Effects of Vitamin E and Selenium Supplements as Nutritional Additives on the Reproductive System: Metabolism of Vitamin E and Se and Their Effects on the Reproduction

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Abstract

There is substantial evidence indicating that selenium and vitamin E supplementation significantly improves reproductive problems associated with free radicals during prepartum (antepartum) and postpartum periods in both females and males. During reproductive periods, increased oxidative stress leads to an elevation in the production of reactive oxygen species (ROS). When this production surpasses the capacity of the antioxidants present in organisms to neutralize these reactive species, the importance of supplementation with antioxidants such as selenium and vitamin E becomes even more pronounced. The supplementation of selenium and vitamin E has been shown to have a significant impact on supporting reproductive performance, particularly in farm animals, by protecting reproductive cells from oxidative damage. In this context, these supplements may serve as an effective strategy to enhance reproductive health and reduce reproductive issues. This section provides a comprehensive overview of the current knowledge regarding the metabolism of vitamin E and selenium, as well as their effects on male and female reproductive systems. Vitamin E reduces cellular damage by maintaining the integrity of cell membranes and preventing lipid peroxidation, while selenium enhances the activity of antioxidant enzymes, thereby reducing oxidative stress.

Keywords: Antepartum, Postpartum, Reproductive periods, Supplementation, Metabolism, Peroxidation

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Introduction

Vitamin E supplementation is used as an effective strategy to improve reproductive functions, support health, and enhance traits in farm animals. After the significant role of vitamin E in animal reproduction was established, reproductive problems identified in different mammalian species were linked to vitamin E deficiency.

As a lipophilic antioxidant, vitamin E is commonly used as a dietary supplement due to its capacity to protect tissues from oxidative stress caused by free radicals. Vitamin E obtained from food and supplements is absorbed in the intestine along with lipids and transported to the liver with the help of alpha-tocopherol transfer protein. The end product of vitamin E metabolism is the water-soluble carboxyethyl hydroxychroman, which can be conjugated with hepatic metabolites and excreted via urine or feces. Vitamin E acts as a peroxyl radical scavenger, reacting with free radicals to form a tocopherol radical, thereby preventing the spread of free radicals in tissues. The tocopherol radical is reduced by a hydrogen donor, incorporating into cell membranes to protect them from oxidative damage.

The effect of vitamin E deficiency on the health of both male and female reproductive systems and reproductive cells has long been investigated. In addition to its positive effects on sperm viability, membrane integrity and motility, studies on various species have shown that vitamin E contributes to the ovarian cycle and fertilisation processes. In addition, the antioxidant properties of vitamin E have been shown to be effective on oocyte maturation, ovulation and luteal function (Mustari et al., 2022).

Minerals, which are naturally occurring inorganic solid substances with a specific chemical composition and crystal structure, function as cofactors in biochemical reactions for the activities of enzymes and hormones. The importance of minerals in maintaining the physiological functions of the female and male reproductive systems has been demonstrated in numerous studies (McDowell, 2003). Selenium, an essential mineral that is naturally found in soil, water, and certain foods, is one of the necessary trace elements, particularly recognized for its role as an important antioxidant in reproductive functions.

Animal diets contain varying amounts of organic selenium (particularly selenomethionine) and inorganic selenium, depending on the species. These selenium forms are absorbed from the intestine through different absorption mechanisms. Research has shown that soluble forms of selenium, including selenite, selenate, selenomethionine, and selenocysteine, are effectively absorbed from the intestine (Johnson et al., 2003; Mirone et al., 2013). Selenite is absorbed through a simple diffusion process, while selenate is actively absorbed in the ileum along with sodium ions. After absorption by the small intestine, selenium diffuses into various tissues and organs of the body and regulates important

biological functions by promoting selenoprotein synthesis. Different selenoproteins such as glutathione peroxidase (GPX), thioredoxin reductase (TrxR) and iodothyronine deiodinases (IDD) act as important antioxidants that prevent oxidative damage within the cell. Studies show that selenium supplementation provides positive effects on both female and male reproductive systems and endocrine functions, contributes to the maintenance of homeostasis in the ovarian cycle and spermatogenesis process, and selenium has therapeutic potential as a powerful antioxidant (Beckett & Arthur, 2005; Mistry et al., 2012; Mirone et al., 2013). Selenium has been noted to alleviate oxidative stress due to its role as a key component of glutathione peroxidase, which eliminates reactive oxygen species, including H_2O_2 and lipid hydroperoxides. It has also been reported to improve reproductive performance during the transition period and postpartum when combined with vitamin E (Kamİloğlu et al., 2005; Khatti et al., 2017).

