

A Guide to the Principles of Animal Nutrition

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Preface

My motivation to write this book came from my interest in helping students to understand and enjoy basic food animal nutrition concepts. I have been teaching principles of animal nutrition (ANS 311), a 3-credit core course in animal nutrition for over a decade at Oregon State University. This book represents the materials I have prepared as a study guide-cum-workbook to facilitate learning for the less-experienced students registered in ANS 311. It is not my intention to provide a comprehensive information on animal nutrition and feeding. My goal is to help students to learn food animal nutrition by focusing on some basic underlying concepts and theories of animal nutrition and feeding rather than providing information on ration formulation. This will be helpful for students with limited animal nutrition background who might otherwise miss important facts or information in the current full-scale textbooks. My intended audience is agricultural science students, students from other disciplines who are planning on a career in animal sciences or veterinary medicine, and faculty members teaching courses related to animal nutrition.

It is my hope that this book will serve as a useful resource for students in the “digital” age who learn and access information through electronic or online venues. There are 20 chapters in this book with a focus on the different fundamental nutrients, their structure, digestion, and metabolism. The first two chapters briefly introduce nutrient analysis and digestive organs and processes. The last two chapters are on feed additives and methods for assessing nutrient utilization. At the beginning of each chapter, I have added boxes that contain the new vocabulary and the major learning objectives. At the end of each chapter, I have “key points” section that highlights the summary followed by review questions. Overall, I have tried to include concepts without unessential details. Through this format, my goal is to give students the tools and help to understand animal nutrition by focusing on some key topics and concepts.

I am indebted to the outstanding group of editors in the Open Educational Resources Unit (formerly Open Oregon State), who have invested so much of their time in providing editorial expertise and exceptional attention to details, sometimes under difficult conditions. A special word of thanks goes to Dr. C.Y. Hu for providing me much of his study materials while taking over teaching of animal nutrition course at Oregon State University. Finally, I would like to express my gratitude to Oregon State University Libraries and Press and Ecampus for honoring me with the open access text pilot program award.

I hope that the book will provide useful basic information on principles of food animal nutrition to students worldwide.

Introduction

The science of nutrition can be defined as the sum of different biochemical and physiological processes which transform food/feed components into body elements that are required for sustaining life, growth, health, and productivity. In farm animals such as livestock, pigs and poultry, nutrition is also important in maintaining food (e.g. meat, milk, eggs) product quality, minimizing the cost of production, and loss of undigested nutrients. Therefore, an understanding of basic nutrition concepts are essential for formulating rations and developing feeding practices for enhancing efficiency of food production while protecting the environment and maintaining the nutritional value of animal-derived foods.

This book entitled “A Guide to Principles of Animal Nutrition” consists of 20 chapters. As the name says, this book is a guide primarily meant to serve students taking animal nutrition courses at the university level and is not a full-scale animal nutrition text. Information digestive anatomy and processes provide the foundation to understand how animals utilize nutrients. The text begins with basic information on feed nutrient analysis, anatomical and physiological bases of digestion in food producing ruminant as well as non-ruminant animals. Description of chemical structure, digestion, absorption, assimilation, and metabolism of energy-producing nutrients followed by vitamins and minerals are emphasized in rest of the chapters. To integrate the basic knowledge of nutrition with practical animal feeding, the book closes with two chapters discussing some of the major feed additives used in animal diets and methods used in assessing feed nutrient utilization.

The most distinguishing characteristic and unique contribution of this book is that it will take advantage of advances in digital teaching technologies to address the current learning trends of a new generation of veterinary/agriculture science students and will enable wide distribution of the educational materials in animal nutritional sciences.

I. Introduction to Nutrition

This chapter provides an introduction to basic nutrition terminology, and discussion of fundamental nutrients in nutrition of food producing animals.

New Terms

Acid Detergent Fiber
Ash
Crude Fiber
Crude Protein
Detergent Fiber System
Dry Matter
Ether Extract
Feed
Fundamental Nutrients
Neutral Detergent Fiber
Nitrogen Free Extract
Nutrient
Proximate Analysis

Chapter Objectives

- To introduce and discuss the basic concepts of nutrition and some basic nutritional terminology
- To introduce and discuss fundamental nutrients in animal diets

Concepts of Nutrition

Nutrition is a relatively new science. It is an applied science that encompasses the principles of other sciences, such as chemistry, biochemistry, and physiology.

Animal nutrition deals with the nutritional needs of food-producing, companion, or service animals. It is the science of preparation or formulation of feed for animals that produce food (e.g., meat, milk) or nonfood materials (e.g., wool). Animal nutrition also is an integrative science, as it deals with the different steps by which the animal assimilates feed, or food, and uses it for its growth, health, and performance (e.g., meat, milk, and egg production and service).

In addition to the health, welfare, or productivity of the animal, food animal nutrition is also very important due to economic (e.g., feed cost) and environmental aspects (manure and undigested, wasted nutrients, such as phosphorus and nitrogen, contaminating air, soil, and water), as well as nutritional quality (eggs, meat, milk).

Nutrients are chemical elements or compounds present in feed that support health, basic body maintenance, or productivity. Fundamental nutrients include water, carbohydrates, protein, fat, vitamins, and minerals.

Why Is Nutrition Important in Livestock?

Nutrition is important for all organisms. However, in food-producing animals, it is especially important due to the nature of the production systems (e.g., confinement), the economics of production, or the products (e.g., meat, eggs, milk) generated.

Feed nutrients, such as nitrogen and phosphorus, are lost into the environment through manure, which if not managed

properly, can lead to environmental pollution. The emission of methane and nitrous oxide from manure is also to some extent dependent on the nature of feed being fed to livestock. Use of good-quality feeds with high digestibility will minimize or reduce environmental pollution.

Feed represents the major expense for raising food animals. For example, feed amounts to more than 65% of the expense in swine or poultry production systems. As world population increases, there is an additional demand for food, land, and energy. As a result, feed production with limited resources will be a challenge in the context of sustainability.

Consumers' perception of the effect of diet on health has increased markedly over the past two decades. This perception has an impact on consumer food choices, especially with regards to certain nutrients in animal products (e.g., saturated fats, cholesterol). Therefore, nutrition is important for producing health-promoting foods for human consumption.

Improper nutrition (under- or overfeeding) can affect animal health. Balanced nutrition can enhance immune health, welfare, productivity, and longevity. Overall, the nutrition of livestock is very important due to their dependence on humans, especially when food animals are raised in confinement. It is also important for economic reasons, to produce human food with limited resources, and to enhance animal productivity, health, and welfare.

Why Nutrition is Important

- Dependence on humans (e.g., confinement)
- Economics
- Environmental protection
- Enhancement of food production with limited resources
- Human health-promotion and food quality enhancements
- Animal health and welfare

Nutrient Analysis of Feedstuffs

The 19th century had a significant impact on modern animal nutrition. Developments during this period include the introduction of fundamental nutrients and the separation of feed into protein, fat, and carbohydrate components. In this respect, proximate analysis, a combination of analytical procedures devised more than 100 years ago by German scientists at the Weende Experiment Station (also known as Weende analysis), paved the way for estimating the nutrient content of feed samples. Although detailed knowledge of different analytical procedures is not required, familiarity with different basic feed analyses will enhance learning and understanding of animal nutrition.

Why Perform Nutrient Analysis of Feedstuffs?

Animal nutrition is the science of feed preparation (formulation) and feeding to meet the needs of animals at different phases of growth, or life stages. Therefore, nutritionists need to know the nutrient components of the feed or the raw materials used in ration formulation. Nutrient analysis serves as a system to analyze the feed and the needs of the animal, enabling producers to optimize nutrient utilization in feed and helping researchers relate to animal performance, tackle issues of underperformance, and reduce food production costs.

Sampling Feed for Analyses

Reasons for Nutrient Analyses in Feed

- Ration formulation and feeding
- Trouble shooting
- Economics

Modern chemical methods and equipment need only a small amount of the feed (2 to 10 g) for analyses. Therefore, sample materials collected and prepared for analyses should represent the best reasonable estimate of the total feed fed to animals. Sample integrity during preparation (e.g., grinding, drying), storage (e.g., temperature), and transportation should be considered. The frequency of feed analysis depends on batches of feed made, variability of feed sources

(e.g., cultivar, location of growth), and cost of analyses. Several core samples should be taken, combined, ground, and subsampled. Avoid taking a sample directly from outside of a bale (use common sense)! Weather patterns should also be considered, as they can affect the moisture content of the sample.

Samples taken for analyses should represent the entire feed, ration, bulk, bale, or load. The bottom line is that analysis will only be as accurate as the sample collected. If a sample is inaccurate, analysis is a waste of money.

Analytical Methods

Traditionally, feedstuffs are subjected to different protocols of laboratory analyses (wet chemistry) for nutrient profiling. These analytical procedures are specific for a given element (e.g., N), compound, or group of compounds. Chemical methods often employ drastic degradation of the sample with different acids or other solvents and may not be true estimates of an animal's ability to utilize them efficiently. However, considering the time and cost of other methods using live animals (e.g., explained in chapter 20) that provide more accurate estimates, laboratory analyses are used widely to get a head start.

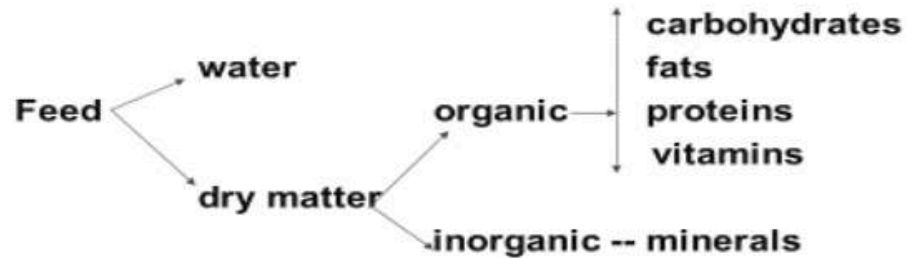
Proximate Analysis

Proximate analyses are a combination of analytical procedures developed in 1865 by Wilhelm Henneberg and Friedrich Stohmann at the Weende Experiment Station in Germany. They are based on the elimination of water from the feed (as shown later) and then the determination of five proximate principles in the remaining dry matter (DM). They are as follows, and their names refer to specific proximate principles:

Proximate Analysis

1. CRUDE PROTEIN
2. ETHER EXTRACT
3. ASH
4. CRUDE FIBER
5. NITROGEN-FREE EXTRACT

Proximate analysis 'Chemistry of Feed'



Dry Matter

The determination of dry matter (DM) is the most common procedure carried out in nutrition laboratories because plant feedstuffs may vary in water content. The amount of water content must be known to permit comparisons of different feeds.

DM is determined by drying the test material at 105° C overnight in an oven. DM is then determined by the following calculation:

$$\text{dry weight} / \text{fresh weight (also called as-fed weight)} * 100 = \% \text{ DM.}$$

Most feeds are around 90% DM, and silages are about 30% to 35% DM. Possible errors in DM analyses include loss of volatile fatty acids (VFAs), essential oils, lactic acid in silage, or any other fermented products. Moisture can also be determined by moisture meters, but results are not as precise as those obtained by drying testing materials in the oven. Freeze-drying or drying at lower temperatures can minimize errors.

The following is an example of DM calculation on a batch of corn silage samples:

Fresh (as-fed) weight = 2 kg
Dry weight = 0.7 kg
DM % = $(0.7 \text{ kg}/2.0 \text{ kg}) \times 100 = 35\%$

A dry matter (DM) test estimates moisture.

The higher the DM, the lower the moisture.

Crude Protein (CP)

The procedure to estimate crude protein was developed by a Danish chemist, Johan Kjeldahl and is commonly known as “Kjeldahl” procedure. The Kjeldahl analysis depend on the measurement of nitrogen (N) in the test material. To convert the measured N content of the test material to crude protein, a calculation factor of 6.25 ($N \times 6.25$) is applied. This is based on the fact that all proteins contain about 16% N ($100/16 = 6.25$) or 16 g of N comes from 100 g protein, or 1 g of N is associated with $100/16 = 6.25$ g of protein.

NITROGEN (N) * 6.25 = CRUDE PROTEIN (CP)

The following is an example of crude protein calculation on a batch of soybean meal samples:

Nitrogen content = 7.35 g

Crude protein = $7.35 \times 6.25 = 45.9$ g

The Kjeldahl procedure measures nitrogen, not protein.

This process of nitrogen determination involves boiling the dried samples in 36 N sulfuric acid (H₂SO₄). This will convert nitrogen to ammonium sulfate ([NH₄]₂SO₄). The mixture is then cooled and neutralized with 12 N sodium hydroxide (NaOH). This will release ionized ammonium. The sample is then distilled, and the distillate containing the ammonium is titrated with 0.02 N sulfuric acid. This analysis is accurate and repeatable but time consuming and involves the use of hazardous chemicals. The information obtained on N content and hence CP content is of limited use to nonruminants, such as pigs and poultry, as it does not indicate the quality of the protein, but it is applicable to ruminant animals that can efficiently utilize all forms of N.

A possible error in the Kjeldahl method is assuming all nitrogen presented in the sample is in protein form. This assumption is not necessarily true because nitrogen could be in nucleic acids (ribonucleic acid (RNA) or deoxyribonucleic acid (DNA)) or can exist as nonprotein nitrogen, such as urea.

Ether Extract

Ether-soluble materials in feed include different organic compounds that are soluble in organic solvents. In animal feeds, ether extract may include fats, fatty acid esters, and fat-soluble vitamins and hence are often referred to as crude fat. The primary goal of ether extracts is to isolate the fraction of the feedstuff that has a high caloric value. A portion of the dried feed sample is boiled in ether (organic solvent) for four hours. Since fats are soluble in ether, ether extract is equivalent to fat. Provided the ether extract contains fats and fatty acid esters, this approach is valid. However, in samples that contain high levels of other compounds soluble in organic solvents, such as plant waxes or resins, it may not give a true estimate of feed caloric value. However, this error is generally small in typical animal feedstuffs. Overall, this test does not indicate anything about the quality of the fat in the feed.

The Ether Extract procedure assumes substances soluble in ether are fats.

Ash

Ash is the residue remaining after all the organic nutrients have been burned off or oxidized completely in an oven at 500° to 600° C for two to four hours. Nutritionally, ash values have little importance, although high values may indicate contamination (e.g., soil) or dilution of the feed sample with limestone or salt. Ash values obtained are cumulative of all the mineral elements combined together. High temperatures used for burning may cause loss of some volatile elements such as chloride, zinc, selenium, iodine, and so on. Consequently, ash values can underestimate mineral contents. However, this error is small. Identifying individual minerals may be more meaningful and useful. If ash values are not very useful, why obtain them? They allow for calculations of nitrogen-free extract compared to DM (see later).

An ash test measures inorganic compounds in feed.

High ash values indicate feed contamination.

Crude Fiber

Crude fiber estimates the indigestible fraction of feed or those fractions of the feed that are fermented in the hindgut by microbes. Crude fiber includes different insoluble carbohydrates that are associated with the cell wall of plants and are resistant to the action of digestive enzymes. Crude fiber is made up of plant cell structural components, including cellulose, hemicellulose, lignin, and pectin. For nonruminant animals, crude fiber is of little value energy-wise. However, it is important for maintaining hindgut health and microbial population. Crude fiber is important in the diets of ruminant animals, which can ferment a large portion of it. Crude fiber is described in detail below.

**Crude fiber measures fermentable components of the feed.
Crude fiber has little energy value but is important for gut health in pigs and poultry.
Ruminant animals can ferment a large portion of crude fiber.**

To determine crude fiber in feed, a sample is dried, boiled in weak sulfuric acid (1.25% H₂SO₄), and filtered. The residue is boiled in a weak alkali (1.25% NaOH) and filtered, and the remaining residue is dried and ashed. The difference between the filtered dried sample and ash is crude fiber. The two boiling processes simulate the pH conditions of the digestive tract, acidic in the stomach and alkaline in the small intestine. However, the enzymatic digestion in the digestive tract is not simulated in the procedure.

Crude fiber tests underestimate true fiber in feed.

A major problem with this procedure is that the acid and base solubilize some of the true fiber (particularly hemicellulose, pectin, and lignin), and some cellulose is partially lost too. Hence crude fiber underestimates true fiber in the test material. The number, or value, obtained in this procedure, therefore, is practically meaningless. Most laboratories have phased out the crude fiber term and replaced it with the detergent fiber system (discussed in detail later)

Nitrogen-Free Extract

The term nitrogen-free extract (NFE) is a misnomer, as there is no nitrogen or extraction process in this procedure. Nitrogen-free extract is not determined analytically in the laboratory, as shown below. NFE supposedly represents the soluble carbohydrates of the feed, such as starch and sugar, and is the difference between the original sample weight and the sum of the weights of moisture (water), ether extract, crude protein, crude fiber, and ash. Therefore, it accumulates the errors of the other analytical systems. It is an overestimate of true NFE.

$$\% \text{ NFE} = (\% \text{ DM} - (\% \text{ ether extract} + \% \text{ crude protein} + \% \text{ ash} + \% \text{ crude fiber}))$$

Nitrogen-free extract is a calculated value and not an analyzed value.

“All Fibers Are Not Created Equal”

Peter J. van Soest (1982) improved methods of crude fiber analyses into the **detergent fiber system**. The concept behind the detergent fiber system is based on the fermentability or digestibility of fiber. Accordingly, plant cells can be divided into cell walls (which contain hemicellulose, cellulose, and lignin and are less digestible) and cell contents (which are mostly digestible, such as starch and sugars) (shown below in detail).

