

Selenium in cattle diseases and reproductive health

Carol Majors,[‡] Andrew Myers,[†] Ramanathan Kasimanickam

College of Veterinary Medicine, Washington State University, Pullman, WA, USA

[‡]Current address: Soda Springs Animal Clinic, Soda Springs, ID, USA

[†]Current address: Verona Veterinary Medical Service, Verona, WI, USA

Abstract

Selenium (Se) is a trace mineral critical for antioxidant activity, immune function, thyroid hormone conversion, fetal development, and reproduction. The goal is to emphasize the importance of preventing Se deficiency in cattle through year-round supplementation, soil analysis, and appropriate treatments to avoid conditions like white muscle disease, nutritional muscular dystrophy, and reproductive issues. Certain US regions have Se-deficient soils that may produce Se-deficient forages. In these areas, Se deficiency-related syndromes are common. Cattle require 0.1 mg/kg of Se for maintenance and more during pregnancy or lactation. Across age groups, deficiency results in various clinical manifestations, including white muscle disease in neonates, nutritional muscular dystrophy in calves and yearlings, and reproductive dysfunction in adults. Diagnosis involves soil and pasture analyses, Se concentrations in blood and serum, erythrocyte glutathione peroxidase activity, liver biopsies, or tissue recovery at slaughter or necropsy. Treatment is generally ineffective for neonates, but can be successful in older cattle, with injectable Se and ongoing supplementation being key. Prevention through year-round supplementation is crucial in Se-deficient regions.

Keywords: Nutrition, selenium, food animal, health, reproduction

Introduction

Selenium (Se) is a trace element with important roles in animal health and performance. In cattle, Se deficiency can have economically important impacts such as reduced fertility, retained fetal membranes (RFM) and increased mastitis and metritis.¹ This review includes the importance of meeting Se requirements for cattle, how to recognize clinical manifestations across age groups, and using year-round Se supplementation to prevent deficiency-related conditions, such as enzootic (nutritional) muscular dystrophy (NMD) and reproductive dysfunction in adult cattle.^{2,3}

Selenium is a naturally occurring trace mineral with a critical role in bovine health; however, its availability in soil and forage varies widely across the US. Areas with Se-deficient soils (e.g. the Pacific Northwest) are at particularly high risk for Se deficiency in cattle, leading to a range of issues, from neonatal diseases to reproductive and metabolic problems in adults.⁴ Understanding the role of Se in cattle, clinical signs of deficiency, and methods for diagnosis, prevention, and treatment, are essential for effective herd management.

There is considerable variation in soil Se concentrations across the continental US (Figure 1),⁵ with markedly lower soil Se concentrations in the Northwest, Northeast, Southeast, and Great Lakes states. Soils with concentrations < 0.05 ppm are considered critically low, whereas concentrations above this threshold are generally considered adequate. However, Se deficiency does not depend solely on soil Se concentrations, as deficiency in cattle occurs because of Se absorbed by plants that is influenced by soil pH and availability of Se compounds in soil.

Critically low soil Se concentrations lead to forages with < 0.1 ppm Se in > 80% of cases, often resulting in deficiencies in unsupplemented cattle. In areas with variable soil Se concentrations (Figure 2),⁶ forages are roughly split between deficient and adequate; therefore, some cattle may still develop Se deficiencies despite adequate soil concentrations.

As forages obtain their Se from soil, the distribution of Se-deficient areas in cattle generally mirrors the distribution of soil Se concentrations, albeit not perfectly. Selenium-deficient soils substantially increase the risk, but do not guarantee Se-deficient cattle.

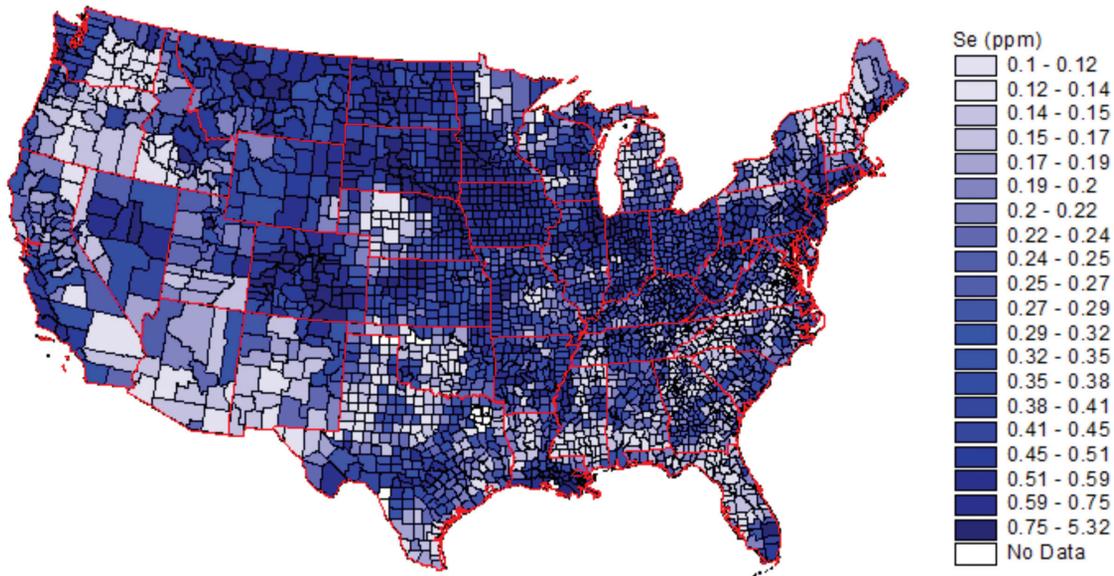


Figure 1. Color-coded map of relative Se concentrations in soil samples for counties in the continental US. Soil Se < 0.05 ppm is critically low; higher values are considered adequate, depending on soil pH and Se availability to plants^{4,9}

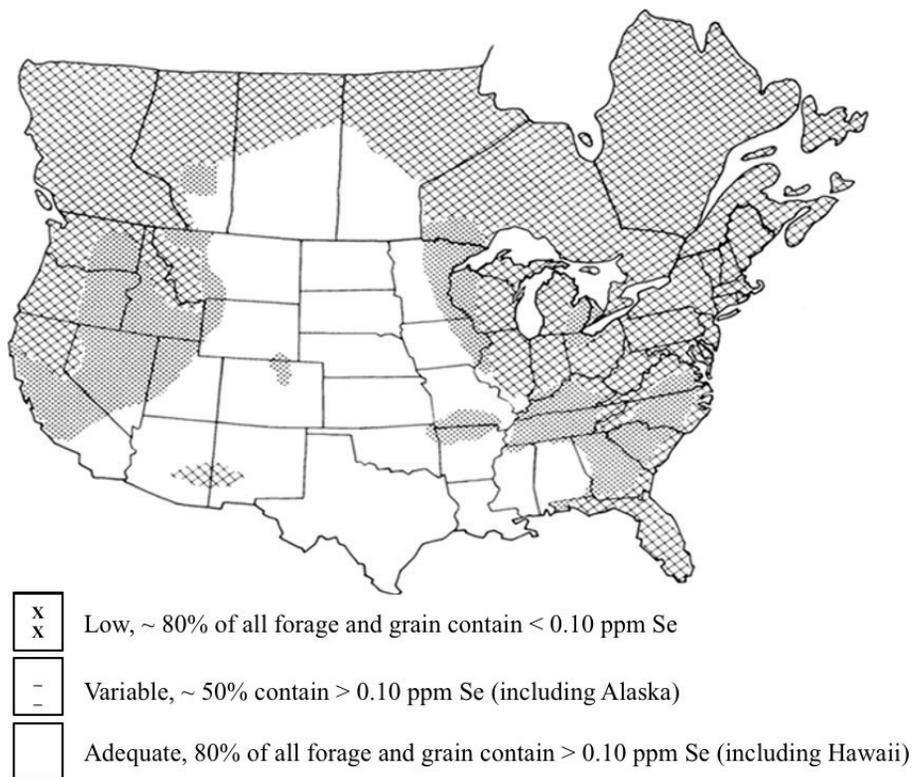


Figure 2. Distribution of forages generally deficient, relatively low, and adequate in Se⁶

Selenium deficiency in south Atlantic seaboard's soil is largely attributed to coastal deposits washed from highly weathered land masses. The northeastern states' soil predates cretaceous period selenization; seleniferous cretaceous shales did not weather in these areas, resulting in low soil Se concentrations. Much of these cretaceous sedimentary shale rocks weathered into the central and northcentral states from Montana and North Dakota, through Wyoming, Colorado, and South

Dakota, resulting in soils that produce forages with variable or adequate Se.⁷

Washington state is riddled with volcanoes; 5 are active and have erupted relatively recently, with volcanic deposits creating very low soil Se concentrations.⁷ Current agricultural practices (e.g. intensive cropping) can further decrease soil Se concentrations.