This section evaluates data from studies regarding the effects of supplemented vitamin E and selenium on the metabolism and reproductive systems of both female and male subjects.

1. Vitamin E

Vitamin E was called 'factor X' or 'anti-fertility factor' because it was a dietary component necessary for reproduction in rats (Evans & Bishop, 1922). Later, this compound was included in the group of fat-soluble vitamins and named 'vitamin E' (Sure, 1924). Vitamin E is a family of compounds consisting of eight isomers:

- Four tocopherols (α-, β-, γ- and δ-tocopherols)
- Four tocotrienols (α-, β-, γ- and δ-tocotrienols).

Tocopherols are saturated forms of vitamin E, while tocotrienols are unsaturated forms with isoprenoid side chains. Since vitamin E cannot be synthesised by the body, it must be taken from outside through food. İts interaction with reactive oxygen species (ROS) resulting from oxidative stress, vitamin E has various positive biological effects in living organisms. With oral intake of vitamin E from natural sources, bile acids, cholesterol, phospholipids and triacylglycerols come together to form micelle structures. These micelles enable vitamin E to reach the intestinal lumen, where absorption takes place. After absorption, vitamin E combines with cholesterol, phospholipids and triacylglycerols to form kilomicrons while being transported through the lacteal vessels. Kilomicrons pass into the systemic blood circulation via the lymph circulation. In studies, it has been reported that the ratio of vitamin E isomers in kilomicrons is very close to the ratio of vitamin E isomers taken with food (Kono & Arai, 2015). Vitamin E reaches the liver and re-enters circulation via very low density lipoproteins (VLDL). α-Tocopherol is bound to α-tocopherol transfer protein (α-TTP) from the liver and enters the bloodstream. Vitamin E that is not bound to α-TTP is then excreted in the feces and urine. After VLDL containing vitamin E is released from the liver, it is stored in the tissues where it exhibits its antioxidant effect. α-Tocopherol is also transported in plasma bound to serum albumin (SA) (Fanali et al., 2013).

Cytochrome P450 (CYP) enzymes, primarily CYP4F2 and CYP3A4, regulate the initial stages of vitamin E metabolism and control the formation of the vitamin's breakdown products. Vitamin E isomers are metabolized by the cytochrome P450 (CYP)-catalyzed omega-hydroxylation process, followed by beta-oxidation of their side chains. Vitamin E is generally excreted from the body via two main routes: bile and urine (Schmölz et al., 2016).

There is no evidence of vitamin E toxicity. However, when taken in large quantities, it can pose serious bleeding risks. High levels of α-tocopherol have the potential to accelerate the lipid peroxidation process by acting as a pro-oxidant and this has been shown by in vitro studies (Schmölz et al., 2016).

1.1. Effects of Vitamin E on the Reproductive System

1.1.1. Effects of Vitamin E on the Female Reproductive System and Offspring Yield

Fertility and reproductive cycle in living organisms consists of complex stages including conception, maintenance of pregnancy and offspring, and problems in any of these stages result in decreased offspring yield or infertility. Researchers have reported that vitamin E may have a promising potential effect that may improve fertility and reproductive health in females (Foote, 2003; Wood, 2017).

Vitamin E, as a strong antioxidant, is a very important nutrient in the early stages of life from the beginning of pregnancy to the postnatal development of the offspring, since it can pass to the foetus through the placenta and to the newborn with colostrum and milk. Lipoprotein receptors such as LDL receptor, VLDL receptor, scavenger receptor class B type-I and lipoprotein lipase play a role in its absorption in placental and mammary tissue. In addition, the alpha-tocopherol transfer protein has an important function in the selective transfer of alpha-tocopherol from the placenta. Umbilical cord blood contains lower concentrations of alpha-tocopherol compared to maternal circulation. Therefore, colostrum containing very high levels of vitamin E is important as an essential means of defence against oxidative stress in the newborn (Beytut et al., 2003; Kamİloğlu et al., 2005).