Use of these methods allows plant components to be divided into **neutral detergent fiber (NDF)** and **acid detergent fiber (ADF)**.

The detergent fiber system includes neutral detergent fiber and acid detergent fiber.

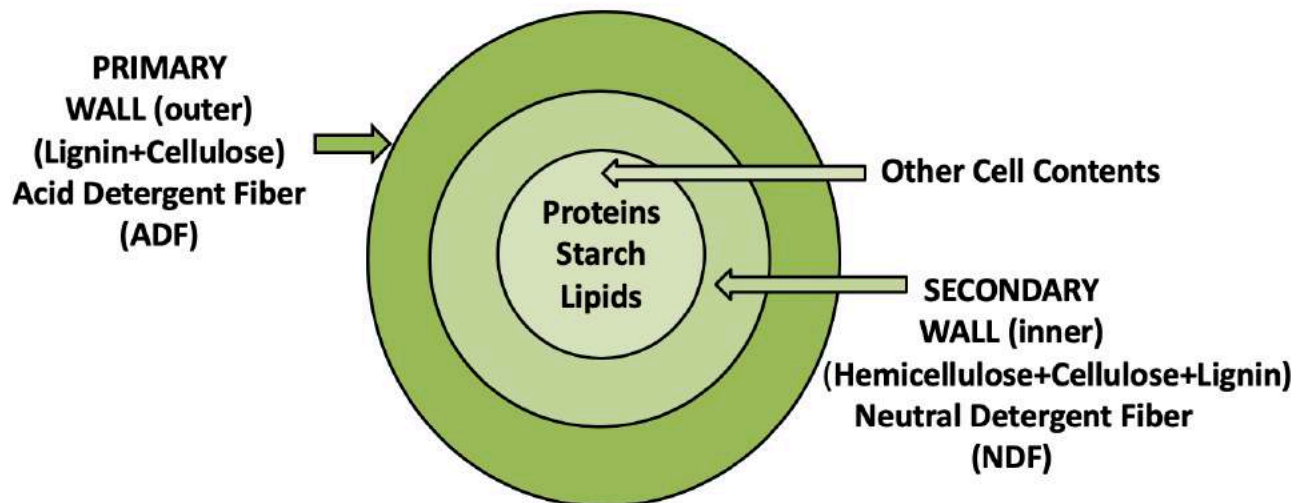
NDF contains the major cell wall components, such as cellulose, hemicellulose, and lignin. It may also contain other very important components, such as cutin, and some proteins too. Hemicellulose, cellulose, and lignin are indigestible in nonruminants, while hemicellulose and cellulose are partially digestible (fermentable) in ruminants.

NDF fractionation is determined by boiling feed samples for one hour in a solution containing sodium lauryl sulfate and ethylene diamine tetra acetic acid (EDTA) at pH 7.0. This detergent extracts soluble components of the feed (protein, sugars, lipids, and organic acids), and the nonsoluble material is called NDF.

$$\text{NDF} = \text{Hemicellulose} + \text{Cellulose} + \text{Lignin}$$

Acid detergent fiber is an estimate of cellulose + lignin in the feed sample. Hemicellulose, therefore, is estimated as NDF – ADF. This is not a perfect system, as there are contaminants in both ADF and NDF terms. ADF does the best job of describing the portion of feed it is designed to estimate (i.e., cellulose + lignin). The ADF and NDF terms have now largely replaced the crude fiber term. By using this method, we can better predict the digestibility of forages for animals. Nowadays, most laboratories use NDF and ADF analysis instead of crude fiber.

Cross Section of Plant Cell



“Crude Fiber” = Neutral Detergent Fiber + Acid Detergent Fiber

Key Points

1. The broad classifications of nutrients are water, protein, fat, carbohydrate, minerals, and vitamins. These classifications are so broad that analysis of these has limited value.
2. For analysis, feed should be sampled as many times as possible and then dried, ground, and mixed for subsampling.
3. Drying is used in the first step of proximate analysis to determine the water content of a feedstuff. Some components of a feed may be lost through volatilization at this time. Usually, this is a small error.
4. To reduce damage to feed, alternatives to drying include freeze-drying or drying at lower temperatures (i.e., 55° C)
5. Ether extract (EE) is determined by extracting the dried sample in organic solvent (ether). It represents the fat content in the sample. It assumes all the substances soluble in ether are fat, which is not true.
6. Crude protein (CP) is determined by the Kjeldahl method. It analyzes the N content of a diet and calculates protein using the assumption that all protein is 16% N. The problems with this are that some proteins are not 16% N and some feed constituents that contain N (i.e., urea, DNA, RNA) are not proteins.

7. Ash is used to determine mineral content. It provides no information on actual amounts of individual minerals. It provides an estimate of the total inorganic component of the diet, which is often interpreted as contamination. Many feed tags indicate a maximum limit for ash as an index of quality.
8. Crude fiber (CF) is an estimate of the cell wall constituent of a feed. Ideally, it should represent cellulose, hemicellulose, and lignin; however, the process of digesting feed with weak acid then weak base solubilizes some of these components (especially lignin and hemicellulose), and as a result, CF underestimates true fiber. It is the major limitation of the proximate analysis system.
9. Nitrogen-free extract (NFE) is designed to provide an estimate of water-soluble polysaccharides (sugars, starch) and is calculated by the difference between the original sample weight and the sum of weights of moisture (water), ether extract, crude protein, crude fiber, and ash. Therefore, it accumulates the errors of the other analytical systems. It is an overestimate of true NFE.
10. Van Soest developed improved methods of fiber analyses (the detergent fiber system). Acid-detergent fiber (ADF) is an estimate of cellulose + lignin, whereas neutral detergent fiber (NDF) is an estimate of cellulose + hemicellulose + lignin. Hemicellulose therefore is estimated as $NDF - ADF$. This is not a perfect system, as there are contaminants in both ADF and NDF terms. ADF does the best job of describing the portion of feed it is designed to estimate (i.e., cellulose + lignin). The ADF and NDF terms have now largely replaced the crude fiber term.

Review Questions

1. How would you define “nutrition”?
2. Why is nutrition important in today’s livestock production?
3. What are the six major classes of nutrients?
4. Proximate analysis of a feed includes the following tests:
5. Crude protein (CP) is determined by the Kjeldahl method. It analyzes the content of _____ in the diet.
 - a. Nitrogen
 - b. Minerals
 - c. Protein
 - d. Water
6. Ether extract is determined by extracting the dried sample in organic solvent (ether). It represents which component of the feed sample?
7. As the dry matter content of a feed increases, the moisture content
 - a. Increases
 - b. Decreases
 - c. Remains the same
8. Among the different proximate analyses, this is a calculated value
 - a. Dry matter
 - b. Crude protein
 - c. Crude fiber

- d. Nitrogen free extract
9. This test measures the inorganic component of feed in proximate analysis
- a. Ether extract
 - b. Moisture
 - c. Crude fiber
 - d. Ash
10. A researcher conducted nitrogen (N) analysis on an unknown feed sample and was found to be 7.0 g. The crude protein (g) content of the feed sample is calculated as follows:
- a. $7.0 + 6.25$
 - b. $7.0 - 6.25$
 - c. $7.0/6.25$
 - d. 7.0×6.25
11. Select the component of forage that is NOT a part of neutral detergent fiber (NDF)
- a. Starch
 - b. Hemicellulose
 - c. Lignin
 - d. Cellulose
12. Differentiate between neutral detergent fiber (NDF) and acid detergent fiber (ADF).

II. Gastrointestinal Tract, Digestive Organs, and Processes

This chapter provides an introduction to the gastrointestinal tract and organs involved in reception, digestion, and absorption of nutrients from feed as it passes through the gastrointestinal tract in livestock.

New Terms

Abomasum
Carnivore
Cecum
Colon
Crop
Duodenum
Foregut/Hind-Gut Fermenter
Gizzard
Herbivore
Ileum
Jejunum
Monogastrics
Omasum
Omnivore
Reticulum
Rumen
Villi

Chapter Objective

- To introduce the different organs of the gastrointestinal tract in omnivores and herbivores that are involved in digestion and absorption.

The nutritional requirements and digestive processes of domestic animals are greatly influenced by the nature of the gastrointestinal (GI) tract, or “gut.” Domestic animals include carnivores (e.g., cats), omnivores (chickens, pigs), and herbivores (cattle, sheep, horses). Herbivores can be either pregastric or postgastric fermenters (based on the site of fermentation in the GI tract). The type of GI tract can also affect the nutrient needs of the animal. For example, carnivores such as cats need animal protein and fats in their diets and require more protein than other animals. Feeding a plant-based diet to cats can lead to nutrient deficiencies and health problems.

Many animals, such as cows, have multiple, compartmentalized stomachs and are commonly referred to as ruminants. Animals such as pigs, dogs, and chickens have simple noncompartmentalized stomachs and are commonly referred to as nonruminants or monogastrics.

The nutritional requirements of animals are greatly influenced by the nature of the “gut.”

Monogastric animals have a single stomach, while ruminants have multiple, compartmentalized stomachs.

Physiologically, monogastric animals are autoenzymatic digesters (auto = self) in the sense that the enzymes secreted by the animal itself are involved in the digestive processes, whereas ruminant animals are alloenzymatic digesters, in which digestion is accomplished by “others” (allo = others) or microbes residing in the gut. Since the microbes are involved in the “processing” of the ingested feed, instead of “digesting,” fermenting may be the more appropriate term to designate digestive processes in ruminant animals. The site of fermentation varies in alloenzymatic digesters. For example, cattle and sheep are foregut fermenters, while horses and rabbits are hindgut fermenters. Overall, food-producing animals

such as pigs and chickens and companion animals (cats, dogs) have a pouch-like, noncompartmentalized stomach, whereas ruminant animals (cows, sheep) have more specialized fermenting chambers. Classification of animals by digestive tract fermentation site is shown in Table 2.1.

Table 2.1. Classification of animals by digestive tract fermentation

Site of Fermentation	Example
Fore-gut	Cattle, sheep, elk, deer
Hind-gut	
Cecal	Rabbit
Colonic	Horse
Ceco-colonic	Elephant

Ruminants

- Multiple compartments
- High storage capacity
- Microbial fermentation
- Microbes provide energy, protein, and B vitamins

Monogastric

- Single compartment
- Limited capacity
- Limited microbial fermentation
- Concentrated feeds and well-balanced diets (e.g., amino acids / vitamins) needed

Monogastric Animals: Gastrointestinal Tract and Digestive Process

The gastrointestinal tract prepares the food (feed) for digestion and absorption. Absorption is the passage of digested nutrients through the gut wall. Digestive processes include mechanical, chemical, and enzymatic processes.

The overall function of the digestive processes is to reduce the feed to a molecular size or increase solubility that allows for absorption and utilization of nutrients from the feed by the cells of the GI tract.

In monogastric animals, digestive processes start with the mouth, tongue, and esophagus and continue through the stomach and small and large intestines. These structures are associated with feed reception, mastication/chewing/swallowing (mouth and tongue), and digestion and absorption (stomach and small and large intestines). The other associated organs involved in the digestive processes are the liver (bile secretion) and pancreas (secretion of several enzymes and hormones).

Features such as the tongue, lips, and/or beak or mouth help in the feed prehension (grabbing) process, and teeth help in chewing and breaking the feed into smaller particles and swallowing. The food is mixed with saliva, which helps in moistening the feed and swallowing. The saliva also contains enzymes such as amylase and lipase (more in newborn animals). The saliva produced by the salivary glands (parotid, submaxillary, sublingual) is added during mastication.

Saliva helps in bolus formation and softening of feed, as well as antibacterial action. The enzyme effects of saliva (e.g., amylase, lipase) are minimal due to the short time feed is present in the mouth.

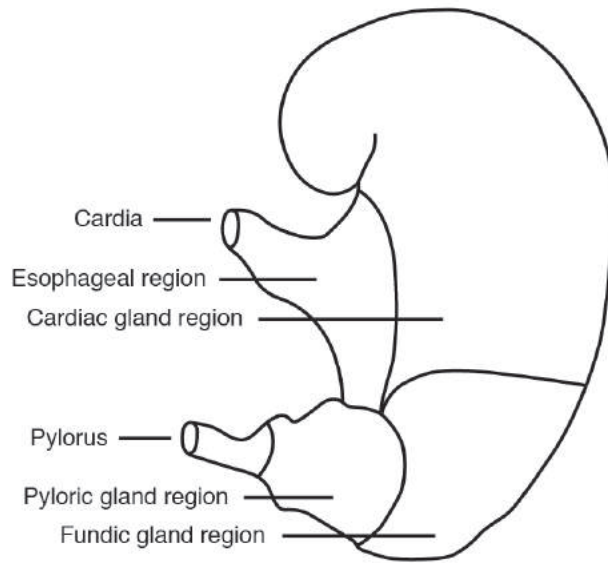


Figure 2.1. Sections of the stomach Source: <http://www.thepigsite.com>

Feed moves down through the esophagus in peristaltic movements through the esophagus into the stomach. There are no glandular secretions in the esophageal region. The stomach is a muscular organ. Functions of the stomach are to serve as a portal or storage of consumed feed and initiate the breakdown of nutrients. The stomach helps in mixing, enzyme secretion, and digestion. The stomach of a monogastric animal includes four functionally distinct zones (Figure 2.1). Adjacent to the esophageal region is the cardiac region, which contains glands that produce mucus. The mucus, a glycoprotein, serves as a protective layer for the stomach wall from acidic secretions and has some antibacterial properties. The fundus gland region and pyloric region are the sites of other secretions such as HCl; gastric secretions, including pepsin; and protein-digesting enzymes. The pH of stomach content varies from one to three due to the acidic action of HCl. The high acidity also provides antibacterial protection to the stomach. The ingested

feed exits from the stomach to the duodenum through the pyloric sphincter, which is under hormonal control to not overload the digestive capacity of the small intestine.

Small Intestine

The major site of digestion and absorption in monogastric animals is the small intestine. It is composed of three distinct regions: the duodenum, jejunum, and ileum. The duodenum is the point of entry for secretions such as bile from the gall bladder and secretions from the pancreas.

A major site of digestion and absorption in monogastric animals is the small intestine.

In order to fit into the small body cavity, the small intestine is highly coiled. In addition to the digestive function, the small intestine is also an immune organ and has special features such as gut-associated lymphoid tissue, Peyer's patches, and Bursa fabricii (in chickens), serving as part of the body's immune defense.

Both digestive and absorptive functions of the small intestine are facilitated by a large surface area. The main increase in surface area is brought about by anatomical features such as villi and microvilli. The villi are small finger-like projections lining the intestinal mucosa and giving it a velvety appearance (Figure 2.2). Each villus is further surrounded by a large number of minute finger-like projections called microvilli. The purpose of these anatomical features is to enhance surface area and thereby absorptive capability (> 60-fold). Each villus contains lymph vessels and capillaries for nutrient transport. The villi are lined with a single layer of cells called enterocytes.

Anatomical features such as villi and microvilli increase the surface area and absorptive capacity of the small intestine.

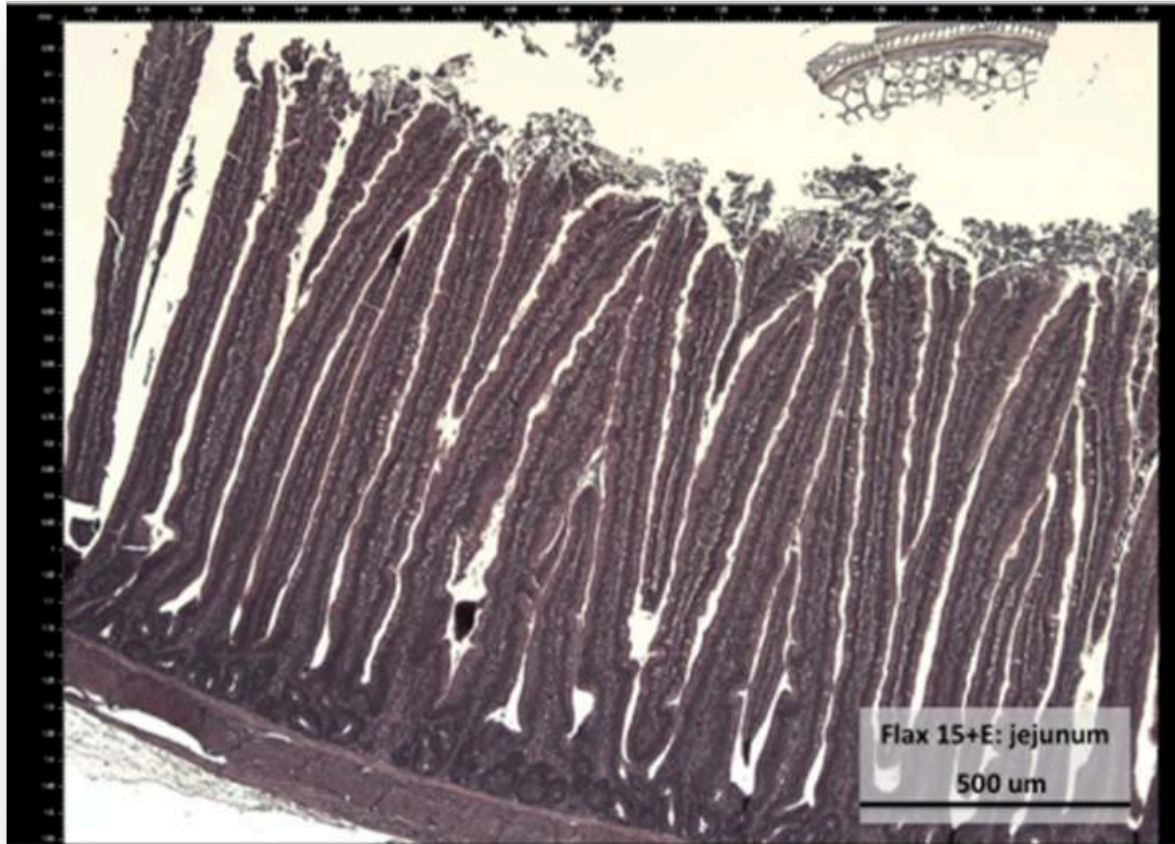


Figure 2.2. Section of jejunum showing villi

The immature enterocytes are formed in the crypts, and as they mature, they move up the villi, acquiring full complements of the digestive enzymes. As they reach the tip of the villi, they are “worn out” and are extruded into the intestinal lumen. These sloughed-off cells are endogenous (coming from within) in origin and contribute to the metabolic fecal nitrogen. Besides enterocytes, villi also have goblet cells involved in mucus production. The mucus functions as a blanket providing protection from bacterial infection as well as lubrication for digesta.

