Nutritional Diseases

Chapter 1 – Minerals and Vitamins

1. Macro-elements

The main major minerals, or macro-elements, are:

- Calcium (Ca), Phosphorus (P), Magnesium (Mg), Potassium (K), Sodium (Na)

Unit of measurement: grams (g)
These represent 99% of the mineral content of the organism.

Importance of Phosphorus and Calcium in Young Animals

The high growth requirements make mineral supplementation essential in young animals. At this stage, the absorption capacities of calcium and phosphorus are maximal. An appropriate supply of these minerals in the diet will promote proper skeletal ossification (ensuring longevity) and prevent the risk of fractures and decalcification problems.

Year-round Mineral Supplementation

It is common for farmers to "neglect" mineral supplementation during the second half of lactation and the dry period, precisely at a time when the cow needs to replenish reserves in preparation for the next lactation.

- **Early lactation:** Calcium demand is high due to intensive milk production. Mineral intake through the diet is insufficient and cannot fully meet the elevated needs; the dairy cow will mobilize bone reserves.
- **Late lactation:** Milk production decreases. At this stage, it is essential to provide an appropriate mineral supplement so the cow can restore bone reserves.
- **Dry period:** Strict calcium rationing is recommended to provide 50–60 g per cow per day to rebuild bone reserves. Excess calcium at this stage can prevent mobilization of reserves at calving.

Table 1: Role and Requirements of Macro-elements in Cattle

Mineral	Main Roles	Risk Situations for Ruminants	Effects of Imbalanced Supply
Calcium	Skeleton, muscle contractions, blood balance, major component of milk	Unprepared dietary transitions around parturition	**Deficiency:** Milk fever (hypocalcemia), chronic lameness, fractures, osteomalacia. **Excess:** reduces absorption of trace elements
Phosphorus	Skeleton, energy metabolism, rumen pH buffer (saliva)	Growth, milk production	Chute of growth, lameness, decreased milk production, reduced fertility. **Excess:** Especially if Ca:P ratio is imbalanced → uterine atony, fertility drop; environmental pollution due to excess excretion in manure

Magnesium	Energy metabolism, muscle contraction	Grazing (pasture)	**Deficiency:** Hypomagnesemic tetany, retained placenta, slow growth
Sodium	Nerve impulses, muscle contractions, blood balance	Insufficient salt intake	Loss of appetite, pica, reduced milk fat. Combined with potassium deficiency → acidosis
Potassium	Nerve impulses, muscle contractions, blood balance	Excess forage (rich in K)	**Excess:** accelerates digestive transit and fat mobilization, alkalosis, increases risk of sodium deficiency; during dry period, increases risk of udder edema
Sulfur	Protein synthesis in the rumen	Excess non-protein nitrogen	Reduced forage intake and rumen protein synthesis → hair loss, production decline, growth defect

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2. Trace Elements (Oligo-elements)

Trace elements, also called micro-elements or oligoelements, are minerals required in very small quantities, usually expressed in milligrams (mg) per kilogram of diet. Despite their small amounts, they play **essential roles** in enzymatic reactions, hormone function, and overall metabolism.

The main trace elements for cattle are:

- Iron (Fe)
- Copper (Cu)
- Zinc (Zn)
- Manganese (Mn)
- Iodine (I)
- Selenium (Se)

Table 2*:* Key Roles and Deficiency Signs

Trace Element	Main Roles	Risk Situations	Deficiency Symptoms
Iron (Fe)	Hemoglobin synthesis, oxygen transport, enzymatic functions	Young animals on milk only, areas with low Fe soil	Anemia, pale mucous membranes, poor growth, lethargy
Copper (Cu)	Cofactor for enzymes (oxidases), connective tissue synthesis, hair pigmentation	High molybdenum or sulfur intake, certain soils	Anemia, depigmentation, bone fragility, infertility
Zinc (Zn)	Skin and hoof health, enzymatic functions, growth	Diets high in calcium or phytates	Parakeratosis, slow wound healing, growth retardation
Manganese (Mn)	Bone formation, reproduction, enzyme cofactor	High calcium intake, soils poor in Mn	Skeletal deformities, poor fertility, weak calves
Iodine (I)	Thyroid hormone synthesis	Iodine-deficient soils	Goiter, weak calves, reproductive issues

