessment of the fertilizing potential of normospermic ejaculates with low selenium concentration is warranted.

An interesting feature of the results reported by *Takasaki et al.* (1987a,b) is that the spermatozoal Se concentration in all fertile men investigated was below  $30 \text{ ng}/10^8$  cells, while one third of the infertile men had a higher cell concentration. As no difference between the 2 groups was found neither between full sperm nor seminal plasma concentrations, it may be assumed that the finding of high spermatozoal concentrations in some infertile men could be due to a defect in incorporation of Se into the MCP during spermatogenesis. Thus a high Se content, per se, is not necessarily the cause of infertility, but could be an indicator of a metabolic dysfunction being the primary causal factor in some types of infertility, since *Takasaki et al.* (1989) also observed increased Se content in spermatozoa from fertile men after denaturation (of whole semen) by heat.

In areas without reported Se deficiency the mean seminal Se concentration is reported to be between 70-80  $\mu\text{g/l}$ . Low as well as high Se concentrations in human sperm have been related to infertility. Higher spermatozoan Se concentrations have been found in some infertile men compared to fertile, which may indicate that a defect in Se incorporation in the sperm cells may be involved in some types of human infertility. From the existing literature no conclusions can be drawn, and more research on the role of selenium in human fertility seems to be needed.

### Conclusions

The essential function of Se as regards to mammalian male reproduction is well-documented with regard to experimental animals, and livestock. Se has an essential role as a component of a selenoprotein in the sperm mitochondria capsula (MCP). Se deficiency leads to deterioration of spermiogenesis and to decreased sperm motility. However, it seems not to influence the ability of the individual spermatozoa to fertilize the ova. In females Se does not seem to influence reproduction in a specific way, but should be looked upon as a nutritional factor, among many others, which may influence dietary status, and, through this, health and reproduction.

In experimental animals and in livestock supplied with Se in amounts commonly accepted as being adequate further supply will not improve the reproductive conditions. Overdosing even at a moderate level has proved to have negative effects.

The need for dietary Se supplementation seems to be much more well-defined for experimental animals and livestock than it is for humans. The diversity of human recommendations found in literature should probably be explained through the fact that, contrary to animals, human dietary

patterns vary to a great extent. As a consequence it may be assumed that also the need for supplements of antioxidants will vary both qualitatively and quantitatively. Furthermore, humans might be occupationally exposed to pro-oxidative components, which will increase the need for antioxidants. With regard to humans it seems likely that a defect incorporation of Se in sperm cells is a characteristic of some forms of male infertility.

There is a need for undertaking studies both in animals and humans to elucidate the relationship between dietary intake, optimal Se status and seminal (spermatozoal) concentration. There is also a strong need for a definition of a spermatozoal Se concentration, which is optimal for spermatogenesis and sperm motility taking into account various noxious environmental conditions. Finally there is a need for a further investigation as to whether high spermatozoal Se concentrations in some infertile men can be due to a defect in synthesis of polypeptides in the sperm mitochondria.

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**Sammendrag***Selen og fertilitet hos dyr og mennesker*

Litteraturen er gennemgået for at vurdere viden om selen i relation til fertilitet hos dyr og mennesker

Selen inkorporeres i et cysteinrigt polypeptid lokaliseret i spermatozoers mitochondriekapsel. Selen må derfor antages at være essentiel for normal spermato-genese hos såvel forsøgsdyr, husdyr som hos mennesker. Der er derimod meget begrænset viden om selen i relation til hunlig fertilitet

Hos forsøgsdyr påvirker lav selenstatus hanlig fertilitet, mens der kun har været begrænset opmærksomhed om selenstatus og hunlig reproduktions adfærd, og data tillader ikke at drage konklusioner. Der er udført talrige undersøgelser på husdyr, hvor effekt af se-

len ofte er bedømt i kombination med andre antioxidanter, men definitive konklusioner kan ikke udledes. Generelt vil tilførsel i henhold til fodernormerne sikre optimal reproduktionsevne hos såvel han- som hunde, mens ekstra tilskud synes at kunne medføre en negativ effekt.

Hos mennesker er der fundet modstridende resultater, idet såvel høje som lave spermakonzentrationer er fundet at have negativ indflydelse på antal af spermatozoer og på deres motilitet. Den optimale sperma selenkonzentration er endnu ikke defineret. Der er indikation for, at en metabolisk defekt i inkorporering af selen i spermatozoer kan relateres til human infertilitet. Der foreligger ingen data, der beskriver selen i relation til infertilitet hos kvinder.

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