The large intestine is made up of the cecum, colon, and rectum. The size of the large intestine varies among species based on the type of feed consumed. Hindgut-fermenting animals, such as horses and rabbits, have relatively large ceca. Most digestion and absorption is complete by the time feed residue reaches the colon. The colon functions primarily in water and mineral absorption. Also, some microbial fermentation of plant fiber occurs in the colon. Generally, hindgut fermentation contributes little energy to the animals. However, hindgut fermentation is very important for maintaining gut health and food safety in meat-producing animals.

Hindgut fermentation provides energy to the microbes and is important for maintaining gut health.

The liver and pancreas are vital to the digestive process. The liver is the largest gland and is a central organ in nutrient digestion and assimilation. Bile produced from the liver is important for lipid digestion and absorption. The liver plays a role in detoxification of different metabolites as well as storage of many vitamins and minerals. The pancreas produces different enzymes that are needed for the digestion of carbohydrates, proteins, and fats.

Ruminant Animals: Gastrointestinal Tract

The mouth of ruminant animals differs from other mammals in that they have an upper dental pad and not incisor teeth. Feed gathering (prehension) is done by the rough tongue. It is followed by preliminary chewing called mastication and mixing with saliva. Ruminant molars are shaped such that they can chew only on one side at a time. An enormous amount of saliva (about 130 to 150 L/day) is produced in ruminant animals (e.g. dairy cows). Ruminant animals' saliva is rich in sodium bicarbonate ions and acts as a buffer, aiding in the fermentation process. Ruminant saliva also provides nitrogen (N; urea), phosphorus (P), and sodium (Na), which are utilized by microbes.

The foregut (stomach) portion of the ruminant animals is divided into four compartments—reticulum, rumen, omasum, and abomasum (Figure 2.3). The reticulum and rumen are partially separated but have different functional purposes.

The foregut of the ruminant animals is divided into four compartments: reticulum, rumen, omasum, and abomasum.

The rumen is the largest compartment, occupying the left side of the abdominal cavity. The rumen acts as a fermentation vat and is subdivided into sacs by thick muscular boundaries known as pillars. In addition, papillae are also present in the rumen. The rumen has a high population of bacteria, fungi, and protozoa.

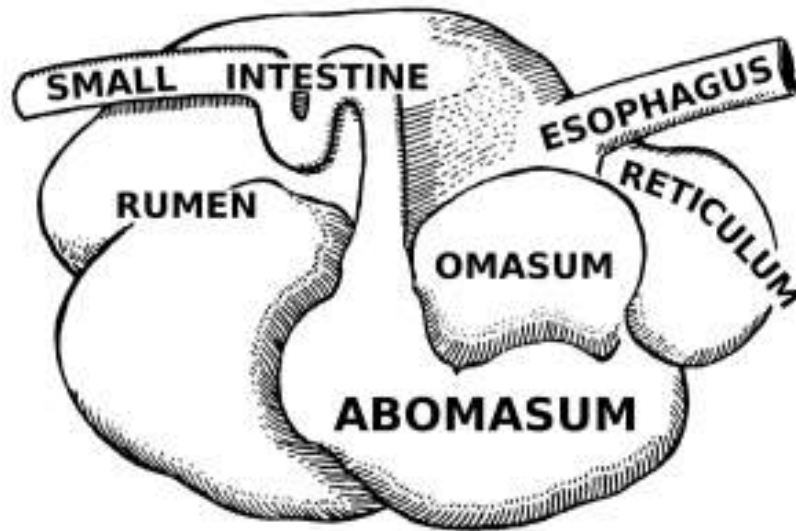


Figure 2.3. Sections of the foregut of ruminant Source: Wikipedia

The reticulum is also called the honeycomb because it is lined with a mucous membrane that subdivides the surface into honeycomb-like compartments (Figure 2.4). The reticulum moves ingested feed into the rumen.

The omasum functions as a food filter and aids in reducing particle size (Figure 2.4). The inside of the omasum is thrown into broad longitudinal folds or leaves. Some absorption may occur in the omasum. The omasum regulates flow to the lower gut and absorbs water and some minerals.

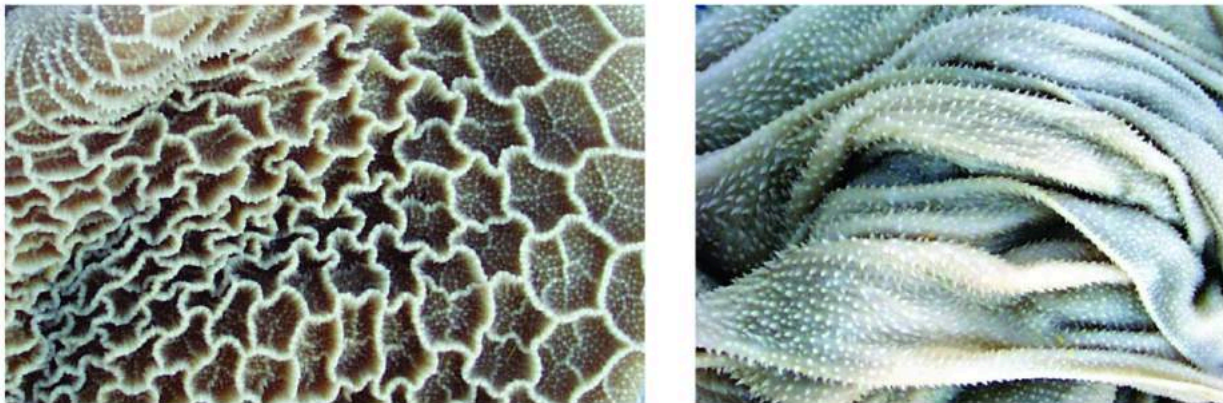


Figure 2.4. Reticulum thrown into folds that form polygonal cells that give it a honeycombed appearance and omasum showing longitudinal muscular folds Source: <http://www.vivo.colostate.edu>

The abomasum functions as the glandular stomach of ruminant animals.

The abomasum is the site where the digestive enzymes are first released in ruminants (e.g., pepsin, mucus, HCl). In young animals, the reticulum, rumen, and omasum are relatively underdeveloped, and exposure to feed, the barn, and other environments enables the growth and maturation of the foregut, and this process takes about six to nine months in large ruminants.

Avian Gastrointestinal Tract

Birds such as chickens are also monogastric animals. As with most birds, chickens obtain their food through the use of their beaks, and their oral cavity does not have teeth but has glands that secrete saliva, making feed easy to swallow.

Food then enters the esophagus, which is a flexible tube that connects the mouth with the rest of the digestive tract. The esophagus includes the crop (expansion of the esophagus), which leads to the proventriculus (also known as the true stomach), or the glandular stomach, where digestion primarily begins (Figure 2.5).

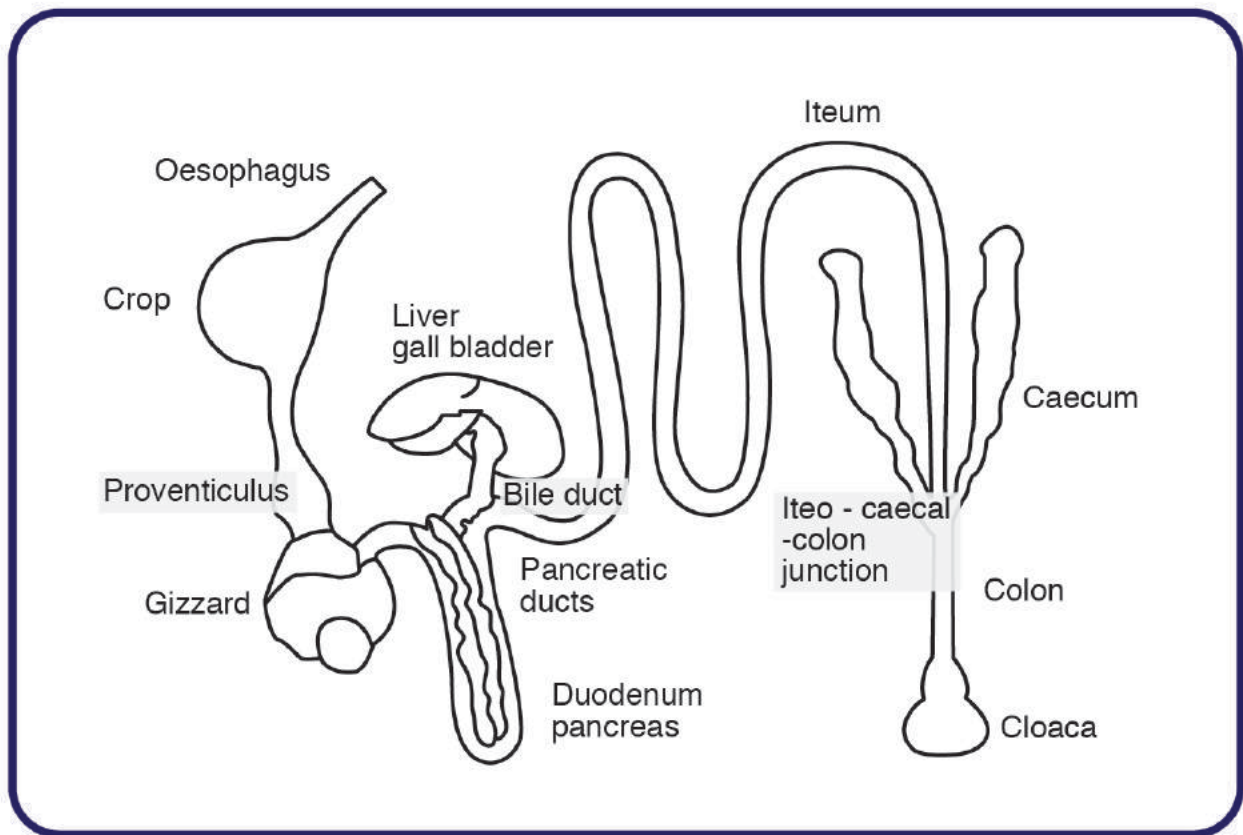


Figure 2.5. Sections of avian gut along with liver and other accessory glands Source: <http://www.thepoutrysite.com>

The gizzard is a part of the digestive tract of birds that is often referred to as the mechanical stomach. The gizzard is made up of two sets of strong muscles that act as the bird's "teeth." Consumed food passes into the gizzard for grinding and mixing. The gizzard acts only as a mechanical organ; therefore, no digestive aids are secreted and absorption of

nutrients does not occur. However, the gizzard is important for mixing ingested feed with water, saliva, hydrochloric acid, and pepsin. The small intestine includes the duodenal loop, which is enclosed by the pancreas. It is also composed of the jejunum and ileum. A remnant attachment of the yolk sac and Meckel's diverticulum are found at the end of the jejunum. Valves at the end of the ileum control passage to two ceca. After some fermentation, digesta are released into a short large intestine that empties into the cloaca and is eventually excreted through the vent.

Key Points

1. Digestive processes include mechanical, chemical, and enzymatic processes.
2. Digestive tract variations and specialization are based on diet, and classification is based on the fermentation process.
3. Digestion starts in the mouth, where chewing and saliva secretion occur. The functions of saliva include lubrication, buffering, and some enzymatic action.
4. The most extensive digestion and absorption in the monogastrics (single stomach) occur in the small intestine.
5. The small intestine is divided into the duodenum, jejunum, and ileum. Pancreatic secretions occur in the duodenum.
6. The three sections of the large intestine are the cecum, colon, and rectum.
7. The four compartments of the ruminant gastrointestinal (GI) tract are the rumen, reticulum, omasum, and abomasum. The abomasum is analogous to the gastric stomach.
8. Functions of the reticulum include receiving food, passing food to the omasum, eructation, and rumination.
9. The bulk of fermentation occurs in the rumen, where anaerobic bacteria, protozoa, and fungi do their work.
10. The omasum regulates flow to the lower gut and absorbs water and some minerals.
11. The abomasum is the site where the digestive enzymes are first released in ruminants (although I should say there are trace amounts of salivary lipase in ruminants). Ruminant saliva does not have amylase. This does not matter because microbes secrete a lot of their own amylases.
12. Hindgut fermenters are those that use the cecum (or colon) for fermentation of plant fiber. They include birds, pigs, and rabbits. Generally, this is less efficient than foregut fermentation.
13. Other accessory organs of the GI tract include the liver, pancreas, and gall bladder.
14. In birds, there is an extension of the esophagus called the crop and proventriculus, functioning as the glandular stomach.
15. The gizzard is a part of the digestive tract of birds, functioning as the mechanical stomach, where most grinding occurs.

Review Questions

1. Compare and contrast the gastrointestinal tract of a swine to that of a cow.
2. List the three sections of the small and large intestine.
3. The small intestine is the primary site of digestion of several nutrients. What are the special anatomical features of the small intestine helping in this process?
4. List the sites of fermentation in foregut fermenting and hindgut fermenting animals.
5. List the different sections of the foregut in ruminant animals along with their functions.

III. Carbohydrates, Structures and Types

This chapter provides an introduction and discussion of carbohydrates that are important in the nutrition of food-producing animals.

New Terms

Amylopectin
Amylose
Cellulose
Disaccharide
Fructose
Galactose
Glucose
Glycogen
Heteropolysaccharide
Homopolysaccharide
Monosaccharide
Oligosaccharide
Polysaccharide
Starch
Trisaccharide

Chapter Objectives

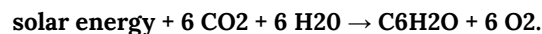
- To present the chemical structure of different types of carbohydrates and their importance in animal nutrition

Carbohydrates

What Are Carbohydrates?

Carbohydrates are the major components of plant tissue, making up to 60% to 90% of the dry matter (DM). Carbohydrates contain carbon, hydrogen, and oxygen in the proportion found in water (CH₂O) and are hence hydrates of carbon. Carbohydrates are the basic energy source in animal cells. Dietary carbohydrates obtained from plant-

based products serve as a major source of energy for the animal. The chlorophyll in plant cells traps solar energy and produces carbohydrates using carbon dioxide and water and gives off oxygen, as shown in the following equation:



Carbohydrates are the major dietary source of energy for animals.

In the plant cell, carbohydrates could be present in the cell content as sugar or starch, or they could be associated with the cell wall structure (e.g., cellulose). When animals eat plant materials (e.g., cereal grains, grass, fodder), energy in the feed's carbohydrates is made available through metabolic processes in the animal cell. Overall, animal metabolism produces energy in a reverse process to that of photosynthesis.

Animal metabolism produces energy in a reverse process to that of photosynthesis in plants.

Structure and Classification

One method of classifying carbohydrates is based on the number of carbon atoms per each molecule of a carbohydrate and on the number of molecules of sugar in the compound. Based on the number of carbon atoms, a carbohydrate can be classified as triose (3 C), tetrose (4 C), pentose (5 C), and hexose (6 C). The suffix “ose” at the end of a biochemical name flags the molecule as a “sugar.” Among these, pentoses (e.g., ribose in ribonucleic acid (RNA)) and hexoses (e.g., glucose, or blood sugar) are the most common sugars in animal tissues. Based on the number of molecules of sugar in the compound, carbohydrates can be classified as (1) monosaccharide, one unit of sugar; (2) disaccharide, two monosaccharides; (3) oligosaccharide, three to fifteen monosaccharides; and (4) polysaccharides, large polymers of simple sugars.

A. Monosaccharides are often referred to as simple sugars (e.g., glucose) and cannot be hydrolyzed into simpler compounds.

Monosaccharides can be subdivided based on the number of carbon (C) atoms. The following list shows the prefixes for numbers of carbons in a sugar.

1. Triose (3 C)
2. Tetrose (4 C)
3. Pentose (5 C; e.g., Xylose and Ribose)
4. Hexose (6 C; e.g., glucose, fructose, galactose, and mannose)

Monosaccharides are the simplest forms of carbohydrate.

Most monosaccharides in animal tissues are of 5 C and 6 C sugars. Simple sugars are also subdivided into aldose, a sugar that contains an aldehyde structure, or ketose, a sugar that contains a ketone group. Both glucose and fructose have the same molecular formula $C_6H_{12}O_6$ and are hexoses (6 C). But glucose is an aldose (also called aldohexose) and fructose is a ketose, or a ketohexose.