Selenium (Se)	Antioxidant function (glutathione peroxidase), muscle and liver health	Low soil Se, high sulfur intake	White muscle disease, retained placenta, immunodeficiency

Interaction Between Trace Elements

- Excess of one trace element may **interfere** with the absorption of another.
  - High molybdenum and sulfur → copper deficiency
  - Excess calcium → zinc and manganese absorption decrease
- Balanced supplementation is therefore crucial, particularly in **growing animals, pregnant cows, and high-producing dairy cows**.

3. Vitamins

Vitamins are **organic compounds required in small amounts** for normal metabolic function. They are classified into **fat-soluble** and **water-soluble** vitamins.

Fat-Soluble Vitamins

- **Vitamin A** (Retinol): Vision, epithelial health, reproduction

  Deficiency: Night blindness, reproductive failure, skin and mucosa lesions

- **Vitamin D** (Calciferol): Calcium and phosphorus metabolism

  Deficiency: Rickets in young animals, osteomalacia in adults

- **Vitamin E** (Tocopherol): Antioxidant, muscle health, immunity

  Deficiency: White muscle disease, retained placenta, decreased immunity

- **Vitamin K**: Blood clotting

  Deficiency: Hemorrhages, prolonged clotting time

Water-Soluble Vitamins

- **B-complex vitamins** (B1, B2, B6, B12, niacin, pantothenic acid, folic acid): Metabolism of carbohydrates, proteins, fats; red blood cell formation; nervous system function
  - Deficiency: Anorexia, poor growth, neurological signs, anemia
- **Vitamin C** (Ascorbic acid): Antioxidant, collagen synthesis, immune support
  - Generally not essential for ruminants, as it can be synthesized in the rumen

4. Vitamin and Mineral Imbalance Syndromes in Cattle

Imbalances of vitamins and minerals can lead to **deficiency or toxicity syndromes**, affecting growth, reproduction, immunity, and production. Understanding these syndromes is critical for veterinary students to **diagnose and prevent metabolic disorders**.

4.1. Trace Element Deficiencies

1. **Iron (Fe) Deficiency**
    - **Risk groups**: Calves fed exclusively on milk, animals in Fe-deficient soils.
    - **Clinical signs**: Anemia (pale mucous membranes), lethargy, decreased growth.
    - **Prevention**: Oral or injectable iron supplementation; inclusion in mineral premixes.
2. **Copper (Cu) Deficiency**
    - **Risk factors**: High molybdenum, sulfur, or iron diets; poor Cu soil content.
    - **Clinical signs**: Depigmentation of hair, anemia, osteoporosis, infertility, diarrhea in young animals.
    - **Prevention**: Copper sulfate supplementation in feed or water, liver inclusion in diet.
3. **Zinc (Zn) Deficiency**
    - **Risk factors**: High calcium/phytate diet, poor soil Zn content.
    - **Clinical signs**: Parakeratosis, hoof lesions, poor wound healing, reduced growth.
    - **Prevention**: Zinc oxide or zinc sulfate in feed.
4. **Manganese (Mn) Deficiency**
    - **Clinical signs**: Skeletal deformities (e.g., shortened limbs), poor fertility, weak calves.
    - **Prevention**: Mn supplementation in mineral mixes.
5. **Iodine (I) Deficiency**
    - **Clinical signs**: Goiter, weak calves at birth, reduced fertility.
    - **Prevention**: Iodized salt or iodine-containing supplements.
6. **Selenium (Se) Deficiency**
    - **Clinical signs**: White muscle disease (degenerative myopathy), retained placenta, immunodeficiency.
    - **Prevention**: Se supplementation via feed, injection, or mineral blocks.