Selenium requirements

Daily Se requirements for cattle are 100 µg/kg dry matter (DM) for beef cattle, 300 µg/kg DM for dairy cows and 100 µg/kg DM for calves.¹ There is a close link between Se and vitamin E status, as well as antioxidant status.⁸ Vitamin E has a key role in Se absorption and utilization.⁹ Selenium and vitamin E have interdependent antioxidant functions; therefore, low vitamin E intake may increase the Se required to prevent some conditions.⁹

The National Academies of Sciences, Engineering, and Medicine (NASEM; formerly National Research Council, the operating arm of the NASEM) recommends a daily intake of 15-60 IU of vitamin E for adult cattle, whereas nursing calves require 40-60 IU.^{10,11} Deficiencies in either Se or vitamin E can impair thyroid metabolism, decrease growth rate, reduce fertility, alter phagocytic response, and lower disease resistance.¹²

Certain dietary components can negatively impact Se absorption in cattle. Diets rich in carbohydrates, nitrates, sulfates, calcium, or hydrogen cyanide (e.g. from clover forage or flax seeds) can interfere with Se utilization. Sulfur competes with Se for absorption at concentrations > 2.4 g/kg DM. Similarly, ferric iron can reduce Se absorption by forming insoluble complexes that are not assimilated by enterocytes.¹³

Calcium also influences Se absorption; concentration of 0.8% DM in the feed supports optimal Se absorption in dairy cows during late pregnancy.¹⁴ However, calcium supplementation in beef cattle, especially at concentrations typically used in intensive production, significantly reduces muscle Se concentrations.¹⁵ Additionally, high concentrations of lead in a calf diet can decrease both serum and tissue Se concentrations.¹⁶ It should also be noted that any lead exposure sufficient to affect Se concentrations is likely to present a food safety risk. The primary concern for the practitioner should be the adulterated meat from these animals, rather than Se deficiency.

Iodine deficiency can exacerbate Se deficiency.¹⁷ In cattle, iodine deficiency leads to marked induction of selenoprotein-D1, accompanied by elevated glutathione peroxidase (GPX) activity. Finally, diets containing adequate levels of crude protein and cellulose can enhance animal's ability to absorb and utilize Se more efficiently.

Selenium intake without supplementation

Most Se for beef cattle is obtained through forage, with soluble Se compounds in water a much poorer source.¹⁵ In areas with moderately low or variable soil Se concentrations, certain plants, (e.g. alfalfa) incorporate more Se than other forages (e.g. red clover, timothy, or brome grasses).¹⁸ Therefore, feeding alfalfa in these regions can reduce the need for supplementation. Additionally, neutral or acidic soils can impair a plant's ability to uptake Se. Forage testing is an important tool in evaluating herd Se status.¹⁹

Organic Se compounds in plant matter are easily absorbed by the gastrointestinal tract and have more prolonged retention. In contrast, inorganic Se (e.g. selenate) in water is readily absorbed but also rapidly excreted. Excess Se is primarily excreted in urine, with lesser amounts excreted in feces and breath. Selenium is mainly stored in the liver and, to a lesser extent, in the kidney.¹⁹

Selenium utilization

Selenium is present in nearly all tissues, but it is most concentrated in tissues that are rapidly breaking down, growing, or dividing, including heart, skeletal muscle, ovaries, and testes. It is also present in immune system tissues (including bone marrow, thymus, liver, intestines, spleen, and lymph nodes), thyroid, and other organs.²⁰ There are many selenoproteins, each with a specific location and function. Selenoproteins involved in reproduction are listed (Table 1).^{21,22}

Selenium has a crucial role as a functional component of a group of antioxidants known as GPX,^{23,24} a water-soluble cytosolic enzyme that protects cellular membranes and membranes of lipid-containing organelles, from irreversible damage caused by peroxides. Vitamin E has a similar function, but as a lipid-soluble antioxidant in cell and organelle membranes. Peroxides can denature cellular proteins, causing cell degeneration and death. Glutathione peroxidase catalyzes breakdown of hydrogen peroxide (H₂O₂) and related hydroperoxides.^{23,24} Based on a cell membrane as the central structure, the role of oxidative stress (OS), generation of reactive oxygen species (ROS), antioxidant defenses, and lipid peroxidation are summarized (Figure 3).

Cell membrane and phospholipids

The cell membrane's phospholipid bilayer, with hydrophilic heads and hydrophobic tails, maintains membrane integrity and selective permeability.²⁵ Unsaturated fatty acids in the tails create bends that promote fluidity, crucial for membrane flexibility, protein movement, and communication. Lipid peroxidation from ROS damages the membrane, triggering inflammation and cell death if uncontrolled.²⁶

Reactive oxidative stress generation

Mitochondria are the main source of ROS during ATP production, leaking electrons that form superoxide anions (O₂•⁻), which convert to H₂O₂.^{28,29} NADPH oxidases and enzymes like cytochrome P450 and lipoxygenases also contribute to ROS production, particularly in immune responses and inflammation.³⁰⁻³²

Enzyme actions in reactive oxidative stress detoxification

Enzymes like GPX, superoxide dismutase (SOD), and catalase protect cells by neutralizing ROS. Glutathione peroxidase reduces lipid peroxides, SOD converts superoxides to H₂O₂, and catalase converts H₂O₂ to water and oxygen, preventing oxidative damage.^{27,33,34}

Micronutrient protection against reactive oxidative stress

Vitamin E, a fat-soluble antioxidant, protects membrane lipids from oxidative damage.^{36,37,38} Vitamin C scavenges ROS and regenerates antioxidants,^{35,36} whereas CoQ10 neutralizes lipid-soluble ROS and supports mitochondrial function and ATP production.³⁸

Oxidative stress and membrane damage

Oxidative stress occurs when ROS production exceeds antioxidant defenses, causing membrane damage, destabilizing fluidity, and impairing function.³⁹ This can lead to protein

Table 1. Selenoproteins, principal location, and functions

Nomenclature	Selenoprotein	Location	Function
GPX1	Glutathione peroxidase 1	Follicle	Antioxidant protection
GPX2	Glutathione peroxidase 2	Embryo	Antioxidant protection
GPX3	Glutathione peroxidase 3	Follicle, endometrium	Antioxidant protection
GPX4	Glutathione peroxidase 4	Embryo, testis, sperm mitochondrial sheath	Antioxidant protection
GPX5	Glutathione peroxidase 5	Epididymis, sperm	Antioxidant protection
TXNRD1	Thioredoxin reductase 1	Embryo	Antioxidant role, redox regulation, cell signaling
TXNRD2	Thioredoxin reductase 2	Embryo	Antioxidant role, redox regulation, cell signaling
TXNRD3	Thioredoxin-glutathione reductase	Sperm	Antioxidant role, redox regulation, cell signaling
DIO1,2 &3	Iodothyronine deiodinase 1, 2, and 3	Utero-placental unit	T3 production
SELENOH	Selenoprotein H	Trophoblast, placenta	Glutathione synthesis
SELENOP	Selenoprotein P	Seminal plasma, utero-placental unit	Se transport, antioxidant
MCSep	Mitochondrial selenoprotein	Sperm mitochondrial capsule	Store for GPX4
TGR	Thioredoxin-glutathione reductase	Spermatid	Antioxidant