Optimal levels of vitamin E to be used in reproductive and gestational animals are not clear due to various factors such as the composition of the diet, the amount of feed consumed, the growth rate of the offspring and environmental or stress conditions. Several studies have shown that increasing dietary vitamin E supplementation during pregnancy or intramuscular injection of vitamin E and/or selenium increases pregnancy incidence, litter size and development, and decreases mortality in the pre-weaning period (Lohmiller et al., 2020; Joshi, 2022).

Embryo implantation is a vital process for the successful conception of a pregnancy and the pregnancy can be lost before, during or after implantation. There are complex hormonal processes between the embryo and the endometrium. Any mismatch between embryo development and the differentiation process of the uterus can lead to implantation not taking place. It is also thought that errors during embryo implantation and early placenta formation can lead to miscarriages and various pregnancy-related complications. Vitamin E may have a protective effect on both mother and foetus during pregnancy and may reduce the risk of pregnancy-related complications because of its radical scavenger activity. In addition, there are findings showing that vitamin E levels in maternal blood increase oxidative balance during healthy pregnancies. Vitamin E deficiency during the reproductive process causes problems such as loss of offspring, premature birth, preeclampsia and restriction of intrauterine offspring growth. It is known that plasma vitamin E levels decrease in recurrent pregnancy loss. Vitamin E supplementation is reported to

improve endometrial thickness, plasma MDA levels, IL-1 and TNF-α gene expression in patients with implantation failure (Hashemi et al., 2019).

Dairy cattle need vitamin E supplements to prevent problems such as placental retention, metritis and cystic ovary development during the prepartum period. 1 g of vitamin E supplement per day can meet the minimum vitamin E requirement during this period.

1.1.2. Effects of Vitamin E on the Male Reproductive System

It has been shown that vitamin E supplementation is an effective strategy for supporting the growth and health of farm animals, as well as improving their qualitative traits and reproductive functions. Germ cells are particularly sensitive to oxidative damage and may require additional antioxidant protection. Vitamin E is a potent exogenous antioxidant that breaks the chain reaction of lipid peroxidation in membranes by scavenging peroxyl (ROO•) and alkoxyl (RO•) radicals. Lipid peroxides (LOO•) are highly cytotoxic to spermatozoa and disrupt the functioning of ion channels in the cell membrane, which is essential for maintaining normal sperm motility (Halliwell & Gutteridge, 2015).

The sperm plasma membrane is specialised for oocyte fertilisation and develops the capacitation, acrosome reaction and fusion response with the oocyte. Oxidative stress in the testes is one of the main causes of apoptosis in germ cells. Therefore, strengthening the defence mechanism against oxidative stress is crucial for the maintenance of spermatogenesis and prevention of testicular atrophy. High-capacity antioxidants such as vitamin E protect germ cells against oxidative DNA damage and play an important role in spermatogenesis. Vitamin E, a powerful antioxidant, is believed to maintain the balance of lipid peroxidation levels to protect the membrane function of sperm. Research has shown that vitamin E improves spermatogenesis, sperm quality, and the feedback regulation of hormones involved in spermatogenesis, while also supporting antioxidant defense and immune response (Amevor et al., 2022).

The protective effect of vitamin E against oxidative damage in sperm cells becomes more significant when the reproductive systems of animals are susceptible to infections due to inadequate hygienic conditions, leading to weakened antioxidant defenses. It has been demonstrated that infection conditions can impair the antioxidant defense of the reproductive system, exacerbating oxidative stress and negatively affecting testicular functions and semen characteristics (Potts & Pasqualotto, 2003).

Although there are studies on the role of vitamin E in male reproductive performance, the results on semen quality and sperm motility still seem to be controversial. Semen contains various antioxidant compounds and defence mechanisms, whereas spermatozoa have very few antioxidant molecules. Therefore, it is considered that vitamin E supplementation may reduce unfavourable stress conditions and maintain sperm quality. Dietary supplementation of vitamin E in rams, rabbits and male pigs has been reported to increase epididymis weight, seminiferous tubule number (Holsinger et al., 2009) and positively affect semen qualitative characteristics and sperm motility (Mahmoud et al., 2013).