4.2. Vitamin Deficiencies

1. **Vitamin A Deficiency**
    - **Clinical signs**: Night blindness, reproductive failure, keratinization of epithelial tissues.
    - **Prevention**: Vitamin A supplementation, use of green forages or concentrates rich in carotenoids.
2. **Vitamin D Deficiency**
    - **Clinical signs**: Rickets in calves (soft bones, limb deformities), osteomalacia in adults.
    - **Prevention**: Sunlight exposure, vitamin D3 supplementation.
3. **Vitamin E Deficiency**
    - **Clinical signs**: White muscle disease, retained placenta, low immunity.
    - **Prevention**: Supplementation with vitamin E-rich feeds or commercial additives.
4. **Vitamin K Deficiency**
    - **Clinical signs**: Hemorrhages, prolonged clotting time.
    - **Prevention**: Vitamin K supplementation, correction of antibiotic-induced gut microbiota disruption.

5. **B-Complex Vitamins Deficiency**
    - **Clinical signs**: Poor growth, anorexia, neurological signs, anemia.
    - **Prevention**: B-vitamin premixes in feed, particularly for stressed or high-producing animals.

4.3. Toxicity Syndromes

Excessive supplementation can also lead to **toxicity**, especially for trace elements:

Element	Toxicity Signs	Risk Situations
Copper	Liver damage, hemolysis, jaundice	High Cu intake with low molybdenum
Selenium	Hair loss, hoof lesions, neurological signs	Excess supplementation in Se-rich areas
Vitamin A	Bone abnormalities, liver damage	Excessive supplementation

4.4. Practical Guidelines for Prevention

1. **Feed Analysis**: Determine mineral and vitamin content of forage, concentrates, and water.
2. **Soil and Region Assessment**: Identify deficiencies common in local soils.
3. **Balanced Supplementation**: Provide minerals and vitamins according to **age, production level, and physiological status**.
4. **Monitoring**: Regularly check **blood parameters** and **clinical signs** to detect early imbalances.

Chapter 2 – Trace Element Deficiency Syndromes in Cattle

2.1. General Diagnostic Approach

When a veterinarian is called to investigate a suspected nutritional disorder in a herd, a **methodical and structured approach** is essential.

1. Collection of History (Anamnesis)

The first step consists of gathering all relevant background information:

- Feeding practices (type of forage, concentrates, mineral and vitamin supplementation)
- Physiological status of animals (growth, lactation, gestation)
- Production level and recent changes
- Geographic location and soil characteristics

This information often provides **early indications of potential deficiencies or imbalances**.

2. Clinical Examination

A **complete and systematic clinical examination** must be performed:

- Both **individual** and **herd-level observation** are necessary
- Trace element deficiencies often affect **only part of the herd**
- Variability in clinical expression is common depending on age, physiological stage, and severity

Careful observation of the entire herd may reveal patterns that are not evident at the individual level.

3. Nutritional Assessment

If a deficiency is suspected:

- The **mineral composition of the diet** must be evaluated
- Attention should be given to **interactions between elements**, such as:
    - Excess molybdenum and sulfur → secondary copper deficiency
    - High calcium intake → reduced zinc and manganese absorption

In some cases, these steps alone may strongly support the diagnosis before laboratory confirmation.

Key Point

Trace element deficiencies are rarely pathognomonic. Diagnosis is often based on:

- A combination of **history, clinical signs, and dietary evaluation**
- Herd-level patterns rather than isolated cases

2.2. Copper (Cu) Deficiency

Onset of Clinical Signs

Copper deficiency develops **gradually** due to significant **hepatic storage** of copper. Clinical signs may take **several months (up to ~170 days)** to appear. Deficiency is **worsened by dietary factors**, particularly:

- High **molybdenum (Mo)** intake
- Excess **sulfur (S)**

Physiological Roles of Copper

Copper is a **cofactor for numerous enzymes** and is essential for:

- **Melanin production** → pigmentation of coat and hair
- **Connective tissue formation** → collagen and elastin synthesis
- **Skeletal and nervous system development**
- **Hemoglobin synthesis** → red blood cell formation
- **Immune function** → resistance to infections
- **Reproductive function** → fertility and placental health