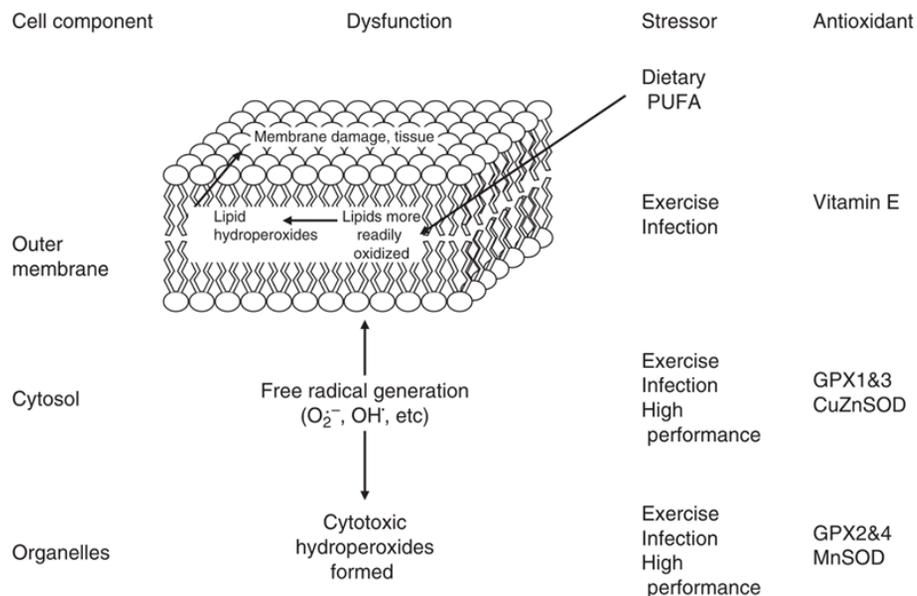


Figure 3. Schematic presentation of cell membrane oxidative metabolism/antioxidant interactions, indicating how GPX 1, 2, and 4 and other micronutrients protect membranes and organelles from peroxidation^{23,27}

dysfunction, increased permeability, and cell death.⁴⁰ Antioxidants and membrane repair mechanisms help prevent and restore membrane integrity.

Role of selenium in immunity

Selenium has a crucial role in maintaining immune function and supporting defenses against infections. As a

key antioxidant, it is integral to several cellular processes that regulate immune responses, particularly through its incorporation into enzymes like GPX that manages OS and inflammation.⁴¹ Given the close relationship between Se and vitamin E, it is not surprising that many Se-responsive diseases may also respond to vitamin E supplementation. Selenium's role in the immune system is summarized below.

Cell-mediated and humoral immunity

Selenium, in conjunction with vitamin E, is essential for both cell-mediated and humoral immunity. Deficiencies in Se compromise resistance to infections, reduce neutrophil function, and decrease antibody production. These deficiencies affect proliferation of T and B lymphocytes in response to mitogens and impair the function of natural killer cells.⁴²

Neutrophil function

Neutrophils have a key role in the innate immune response and rely heavily on Se to maintain high GPX enzyme activity.⁴³ This enzyme is vital for managing OS during phagocytosis (process by which neutrophils engulf and kill pathogens). Without adequate Se, neutrophils have a reduced ability to eliminate pathogens and manage peroxides generated during this process.^{43,44}

Selenium supplementation in colostrum significantly increased IgG concentrations in calves⁴⁵; a single feeding of colostrum supplemented with 3.0 ppm Se caused a 40% increase in peripheral IgG that persisted at least 2 weeks, implying a prolonged benefit to immunity. Increases in IgG were attributed to Se's ability to promote pinocytosis (the process by which cells absorb antibodies from colostrum) within intestinal epithelial cells.⁴⁶ This mechanism ensures more efficient absorption of immunoglobulins, providing calves with better passive immunity.

Improved humoral response

Selenium supplementation enhanced the humoral immune response in cattle experimentally infected with the infectious bovine rhinotracheitis virus.⁴⁷ Selenium-supplemented cattle produced more antibodies and had a stronger immune response compared to control groups.

Although Se deficiency may increase susceptibility to infections, naturally Se-deficient herds have not had a consistent increase in infection rates or severity.^{45,48} In addition to Se, other factors also influence disease incidence.

Selenium is transferred from dam to fetus during pregnancy, ensuring a calf is born with adequate Se. Prenatal Se supplementation in dams helps produce Se-sufficient calves and improves immune status at birth.⁴⁹ After birth, Se concentrations in calves deplete rapidly, typically within 18 days. However, the initial boost in Se from the dam may provide early-life protection against neonatal morbidity and disease. Milk Se concentrations are highly correlated with maternal blood Se concentrations. Selenium-adequate dams can provide Se-sufficient milk to their calves until weaning, supporting calf's immune system until they are able to consume solid feed.^{50,51} In cattle, Se supplementation can enhance passive immunity (IgG transfer), improve immune responses during infections, and provide essential nutrients to offspring through placenta and milk. Ensuring dams and calves are sufficient to promote a strong immune system, reduces susceptibility to infections and improves herd health.

Role of selenium in thyroid function

In cattle, Se has a critical role in thyroid function by supporting synthesis, metabolism, and regulation of thyroid

hormones.⁵² Thyroid gland produces hormones, primarily thyroxine (T4) and triiodothyronine (T3) that regulate metabolism, growth, and development. Selenium is essential for enzymes involved in the activation and deactivation of these hormones. Selenium is incorporated into several selenoproteins that are crucial for thyroid function, most notably the deiodinase enzymes, in cattle, mice, and humans.^{52,53} These enzymes are responsible for the activation and inactivation of thyroid hormones:

Type 1 deiodinase: This enzyme converts T4 into the more active T3 that is essential for regulating metabolism and energy balance; it is highly active in liver and kidneys.

Type 2 deiodinase: This enzyme converts T4 to T3, but it is mainly active in the brain, pituitary, and brown adipose tissue. In the brain, it has an important role in regulating local concentrations of T3 to support neurodevelopment and cognition.

Type 3 deiodinase: This enzyme converts T4 and T3 to inactive forms; it has an important role in regulating thyroid hormone action, especially in placenta and brain.

In cattle, inadequate Se reduces the efficiency of these enzymes and the conversion of T4 into active T3.⁵²

Antioxidant defense in the thyroid

Selenium is a key component of GPX, an enzyme that helps protect the thyroid from OS⁵⁴ when it generates H₂O₂ during thyroid hormone synthesis. Excess H₂O₂ can cause oxidative damage to thyroid cells.⁵⁵ Glutathione peroxidase, with the help of Se, neutralizes this peroxide, prevents damage and ensures thyroid function.⁵⁴ The antioxidant role of Se protects the thyroid from damage during high hormone production or stress.

Thyroid hormone synthesis

Selenium is involved in synthesis of thyroid hormones; adequate Se concentrations ensure that the thyroid can produce sufficient T4 and T3.^{52,56} Although iodine is a key component for thyroid hormone synthesis, Se promotes thyroid function, contributing to activation of thyroid hormones. In cattle, selenium affects pituitary release of thyroid-stimulating hormone (TSH) that stimulates thyroid to produce T4 and T3.⁵⁸ Further, Se deficiency can increase TSH concentrations to compensate for low thyroid hormone concentrations, potentially causing subclinical hypothyroidism (marginal thyroid hormone concentrations).⁵⁷

Selenium deficiency and thyroid hormone imbalance

When Se concentrations are inadequate, the conversion of T4 to T3 is impaired due to reduced activity of the deiodinase enzymes; this can reduce the T3/T4 ratio that can affect various metabolic processes. A Se-deficient diet reduces T3 concentrations, although T4 concentrations may increase. This imbalance can lead to suboptimal metabolic function and growth issues, as T3 is the active hormone responsible for regulating energy expenditure, cell growth, and development.⁵⁸ Selenium deficiency in calves is often associated with poor growth and performance,^{12,59} due in part due to reduced thyroid function and hormonal imbalance. Since T3 is involved

in regulation of growth, Se deficiency could impair growth rates and developmental processes in young animals.^{1,12,59} Selenium-deficient calves often have slower growth rates, likely due to impaired thyroid function and reduced T3.^{12,59} In these calves, Se supplements can restore normal thyroid function, improve T3 production, and promote growth. However, Se supplementation typically does not affect growth in well-nourished calves.