The three hexoses that are nutritionally and metabolically important are glucose, fructose, and galactose (see Figure 3.1).

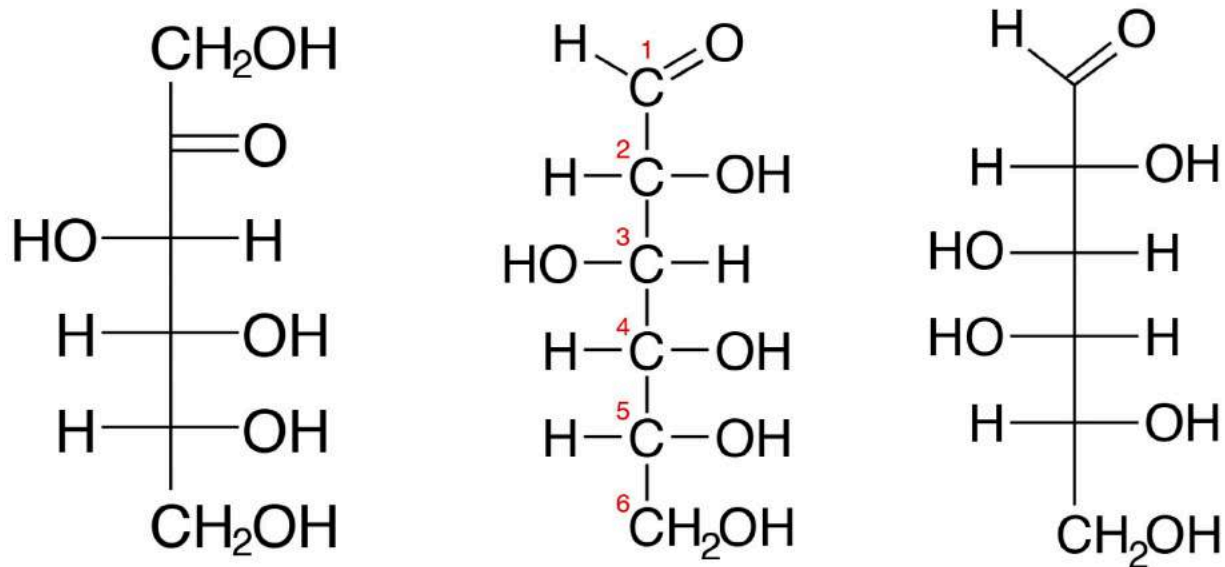
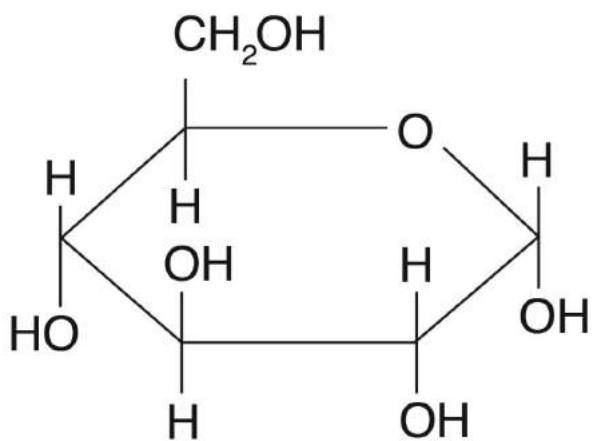


Figure 3.1. Structure of simple sugars (Source: Wikipedia)



The chemical structure of glucose can be represented as a straight chain form (Figure 3.1) and in cyclic form (also shown in Figure 3.1). In a biological system, glucose exists primarily as a cyclic form and very rarely in a straight form (in aqueous solution). Glucose is the form of carbohydrates found in circulating blood (blood sugar) and is the primary carbohydrate used by the body for energy production. Fructose, or “fruit sugar,” is found in ripened fruits and honey and is also formed by digestion of disaccharide sucrose. Galactose is found along with disaccharide lactose in mammalian milk and is released during digestion.

Most nutritionally important sugars are pentoses or hexoses.

Glucose can exist as α and β isomers and has immense animal nutritional implications. These two isomers differ in their orientation of OH on C #1 (shown in red in Figure 3.2).

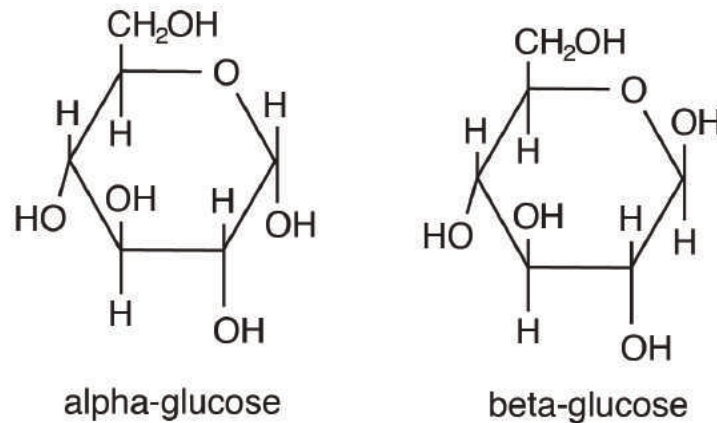


Figure 3.2. Alpha- and beta-glucose structure Source: Wikipedia

For example, starch contains α -D-Glucose, while cellulose has rigid polymers with β -D-Glucose. Nutritionally important sugars are of the D-form (not the L-form). D and L refer to stereo-orientation at asymmetric carbon position 5 in a hexose or carbon position 4 in a pentose.

Nutritional important sugars are of the D-form.

B. Disaccharides are made up of two monosaccharides bonded together by a glycosidic (covalent) bond. The following are some of the common disaccharides:

1. Sucrose-glucose + fructose (e.g., table sugar)
2. Lactose-glucose + galactose (milk sugar)
3. Maltose- α -D-Glucose + β -D-Glucose (malt sugar)
4. Cellobiose- β -D-Glucose + β -D-Glucose (cellulose)

Among the different disaccharides, lactose (milk sugar) is the only carbohydrate of animal origin. However, cellobiose as a component of cellulose is important in animal nutrition. Monogastric animals cannot digest cellulose because they do not produce the cellulase enzyme that can split β -D-Glucose.

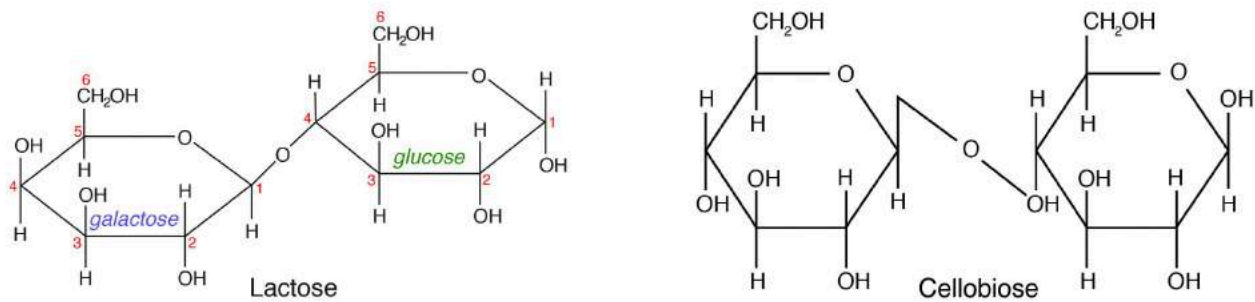


Figure 3.3. Important disaccharides in animal nutrition and feeding, lactose and cellobiose Source: Wikipedia

C. Oligosaccharide are made by bonding together three or more (3 to 15) monosaccharides bonded together.

1. Raffinose (glucose + fructose + galactose; 3 sugars)
2. Stachyose (glucose + fructose + 2 galactose; 4 sugars)

In animal diets, oligosaccharides are commonly found in beans and legumes. Some oligosaccharides are used as substances to enhance the growth of good microbes (prebiotics). Recently, there has been an increased interest in the use of different oligosaccharides as feed additives to enhance hindgut health (e.g., fructooligosaccharides, mannan oligosaccharides).

D. Polysaccharides, as their name implies, are made by joining together large polymers of simple sugars.

Polysaccharides are the most important carbohydrate in animal feed. Polysaccharides are composed of many single monosaccharide units linked together in long, complex chains. The functions of polysaccharides include energy storage in plant cells (e.g., seed starch in cereal grains) and animal cells (e.g., glycogen) or structural support (plant fiber). Components of cell wall structure are also called nonstarch polysaccharides, or resistant starch, in animal nutrition, as they cannot be digested by animal enzymes but are fermented by hindgut and rumen microbes.

Polysaccharides can be homopolysaccharides or heteropolysaccharides.

- a. Homopolysaccharide
- b. Heteropolysaccharide

a. **Homopolysaccharide:** Contains only one type of saccharide unit.

Examples of homopolysaccharides that are important in animal nutrition include starch (nonstructural form), glycogen (animal form), and cellulose (plant structural form).

1. **Starch:** Principal sugar form of carbohydrate in cereal grains (seed energy storage). The basic unit is α -D-Glucose. Forms of starch in cereal grains include
 - a. Amylose- α 1,4 linkage-straight chain, nonbranching, helical structure
 - b. Amylopectin- α 1,4 linkage with alpha 1,6 linkage at branch points

Amylose is the simplest of the polysaccharides, being comprised solely of glucose units joined in an alpha 1,4 linkage (Figure 3.4). Amylose is water soluble and constitutes 15% to 30% of total starch in most plants.

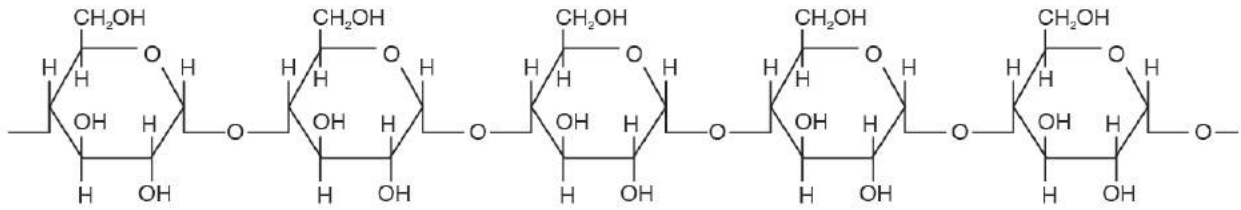


Figure 3.4. Amylose structure showing straight α 1,4 linkage Source: Wikipedia

Amylopectin differs in how the glucose units are joined together. Alpha 1,4 linkages predominate, but a “branch” arises from an alpha 1,6 linkage. Such branches make the structure of amylopectin more complex than that of amylose. Amylopectin is not water soluble and constitutes 70% to 85% of total starch in plant cells.

Starch is the chief carbohydrate source in the diet of monogastric animals.

Amylopectin is the major form of starch in plant cells.

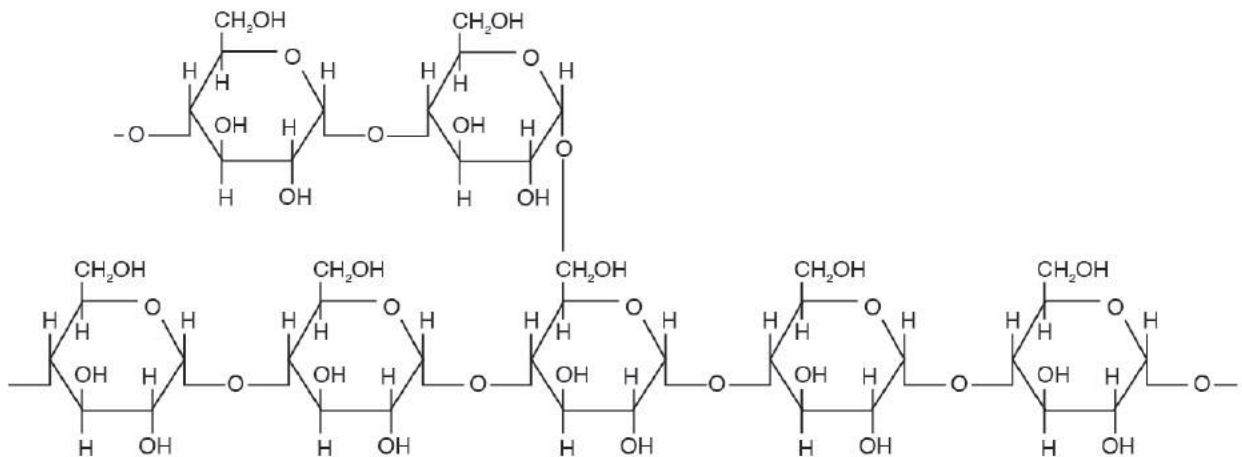
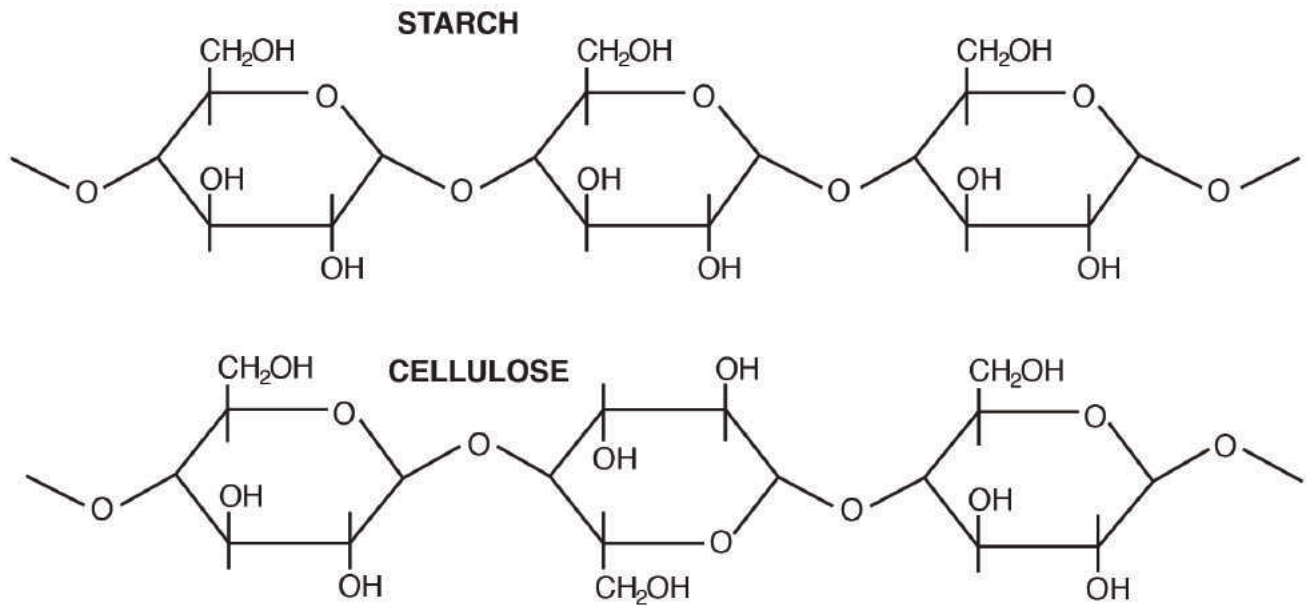


Figure 3.5. Amylopectin structure showing straight α 1,6 linkage Source: Wikipedia

Glycogen is a form of starch found in animal tissue and is hence called animal starch. Glycogen is a polysaccharide that is physically related to amylopectin with basic α -D-Glucose but has a mix of α 1,4 and α 1,6 bonds. Glycogen exists in a small amount (< 1%) in liver and muscle tissue.

Cellulose is the most abundant carbohydrate in nature. It provides structural integrity to plant cell walls. The basic unit is β 1,4 linkage, straight chain, nonbranching (Figure 3.3). Cellulose is highly stable. No animal enzyme can break it; only microbial cellulase can degrade it. Ruminant animals such as cattle, however, have bacteria in their rumen that contain the enzyme cellulase. It breaks the beta 1,4 links of the glucoses in cellulose to release the sugar for energy.



b: Heteropolysaccharide: A component of plant cell walls with a mix of 5 C and 6 C sugars (e.g., hemicellulose and pectin, a mixture of pentose and hexose units).