Clinical Signs

1. Coat and Pigmentation Disorders

- Hair becomes **dull and rough**

- Depigmentation occurs, especially around the **eyes ("spectacles")**, **muzzle**, and **patches of hair**
- Black hair may become **reddish**, red hair may become **yellowish**
- Coat abnormalities are observed in **both young and adult animals**

2. Skeletal Disorders

- **Epiphyseal enlargement**
- **Spontaneous fractures**
- **Bone fragility**

3. Hematological Disorders

- **Variable anemia**:
  - Microcytic, hypochromic in young animals
  - Macrocytic in adults
- Severity depends on degree and duration of deficiency

4. Cardiovascular and Respiratory Disorders

- Rare sudden death from **cardiovascular elastin defects**
- **Dyspnea, tachypnea**
- Jugular distension due to vascular compromise
- Cardiac arrhythmias may occur

5. Digestive Disorders

- Chronic diarrhea **resistant to antibiotics or antiparasitic treatment**
- Reduced appetite (anorexia)

6. Production Effects

- **Weight loss**
- **Reduced milk production**
- **Growth retardation** in young animals

Key Point

Copper deficiency often **presents with multiple, subtle signs**, making early detection difficult. Veterinary evaluation must include **diet analysis, herd observation, and clinical assessment**.

2.3. Zinc (Zn) Deficiency

Onset of Clinical Signs

Zinc deficiency can develop **rapidly**, often within **10–14 days**, depending on dietary intake and antagonists (e.g., high calcium or phytate content).

Physiological Roles of Zinc

Zinc is a **cofactor for multiple enzymes** and is essential for:

- **Protein and carbohydrate metabolism**
- **Skin and hoof health**
- **Hormone regulation**, including steroid hormones
- **Immune system function**
- **Growth and development**

Clinical Signs

1. General and Production Effects

- **Loss of appetite** (often intermittent or cyclical)
- **Weight loss and growth retardation**
- **Reduced productivity** (milk and weight gain)
- These effects are often **the first signs** noticed in deficiency

2. Skin and Appendage Disorders

- **Parakeratosis**: chronic, non-inflammatory thickening and cracking of the epidermis
- **Alopecia**, particularly around the **eyes, muzzle, and ears**
- Hair may become **rough, brittle, or sparse**

3. Dermatological Complications

- Increased susceptibility to **secondary infections**:
    - Parasitic (mange, ringworm)
    - Bacterial (dermatitis, foot rot)
    - Viral (papillomatosis)
- Poor healing of skin lesions

4. Hoof and Horn Abnormalities

- Hooves and horns become **soft, deformed, sometimes cracked**
- May lead to **lameness** and reduced mobility

5. Musculoskeletal Effects

- Joint stiffness, particularly in distal joints
- In young animals, **rigidity of hocks** may occur
- Gait may appear **stiff or awkward**

6. Other Signs

- In severe deficiency, **hypersalivation**
- Secondary issues due to **reduced feed intake and weakened immunity**

Key Point

Zinc deficiency is often **easily recognizable from skin and hoof lesions**, but early signs like reduced appetite and growth retardation may appear **before visible dermatological symptoms**.

2.4. Cobalt (Co) Deficiency

Onset of Clinical Signs

Cobalt deficiency usually develops **slowly**, as cobalt is stored in the liver for vitamin B12 synthesis. Clinical signs may appear **after several months**, depending on:

- Age of the animal
- Previous dietary cobalt intake
- Degree of deficiency in the current ration

Physiological Roles of Cobalt

Cobalt is essential for the **synthesis of vitamin B12 by ruminal microflora**, which is critical for:

- **Energy metabolism** (carbohydrate and fat utilization)
- **Protein metabolism**
- **Cellular growth and division**
- **Red blood cell formation**

Clinical Signs

1. Production and Growth

- **Decreased appetite** (anorexia), leading to reduced feed intake
- **Weight loss** progressing to cachexia in severe deficiency
- **Delayed or disproportionate growth** in young animals (e.g., large head relative to body)

2. Hematological Signs

- **Normocytic, normochromic anemia**, typically appearing in the later stages of deficiency
- Pallor of mucous membranes

3. Coat and Skin Disorders

- Hair becomes **rough, long, and erect**, especially over the withers
- Coat may appear dull and less healthy