Selenium supplementation and improved thyroid function

Milk selenium and neonatal growth

The correlation between milk Se concentrations and neonatal T3 concentrations highlights the importance of Se-rich milk for supporting newborn development. Since T3 has a critical role in growth, adequate Se intake during the neonatal period is essential for promoting growth and development in offspring.

Positive effects in ewes and lambs

Selenium supplementation can increase T3 in both ewes and their lambs. For example, Se-supplemented ewes had higher T3 in their serum and milk that positively affected thyroid hormone concentrations in their lambs. In these lambs, there was a strong positive correlation between milk Se concentration and serum T3 ($r = 0.84$, $p < 0.01$).^{60,61} In contrast, a negative relationship was observed in the control (unsupplemented) group; lower Se intake reduced T3 and increased the T4/T3 ratio.

Role in fetal development

Selenium transfer from dam to fetus

Selenium is primarily incorporated into selenoproteins (e.g. GPX and thioredoxin reductase) that have critical roles in reducing OS and protecting cells. The fetus relies on maternal Se to ensure proper development and to establish its own antioxidant defenses. Selenium is actively transported across the placenta from maternal blood to the fetus.⁶² The placenta has specialized transport systems to facilitate transfer of various nutrients and trace elements, including Se, to support fetal development. Selenium is transported in the blood as selenomethionine or selenate and transferred from dam to fetus via the placenta, facilitated by placental transporters, including sodium-dependent active transport mechanisms.^{63,64} Fetal Se concentrations largely depend on maternal Se intake.^{62,64} If a dam has adequate Se, the fetus is typically well supplied. However, maternal Se deficiency can reduce Se transfer to the fetus, with detrimental effects on fetal development.⁶⁵ Selenium, through its role in selenoproteins (GPX), protects fetal tissues from oxidative damage, crucial during rapid cell division and organ development.⁶⁶

Selenium's role in embryonic development and maternal health is critical, especially during pregnancy, where it influences antioxidant defense, hormonal regulation, and fetal growth.⁶⁶⁻⁶⁸ Although much of Se's function is understood in terms of postnatal development, its importance during pregnancy is equally important, particularly in protecting the fetus from OS and supporting placental function. The role of Se in embryonic development, fetal health, and maternal nutrition during pregnancy is as follows:

Selenium's role in embryonic development and antioxidant protection

- DNA methylation and gene expression: Selenium affects DNA methylation and mRNA expression during embryonic development. These processes are essential for cell differentiation, proliferation, and apoptosis, critical in early development.⁶⁹ Disruptions in these processes, especially those involving OS, can negatively affect embryo development and survival.
- Oxidative stress in embryonic development: During embryonic development, OS is an important concern. The embryo, despite developing in a relatively low-oxygen environment, has low antioxidant capacity and is particularly vulnerable to oxidative damage. Selenium added to the culture media can improve embryonic development.⁷⁰ Cocultured 2-cell stage embryos and the oviductal epithelial cells had a significantly increased cleavage rate and enhanced blastocyst development,⁷¹⁻⁷³ suggesting Se may promote embryogenesis (Figure 4).
- Selenium's antioxidant role: Selenium has a crucial role in mitigating OS by being a key component of selenoproteins (e.g. GPX) that neutralize ROS. Selenium supplementation during pregnancy ensures the proper balance of antioxidants in both the dam and fetus, reducing cellular damage that can impair development.

Selenium and pregnancy complications

- Low Se and pregnancy complications: In small ruminants and humans, low Se concentrations during pregnancy are linked to low birth weight, impaired fetal growth, and increased likelihood of a small for gestational age (SGA) fetus (Figure 4).^{74,75} In humans, Se's antioxidant properties help protect against oxidative damage in the developing fetus, and insufficient Se during pregnancy can lead to OS that harms both maternal and fetal health.⁷⁶
- Progesterone production: Selenium also has a role in progesterone production that is essential for maintaining pregnancy (critical for maintaining uterine conditions conducive to fetal development and for preventing premature labor). In a study with heifers, Se supplementation increased plasma progesterone concentrations during the late stages of pregnancy (29-39 weeks), indicating that Se supports the function of the corpus luteum (CL) and the placenta during pregnancy (Figure 4).⁷⁷

Placental development and selenium's influence on oxidative balance

- Oxidative stress and placental function: During pregnancy, the placenta is the interface between the dam and fetus, facilitating nutrient and oxygen exchange. During placental development, OS can affect placental function (Figure 4).⁷⁸ Placenta changes over time, including increased NADPH oxidase-mediated ROS generation, that affects trophoblast proliferation, spiral artery remodeling, and placental growth.⁷⁹ Excessive ROS can compromise placental function, especially if antioxidant function is inadequate.
- Complications from oxidative imbalance: Imbalances in ROS and antioxidant capacity can lead to insufficient remodeling of spiral arteries by extra-villous trophoblasts that is essential for proper blood flow and nutrient exchange.⁸⁰ In humans, disruptions in this process may result in pregnancy complications such as preeclampsia and intrauterine growth restriction (IUGR), both are associated with fetal hypoxia.⁶⁵

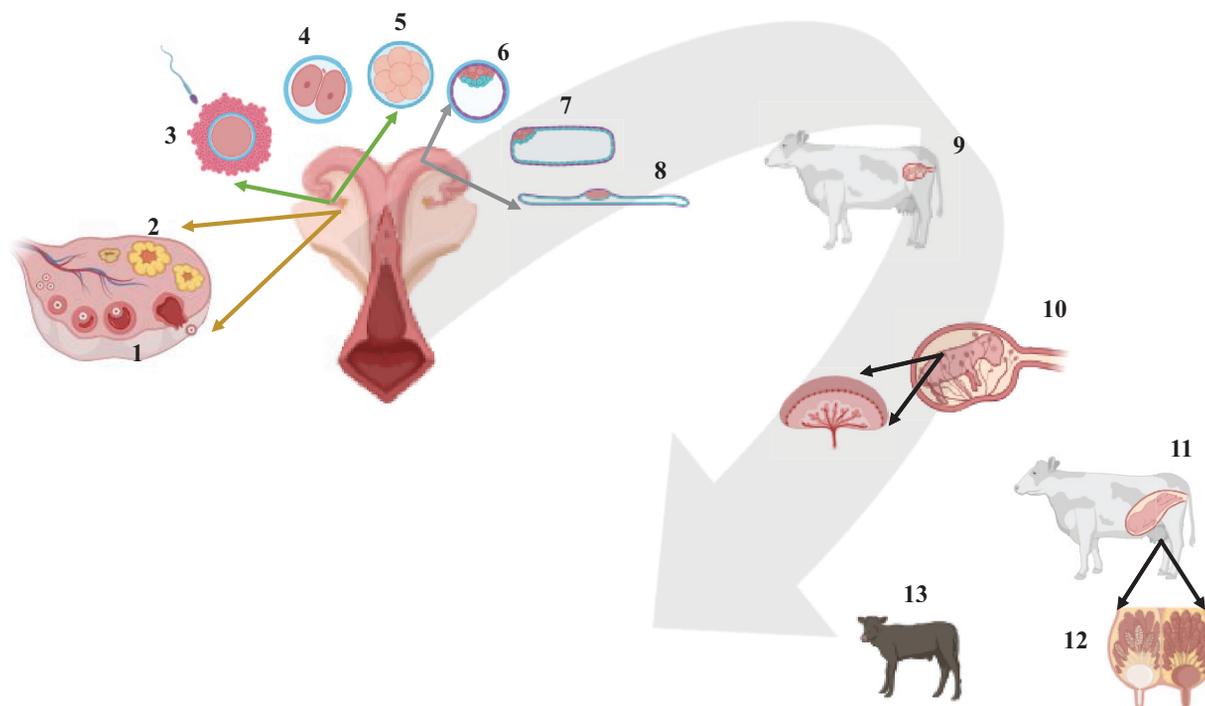


Figure 4. Roles of selenium in female reproduction

Effects of selenium supplementation on maternal health and fetal development

- **Selenium and maternal health:** Selenium supplementation during pregnancy can promote antioxidant balance and support placental function, reducing pregnancy complications.^{65,81} It is particularly beneficial in preventing conditions related to OS, protecting maternal immune system and tissues.
- **Impact on fetal development:** In humans and animals, adequate Se intake during pregnancy supports fetal growth and development, reduces the risk of IUGR and promotes development of critical tissues, e.g. brain and lungs.^{8,82} Selenium’s antioxidant properties protect fetal tissues from oxidative injury, promoting organ development.^{8,65,82}