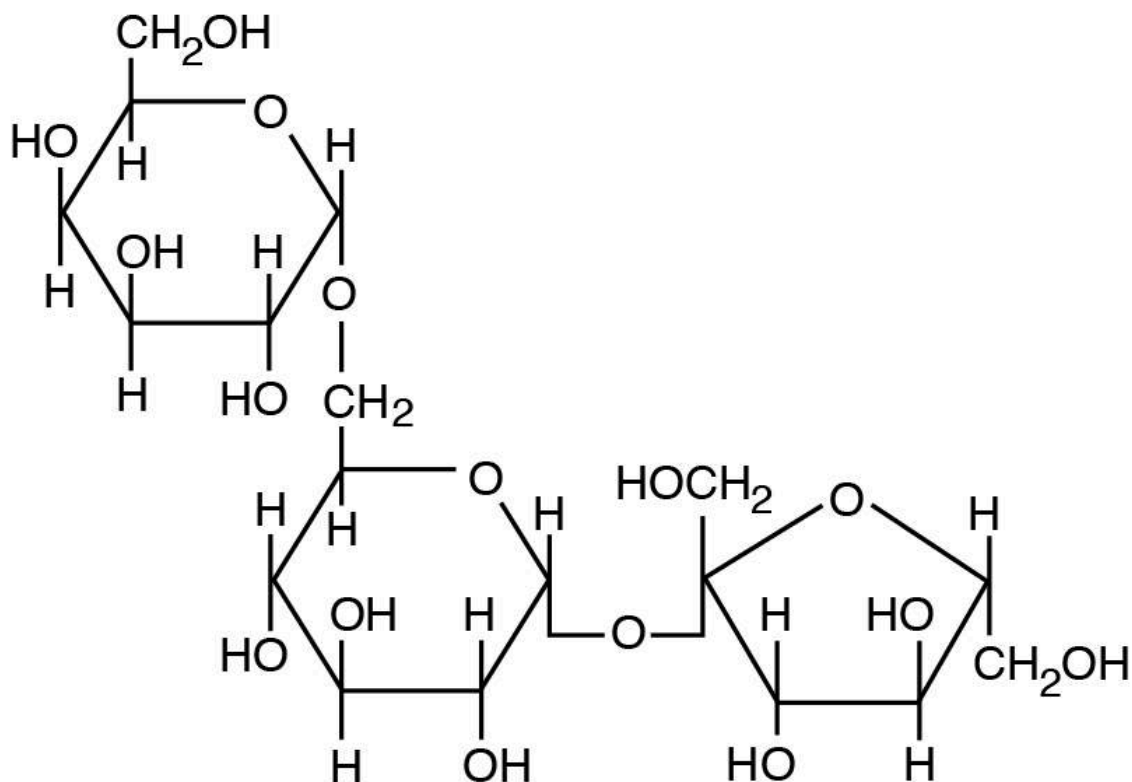


Figure 3.7. Heteropolysaccharide structure showing a mix of 6 and 5 C sugars Source: Wikipedia

Key Points

1. Carbohydrates are “hydrates of carbon” and have the generic structure of $C(n)H(2n)O(n)$.
2. A single sugar unit is a monosaccharide. These can consist of 3-carbon moieties (triose), 4-carbon units (tetrose), 5-carbon moieties (pentose), and 6-carbon moieties (hexose).
3. Most nutritionally important sugars are pentoses or hexoses.
4. Further classification of sugars is a definition of either aldose (having an aldehyde group) or ketose (having a ketone group). Glucose, mannose, and galactose are aldoses, whereas fructose is a ketose.
5. Nutritionally important sugars are of the D-form (not the L-form). D and L refer to stereo-orientation at asymmetric carbon position 5 in a hexose or carbon position 4 in a pentose.
6. Sugars link together via a glycosidic bond to form di- (two monosaccharides) or oligo- (3 to 15 monosaccharides), and polysaccharides.
7. The nature of glycosidic bonds influences the structural and chemical properties of the sugars and influences their ease of digestion. Sugars that bond via an alpha 1,4 linkage may be digested by mammalian enzymes. Sugars that are linked via the beta 1,4 linkage are resistant to digestion.
8. Nutritionally significant disaccharides are sucrose and lactose.

9. Starch from plants serves as a major energy source in animal diets. Starch consists of two types of molecules: amylose (alpha 1,4 linked glucose) and amylopectin (alpha 1,4 and alpha 1,6 linked glucose).
10. Glycogen, a storage form of carbohydrates in the liver and muscles, is very similar to starch also called animal starch.
11. Plant polysaccharides also include cellulose and hemicellulose and pectin (nonstarch polysaccharides). Mammalian enzymes cannot degrade these polysaccharides to free sugars, but microbial enzymes can handle them.

Review Questions

1. In what important ways do starch and cellulose differ?
2. What are the disaccharides of nutritional significance?
3. Nutritional important sugars are of the D-form or the L-form?
4. The most important sugar in nutrition
5. List the two forms in which starch exist
6. The forms of starch in the animal body is?
7. A structural homopolysaccharide made of glucose is
 - a. cellulose
 - b. hemicellulose
 - c. pectin
 - d. raffinose
8. Among these different sugars, the primary source of energy for a broiler chicken is
 - a. fructose
 - b. sucrose
 - c. glycogen
 - d. glucose
9. Two molecules of sugar are linked together by this bond
 - a. peptic bond
 - b. glycosidic bond
 - c. diglyceride bond
 - d. both a) and b)
10. Among the two forms of starch, this is the major component of cereal grains
 - a. amylose
 - b. amylopectin
 - c. cellulose
 - d. glycogen

IV. Carbohydrates, Digestion and Absorption

This chapter provides an introduction to the different processes that are involved in the digestion or fermentation of carbohydrates in monogastric and ruminant animals.

New Terms

Amylase
Dextrins
Disaccharidase
Maltase
Nonstarch polysaccharides
Sucrase
Volatile fatty acids

Chapter Objectives

- To discuss the digestion and/or fermentation carbohydrates in food-producing animals
- To discuss carbohydrate fermentation-related disorders in ruminant animals

The primary site of carbohydrate digestion is in the lumen of the small intestine, where pancreatic amylase begins the digestion of starch granules (amylose and amylopectin). In some birds, there is some salivary amylase action in the mouth, but not in farm animals.

There are two forms of amylase, one that cleaves α 1,4 bonds in a random fashion, while the other removes disaccharides units (maltose) from the polysaccharide chain. Pancreatic amylase does not act on α 1,6 bonds that form the branch points in the structure of amylopectin. The end products of amylase digestion include a mixture of glucose, maltose, and dextrins (residues containing α 1,6 branch points). Dextrins are acted upon by α 1,6 glucosidase.

The small intestine is the site of the digestion of carbohydrates in farm animals.

Dietary simple sugars, such as glucose and fructose, do not need to be digested, as they can be absorbed through the intestinal epithelium directly. The end products of starch digestion diffuse into the brush border, where the final digestive processes occur. Disaccharides such as maltase and isomaltase on the intestinal brush border then complete the degradation and are hydrolyzed to their constituent monosaccharides by enzymes on the brush border, and the monosaccharides released are absorbed into the enterocyte. Sucrose is acted upon by sucrase to yield glucose and fructose for absorption. In young animals kept on milk (preweaning), lactose is acted upon by lactase to yield glucose and galactose. Amylase activity is very low in young animals consuming milk and is stimulated by solid food consumption.

The end product of carbohydrate digestion in monogastric animals is mainly glucose.

Monosaccharides are absorbed both by simple diffusion and adenosine triphosphate (ATP)-dependent active transport. A sodium-dependent glucose transport protein binds glucose and Na⁺ and transports them through the enterocyte and releases them in the cytosol.

Carbohydrate-Digesting Enzymes

- Amylase
- Disaccharidase
- Maltase
- Sucrase
- Lactase

Table 4.1. Overview of carbohydrate digestion: Site, enzymes, and end products produced in monogastric animals

Site	Enzyme	Product
Mouth	Salivary Amylase	Not too significant
Small Intestine	Pancreatic Amylase Di/oligosaccharidases	Monosaccharides
Large Intestine	None	Some volatile fatty acid

Monogastric animals do not secrete enzymes that digest the complex carbohydrates (β 1,4 linkages; e.g., nonstarch polysaccharides [NSP], glucans, cellulose) that are components of plant fiber (e.g., wheat, barley) and are acted upon by hindgut microbes to yield volatile fatty acids (VFAs). High levels of NSP and glucans in a monogastric diet can cause viscous digesta and can interfere with digestion processes leading to malabsorption. In poultry, high-NSP-containing diets (e.g., barley, rye) can produce wet litter, dirty eggs, and diarrhea.

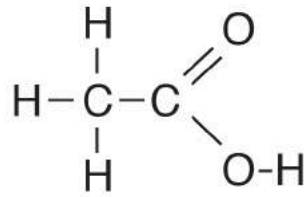
Carbohydrate Digestion in Ruminants

Carbohydrate digestion in ruminant animals is through microbial fermentation in the rumen. Dietary carbohydrates are degraded (fermented) by rumen microbes (bacteria, fungi, protozoa). The purpose of rumen fermentation is to produce energy as ATP for the bacteria to use for protein synthesis and their own growth. VFAs, also known as short-chain fatty acids (shown below), are produced as a product of rumen fermentation and are absorbed through the rumen wall and are utilized by the animal as an energy source.

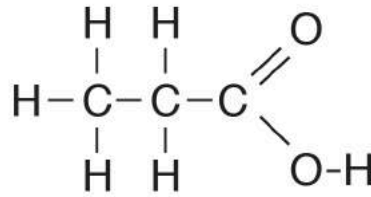
The three major VFAs are acetic (C2), propionic (C3), and butyric acid (C4; shown below).

Major Volatile Fatty Acids Produced in the Rumen

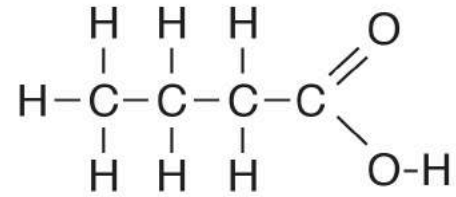
- Acetic acid
- Propionic acid
- Butyric acid



Acetic acid



Propionic acid



Butyric acid

The end products of digestion in ruminants are volatile fatty acids and some monosaccharides.

In young ruminants, rumen and the reticulum are not fully developed and are relatively small. The reticular/esophageal groove reflex, a tube-like fold of tissue, channels milk or water that is sucked from a nipple directly through the omasum to the abomasum. This is a reflex, stimulated by sucking. When the animal is weaned, it normally loses this reflex. Solid food, such as creep feed, passes into the small rumen and fermentation starts. The neonatal ruminant animal has no ruminal bacterial population but from birth, it starts to pick up bacteria from the mother and environment, particularly through contact. Solid food is then fermented forming VFAs, which stimulate the growth and development of the rumen, particularly the growth of the papillae for absorption.

All the digested and absorbed monosaccharides and volatile fatty acids enter into the liver.

The end products of rumen fermentation are microbial cell masses, or microbial protein-synthesized VFA, and gases such as carbon dioxide, methane, hydrogen, and hydrogen sulfide. The products of fermentation will vary with the relative composition of the rumen microflora. The microbial population also depends on the diet, since this changes the substrates for fermentation and subsequently the products of fermentation. For example, starch is the major dietary constituent in concentrate-fed ruminants (e.g., feedlot cattle). The rumen of such animals will have higher amylolytic bacteria than cellulolytic bacteria present in the rumen of roughage- and pasture-fed animals. Factors such as the forage:concentrate ratio, the physical form of the diet (ground vs. pelleted), feed additives, and animal species can affect the rumen fermentation process and VFA production.

Molar ratios of VFAs are dependent on the forage:concentrate ratio of the diet. Cellulolytic bacteria tend to produce more acetate, while amylolytic bacteria produce more propionic acid. Typically three major VFA molar ratios are 65:25:10 with a roughage diet and 50:40:10 with a concentrate-rich diet. Changes in VFA concentration can lead to several disorders of carbohydrate digestion in ruminants. Rumen acidosis occurs when animals are fed high-grain-rich diets or when animals are suddenly changed from pasture- or range-fed to feedlot conditions.

Key Points

1. Very little digestion occurs in the mouth in farm animals.
2. The small intestine is the site of carbohydrate digestion in monogastrics.
3. Pancreatic amylase acts on alpha 1,4 links, and other disaccharidases and remove disaccharide units.
4. The end product (mainly glucose) diffuses into the brush-border using ATP-dependent glucose transporters.
5. Undigested (fiber, nonstarch polysaccharides [NSP]) in the hindgut can serve as an energy source for hindgut microbes in monogastrics.
6. Ruminant carbohydrate digestion is very different from monogastrics. First, there is no amylase secreted in the saliva and then most carbs are fermented in the rumen by microbial enzymes.
7. Carbohydrates are fermented to volatile fatty acids (VFAs) in the rumen. These include acetic acid, propionic acid, and butyric acid.
8. VFAs are absorbed through the rumen wall into the portal vein and are carried to the liver.
9. Ratios of the VFAs change with the type of diet. Roughage diets favor microbes that produce more acetic acid, whereas concentrate diets favor microbes that produce more propionic acid.
10. Carbohydrate fermentation disorders in ruminants include rumen acidosis (grain overload), when cattle are fed high-starch-based cereal or grain-rich diets or when there is a sudden change from pasture to feedlot.

Review Questions

1. List the enzymes involved in carbohydrate digestion in monogastric animals.
2. What are the end products of carbohydrate digestion in monogastrics and in ruminants?
3. What are the major volatile fatty acids (VFAs) produced by rumen fermentation?
4. What causes rumen acidosis?
5. Feeding too much barley to broiler chickens can cause sticky feces and digestibility problems. Why?
6. In ruminants fed concentrate-rich diets, the major VFA produced in the rumen is?
7. In ruminants fed roughage-rich diets, the major VFA produced in the rumen is?
8. Fill in the monosaccharides that compose each disaccharide listed below and the enzyme required to break the bond between them.

Sugar	Enzyme	Monosaccharide 1	Monosaccharide 2
Sucrose			
Maltose			

V. Carbohydrates, Metabolism

This chapter provides an introduction to the different metabolic pathways by which absorbed glucose and volatile fatty acids are converted to energy in the animal body.

New Terms

Adenosine Triphosphate (ATP)
Acetyl Coenzyme A (CoA)
Electron transport chain
Flavin Adenine Dinucleotide (FAD)
Glycolysis
Nicotinamide Adenine Dinucleotide (NAD)
Pyruvate
Pyruvate dehydrogenase
Tricarboxylic Acid (TCA) cycle

Chapter Objectives

- Introduce the fate of absorbed glucose and volatile fatty acids
- To provide information on bioenergetics of glucose catabolism in the animal body

Why Do Animals Need Energy?

Energy is defined as the “ability to do work”. Animals need energy to carry out all the body processes (e.g., nutrient transport, synthesis, muscle contraction) required to maintain life. Without energy, an animal is unable to move, to digest its food, to reproduce, to grow, or even to breathe. Energy requirement and balance are more important in food-producing animals with their need to synthesize nutrients (e.g., proteins, fat) for deposition into muscle, milk, and eggs. Carbohydrates are the major energy source in the diet of farm animals.

Carbohydrates are the major source of energy in the animal's diet.

Forms of Energy

Hydrogen plays a prominent role in energy metabolism. During the catabolism of glucose ($C_6H_{12}O_6$) by the animal, hydrogen is transferred from glucose to hydrogen receptors, such as nicotinamide adenine dinucleotide (NAD^+) and flavin adenine dinucleotide (FAD). These hydrogen acceptors (reducing equivalents) are oxidized in the reactions of the respiratory chain inside the mitochondria to release energy. In biological systems, oxidation of hydrogen is coupled with the synthesis of adenosine triphosphate (ATP). ATP is the readily available form of energy (“molecular energy currency unit”) in the cell. ATP has three components: a nitrogenous base (adenine), the sugar ribose, and the triphosphate (figure 5.1). Energy is stored within the PO_4 bonds, and the release of each phosphate bond generates eight kcal of energy.

Forms of Energy

- 1 mole ATP = 8 kcal/mol
- Reducing equivalents
- 1 mole NAD, NADH = 3 ATP
- 1 mole FAD, FADH = 2 ATP

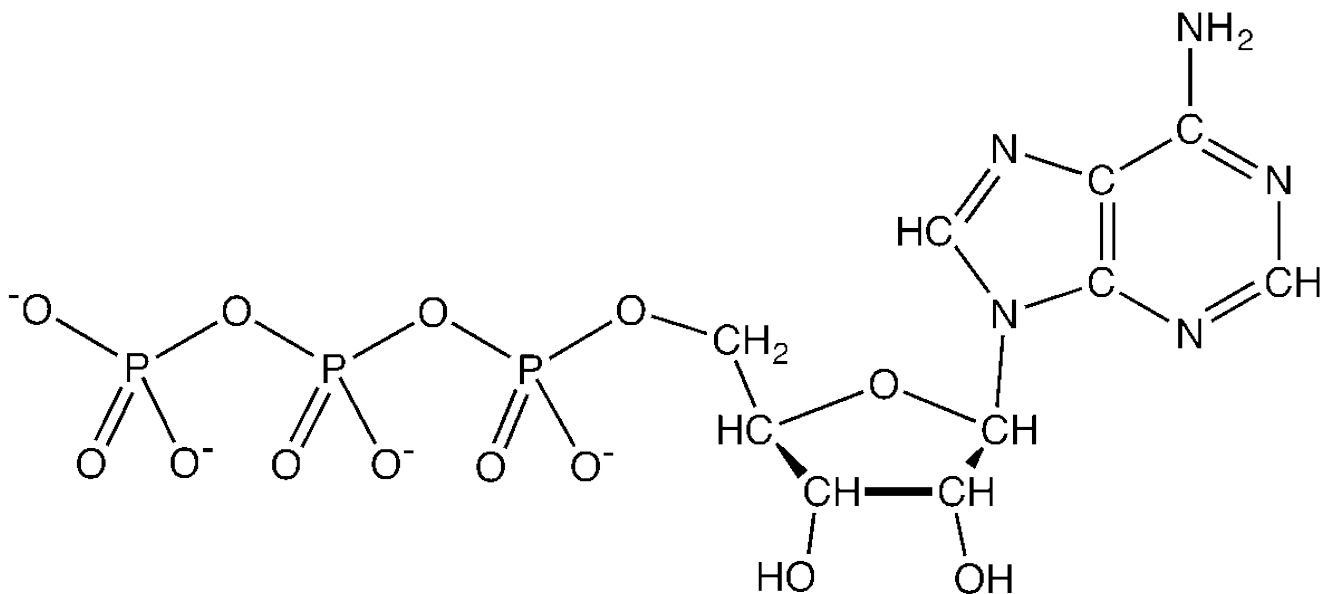


Figure 5.1. ATP structure Source: Wikipedia

ATP is the compound used as an energy source in biochemical reactions.