4. Digestive Disorders

- **Pica** (abnormal eating behavior)
- **Bloating (meteorism)**
- Occasional diarrhea

5. Ocular Signs

- Mild **lacrimation** in some cases

Key Point

Cobalt deficiency manifests primarily through **slow-onset metabolic, hematological, and growth disturbances**, rather than acute or dramatic clinical signs. Early detection relies on:

- Herd observation for reduced growth and production
- History of low cobalt intake or antagonistic dietary factors
- Blood or liver analysis for vitamin B12 and cobalt status

2.5. Selenium (Se) Deficiency

Selenium deficiency in cattle is often **associated with vitamin E deficiency**, as both act synergistically in **antioxidant defense** and **muscle protection**. Deficiency can affect **growth, immunity, reproduction, and muscular health**.

Physiological Roles of Selenium

- **Antioxidant protection** (component of glutathione peroxidase)
- **Muscle and liver function**
- **Immune system support**
- **Reproductive performance**

Major Clinical Syndromes

1. Nutritional Muscular Dystrophy ("White Muscle Disease")

a. Dyspnea–Myopathy Syndrome (Young Calves)

Age of onset: Typically 2 weeks to 3 months
Predisposing factors: Rapid growth, high nutritional demands, low Se intake

Clinical Features:

- Stiff gait, reluctance to move
- Difficulty standing; prolonged recumbency
- Muscle tremors, localized or generalized
- Tachypnea (up to 80–100 breaths/min), exaggerated respiratory movements
- Painful standing and recumbency positions
- Difficulty suckling and swallowing
- Animals often appear **thirsty despite maintained appetite**

Outcome:

- Rapid progression (hours to days)
- Death usually due to **cardiac failure**, sometimes with secondary infections (bronchopneumonia, enteritis)

Post-mortem Findings:

- Skeletal muscles pale, streaked, or soft ("fish or chicken flesh" appearance)
- Myocardial degeneration, especially in the left ventricle
- Pulmonary edema, spumous exudate in airways
- Possible renal congestion or discoloration

b. Stiffness Disease (Older Young Cattle)

Age of onset: 6–24 months (occasionally up to 26 months)
Context: Often after turnout to pasture; sometimes in housed animals

Clinical Signs:

- Stiff, slow, or uncoordinated gait
- Ataxia with asymmetry; severe cases may prevent standing
- Muscle hypertrophy, swelling, firmness, heat, and tenderness
- Respiratory signs present but less severe than in calves
- Constipation may occur; appetite and rumination generally preserved
- Animals remain **alert**, but movement is laborious

Outcome:

- Death can occur due to **acute cardiopulmonary failure** or secondary complications
- Myoglobinuria may be present but is inconsistent

Post-mortem Findings:

- Muscular lesions vary in location and severity
- Pale streaks or yellowish-white discoloration in muscles of **back, croup, shoulders**
- Bilateral symmetry common, but unilateral lesions can occur
- Diaphragm, intercostal muscles, tongue, myocardium, and limb muscles may also be affected

Key Point

Selenium deficiency is **clinically variable**, but early recognition is critical. It primarily affects **muscle and cardiac function**, and is especially dangerous in **rapidly growing calves** or animals with **high metabolic demands**.

2.6. Iodine (I) Deficiency

Onset of Clinical Signs

Cattle are **less sensitive than sheep and goats** to iodine deficiency. Short-term reductions in dietary iodine are often **buffered by recycling and storage mechanisms** in the cow.

In adult cows, clinical signs are usually **mild or absent**, whereas **newborn calves from iodine-deficient dams** may show **prominent clinical signs**, the most notable being **goiter**.