Effects on lactation and offspring growth

- **Selenium and colostrum:** Selenium supplementation during pregnancy and lactation increases GPX activity and reduces lipid oxidation in colostrum, improving antioxidant capacity.⁶⁰ This provides the newborn with essential antioxidants, prevents oxidative damage and promotes immune function (Figure 4).
- **Selenium and growth of calves and piglets:** Supplementing Se in cattle and pigs improved milk composition and enhanced transfer of immunoglobulins (IgA, IgM) to calves and piglets.^{1,81,83} This enhanced preweaning survival, immune function, and piglet growth and development.
- **Selenium and heat stress:** Selenium supplementation of heat-stressed cows and sows, improved antioxidant

capacity and immunoglobulin transfer.^{81,83} This promoted maternal health and fetal development, despite exposure to high temperatures.

Selenium is vital for supporting healthy fetal development, placental function, and maternal health during pregnancy. Its role in reducing OS and supporting hormone production is indispensable to support pregnancy and promote growth and development of the offspring.

Role in female reproduction

Selenium offers a broad range of health benefits for livestock, especially cattle, having a crucial role in reproductive health. Deficiencies in Se can cause large economic losses, due to impaired fertility, increased incidence of mastitis, metritis, RFM, and others.^{13,84}

Selenium has a vital role in oocyte and ovary function. In an *in vitro* study using a buffalo oocyte cell line, Se significantly improved the rate of nuclear maturation and reduced the number of cells in the germinal vesicle stage. Although Se did not affect cell development at other meiotic stages, it altered expression of several genes involved in oocyte development, including downregulating Casp3 and Amh, and upregulating GPX4 and SOD.⁸⁵ Selenium also reduced transcript levels of Pla2g3, a phospholipase involved in lipid metabolism.^{85,86} Similarly, in a study on primary luteinized granulosa cells from goat ovarian follicles, Se stimulated cell proliferation and increased markers associated with estradiol production, such as 3β-HSD, p-Akt, cAMP, and steroidogenic acute

regulatory protein.⁸⁷ Therefore, Se may regulate the hypothalamic-pituitary-gonadal (HPG) axis by influencing sex hormone production through its effects on redox balance and lipid metabolism.

In terms of reproductive health, Se is critical for fertility and early embryonic development. Adequate Se reduces OS during ovarian follicular and CL development, thereby protecting reproductive organs from ROS (Figure 4).⁷⁷ Selenium also has a key role in maintaining progesterone production in heifers that is essential for pregnancy maintenance (Figure 4).⁷⁷ Without sufficient Se, the CL cannot function properly that can lead to reproductive failure. Selenium supplementation reduces the incidence of RFM, metritis, endometritis, and ovarian cysts, all negatively affect cow health and herd productivity. Ensuring Se adequacy before and after calving is crucial to optimize reproduction.⁸⁸⁻⁹¹ Cows with adequate Se also have fewer services per conception, a higher pregnancy rate at first service, and a shorter interval to conception,^{90,91} all improve reproductive efficiency.

Selenium also impacts mastitis and metritis. Selenium deficiency can impair milk quality, alter colostrum composition, and hinder immune transfer to the offspring.^{81,92-97} Inadequate Se increases the risk of mastitis.⁹⁸ Selenium's antioxidant properties protect the mammary gland from oxidative damage, reducing mastitis incidence (Figure 4).⁹⁹ Selenium supplementation also reduces the risk of metritis, a uterine infection that can follow calving^{13,91} and lead to prolonged postpartum anestrus, delaying conception. By improving immune function and reducing inflammation, Se helps prevent these conditions, enhancing reproductive health.^{100,101}

Regarding Se supplementation sources, the form of Se (sodium selenite or selenized yeast) has little impact on conception rates or calving intervals.¹⁰² However, sources may vary in bioavailability and effectiveness in various farming systems. Regardless of the source, ensuring adequate Se in the diet is essential for maintaining overall reproductive health in cattle.

From an economic perspective, Se supplementation improves reproductive efficiency by reducing the number of services per conception, improving first-service success rates, and shortening calving intervals.¹⁰³ These benefits increase herd productivity and profitability. Selenium also lowers the incidence of metritis, mastitis, and RFM, reducing veterinary costs and leading to healthier cows^{1,56,88,103} with greater milk yields, longevity, and long-term profitability.

Further, the bovine ovary contains high levels of Se that are localized to healthy preovulatory follicles, but not atretic follicles.⁷² The presence of Se in healthy follicles implies involvement in preparing the oocyte for fertilization, embryo development, and postnatal life (Figure 4). Adding Se to *in vitro* fertilization (IVF) cultures for cattle, dogs, pigs, and yaks improved embryo development, reduced ROS, and decreased DNA damage.¹⁰⁴⁻¹⁰⁸ Cattle fed organically bound Se have better conceptus length and differential expression of genes related to pregnancy recognition (Figure 4).^{109,110} Furthermore, ewes supplemented with Se throughout pregnancy have increased cell density and cell proliferation in the placenta during late pregnancy that correlates with improved lamb birth weights.¹¹¹ These effects

on placental development may partially explain effects of Se on fetal growth and development.¹¹¹⁻¹¹⁴ In humans, low Se in plasma, follicular fluid, or amniotic fluid, as well as low tissue GPX, are associated with infertility, miscarriage, preterm birth, gestational diabetes, and SGA fetuses,^{75,115-118} emphasizing the importance of Se for reproductive success across species.

1. Regulates preantral and preovulatory follicle function, enhancing fertility competence of oocytes;
2. Enhances CL function, ensuring sufficient progesterone to support pregnancy;
3. Promotes successful fertilization;
- 4 and 5. Improves early embryonic development in the oviduct by increasing the cleavage rate and 8-cell embryo development;
6. Enhances blastocyst rate;
- 7 and 8. Increases conceptus length by enhancing the expression of genes related to maternal recognition of pregnancy, including interferon-stimulated genes and progesterone-stimulated genes;
9. Promotes early embryo and fetal development;
10. Increases cell density and cell proliferation in the placenta;
11. As the placenta is the primary site for the exchange of nutrients, respiratory gases, and metabolic wastes, Se promotes fetal growth and development;
12. Protects mammary gland from oxidative damage and reduces incidence of mastitis;
13. Enhances immune function in neonate.

Selenium is an essential micronutrient that has a crucial role in immune function and reproductive health in cattle. It helps protect against OS and inflammation, supports fertility, and improves milk quality. Supplementing cattle with Se, especially in areas where the soil is deficient, can improve reproductive efficiency, health outcomes, and overall economic performance. Ensuring adequate Se before and during pregnancy, as well as in the postpartum period, can significantly reduce the incidence of reproductive disorders such as RFM, metritis, and ovarian cysts, while also enhancing immune function and disease resistance.

Role in male reproduction

Selenium has a crucial role in sperm health, sperm parameters and fertility,¹¹⁹ particularly in bulls (Figure 5). Selenium, through its incorporation into selenoproteins like GPX4,¹²⁰ has several protective and structural functions in sperm, promoting viability, motility, and integrity that are critical for fertility. Selenium is an essential component of GPX4, a key antioxidant enzyme protecting sperm from oxidative stress-induced damage.¹¹⁹ Selenium (via GPX4) contributes to the structural integrity of the sperm's midpiece, critical for mitochondrial function and motility.¹²¹ Oxidative stress caused by ROS can damage sperm membranes and impair motility.¹²² Adequate Se protects sperm mitochondria producing energy that is essential for sperm motility.^{123,124} Poor motility reduces sperm fertilizing ability and fertility.