The Fate of Absorbed Glucose and Metabolic Conversions in the Animal Body

Absorbed glucose could be utilized by the animal for several functions. The first priority is the formation of glycogen in the liver, which is stored in muscle and hepatic tissue. However, the body stores very little as glycogen (a starch-like compound), and glycogen could be rapidly hydrolyzed back to glucose through a process called glycogenolysis. This helps in maintaining blood glucose at a narrow range in normal healthy animals. The second priority is oxidation to form energy (a major function) and is described in the next sections. Since glycogen storage is limited, excess glucose is

converted to fat and is stored in adipose tissue. This is accomplished by the breakdown of glucose to pyruvate through a process called glycolysis, which is then available for fat synthesis.

The Fate of Absorbed Glucose

- Storage as glycogen in liver and muscle (small amount)
- Oxidization for energy
- Fat synthesis and storage as fat in adipose tissue

How Cells Derive Energy from Glucose: Metabolic Pathways

Cells use different steps to break down the absorbed glucose to carbon dioxide and water through different enzymatic reactions. The catabolism of glucose occurs in two metabolic pathways: glycolysis and the tricarboxylic acid (TCA; also called citric acid or Krebs') cycle.

Two major pathways of glucose catabolism are glycolysis and the TCA cycle.

Glycolysis: Enzymes for glycolysis are located in the cytosol of the cell, and glycolysis occurs in this part of the cell. Glycolysis is the breakdown of 6 C glucose into two 3 C end product pyruvates in aerobic metabolism and lactic acid in anaerobic metabolism. It is a catabolic pathway involving oxidation and yields ATP and NADH (reduced NAD) energy. Glycolysis is the pathway by which other sugars (e.g., fructose, galactose) are catabolized by converting them to intermediates of glycolysis. Fructose can be converted to fructose-6-phosphate by hexokinase. Galactose can enter glycolysis by being converted to galactose-1-phosphate followed by conversion (ultimately) to glucose-1-phosphate and subsequently to glucose-6-phosphate (G6P), which is a glycolysis intermediate.

Energy Production Process through Glycolysis: Glycolysis has two phases: an energy investment phase requiring the input of ATP (preparatory phase) and an energy realization phase (pay off) where ATP is made (Figure 5.2). Cells that utilize glucose have an enzyme called hexokinases, which use ATP to phosphorylate the glucose (attaches a phosphorus group) and changes it into G6P. At this point, the cellular “machinery” can begin to process the glucose. Briefly, in the first reaction of glycolysis, hexokinase catalyzes the transfer of phosphate to glucose from ATP, forming glucose-6-phosphate. Thus this step uses ATP, which provides the energy necessary for the reaction to proceed. Glucose-6-phosphate is converted to fructose-6-phosphate and subsequently to fructose-1,6-biphosphate, which is cleaved to dihydroxy acetone phosphate (DHAP) and glyceraldehyde-3-phosphate (G3P). During this process, an additional ATP is required to phosphorylate the intermediate fructose-6-phosphate. Therefore, the “preparation” of glucose results in two molecules of ATP being used for every glucose molecule processed.

During the payoff phase, G3P is further processed to produce pyruvate. During this phase, one NADH and two ATP are produced during the intermediate steps. The DHAP produced can be simply converted into G3P and processed in a similar manner as the first G3P. Therefore, one glucose molecule will result in the production of two NADH, four ATP, and two pyruvate molecules.

Glycolysis: Net Gain of ATP

- Input = 2 ATP
- Produces = 4 ATP and 2 NADH
- Net gain = 8 ATP (aerobic)

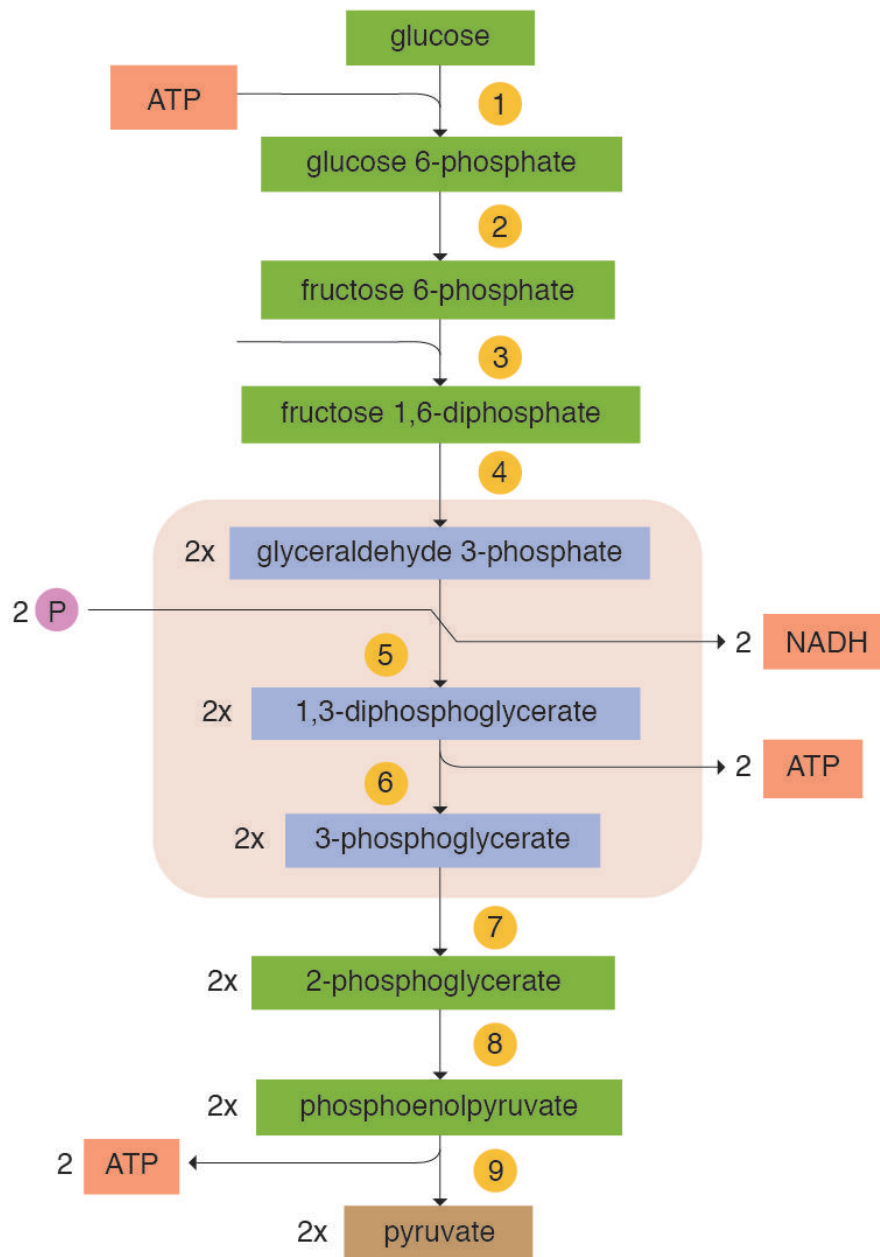


Figure 5.2. Glycolysis pathway in cytosol Source: Wikipedia

Glycolysis (Aerobic)

Input: = 1 glucose molecule

Requires = - 2 ATP (Activation)

Produces:

+ 2 pyruvate molecules

+ 4 ATP

+ 2 NADH (= 6 ATP)

Total = 6 + 4 = 10 ATP

Net gain = 10 - 2 = 8 ATP

Glycolysis: Overall Functions

ATP Production:

For each molecule of glucose, 2 ATP (preparatory phase) were used and 2 NADH, 4 ATP, and 2 pyruvate molecules (payoff phase) were generated; which equals a net production of 2 NADH, 2 ATP, and 2 pyruvate molecules, and the net gain of ATP is 8 per mole of glucose.

Production of Other Intermediates:

Glycolysis provides pyruvate for the TCA cycle, amino acid synthesis through transamination, glucose-6-phosphate (glycogen synthesis), nicotinamide adenine dinucleotide phosphate, (NADPH) (fatty acid synthesis; triglyceride synthesis), and dihydroxyacetone phosphate for glycerol synthesis (the backbone of fat).

Fates of Pyruvate in the Animal Body

It is important to discuss the fate of pyruvate generated through glycolysis. Pyruvate has different fates, depending on the conditions of the animal and the cell type.

Fates of Pyruvate

- Lactic acid production
- Acetyl CoA production

Lactic Acid Production: When oxygen is present, there is plenty of NAD⁺, so aerobic cells convert pyruvate to acetyl coenzyme A (CoA) for oxidation in the citric acid cycle. When oxygen is absent, NAD⁺ levels can go down, so to prevent that from happening, lactate dehydrogenase uses NADH and pyruvate is converted to either lactate (animals) or ethanol (bacteria/yeast). Anaerobic conversion of NADH to NAD⁺ provides much less ATP energy to cells than when oxygen is present. Anaerobic metabolism of

glucose generates only two ATP per glucose. Once oxygen is depleted for the cell, another system will convert the lactic acid back to pyruvate and produce glucose.

Anaerobic metabolism of glucose produces two ATP per glucose molecule.

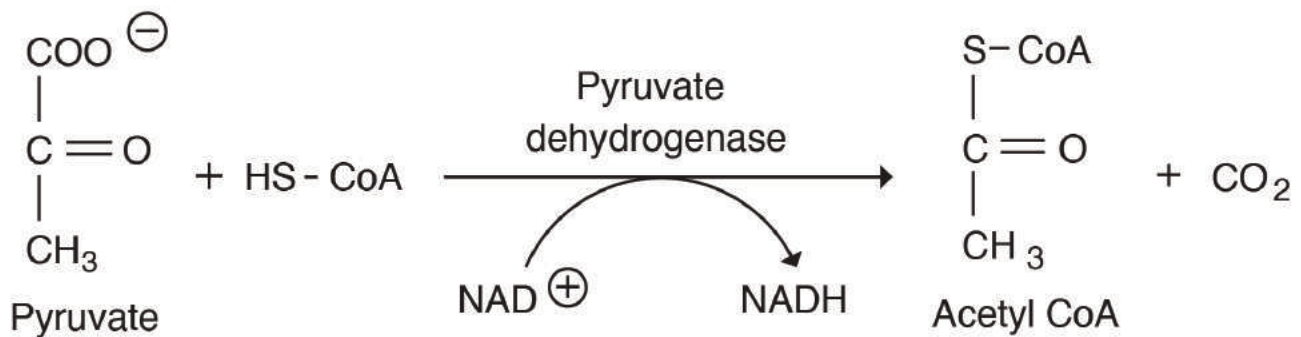
Acetyl CoA Production:

Acetyl CoA Production: Acetyl CoA production occurs in the aerobic state and serves as the main precursor for the TCA cycle, lipogenesis, and ketogenesis (during negative balance). Acetyl CoA is converted to ATP through different steps in the TCA cycle. During this conversion, the enzyme pyruvate dehydrogenase and different B vitamin-containing coenzymes (thiamine, riboflavin, niacin, pantothenic acid) function through a series of condensation, isomerization, and dehydrogenation reactions and produces several different intermediates that are used for fat or amino acid synthesis.

Pyruvate dehydrogenase links glycolysis with the TCA cycle by converting pyruvate into acetyl CoA.

To generate more energy from the glucose molecule, further biochemical processes occur within the animal body. These include the enzymatic step pyruvate dehydrogenase (PDH), which connects glycolysis (cytosol) with the TCA cycle in the mitochondria. During this step, 3 C pyruvate is converted to an active form of acetic acid called acetyl CoA, and CO₂ is produced.

Pyruvic acid is decarboxylated and the 2 H ions are picked up by NAD⁺ and thus it provides two mole of NADH for each mole of glucose (net = 6 ATP produced). This enzymatic step needs coenzyme A and its activity is highly regulated by the concentration of acetyl CoA, ATP, and NADH.



Through PDH, one mole of glucose produces 2 NADH or 6 ATP.

TCA, Citric Acid, or Krebs' Cycle

Functions of the TCA Cycle

- Recover more chemical energy
- Provide metabolic intermediates (e.g., citrate, α -ketoglutarate, oxaloacetate)

TCA cycle, citric acid cycle, or Krebs' cycle (named after Hans Krebs who discovered the pathway in 1937) includes a series of enzyme-catalyzed chemical reactions that occur in the matrix of the mitochondria. Several B vitamin-containing enzymes function as coenzymes in the pathway (e.g., thiamine, riboflavin, niacin). The TCA cycle is the key part of aerobic respiration in cells. The TCA cycle also serves as a source of precursors for storage forms of fuels (lipids) and building blocks, such as amino acids, in the animal body.

The TCA cycle is the central metabolic hub of the cell and occurs in the mitochondria.

Prior to entrance into the cycle, pyruvic acid is converted to acetyl CoA through PDH as described previously. Acetyl CoA (2 C) enters the cycle by combining with a 4 C compound called oxaloacetate and forms a 6 C citric acid. This reaction "pulls" the cycle forward. Citric acid undergoes a series (about 10) of enzyme-catalyzed conversions producing different intermediates (e.g., α -ketoglutarate, succinate, fumarate, malate; shown in Figure 5.3).

In many of these steps, high-energy electrons are released to NAD, and NAD molecules also acquire H^+ and become NADH. In one of the steps, FAD serves as the electron acceptor, and it acquires H^+ and becomes FADH₂. In one of the reactions, one ATP is also synthesized from one acetyl CoA. At the end of the cycle, the final product is oxaloacetic acid, which is identical to the oxaloacetic acid that begins the cycle and will pick up another acetyl CoA to begin another turn of the cycle. Altogether, the TCA cycle produces (per one mole of glucose or two moles of pyruvic acid), two ATP molecules, six NADH, and two FADH₂. Both NADH and FADH are used in the electron transport chain to generate ATP. Overall, a net gain of 24 ATP per mole of glucose is obtained through the TCA cycle.

One mole of glucose produces 24 ATP through the TCA cycle.

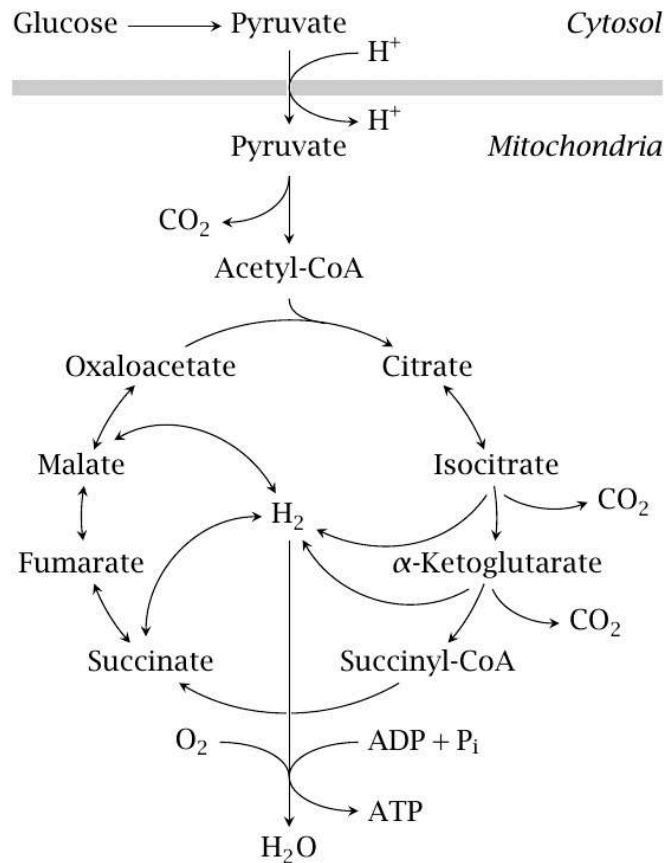


Figure 5.3. TCA cycle in the mitochondria Source: <http://watcut.uwaterloo.ca/webnotes/Metabolism/TCAcycle.html>

Overall, for every glucose molecule fully metabolized to CO₂ and H₂O, we receive 38 ATP.

There are eight kcal of energy in every ATP high-energy phosphate bond. Hence net recovery of energy is $38 \times 8 = 304$ kcal.

The efficiency of converting glucose bond energy into ATP high-energy P bond is therefore $304/674^{*1} \times 100 = 45\%$.

The TCA cycle is the final common pathway for oxidation of carbohydrates, fats, and proteins that provides energy.

1. *=gross energy of 1 mole of glucose.

The rate-limiting step in the TCA cycle is the combination of oxaloacetate and acetyl CoA to produce citrate. Because of this, it is essential to have adequate quantities of oxaloacetate in order to produce ATP and maintain cell viability. Nutritional diseases such as ketosis result from a deficiency in energy production and therefore require oxaloacetate as precursors to “jump-start” the TCA cycle. The TCA cycle also produces intermediates that serve as a precursor for the synthesis of fatty acids and amino acids. For example, citrate can be used for fatty acid synthesis, while oxaloacetate can be used for nonessential amino acids (glucogenic amino acids or most of the nonessential amino acids, e.g., alanine, serine, cysteine, glycine). During low glucose conditions, these amino acids can produce glucose and α -ketoglutarate can be used for glutamic acid synthesis.

Altogether, one mole of glucose produces the equivalent of 38 ATP (8 + 6 + 24), or 304 kcal, through the different steps explained.