Physiological Roles of Iodine

- Essential for **thyroid hormone synthesis** (T3 and T4)
- Regulates **metabolic rate, growth, and development**
- Influences **reproductive performance and immunity**

Clinical Signs

1. Goiter in Calves (Most Common Sign)

- Visible or palpable enlargement of the **thyroid gland**
- Result of **thyroid hyperplasia** due to insufficient hormone synthesis
- Often occurs when the **dam has prolonged iodine deficiency**
- Mild deficiency in adults may **not produce detectable thyroid enlargement**

2. Reproductive and Neonatal Effects

- Weak calves at birth
- Increased risk of **placental retention**
- Reduced fertility in adult cows

Diagnostic Considerations

- Goiter is **not always present**, especially in moderate deficiency
- Other causes of thyroid enlargement (excess iodine, congenital anomalies, inflammation) must be excluded
- Histological analysis of thyroid tissue may be required for definitive diagnosis
- In calves born to deficient dams, **thyroid weight and histology** provide reliable confirmation

Key Point

Iodine deficiency in cattle is **often subtle**, primarily manifesting in **newborn calves**. Veterinarians must assess:

- Maternal dietary history
- Regional soil iodine content
- Clinical examination of calves for goiter or weakness

2.7. Manganese (Mn) Deficiency

Onset of Clinical Signs

Manganese deficiency can affect **both the dam and her offspring**.

- In pregnant cows, **maternal deficiency** may result in **congenital malformations** in calves.
- In young and adult cattle, deficiency signs appear **relatively quickly** when dietary intake is inadequate.

Physiological Roles of Manganese

- **Cofactor for numerous enzymes** involved in metabolism
- **Bone formation** and **cartilage development**
- **Reproductive hormone synthesis** (steroid hormones)
- Supports **growth and overall development**

Clinical Signs

1. Skeletal Abnormalities

- Most prominent and **early indicator** of Mn deficiency
- **Chondrodystrophy of limbs** in newborn calves (twisting or shortening of limbs)
- Joint deformities, particularly in **hind limbs**
- Decreased bone strength → risk of fractures
- Swelling of joints may occur

2. Growth Retardation

- Reduced body growth and poor weight gain
- Disproportionate limb development relative to body size

3. Reproductive Effects

- Infertility or subfertility in both sexes
- Potential disruption of **steroid hormone synthesis**

4. Other Signs

- Lameness due to skeletal weakness
- Rarely, secondary complications due to poor mobility

Key Point

Manganese deficiency is most evident in **skeletal malformations and impaired growth**, particularly in calves born to deficient dams. Early recognition depends on:

- **Observation of limb conformation in newborns**
- Assessment of **dietary manganese intake**
- Differentiation from other causes of skeletal deformities

2.8. Subclinical Deficiencies (Subcarences) and Combined Deficiencies

Overview

With modern feeding practices and widespread use of **mineral-vitamin supplements**, **acute trace element deficiencies** have become rare in cattle. However, **subclinical deficiencies** (subcarences) are increasingly important. These are **mild, chronic deficiencies** that may not

produce obvious individual symptoms but can significantly affect herd health and productivity.

Key Concepts

1. Subclinical Deficiency

- Occurs when **dietary intake barely meets minimum requirements**
- Clinical signs are **subtle or absent at the individual level**
- Herd-level effects include:
  - Reduced growth rates
  - Lower milk yields
  - Poor reproductive performance
- Often discovered **incidentally** during herd investigations for production issues

2. Combined Deficiencies

- Deficiencies of one element can **interfere with the absorption or metabolism of others**
  - Example: Excess zinc can impair cobalt absorption
  - Excess molybdenum or sulfur can induce secondary copper deficiency
- Multiple mild deficiencies may occur simultaneously, **amplifying negative effects**

Causes

- Selection of **high-yielding breeds** with higher nutritional demands
- Poor quality or low-mineral content of **forages** due to:
  - Soil depletion
  - Monoculture and plant selection for high yields
- Inadequate or irregular distribution of **mineral-vitamin supplements**
- Economic or logistical constraints limiting **balanced feeding**

Clinical and Herd-Level Indicators

- **No overt individual symptoms**
- **Recurrent, multifactorial health problems** in the herd, such as:
  - Respiratory infections
  - Digestive disorders
  - Poor wound healing
  - Delayed growth in calves
- **Subtle production losses** often the first sign:
  - Reduced milk fat or yield
  - Slower weight gain in growing stock

Key Point

Subclinical deficiencies are **economically significant** and may be overlooked because clinical signs are subtle. Veterinarians must assess:

- **Herd performance trends**

- **Feed and supplement analysis**
- **Blood or tissue assays** for trace elements and vitamins

2.9. Trace Elements and Disease Resistance

Trace elements play a **critical role in the immune system** of cattle. Even marginal deficiencies can reduce the animal's ability to resist infections and respond effectively to immunological challenges.