Although GPX4 activity is not significant in sperm, it is crucial for protecting sperm at various stages of development. In the early stages of spermatogenesis, GPX4 protects sperm DNA from oxidative damage. GPX4 is strongly expressed in seminiferous tubules of healthy bulls, particularly in spermatogonia, round spermatids, and elongated spermatids, suggesting a critical role in sperm maturation. Immuno-signals for GPX4 are also detected in the acrosomal region of epididymal and ejaculated sperm, further emphasizing its importance in sperm function.¹¹⁹

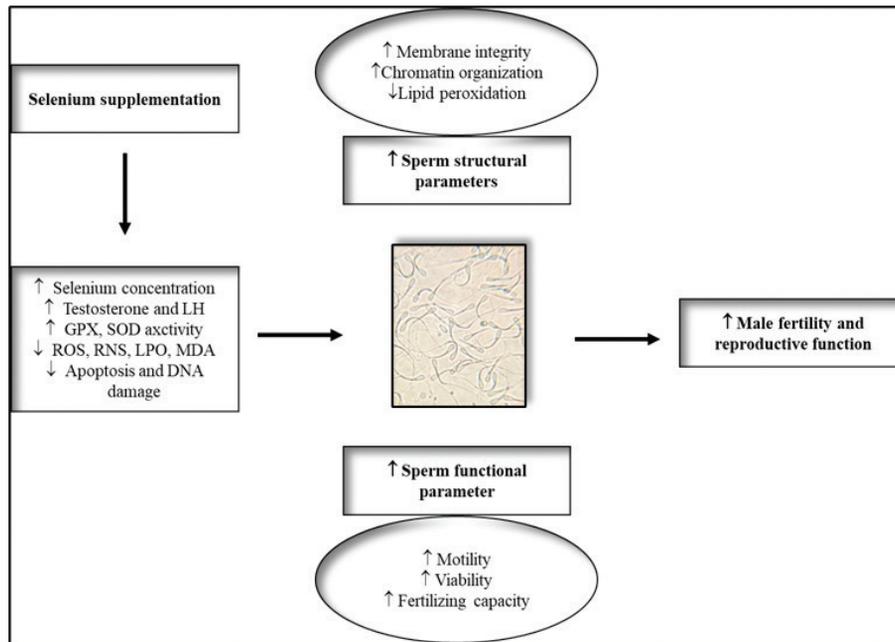


Figure 5. Effects of selenium supplementation on sperm and fertility

Impact on sperm quality

Selenium supplementation of Holstein bulls improved various sperm quality parameters, including viability, motility, membrane integrity, and postthaw quality. These improvements are particularly notable after semen cryopreservation.¹²⁵ Supplementation also reduced lipid peroxidation and apoptosis/necrosis rates in sperm that are commonly associated with oxidative damage. Selenium supplementation elevates antioxidant biomarkers in seminal plasma, including total antioxidant capacity (TAOC) and malondialdehyde (MDA), markers of OS. Selenium reduces oxidative damage to the sperm, particularly in winter and autumn, when GPX concentrations in the testes naturally increase.¹²⁶ Selenium deficiency can increase abnormal sperm morphology (e.g. deformed heads or tails) that reduces fertilizing ability.¹²⁷ Selenium, through its antioxidant properties, helps prevent oxidative damage to sperm DNA that is crucial for fertilization and development.¹²⁸

Selenium has a multifaceted role in protecting sperm from oxidative damage, improving sperm quality, and contributing to overall fertility and reproductive health in bulls. By incorporating Se into selenoproteins (e.g. GPX4), sperm maintain structural integrity, motility, and viability, making Se supplementation a useful tool for improving sperm health and fertility.

Selenium nanoparticles (SeNPs) protect against aflatoxin B1 induced toxicity, reducing morphologically abnormal sperm and DNA fragmentation¹²⁹ and preserving testicular architecture, mitigating atrophy of the seminiferous tubules.¹³⁰ Furthermore, SeNPs protected against deltamethrin-induced reproductive toxicity and negative effects such as sperm characteristics, TST, and antioxidant biomarkers, as well as behavioral and histopathological alterations. Bulls given SeNPs had improved semen parameters, antioxidant status, and sexual performance.¹³¹ In addition, SeNPs improved the quality of semen in rats and goats.¹³¹⁻¹³³

Selenium's role in testosterone production

Hormonal regulation: Selenium has an indirect but essential role in testosterone (TST) production by supporting testicular function. Testosterone is a key hormone for spermatogenesis and overall bull fertility.

Selenium and the hypothalamic-pituitary-gonadal axis: Selenium is involved in the proper functioning of the HPG axis that regulates the release of hormones, including LH and Follicle stimulating hormone (FSH).¹³⁴ These hormones stimulate the testes to produce TST and sperm. Selenium also helps regulate the enzymes involved in testicular function.

Selenium is also important for maintaining production of sex steroid hormones, including TST, estradiol, and progesterone. Selenium depletion lowered TST concentrations while decreasing testicular mass and adversely affecting testicular morphology.¹³⁵ Although testicular mass of first-generation selenium-deficient rats was not significantly changed, each successive generation had increasingly worsening symptoms. Exogenous GnRH or LH induced a reduced TST secretion in Se-deficient rats; perhaps TST production was impaired or receptors or signaling pathways for GnRH and LH were affected. Selenium supported TST synthesis by activating the extracellular signal-regulated kinase (ERK) signaling pathway in ovine Leydig cells.¹³⁶

Selenium affects proliferation and apoptosis of Leydig cells by regulating OS and expression of cell cycle and apoptosis-related genes.^{131,132} In addition, Se can enhance TST production by activating the ERK signaling pathway and expression of downstream genes (StAR and 3 β -HSD) that could be related to regulatory roles of Se in male fertility and spermatogenesis.^{136,137}

Selenium has an important role in immune system function, enhancing the ability of white blood cells (e.g. neutrophils

and lymphocytes) to respond to infections.¹³³ A healthy immune system is crucial for maintaining reproductive health in bulls, as it reduces risk of reproductive infections (seminal vesiculitis, epididymitis, or orchitis) that can impair sperm production and quality.¹³⁸ Selenium deficiency can suppress immune responses, increasing susceptibility to reproductive tract infections that reduce sperm quality.

Seminal plasma

Selenium is present in the seminal plasma ($457.4 \pm 108.7 \mu\text{g/liter}$) and its antioxidant activity protects sperm from oxidative damage during ejaculation and transport through the female reproductive tract.^{136,137} Selenium is also involved in production of seminal plasma proteins that support sperm viability and function. Selenium-dependent enzymes promote the health of sperm and improve their ability to survive in the female reproductive tract.

Fertility and in vitro findings

Selenium supplementation enhances sperm function in IVF. It significantly improves mitochondrial activity in sperm and sperm acrosome integrity and promotes sperm binding to the zona pellucida (ZP), critical for fertilization.^{21,139}

Beef cattle selenium requirements

Concentrations of blood Se $> 100 \text{ mg/liter}$ are required to maintain optimum immunocompetence in growing beef cattle.¹⁴⁰ Dietary requirements to avoid NMD are 0.1 mg/kg DM . Regular Se analysis of forages and feeds is necessary to detect deficiencies, and supplementation may be needed, in the form of salt blocks, loose minerals, or selenium-enriched yeasts.³ The FDA permits selenium to be included in the total diet of beef cattle at a maximum level of 0.3 ppm of dry matter. In areas where deficiencies are common, the maximum allowable level should be used (<https://www.ecfr.gov/current/title-21/chapter-I/subchapter-E/part-573/subpart-B/section-573.920> [cited April 8 2025]). Additionally, the FDA allows up to 120 ppm of Se to be incorporated into a salt-mineral mixture for free-choice feeding. Selenium deficiency is unlikely if adequate amounts are consumed through the mineral supplement. However, the Se concentration in the supplement and the labeled intake must ensure that the total daily intake does not exceed 3 mg . For example, a mineral supplement with a recommended intake of $4 \text{ ounces per head per day}$ cannot contain more than 26 ppm of Se.