TCA Cycle Intermediates: Roles

- Citrate for fatty acid synthesis
- α -ketoglutarate for glutamic acid (amino acid) synthesis
- Oxaloacetate for non essential amino acid synthesis

Electron Transport System, Respiratory Chain, or Oxidative Phosphorylation:

The electron transport chain permits recovery of redox energy associated with NADH and FADH. H ions react with O₂ and form water, and thus the electron transport system serves as a source of metabolic water. Electrons are carried to the electron transport system in the mitochondrial wall by NADH and FADH₂. Coenzyme Q accepts a pair of electrons and passes electrons singly to cytochrome C and acts as a “traffic cop” for electrons. Oxygen is the terminal electron acceptor and if oxygen is not available, electrons will not pass through the electron transport system and NADH and FADH₂

will not be reoxidized. For these reasons, the citric acid cycle will not run either. This is part of metabolic control. Interruption of electron flow can result in production of reactive oxygen species (free radicals). Cellular enzymes, such as superoxide dismutase and catalase, help deactivate reactive oxygen species.

Ruminant Carbohydrate Metabolism

Volatile fatty acids (VFAs) are produced in large amounts through ruminal fermentation and are of great importance in that they provide greater than 70% of the ruminant’s energy supply. The rumen epithelium performs efficient absorption of VFAs through diffusion through a concentration gradient. As they pass through the epithelium, the different VFAs undergo different degrees of metabolism.

The Fate of Volatile Fatty Acids

- Acetate—precursor to Acetyl CoA (TCA cycle; fat synthesis)
- Propionate—precursor to glucose (glycolysis; milk lactose)

Acetate and propionate pass through the epithelium largely unchanged, but almost all the butyric acid is metabolized in the epithelium to beta-hydroxybutyric acid, a type of ketone body.

The three major VFAs (acetic, propionic, butyric) absorbed from the rumen have somewhat distinctive metabolic fates:

Acetic acid is utilized minimally in the liver and is oxidized throughout most of the body to generate ATP. Another important use of acetate is as the major

- Butyrate—precursor to Acetyl CoA (TCA cycle)

source of acetyl CoA, and it enters the TCA cycle. Acetic acid is used for the synthesis of lipids (e.g., milk or body fat). A high-roughage diet favors the production of acetic acid.

Propionic acid is almost completely removed from portal blood by the liver. Propionate is converted to succinyl CoA, and it enters the TCA cycle. Within the

liver, propionate serves as a major substrate for gluconeogenesis, which is absolutely critical to the ruminant because almost no glucose reaches the small intestine for absorption. For example, in a dairy cow, all the glucose in the milk lactose was synthesized in the liver and most of that synthesis was from propionic acid. A high-concentrate diet favors the production of propionic acid.

For **butyric acid**, butyrate is split into two acetyl CoAs, and it enters the TCA cycle. Most of the butyric acid that comes out of the rumen as the ketone beta-hydroxybutyric acid which oxidized in many tissues for energy production.

Key Points

1. One mole of glucose, if burned in a flame, liberates 674 kcal of heat. Life is equivalent to this process (i.e., respiration is combustion); hence oxidation of glucose in an animal's body allows for the recovery of the chemical bond energy of glucose in a useable form. Energy is needed for life processes including heart work, protein synthesis, fat synthesis, milk synthesis, or meat and egg production.
2. Different metabolic pathways such as glycolysis, tricarboxylic acid (TCA), and the electron transport system, plus one enzyme, pyruvate dehydrogenase (PDH), allow efficient conversion of glucose chemical bond energy into useable energetic intermediates such as ATP.
3. Glycolysis yield two net ATP / glucose molecule plus two NADH. Because NADH can be metabolized to ATP and because we get two NADH from each glucose molecule, the net ATP yield per glucose in glycolysis is eight.
4. Pyruvate dehydrogenase connects glycolysis with the TCA cycle. The TCA cycle occurs in the mitochondria. PDH generates one NADH and one CO₂ for each pyruvate and also generates an acetyl CoA, which is the starting material for the TCA cycle.
5. TCA cycle produces two ATP, six NADH, and two FADH₂ per each mol of glucose.
6. The electron transport within the wall of the mitochondria permits recovery of redox energy associated with nicotinamide adenine dinucleotide and flavin adenine dinucleotide reduced forms (NADH and FADH).
7. Other sugars besides glucose are metabolized by these pathways. Fructose may be metabolized into the glycolytic pathways either via fructose-6-phosphate and galactose, a constituent of milk sugar, may be metabolized to glucose-6-phosphate, a glycolytic intermediate.
8. For every glucose molecule fully metabolized to CO₂ and H₂O, we receive 38 ATP. There are eight kcal of energy in every ATP high-energy phosphate bond. Hence the net recovery of energy is $38 \times 8 = 304$ kcal. The efficiency of converting glucose bond energy into ATP high-energy P bond is therefore $304/674 \times 100 = 45\%$.

9. In ruminants, volatile fatty acids (VFAs) serve as a major energy source. Through entry into the TCA cycle, acetate—mainly used for fat synthesis and propionic acid—serves as a major substrate for gluconeogenesis.
10. A high-fiber diet favors acetate, and a high-concentrate diet favors propionic acid.
11. In dairy cows, milk fat and milk yield are highly affected by the type of carbohydrate fed.

Review Questions

1. What are the three principal fates of glucose after absorption?
2. How much ATP is generated during the complete oxidation of glucose under aerobic conditions?
3. What is the readily available form of energy for cells?
4. The site of glycolysis and the tricarboxylic acid (TCA) cycle in the cell is _____ and _____.
5. In ruminants fed concentrate-rich diets, _____ is the major volatile fatty acid (VFA).
6. What are the three principal fates of glucose after absorption?
7. This VFA is the precursor of glucose in ruminants_____.
8. _____ links glycolysis with the TCA cycle.
9. In ruminants fed roughage-rich diets, _____ is the major VFA.
10. A 2 C chemical that enters the TCA cycle to be oxidized to provide energy is _____.
11. The end product of glycolysis under aerobic conditions is _____.
 - a. lactic acid
 - b. pyruvic acid
 - c. acetic acid
 - d. citric acid
12. Acetyl CoA enters the TCA cycle and condenses with this 4 C compound to form citrate _____.
 - a. malate
 - b. oxaloacetate
 - c. fumarate
 - d. pyruvate
13. Complete the following table for total ATP and kcal produced through the three different metabolic pathways for a single glucose molecule under aerobic conditions.

	Glycolysis	Pyruvate to Acetyl CoA	TCA/Citric Acid	Total ATP	Total kcal/ glucose molecule
Total ATP					

VI. Lipids, Structure

This chapter provides an introduction and discussion of lipids (fats) that are important in the nutrition of food-producing animals. After carbohydrates, lipids serve as a major source of energy in animal diets.

New Terms

Cholesterol
Conjugated linoleic acid
Essential fatty acid
Fatty acid
Glycerol
Lipid
Monounsaturated fatty acid
Omega-3 fatty acid
Omega-6 fatty acid
Polyunsaturated fatty acid
Saturated fatty acid
Triglyceride

Chapter Objectives

- To present the chemical structure of lipids and fatty acids of importance in animal nutrition

Lipid Structure

What Are Lipids?

Lipids (also known as fats) are components of plant (e.g., vegetable oils) and animal tissues (e.g., meat, eggs, milk). On a physical nature, lipids are relatively insoluble in water and are soluble in organic

solvents, such as hexane, ether, and chloroform.

Chemically, lipids are organic compounds and esters of fatty acids and glycerol (a 3 C compound) or some other alcohol.

Fats are the primary storage form of energy (e.g., oil in seed) and serve as an animal's body's "savings account." For example, the abdominal fat pads in chicken and back fat in pigs are mostly triglycerides.

Lipid Classifications

1. Simple lipid = Esters of fatty acid with alcohol, e.g. 1 glycerol + 3 fatty acids (commonly called triglyceride or triacylglycerol)
2. Compound
 - a. Glycolipid
 - b. Lipoproteins
 - c. Phospholipids
3. Derived Lipids

Simple lipids like triglycerides are more common and are an important component in animal rations (e.g., vegetable oil and animal fats such as tallow or lard).

Compound lipids are composed of a lipid plus a nonlipid molecule (e.g., protein). Lipoprotein (lipid + protein) are examples of compound lipids and are used for lipid transport (like a courier). Within the animal body, compound lipids are more important in physiology and metabolism (e.g., lipid transport, phospholipids as part of cell membranes).

As implied by their name, derived lipids originate from simple or compound lipids through hydrolytic processes. Examples of derived lipids include sterols, fatty acids, and fat-soluble vitamins.

Why Add Fats to Animal Diets?

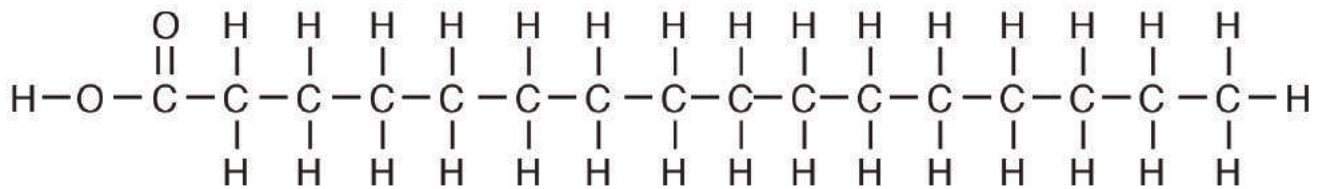
Nutritionally, fats are excellent sources of energy and are essential to the survival of animals. Fats are the sole source of **essential fatty acids** (those that cannot be made by the body) for animals. Fats can also provide fat-soluble vitamins. However, this role is very minimal in livestock as feeds are supplemented with vitamins.

The most important role of dietary fat is to provide essential fatty acids.
As the fat content of the diet increases, the energy density of the diet goes up.

Physically, the addition of fats is associated with the improvement of feed quality, the reduction of dust in feed, the reduction of feed particle separation during processing, an increase in palatability, an increase in digestive lubrication (i.e., emulsification and rate of passage), and an increase in feed digestibility.

Fatty Acids: What Are They?

Fatty acids are the main players in lipid nutrition. This is due to their diversity in structure, composition, and metabolizability. The molecular composition of a fatty acid includes a hydrophilic carboxyl group (-COOH) and a hydrophobic methyl group (-CH₃) at opposite terminals of a hydrocarbon backbone (see Figure 6.1).



Palmitic Acid

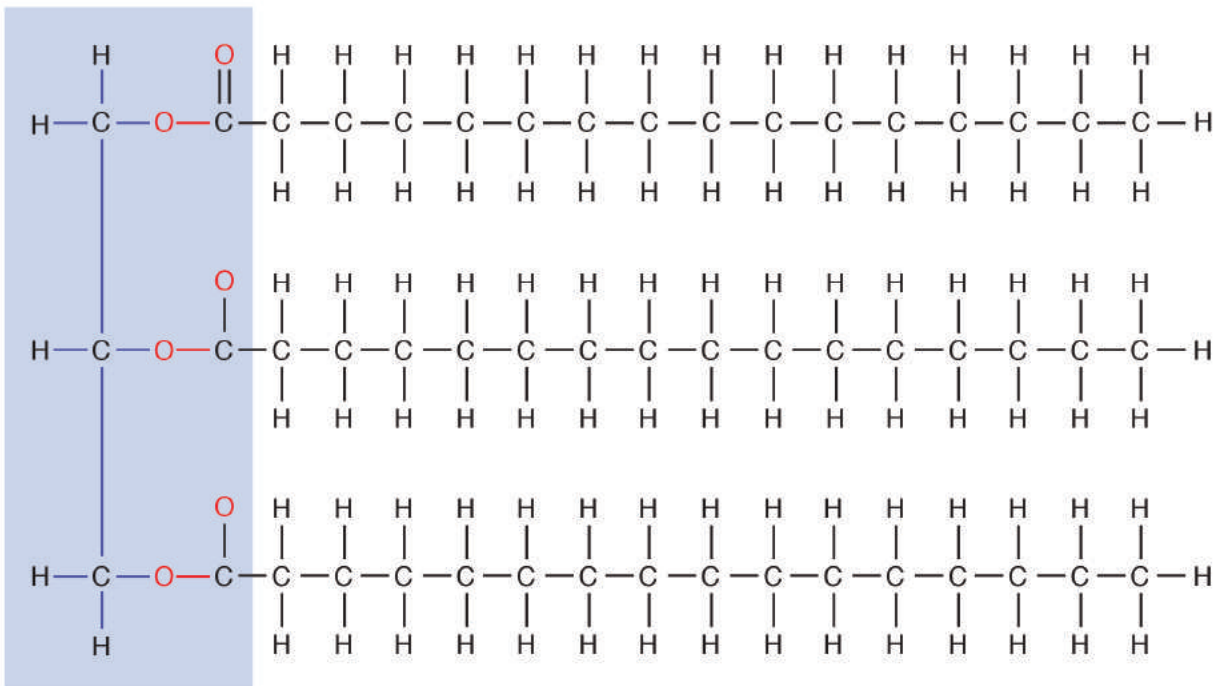
Figure 6.1. Fatty acid structure with hydrocarbon chain showing carboxyl and methyl end

In most cases, three fatty acids are attached to the glycerol molecule and are called triacylglycerol. The three fatty acids in triacylglycerol can differ in chain length (i.e., total carbons in the fatty acid molecule) as well as in the number of double bonds.

A schematic representation of a triacylglycerol structure with three fatty acids on a glycerol backbone is shown below.



Triglyceride



Fatty acid composition and structure determine the physical property and nutritional quality of fats. For example, when there is a predominance of saturated fats in the triacylglycerol, fat tends to solidify (e.g., fat around a piece of meat), and when there is a predominance of unsaturated fats, fat tends to liquefy (e.g., salad oil).

Physical Properties: Fatty Acids

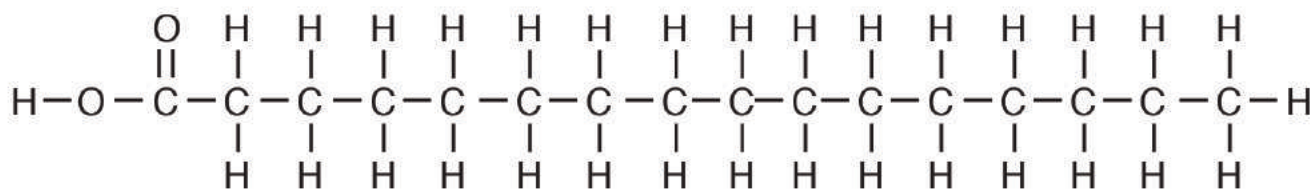
- An increase in saturation makes fats more solid.
- An increase in unsaturation makes fats more liquid or decreases their melting point.

Fatty acids are classified into three families based on the presence (or absence) of double bonds in the hydrocarbon chain. These include saturated fatty acids, monounsaturated fatty acids, and polyunsaturated fatty acids (PUFAs).

Acids

- Saturated = no double bonds
- Unsaturated = presence of double bonds (could be one or two)
- Polyunsaturated = more than two double bonds

Saturated fatty acids are “saturated” with hydrogen or straight chains with no double bonds (e.g., palmitic acid, C16:0). When there is a predominance of saturated fats in the glycerol moiety, the triacylglycerol tends to be solid. This is because due to their straight chain nature, they tend to “pack” very tightly in the membrane (e.g., tallow or beef fat; Figure 6.2).



Palmitic Acid

Figure 6.2. Saturated fatty acid with no double bonds

Unsaturated fatty acids contain one or more double bonds between adjacent carbon atoms in the hydrocarbon chain. Unsaturated fatty acids may be either mono (one double bond) or polyunsaturated (more than two double bonds). When there is a predominance of unsaturated fats, the triglyceride tends to be liquid because unsaturation gives a “bend” in their structure and they cannot pack as tightly as saturated fats (e.g., vegetable oil).

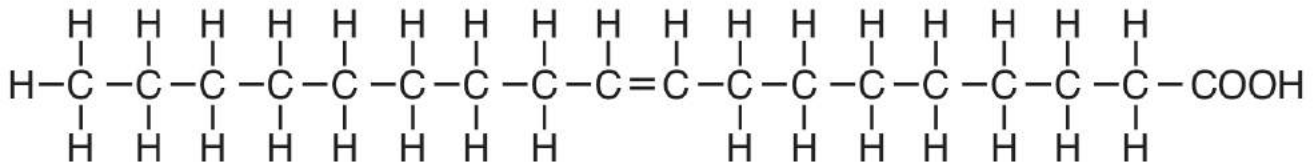


Figure 6.3. Unsaturated fatty acid

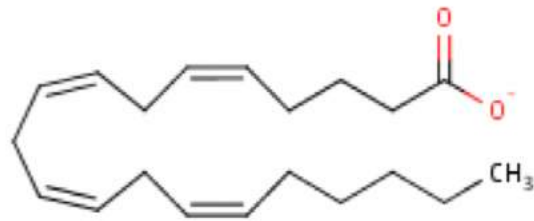


Figure 6.4. Polyunsaturated fatty acid

Polyunsaturated fatty acids are commonly called “PUFA” and contain two or more double bonds. Due to these extra double bonds, PUFAs tend to be more “round” when compared to the straight chain structure of a saturated fat (e.g., Figure 6.2 vs. Figure 6.4). These double bonds also change the physical nature of the fat, making it more liquid than straight chain saturated fat. In addition to the number of double bonds, the position of double bonds in the carbon-carbon chain is also important in nutrition and in the metabolism of lipids; this is explained below.