Physiological Roles in Immunity

1. Zinc (Zn)

- Essential for **lymphocyte proliferation** and **cell-mediated immunity**
- Deficiency may reduce **antibody production** and **resistance to infections**
- In calves receiving zinc-deficient rations, **delayed skin response** to intradermal phytohemagglutinin tests indicates impaired immune function

2. Copper (Cu)

- Cofactor for enzymes involved in **oxidative defense** and **white blood cell function**
- Deficiency leads to:
    - Impaired **neutrophil activity**
    - Reduced **macrophage function**
    - Increased susceptibility to **respiratory and digestive infections**

3. Selenium (Se)

- Component of **glutathione peroxidase**, protecting cells from oxidative damage
- Enhances **antioxidant defense** in neutrophils and other immune cells
- Deficiency increases **risk of mastitis, metritis, and muscular dystrophy**

4. Cobalt (Co)

- Supports **vitamin B12 synthesis** in the rumen
- Vitamin B12 is important for **red blood cell production** and **cellular energy metabolism**
- Deficiency may contribute to **anemia and reduced immune efficiency**

5. Iodine (I)

- Required for **thyroid hormone synthesis**
- Thyroid hormones influence **metabolic rate and immune function**
- Deficiency may indirectly impair **resistance to infections**

6. Manganese (Mn)

- Cofactor for **antioxidant enzymes** and **immune-regulating enzymes**
- Deficiency can contribute to **poor growth, skeletal anomalies, and reduced disease resistance**

Clinical Implications

- Herds with **marginal trace element intake** may appear healthy but have **higher incidence of infections**
- Performance losses may include:
  - **Delayed growth** in calves
  - **Lower milk production** in dairy cows
  - **Reproductive inefficiency**
- Preventive strategies rely on **balanced supplementation** tailored to herd needs

Key Point

Maintaining **adequate trace element intake** is essential not only for growth and reproduction but also for **optimizing immunity** and reducing the incidence of infectious diseases in cattle.

.10. Practical Guidelines for Prevention and Monitoring of Mineral and Vitamin Imbalances

Effective prevention of trace element and vitamin deficiencies requires a **systematic, herd-level approach** that combines feed analysis, supplementation, and monitoring.

1. Feed and Water Analysis

- Determine the **mineral and vitamin content** of:
  - Forages (fresh, hay, or silage)
  - Concentrates and commercial feeds
  - Water sources
- Identify **deficient or imbalanced elements** before supplementation

Key Tip: Seasonal variation in forage composition should be considered, as mineral content may **decline during storage**.

2. Soil and Regional Assessment

- Evaluate local **soil mineral content** to predict deficiencies in grazing animals
- Certain regions are naturally low in **Se, I, Cu, or Mn**
- Adjust **supplementation strategies** accordingly

3. Balanced Supplementation

- Provide **minerals and vitamins according to age, production stage, and physiological status**
- Methods of supplementation include:
  - **Mineral blocks or licks** (free access)
  - **In-feed premixes**
  - **Injectable preparations** for high-demand situations (e.g., late gestation or early lactation)
- Monitor for **over-supplementation**, which may cause toxicity (especially Cu, Se, and vitamin A)

4. Monitoring and Clinical Observation

- Regularly check **blood or tissue levels** of key elements in representative animals
- Observe herd-level signs:
  - Reduced growth rates
  - Lower milk production
  - Recurrent infections
  - Subtle clinical signs (coat, hooves, or skeletal anomalies)
- Maintain **records of supplement usage, herd health, and production data**

5. Herd Management Considerations

- Ensure **adequate access** to minerals in both stall-fed and pasture systems
- Adjust **quantities based on herd size and individual intake variability**
- Maintain **clean, accessible mineral sources**, especially in group housing or pasture systems
- For high-yielding animals, pay particular attention to **trace element interactions** (e.g., Mo–S–Cu, Ca–Zn)

Key Point

A **proactive, preventive approach** combining **diet analysis, supplementation, and herd monitoring** is essential for maintaining **optimal health, production, and reproductive performance** in cattle. Early detection of deficiencies—even subclinical—is crucial to prevent economic losses and disease susceptibility.