Clinical presentation of selenium deficiency in various age groups

Selenium deficiency, often paired with a lack of vitamin E, can lead to various NMD in cattle, with clinical signs that can range from acute to subacute.¹⁴¹ These disorders affect a wide array of conditions, including poor growth in pastured calves, lowered milk production and subclinical mastitis in dairy cows, decreased fertility, embryonic death, RFM, metritis, poor uterine involution, cystic ovaries, impaired immune function, and even premature death, especially in cases of marginal Se deficiency.³

In neonates, Se is crucial for fetal development, and its deficiency in calves can lead to severe health issues, as Se crosses the placenta from dam to fetus. Selenium-deficient calves

may suffer from myocardial necrosis, heart failure, and other systemic issues due to insufficient antioxidant activity from GPX, an enzyme dependent on Se. Clinical signs include premature birth, perinatal death, heart or respiratory failure, and spontaneous abortion. Postmortem findings will typically include myocardial and skeletal muscle damage. Timely Se supplementation to both dams and calves is essential for preventing further damage.^{8,20}

In beef calves aged 2-8 months, Se deficiency often manifests as acute or subacute NMD after unaccustomed exercise. Calves raised indoors and then exposed to pastures or those moved long distances may develop NMD due to Se-deficient diets. The OS from exercise accelerates muscle degeneration, leading to muscle calcification and widespread tissue damage. Acute NMD can lead to death from heart failure or respiratory distress, with affected calves being weak, recumbent, and unable to stand.^{8,141} Subacute NMD, more common in rapidly growing calves, typically affects skeletal muscles, causing weakness and tremors but often resolves with treatment.^{3,142} Muscle degeneration releases several enzymes, including creatinine kinase (CK); this can be useful in supporting a diagnosis of NMD.^{1,136} Subclinical Se deficiency in older calves may manifest as ill thrift, with stunted growth, poor coat condition, and poor nutrient absorption, potentially leading to death.^{1,20}

Yearling cattle, particularly those raised on Se-deficient diets or grain-based feeds that cause vitamin E degradation, are also susceptible to NMD.³ Subclinical cases often present as weakened immune systems, with an increased susceptibility to diseases like respiratory infections. Subacute NMD in yearlings can cause muscle degeneration, similar to clinical signs in older calves. Severe cases may lead to 'flying scapula'¹⁴³ or a spreading of toes, and muscle weakness may impair feeding or swallowing.¹ Acute cases can cause myoglobinuria, stiffness, and even sudden death.³ Selenium supplementation alleviates some clinical signs, especially in beef steers, where it can improve prolactin and mitigate fescue toxicosis.¹⁴⁴⁻¹⁴⁹

In adult cattle, Se deficiency often manifests as reproductive issues, due to its crucial role in follicular development, progesterone production, and antioxidant defense during high metabolic stress (e.g. pregnancy and lactation). A Se-deficient herd may have higher rates of RFM, infertility, metritis, cystic ovaries, and early embryonic death, even with marginal deficiencies.^{3,20,150} Bulls are also affected, with reduced sperm motility and viability due to impaired selenoproteins in sperm tails and acrosomes, leading to decreased fertility.^{124,125}

Selenium toxicosis

Selenium deficiency is more common than toxicosis, but the latter does occur. Mining operations can expose Se-bearing elements to air and water, mobilizing Se into aquatic systems where it bioaccumulates in the ground water, soil, grasses and forages, amplifying hazardous effects.¹⁵¹ Selenium toxicosis occurs when an animal's diet exceeds $3\text{-}8 \text{ ppm}$ of Se (threshold varies by species).¹⁵² Some animals can tolerate higher concentrations. Forage plants that lead to toxicosis typically contain 30-160 times the amount of Se required for nutrition. Poisoning can also result from consuming grains with elevated Se, affecting livestock such as poultry and swine.¹⁵²

Selenium poisoning is classified into acute and chronic forms.^{153,154} Acute poisoning typically results from short-term ingestion of highly seleniferous plants, particularly when animals are hungry and consume low-palatable plants with high Se content. Symptoms of acute poisoning include abnormal posture, unsteady gait, abdominal pain, and, in severe cases, death.^{153,154} Chronic selenosis is associated with hair and hoof abnormalities. Bilateral alopecia or hair fragility and breakage along the mane, tail, and the nape are described in production animals. Chronic Se poisoning is further categorized into alkali disease and blind staggers.^{153,154} Alkali disease occurs when animals consume seleniferous forages (typically 5-40 ppm of Se) over extended intervals. Symptoms of alkali disease include dullness, emaciation, rough coats, hair loss, and lameness. Blind staggers result from grazing Se indicator plants containing water-soluble Se, with impaired vision, weakness, paralysis, and death from respiratory failure.

There is debate whether blind staggers is caused solely by Se, with other factors or toxins in plants suspected as having a role.¹⁵⁵ Similar symptoms have been observed in conditions such as forage poisoning in Colorado, tansy mustard poisoning in the Southwest, and kochia poisoning.¹⁵⁶ High sulfate concentrations in water or feed may also contribute to these symptoms, implying blind staggers may have multiple causes. Ultimately, alkali disease appears to be the primary chronic manifestation of Se poisoning, whereas blind staggers remain a more ambiguous condition that could involve additional toxins or unknown factors.

Reproductive effects of Se toxicity

Selenium toxicity can negatively affect reproduction in cattle in several ways. At higher concentrations, Se can become prooxidant, leading to OS and damage to cellular

components. Elevated Se can impair sperm quality¹⁵⁷ in bulls. Selenium controls development of granulosa cells and biosynthesis of estradiol-17 β in adult ovaries in vitro.¹⁵⁸ Selenosis can disrupt oocyte development leading to reduced fertility. High Se concentrations can cause spontaneous abortions, particularly in later stages of pregnancy.¹⁵⁹ Calves born to cows with high Se exposure may have birth defects, particularly musculoskeletal issues.¹⁶⁰ Selenium toxicity can decrease milk yield, possibly due to damage to mammary tissue or an overall decline in health.

Diagnosis of selenium deficiency

Testing soil, feed, and live cattle

If herd problems are primarily reproductive, general unthriftiness or subtle and deficient Se status is suspected, a diagnosis is typically made based on clinical signs and response to Se therapy. Testing is an important adjunct for diagnosis and is vital for future supplementation planning. Deficient, marginal, and normal Se concentrations standards for the analysis of different samples are provided (Table 2). Representative analysis of pasture/forage/herd samples are critical.

Diagnosis of selenosis

Diagnosis generally involves observation of symptoms such as hoof abnormalities, hair loss, and reproductive problems.^{161,162} Blood, serum samples or liver tissues are tested for Se.¹⁶³ Normal Se concentrations in cattle are generally 0.05-0.10 ppm, with > 0.25 ppm considered toxic. Testing for Se content in the forage and feed is also critical.¹⁶⁴ Liver and kidney are the most common postmortem samples used for Se analysis.¹⁶⁵

Table 2. Selenium concentration standards: deficient, marginal, and normal levels for various sample analyses

Sample types for testing	Selenium level		
	Deficient	Marginal	Normal
Soil	0.5 mg/kg	-	-
Pasture	< 0.02 mg/kg DM of pasture	-	Forages and grains with 0.1 mg/kg DM are considered adequate ³
Serum [†]	0.002-0.025 mg/ml	0.03-0.07 mg/ml	0.08-0.3 mg/ml ³
Whole blood [‡]	< 0.05 ppm	0.05-0.1 ppm	> 0.11 ppm*
Serum erythrocyte GPX activity**	0.2-10	10-19	19-36
Serum CK levels***	-	-	21-31 IU/L [§]
Liver biopsy	0.07-0.6 mg/ gram DM	0.45-0.9 mg/ gram DM	0.9-1.75 mg/ gram DM ³

[†]It responds more rapidly to Se treatment than whole blood Se;

[‡]There is a delayed response to Se treatment compared to serum Se;

*(> 5 ppm high; Se toxicity)²⁰

**This test is faster and more economical than testing for Se directly; however, the enzyme is unstable, and improper handling can significantly skew the results. The units are expressed as micromoles per minute at 37 °C per gram of hemoglobin;

***It is the most common diagnostic aid for NMD, though it is not specific to Se deficiency syndromes. Its half-life is 2-4 hours, rapidly declining after myodegeneration, but it can remain elevated for up to 3 days following unaccustomed exercise. Levels typically return to normal within a few days of treatment;

§After a myodegeneration event, levels are commonly > 5,000-10,000 IU/liter³.