2. Selenıum

Selenium is a vital trace element. It fulfils many critical functions by being involved in the structure of more than 30 selenoproteins found in the body. These selenoproteins help prevent the formation of various chronic diseases by inhibiting cellular damage caused by free radicals. Selenium can exist in four different oxidation states (Brown & Arthur, 2001):

- Elemental Selenium (Se^0),
- Selenide (Se^{-2}),
- Selenite (Se^{+4}) and
- Selenate (Se^{+6})

These oxidation forms are present in various organic and inorganic matrices. Selenite and selenate, which are naturally soluble inorganic species, account for a large proportion of the total selenium in the environment (Oldfield, 2002). The main source of selenium is rocks and soils on earth. The amount of selenium in soils generally varies between 0.1 and 2 µg/g; however, this distribution is quite heterogeneous. Some areas have selenium-rich soils (about 100 µg/g), while in other areas there is little or no selenium in the soil (0.1 µg/g or less) (Mangiapane et al., 2014). Selenium compounds in organic form consist mainly of selenium-containing amino acids. Selenium is contained in the structure of the amino acids selenocysteine and selenomethionine. Selenium-containing amino acids are vital for the synthesis of selenium-containing proteins and peptides. While selenocysteine is the most common form of selenium in animal tissues, selenomethionine is the main compound used in the synthesis of selenium-containing peptides (SePs) in plants, algae, yeasts and bacteria. Regulation of SeP synthesis is important for understanding selenium homeostasis and disorders resulting from its disruption. Cellular selenium concentration is the primary regulator of selenium incorporation into SePs and occurs primarily in response to changes in selenium availability. Ingested selenomethionine can be incorporated into the protein structure in an incidental manner by substituting methionine, or it can be converted to selenocysteine by trans-sulphurisation in the liver and kidney and used in the biological selenium pool. Among the functionally active selenoproteins (SeP) in humans and animals, no selenomethionine-containing proteins are found. Only genetically encoded proteins that fulfil basic biological functions are considered as selenoproteins (SeP). Some of these SePs are glutathione peroxidase, thioredoxin reductase and deiodinase, which are antioxidant enzymes involved in the activation of thyroid hormones. In addition, selenoprotein P acts as a carrier protein that transports selenium between the liver and other organs (Sobolev et al., 2018). Selenium metabolism in living organisms is a complex process of selenium uptake, absorption, metabolic transformation and excretion. Dietary selenium is absorbed in the small intestine, where it is converted into components such as selenomethionine and selenocysteine. These forms help prevent cellular damage, especially in the structure of antioxidant enzymes. The selenium requirement of animals varies depending on species, age and dietary conditions. Even a slight deviation from normal Se levels can have serious consequences. Ruminants generally require 0.1 mg/kg Se, while poultry require 0.05-0.15 mg/kg and pigs require 0.1-0.3 mg/kg (Council et al., 2012; Ahsan et al., 2014).

Selenium from dietary organic and inorganic sources can be used in the synthesis of selenoproteins, stored or excreted from the body after absorption from the intestine. Approximately 20 different biologically active selenoproteins have been identified and these are usually redox enzymes containing selenocysteine residues at their active sites (Spears, 2003). Selenium is stored in liver, kidney and muscle tissues

and excess selenium in the body is excreted in the urine.

The ability of ruminants to absorb selenite or selenate from their diet is very low due to the highly reducing rumen environment. Since rumen microorganisms convert most of the dietary inorganic selenium into unabsorbable elemental or inorganic selenide forms, the absorption rate of selenium from selenite is about 25-30%. The role of rumen microorganisms in selenium metabolism is similar in many ways to how microbes affect dietary protein quality. Bacteria have the ability to synthesize methionine, cysteine, and selenium analogs such as selenomethionine and selenocysteine; these selenoamino acids are incorporated into microbial protein. This process enriches the selenium content of rumen microorganisms. It has been shown that ruminants can convert inorganic selenium supplements into a metabolizable form and that rumen microorganisms tend to utilize the organic form of selenium (Mehdi & Dufrasne, 2016; Hendawy et al., 2021).

Selenium deficiency leads to physiopathological changes in farm animals, resulting in significant financial losses each year. In particular, selenium deficiency can cause health issues such as muscular diseases and immune system disorders, especially in ruminants. Therefore, adequate selenium intake is critical for the healthy development of animals and the strengthening of their immune systems (Jacques, 2001).

Selenium (Se) plays structural and enzymatic roles, affecting reproductive function through its catalytic and antioxidant activities in both males and females. Selenoproteins are essential for reproduction, and silencing of the selenocysteine-tRNA gene in females causes embryonic lethality (De Rosemond et al., 2005).