Element / Vitamin	Main Roles	Risk Situations	Deficiency Signs	Excess / Toxicity
Calcium (Ca)	Skeleton, muscle contraction, blood balance, milk component	Unprepared dietary transitions around calving	Hypocalcemia, milk fever, fractures, osteomalacia, chronic lameness	Reduces absorption of trace elements
Phosphorus (P)	Skeleton, energy metabolism, rumen pH buffer	High growth, milk production	Growth retardation, lameness, reduced fertility, low milk production	Imbalanced Ca:P → uterine atony, fertility drop; environmental pollution
Magnesium (Mg)	Energy metabolism, muscle contraction	Grazing on Mg-poor pastures	Hypomagnesemic tetany, retained placenta, slow growth	Rare, but excess can affect Ca absorption
Sodium (Na)	Nerve impulses, muscle contraction, blood balance	Insufficient salt intake	Loss of appetite, pica, reduced milk fat; with K deficiency → acidosis	Rare in cattle
Potassium (K)	Nerve impulses, muscle	Excessive high-K forage	Accelerated digestive transit, fat mobilization,	Rare; may increase Na deficiency

	contraction, blood balance		alkalosis, udder edema risk	
Sulfur (S)	Protein synthesis in rumen	Excess non-protein nitrogen	Reduced forage intake, hair loss, production decline, growth defects	Rare in cattle
Copper (Cu)	Enzymatic cofactor, pigmentation, connective tissue, hemoglobin, immunity	High Mo/S intake, low soil Cu	Hair depigmentation, skeletal fragility, anemia, infertility, diarrhea in young	Liver damage, hemolysis, jaundice
Zinc (Zn)	Skin & hoof health, enzyme cofactor, growth	High Ca/phytate diets	Parakeratosis, alopecia, hoof deformities, poor wound healing	Rare; chronic excess → Cu deficiency
Manganese (Mn)	Bone & cartilage formation, reproduction, enzyme cofactor	Low Mn soil, high Ca intake	Skeletal deformities, joint swelling, poor fertility	Rare; high intake may interfere with Fe absorption
Iodine (I)	Thyroid hormone synthesis	Iodine-deficient soils	Goiter, weak calves, reproductive failure	Thyroid dysfunction in excess
Cobalt (Co)	Vitamin B12 synthesis in rumen	Low dietary Co	Growth retardation, weight loss, anemia, rough coat, diarrhea	Rare; may interact with Zn
Selenium (Se)	Antioxidant (glutathione peroxidase), muscle & liver health	Low Se soil, high S intake	White muscle disease, retained placenta, immunodeficiency	Hair loss, hoof lesions, neurological signs
Vitamin A	Vision, epithelial health, reproduction	Winter with low green forage	Night blindness, keratinization, reproductive failure	Bone abnormalities, liver damage
Vitamin D	Ca and P metabolism	Animals indoors with low sun exposure	Rickets, osteomalacia, skeletal fragility	Calcification of soft tissues (kidney, aorta, joints)
Vitamin E	Antioxidant, muscle & immune health	Low forage vitamin E	White muscle disease, retained placenta, low immunity	Rare; usually safe
Vitamin K	Blood clotting	Antibiotic-induced gut disruption	Hemorrhages, prolonged clotting time	Rare
B-Complex Vitamins	Metabolism (carbs, proteins, fats), RBC	Stress, high-producing animals	Anorexia, poor growth, neurological signs, anemia	Rare

	formation, nervous system			
Vitamin C	Antioxidant, collagen synthesis, immunity	Generally synthesized in rumen	Deficiency rare in ruminants	Excess generally safe