Postmortem testing

Necropsy should be done on aborted fetuses or acute deaths, especially if NMD is suspected. The earlier the diagnosis and intervention, the more subclinical and subacute cases can be remedied. Because NMD is not an inflammatory process but rather degeneration of hyaline followed by coagulation necrosis +/- mineralization, there will be no muscle inflammation.¹ No specific skeletal muscle is always affected, but groups of muscles are always affected bilaterally and symmetrically. They contain localized degenerative and necrotic areas that are white to gray. The affected area is often a streak through the center of an otherwise normal muscle or may be flanking either side of a normal piece of muscle.³

Diaphragm also has a streaked appearance but in a radiating pattern with a central focus of muscle degeneration/necrosis. The diaphragm will be very friable and edematous, with or without mineralization.³ If the muscles of the throat or chest are affected, secondary pneumonia is common.³ In calves with myocardium degeneration, the endocardium of the left ventricle is commonly affected. White to gray lesions may extend to involve interventricular septum and papillary muscles. Pulmonary congestion and edema are common with heart involvement.³

Treatment of selenium deficiency

Due to the close relationship vitamin E and Se in protecting cells and having difficulties in determining, which is the primary deficiency, Se and α -tocopherol combinations are recommended.³

Treating NMD in calves is best done with an injectable Se (3 mg) and DL alpha-tocopherol acetate (150 IU/ml) injection at 2 ml/45 kg BW IM. Once is usually sufficient unless it is the acute myocardial form where mortality can reach 90%. If there is at least 1 case of Se deficiency, treatment of the entire herd is recommended. As unaccustomed exercise is a predisposing factor for clinical NMD, cattle should be carefully handled during treatment. Subacute cases of NMD should improve by 3 days and regain mobility within 1 week.³

Treatment of selenosis

If Se toxicity is suspected, stop the use of any Se-containing feed supplements. In recovering steers, half-lives for Se elimination from serum, whole blood, liver, and muscle were 40.5 ± 8.2 , 115.6 ± 25.1 , 38.2 ± 5.0 , and 98.5 ± 19.1 days, respectively (relatively long).¹⁶⁶ Treatment may involve fluids, antioxidants, and liver-protective substances, but recovery can be slow, and severe cases may have permanent damage.¹⁶⁷ Medications or supplements like vitamin E and antioxidants may reduce OS in the liver.

Preventing selenium deficiency with routine mineral supplementation

Selenium supplementation needs to be tailored to each specific operation. Current Se status must be known to determine how and when to supplement the herd.¹⁶⁸ First, determine if the area in question is considered Se deficient; this can be gained from local extension offices or the US

Geological Survey website.⁵ Consider testing pasture or feed and check labels of any packaged products.¹⁵² Are there any signs of Se deficiency, as described above? If so, consider blood testing for serum Se concentrations, whole-blood Se concentrations, or erythrocyte GPX concentrations, on a representative sample or all cattle.¹⁶⁸

When developing a supplementation program, consider economics and logistics. There are inorganic and organic forms of Se. The inorganic form is usually sodium selenite that gives a short-duration increase in Se but does not persist. However, if applied to soil, it is readily taken up by plants and incorporated as organic Se. Organic Se is usually selenomethionine and typically provides a long-standing boost in Se status.¹⁶⁸

Selenium injections, typically paired with vitamin E or copper, utilize inorganic sodium selenite, and are therefore best used as prevention in young livestock. In heifers and cows, injecting a Se and vitamin E supplement 3 weeks before calving can greatly decrease fertility problems, RFM, poor uterine involution, and cystic ovaries.³

Oral Se supplementation during pregnancy provides protection for the fetus due to placental transfer and may decrease neonatal morbidity and mortality. Selenium concentrations in colostrum and milk reflect the dam's Se status. However, milk Se concentrations decrease 7 days after calving, so treatment of calves is often needed.³

Mineral supplementation is commonly used, typically in loose or block form. Loose is more common as it is more readily consumed, but Se can be leached by rain. Due to individual differences in intake and potential losses due to rain, results are often inconsistent.¹⁶⁹

For calf NMD prevention, loose salt and mineral mix can be fed ad libitum. The recommended 0.1 mg/kg Se and 1 gram/day/head alpha-tocopherol can be achieved with 14.8 mg/kg of mixture and 2,700 IU/kg mixture of vitamin E. Cow and calf health and productivity are optimized by using this for the last 2 trimesters of pregnancy and first month of lactation.^{3,169}

Fortified feed supplements have inorganic and/or organic sources of Se; the latter will produce a more prolonged increase in blood and liver Se concentrations than the former. Furthermore, organic sources result in more consistent intake on an individual basis.¹⁶⁷ Seleno-yeast is an excellent option for supplementation in beef cattle. In pregnant cattle, seleno-yeast increased GPX activity better than sodium selenite when offered as a free-choice mineral.³ Selenium pellets are available as 10% Se and 90% iron grit. They can increase GPX activity for > 2 years and are effective when fed to pregnant cows in the last 3 months of pregnancy, increasing Se milk content to prevent calf NMD.³ These pellets were also used for growing cattle and did not cause toxicosis at 2-4 times the recommended dose. Alternatively, to ensure no problems upon turn out to pasture in the spring after being fed hay, straw, or high-moisture grain through the winter, supplementing with 0.1 mg/kg DM Se and 150 mg/day/head alpha-tocopherol can be used.³ Selenium boluses are only approved in California. They provide a consistent amount of Se that is slowly released from the bolus into the rumen, making them a good option for grazing animals where supplementation is not feasible.⁶⁹ These supplementation options should be used year-round in Se-deficient areas to improve reproductive and immune health.

Agricultural practices can also increase soil Se concentrations, though they very rarely make a large impact. Applying manure of farm animals fed imported Se-adequate feed can slowly increase soil concentrations, although this can be a lengthy and expensive process.³ Finally, there are licensed products to fertilize Se-deficient soils. It is typically recommended to fertilize 5-10 grams of actual Se per acre; an application rate of 12-24 grams of sodium selenate per acre will provide the recommended 5-10 grams of actual Se per acre.¹⁶⁷

Prevention of selenosis

Ensure that Se supplementation is done within the recommended levels; total Se intake for cattle should not exceed 0.3 mg/kg body weight per day. Regularly test forage and pasture soil for Se content,¹⁷⁰ especially in areas known to have Se-rich soils. Forage or feed that contains 2-5 ppm Se poses a marginal threat to livestock. Forage with > 5 ppm Se will cause acute toxic conditions in livestock and should be avoided.¹⁷⁰ High Se forage can be mixed with low Se forages to balance total Se content.¹⁷¹ In seleniferous areas with high Se concentrations, cattle should not be allowed to drink water with a Se concentration > 0.5 ppm.¹⁷⁰

Conclusion

Selenium is a critical micronutrient with an indispensable role in cellular function; therefore, its importance cannot be overstated. Selenium, a powerful antioxidant, supports a variety of physiological processes, including immune function, thyroid hormone metabolism, and reproductive health. In the northwestern US, where soil and forage Se concentrations can be low or highly variable, this nutrient is particularly important. Deficiencies in Se are common in these regions, making it essential to ensure that animal health and productivity are not compromised. Inadequate Se intake can reduce fertility and the immune response, increase susceptibility to infectious diseases, and compromise growth rates in calves. Furthermore, Se deficiency can lead to more severe conditions, including NMD and cause death. Therefore, it is crucial to thoroughly assess Se status of animals and the local environment to determine potential deficiencies. Understanding how Se concentrations in the soil, forage, and livestock affect herd health will enable targeted supplementation strategies that improve livestock productivity, enhance disease resistance, and ensure long-term sustainability. Well-informed, proactive approach to Se supplementation can prevent costly health issues and optimize herd performance.

Conflict of interest

None to declare.

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