Glutathione peroxidase is known to play a significant role in female reproductive function, influencing follicle growth, maturation, and dominance. Additionally, through its potential antioxidant role, it protects the growing follicle from increased reactive oxygen species (ROS), preventing cell apoptosis due to oxidative stress and contributing to the determination of the dominant follicle. Glutathione peroxidase is a key enzyme that reduces H_2O_2 in the endometrium during the implantation process in the postovulatory phase, serving as a defense against oxidative stress. It also plays a role in protecting the embryo and extra-embryonic tissues against ROS and is involved in maternal-fetal selenium transfer mechanisms (Colakoglu et al., 2017).

Selenium is found in high concentrations in the testis, where it is important for sperm motility and viability. It plays a role as an antioxidant and H_2O_2 scavenger in maintaining the integrity of the sperm cells during sperm maturation and ultimately protects the embryo from oxidative damage that could jeopardise its viability. In male reproduction, the cysteine-rich structural protein of mitochondrial capsules, which stabilises the keratinous outer layer of mitochondria in the sperm midpiece was thought to be a selenoprotein. However, it is also considered that this mitochondrial capsule selenoprotein is not responsible for selenium-dependent sperm function (Ahsan et al., 2014).

Selenium is an important micronutrient in the body and plays a critical role, especially for male reproductive health. This element has a significant impact on sperm production and quality; in the case of selenium deficiency, sperm motility may be reduced, which negatively affects the ability to fertilise. Selenium is also associated with testosterone production and low levels can lead to hormonal imbalances, causing problems such as sexual reluctance. Thanks to its antioxidant properties, selenium protects cells from oxidative damage by being present in the structure of enzymes that neutralise free radicals (e.g. glutathione peroxidase), which supports the health of reproductive cells. Furthermore, selenium's positive effects on the immune system indirectly strengthen reproductive health by increasing resistance to infections. It also has positive effects on embryonic development and foetal growth, and selenium deficiency can lead to birth defects and low birth weight. Therefore, ensuring adequate selenium intake is vital to support reproductive health (Rayman, 2000; Combs Jr, 2001).

Low selenium levels are associated with problems such as labour problems due to reduced tension of the uterine muscular layer, postpartum paraplegia, placental retention and purulent inflammation of the uterine lining. In addition, selenium deficiency leads to the formation of ovarian cysts and increased embryonic mortality in the first 3-4 weeks after insemination.

Selenium poisoning is rare and is usually caused by an overdose of selenium supplements. The most common forms of selenosis are chronic selenosis, known as alkalosis, and acute selenosis, colloquially referred to as blind intoxication (Zarczynska et al., 2013).

2.1. Selenium in the Female Reproductive System and Offspring Yield

Selenium is a micronutrient that has important effects on the female reproductive system and offspring production. In the female reproductive system, selenium deficiency has been reported to adversely affect the ovulation process and reduce egg quality, which in turn reduces fertilisation rates and weakens the pregnancy rate. In addition, selenium is critical for foetal development; selenium deficiency is reported to cause birth defects and low birth weight. Selenium's activation of antioxidant enzyme systems is also known to support the pregnancy process by reducing cellular damage in both mother and foetus. In addition, it has been shown that selenium can strengthen the immune system, reduce the risk of infection and thus contribute to the healthy progress of the pregnancy process. In this context, adequate levels of selenium have an important role in increasing female reproductive health and offspring yield (Rayman, 2000; Arthur et al., 2003; Rutigliano et al., 2008). It has been shown that 5 ng/mL supplementation of sodium selenite triggers proliferation of bovine granulosa cells and improves the proliferation rate, as well as increasing oocyte growth and stimulating E2 synthesis (Basini & Tamanini, 2000).

In vitro, it has been shown that selenium reduces the harmful effects of reactive oxygen species (ROS) in bovine luteal cells, and GPX mimics the effect of follicle-stimulating hormone (FSH) by suppressing apoptosis in rat ovarian follicles. In addition, selenium deficiency causes degeneration in ovarian tissue and follicular atresia in rats. In bovine ovarian tissue, selenium has been found to be concentrated particularly in the granulosa cell layer of large follicles. It has been reported that there is approximately ten times more selenium in bovine follicle walls compared to the corpus luteum, and that the selenoprotein gene GPX1 in granulosa cells is up-regulated in large follicles compared to small or atretic follicles (Ceko et al., 2015).

2.1.2. Selenium in the Male Reproductive System

Among the factors affecting animal reproductive performance, such as genetics, nutrition, management, and environment, selenium holds significant importance (Mistry et al., 2012). Selenium is critical for testosterone biosynthesis and the normal development of spermatozoa (Flohé, 2007). High levels of selenium found in testicular tissue are primarily in the form of GPx4 (glutathione peroxidase 4). This establishes

a significant link between selenium and sperm quality as well as male fertility, since GPx4 is a key component that determines the structure of the midpiece of the sperm cell and protects developing sperm cells from oxidative DNA damage.

Reactive oxygen species (ROS) can damage the sperm membrane, reducing sperm viability and leading to male infertility (Beckett & Arthur, 2005; Safarinejad & Safarinejad, 2009). Research has shown that increasing dietary selenium intake can enhance male fertility by boosting antioxidant GPx activity. It has been reported that in human semen, the optimal range for selenium levels is between 50-60 µg/mL, and there is a positive relationship between sperm count and semen selenium concentration (Shahin et al., 2021).

In quails, selenium supplementation has been shown to increase accumulation in testis and ovarian tissues in a concentration-dependent manner. Nano-Se supplementation significantly enhanced sexual behaviors and markedly increased plasma testosterone levels and sperm quality characteristics (sperm count, motility, and viability) even at low supplementation concentrations of 0.1 mg/kg (El-Kazaz et al., 2020).

Furthermore, both organic and inorganic selenium supplementation at 10 mg has been demonstrated to have significant effects on sperm parameters in buffalo bulls over a three-month supplementation period, resulting in increases in parameters such as ejaculate volume, mass motility, progressive motility, sperm concentration/mL, and sperm output per ejaculate (El-Sharawy et al., 2017).

There is a favourable interaction between selenium and vitamin E that supports each other. Both nutrients act as important cellular antioxidants that protect cells from the harmful effects of hydrogen peroxide and other peroxides derived from fatty acids. Selenium, as part of the glutathione peroxidase (GSH-Px) enzyme in the cytosol, is involved in the reduction of peroxides, whereas vitamin E functions as a specific lipid-soluble antioxidant in cell membranes. Therefore, GSH-Px neutralises peroxides before they damage the cell membrane, while vitamin E prevents chain auto-oxidation that may occur in membrane lipids and creates a protective effect on the cell membrane (Kamİloğlu et al., 2005). The relationship between vitamin E and selenium is not limited to their antioxidant function. Deficiency of both nutrients can lead to the emergence of various diseases. There is also scientific evidence that some diseases associated with selenium deficiency respond favourably to vitamin E supplementation or treatment, and that some conditions associated with vitamin E deficiency can be improved by selenium supplementation (Johnson et al., 2003).

In all farm animals, problems in reproductive performance in both males and females are associated with selenium deficiency. Reproductive problems due to selenium deficiency in farm animals can be listed as follows:

- Irregular, weak or silent heat periods, delayed fertilisation, low pregnancy rates and cystic ovary formation (Corah & Ives, 1991).
- Decreased sperm motility (McKenzie et al., 1998).
- Decreased uterine contractions (Segerson & Libby, 1982).
- Increased incidence of mastitis (Olson, 1994).
- Failure to expel foetal membranes (RFM) (Trinder et al., 1969; Campbell & Miller, 1998).

Among these reproductive problems, retained foetal membranes (RFM) is one of the most frequently reported disorders (Olson, 1996).

Conclusion

Reproductive problems seen in both male and female farm animals have revealed the importance of some micronutrients in their solution. Vitamin E and Se are essential micronutrients for reproductive system. Positive correlations have been reported between the deficiency of micronutrients such as vitamin E and selenium and female problems such as irregular estrus, decreased fertilisation rate, cystic ovary, early embryo mortality, impaired uterine movements, problems in sperm production, testosterone synthesis and transport, and impaired secretory activities of appendage glands. For these reasons, eliminating micronutrient deficiencies in animals is important for farm management. Micronutrient strategies of farms are important for both protecting the farm and achieving optimum levels in production quality. Studies on the positive or negative effects of micronutrient intake or lack of micronutrients by living organisms have revealed their reproductive relationship in many aspects, but there are still some points that have not yet been determined. This review may contribute to the research on the place and importance of micronutrients in male and female reproductive systems.

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