Nutritionists designate the term *omega* (ω) or “n” to denote the position of double bonds in the carbon chain in a PUFA. The omega carbon is the first carbon with a double bond counting from the methyl end (CH_3) of the carbon chain. The two PUFA classifications are omega-6 (also called n-6, or ω -6) or omega-3 (n-3, or ω -3). For instance, omega-3 fatty acid will have the first double bond at the third carbon when counted from the methyl (CH_3) end (Figure 6.5a) and omega-6 fatty acids will have the first double bond at the sixth carbon when counted from the methyl (CH_3) end (figure 6.5b). The locations of the double bonds are also indicated by the Greek letter Δ , “delta,” in some chemistry or biochemistry textbooks. The delta term denotes the position of double bonds from the carboxyl end. However, the term omega, or “n,” is the one that is commonly used by nutritionists.

Two Types of PUFA

1. Omega-6 (n-6, or ω -6) fatty acid
2. Omega-3 (n-3, or ω -3) fatty acid

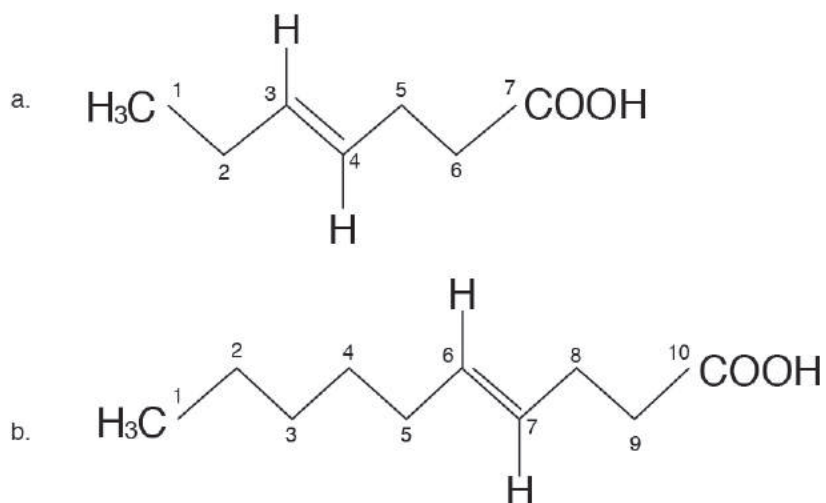


Figure 6.5. Basic structure of an omega-3 (a) and an omega-6 (b) fatty acid

Essential Fatty Acids

In nonruminant, or monogastric, animals such as pigs, two fatty acids (α -linolenic acid, C18:3 n-3) and linoleic acid (C18:2 n-6) have to be supplied in the diet and are called essential fatty acids. This essentiality is due to the inability to insert double bonds at the third and sixth carbon from CH₃ end in n-3 and n-6 locations. In addition to these two essential fatty acids, carnivores such as cats need arachidonic acid (C20:4 n-6) in their diets.

In nutrition, the term “essential” means animals cannot synthesize it to meet their requirements. Essential fatty acids include the following:

1. Linoleic acid (C18:2 n-6)
2. Linolenic acid (C18:3 n-3)
3. Arachidonic acid (C20:4 n-6; in true carnivores, e.g., cats)

Fatty Acid Nomenclature

Fatty acids are commonly expressed by their trivial names (e.g., linoleic acid) or their associated shorthand notations (C18:2 n-6). The shorthand nomenclature of a fatty acid includes the number of carbon atoms and double bonds. For instance, in linolenic acid, C18:2 n-6 stands for 18 carbon atoms and two double bonds, of which the first double bond is at the sixth carbon atom from the methyl carbon. Some of the common fatty acids in animal foods, such as chicken or pork, and their trivial names and shorthand notation are shown in Table 6.1.

Cis and Trans Fatty Acids

Unsaturated fatty acids can form geometric isomers, with either cis or trans, depending on the stereo-conformation of groups around a double bond. Most natural fatty acids of animal and plant origin are of the cis type, whereas those of bacterial origin contain both cis and trans types.

Table 6.1. Names and abbreviations of some of the common fatty acids in animal tissues.

Palmitic Acid	C16:0
Palmitoleic	C16:1
Stearic acid	C18:0
Oleic acid	C18:1
Linoleic acid	C18:2 n-6
Linolenic acid	C18:3 n-3
Arachidonic acid	C20:4 n-6
Docosahexaenoic acid	C22:6 n-3

For example, conjugated linoleic acid (CLA) is a trans fatty acid present in cow's milk or other ruminant food like beef and is produced by rumen microbes during the biohydrogenation process. In CLA, the two double bonds lack a methylene group separating them, have a conjugated arrangement, and are called natural trans fats. Trans fats such as CLA have received considerable attention due to their several health-promoting (e.g., anticancer, immune health enhancing, lean body mass enhancing) effects. There are other trans fats that are produced during the hydrogenation process (the addition of hydrogen) when liquid vegetable oil is made into solid fats such as margarine. These are synthetic trans fats and have different health effects when compared with "natural" trans fats such as CLA.

CLA is an intermediate conjugated fatty acid formed during biohydrogenation, or conversion from unsaturated to saturated fatty acid.

Essential fatty acid

Linoleic acid (9c, 12c-18:2)



α -Linolenic acid (9c, 12c, 15c-18:3)



Δ -9 (counted from carboxyl end of the hydro carbon chain is shown as the position of the first double bond in linoleic (18:2 Δ -9,12) and linolenic (18:3 Δ -9,12,15) acid.

Conjugated linoleic acid (CLA)

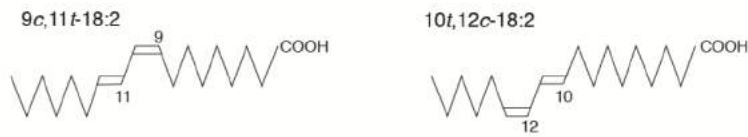


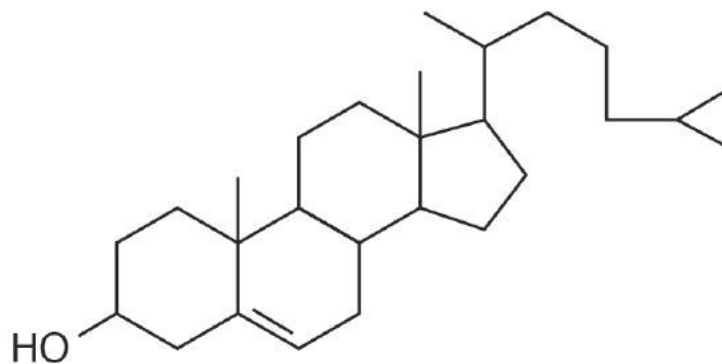
Figure 6.6. Fatty acid structure showing cis bonds in essential n-6 and n-3 fatty acid versus conjugated trans bonds in CLA isomers. Source: Google

Cis versus Trans Fatty Acids

- Most natural fats occur in the cis form.
- The exception is a trans fat called conjugated linoleic acid (CLA; C18:2 n-6), which is produced by rumen microbes.

Cholesterol

Sterols (lipids with phenanthrene ring-like structures) are the most abundant steroid in the human diet. Cholesterol is the best known steroid (fat-soluble substance containing a steroid nucleus) and is the precursor of many other substances such as vitamin D, bile acids, sex hormones, and corticosteroid hormones.



An important component of animal tissues, egg yolks, and cell membranes, cholesterol synthesis is partly by dietary intake and partly by biosynthesis from acetyl CoA. Excess cholesterol is stored in arteries and can lead to atherosclerotic plaque formation and cardiovascular disorders. Excretion of cholesterol is through bile acid formation. Plant cells do not contain cholesterol but instead contain other sterols called phytosterols.

Key Points

1. The lipid constituent of a feed is that portion that is soluble in organic solvents. Chemically, it is defined as an ester of fatty acids and glycerol. The most common form of lipids in plants is the triglyceride, but some parts of plants also contain compound lipids.
2. Fats are made up of a glycerol backbone with fatty acids attached. We call these triglycerides, or more correctly, "triacylglycerol."
3. Triglycerides serve as energy reserves for the plant (seeds) or animal (fat depots).
4. As the fat content of feed goes up so does its energy value.
5. Functions of fats include providing energy, being components in the plasma membrane of all cells, being carriers for fat-soluble vitamins, and providing insulation and lubrication.
6. Fatty acids may be saturated, unsaturated, or polyunsaturated. Palmitic and stearic acids are saturated, oleic acid is unsaturated, and linoleic acid is polyunsaturated.
7. There are two essential fatty acids. These are linoleic (C18:2) and linolenic (C18:3).
8. Arachidonic acid requirements may be met with linoleic acid (except in cats). Essential fatty acid can be omega-6 and omega-3 based on the position of the first double bond from the methyl (CH₃) end.
9. Conjugated linoleic acids (CLA) are a group of various fatty acid isomers synthesized by the rumen bacteria.
10. CLA is 18 carbon atoms long with two double bonds separated by only one carbon, thus the name "conjugated." Most recently, CLA has been discovered as a potent inhibitor of fat deposition. Other effects such as cancer prevention and promotion of immune health are also reported.
11. Cholesterol is the most abundant steroid present in animal tissue and serves as a precursor for vitamin D, bile acids and steroid hormones.
12. Cholesterol synthesis in the body is regulated by intake and by excretion through bile acid formation.
13. Cholesterol deposits in arteries can lead to pathological disorders.

Review Questions

1. What are the functions of lipids in animal diets?
2. What is the difference between saturated, unsaturated, and polyunsaturated fatty acids?
3. What is the difference between omega-3 and omega-6 fatty acids? Give an example of each.
4. C_{20:5} n-3 is a fatty acid present in fish oil. Write three things about this fatty acid from its scientific notation.
5. What is a conjugated fatty acid? Give an example.
6. What is the difference between cis and trans fatty acids? Give an example of each.

7. Why is it that we can pour salad dressing, while we need a knife to cut the fat around a steak?
8. What are the essential fatty acids, and why are they essential?
9. Which of the fatty acids are considered essential for cats?

VII. Lipids, Digestion

This chapter provides an introduction to the different processes that are involved in the digestion of lipids in monogastric and ruminant animals.

New Terms

Bile
Biohydrogenation
Chylomicron
Emulsification
Micelles
Pancreatic lipase

Chapter Objectives

- To discuss the digestion of lipids in food-producing animals
- To discuss the biohydrogenation and conjugated linoleic formation in ruminant animals

Monogastric Animals

The digestion process involves the breakdown of lipid molecules into smaller ones that are eventually absorbed into the blood. Lipids are not soluble in water, which is the aqueous medium of the digestive tract (lipids are hydrophobic). Therefore, the initial step in lipid digestion is to make them dissolve in water. How? Through a process called emulsification, or the dispersion of lipids in small droplets.

Emulsification is the dispersion of lipids in small droplets.

Dietary lipids (mostly triglycerides), upon their entry into the small intestine, are emulsified by bile salt (also called bile acid) released from the gall bladder. Bile salt functions as a detergent (due to their OH and COOH groups), and large lipid molecules form smaller lipid droplets surrounded by a layer of bile. Emulsified lipids are acted upon by enzyme pancreatic lipase and converted into fatty acids, monoglycerides and glycerol.

The lipid digestion products are assembled into micelles. These are temporary combinations of bile salt, fatty acids, monoglycerides, and other fat-soluble substances such as vitamins and cholesterol. The micelles are water soluble and enable the lipid digestion products to be transported to the small intestinal surface for absorption. At the site of absorption, the micelle breaks down and the bile salt returns to the intestine for continuing emulsification processes (**bile salt recycling**). The components are absorbed into the small intestine by passive diffusion. In a nutshell, the ability to form micelles and the presence of bile salt are very important for lipid digestion, and the lack of it can affect digestibility. For example, saturated fatty acids are less efficient than unsaturated fatty acids in forming micelles. So a blend of saturated and unsaturated fatty acids is used in animal rations.

Micelles and chylomicrons are temporary compounds formed during lipid absorption.

Once inside the intestinal cell (or enterocyte), the monoglycerides and fatty acids are reesterified, and together with free and esterified cholesterol, lipoproteins and phospholipids are assembled into **chylomicrons**. The chylomicrons are secreted into the lymphatic system.

Ruminant Animals

In ruminant animals, the lipid content of the diet is low (under 5%) and comes from different sources such as grass, leaves, oil seeds, or cereal grains. Leaf or grass lipids are mainly galactolipids, phospholipids, waxes, pigments, and essential oils, and oil seed or grain lipids are mainly triglycerides.

In the rumen, there is no emulsifying agent or pancreatic lipase enzyme. Instead, there are rumen microbes producing microbial lipases. When dietary lipids enter the rumen, the initial step is the hydrolysis of the ester linkages in triglycerides, phospholipids, and glycolipids. Hydrolysis of dietary lipids is done by microbial lipases, which releases glycerol and fatty acids (free fatty acids) from the lipid backbone. Glycerol is readily metabolized by the rumen bacteria to form propionic acid. Feeding of supplemental fat increases the proportion of propionic acid (one of the volatile fatty acids, or VFAs) and the propionate:acetate ratio in ruminants. Hydrolysis is a prerequisite for the next step.

Biohydrogenation of unsaturated fatty acids is the second major transformation that dietary lipids can undergo in the rumen. Fatty acids with double bonds are altered by microbes to form more stable fatty acids. Fatty acids such as linoleic acid are converted “conjugated” fatty acids (e.g., conjugated linoleic acid, or CLA) in which the double bonds are not separated by methylene (CH₂) groups. The position of double bonds is altered, and the fatty acids are converted to more stable “trans” fats. Some odd-numbered (e.g., C19:0) and branched-chain fatty acids are also created during this process. For example, linoleic acid (C18:2 n-6), where the double bonds are in the cis position (cis9-cis12), is converted to several isomers of CLAs during this conversion step.

Lipids undergo hydrolysis, biohydrogenation, and conjugated fatty acid formation in the rumen.
Why biohydrogenation?
Too much unsaturated fatty acids can be toxic to rumen microbes.

Lipid digestion in the ruminant small intestine is very similar to lipid digestion in monogastric animals. The two key secretions enabling this process are bile and pancreatic juices. These secretions enable the lipids to form micelles for absorption. Bile supplies bile salts and pancreatic juice and enzymes. These compounds desorb the fatty acids from feed particles and bacteria, allowing the formation of micelles. Once micelles are formed, they facilitate the transfer of water-insoluble lipids across the intestinal epithelial cells of the jejunum, where the fatty acids are absorbed. Within the intestinal epithelial cells, the fatty acids are reesterified into triglycerides and then packaged into chylomicrons for transport in lymph to the blood.

Key Points

1. Digestion of fat is mainly done in the small intestine.
2. Bile salts emulsify the fat and pancreatic lipase hydrolyzes them to release fatty acids and glycerol.
3. Micelles are temporary compounds formed during the fat digestion and absorption process.
4. Micelles are water soluble and enable the lipid digestion products to be transported to the small intestinal

surface for absorption.

5. At the site of absorption, micelle breaks down and components are absorbed into the small intestine by passive diffusion.
6. Bile salt returns to the intestine for continuing emulsification processes.
7. Reuse of bile salt for fat digestion is called bile salt recycling.
8. In ruminant animals, lipids undergo hydrolysis, biohydrogenation, and conjugated fatty acid formation in the rumen.
9. All these processes are done for the survival of rumen microbes because too much unsaturated fatty acids can harm microbial survival.
10. During biohydrogenation, several intermediate trans fatty acids are formed. Conjugated linoleic acid (CLA) is an example of such an intermediate.
11. Lipid digestion in the ruminant small intestine is very similar to that in monogastric animals.
12. Chylomicrons are temporary compounds (e.g., micelles) formed during lipid absorption.

Review Questions

1. What is meant by biohydrogenation?
2. Give an example of a conjugated fatty acid.
3. What are the primary compounds involved in the digestion and absorption of fats, where do these compounds come from, and what are their primary functions?
4. How do micelles differ from chylomicron?
5. How do cows differ from pigs in fat digestion and absorption upon consuming diets containing canola oil?

VIII. Lipids, Transport, Deposition, and Metabolism

This chapter discusses the transport of digested and absorbed fatty acids in the animal body, depositing in tissues, and synthesis and oxidation of fatty acids for energy production.

New Terms

Fatty acid synthesis
High-density lipoprotein (HDL)
Ketone bodies
Ketosis
Lipogenesis
Lipoproteins
Low-density lipoprotein (LDL)
Lipoprotein lipase
Malonyl CoA
 β -oxidation
Very-low-density lipoprotein (VLDL)

Chapter Objectives

- To discuss the role of lipoproteins in fatty acid transport, storage and metabolism.
- To introduce the transport of digested fatty acids in the body
- To introduce how digested and absorbed fatty acids are mobilized and can serve as energy sources during times of need

Lipid Transport: Blood lipids consist of chylomicrons formed within the intestinal mucosal cells during absorption as well as lipids derived from storage depots, such as liver and adipose tissue. Blood lipids are transported as lipoproteins due to their hydrophobic nature.

Lipids are transported as lipoproteins in the blood.

Lipoproteins: Lipoproteins consists of an inner core of hydrophobic lipids surrounded by a surface layer of phospholipids, cholesterol, and outer proteins (apolipoprotein).

Lipoproteins are a lipid + a protein (compound lipid). Because lipids are less dense than water, the density of lipoproteins decreases as the proportion of lipid to protein increases. Lipoproteins have a major role in lipid and cholesterol transport and metabolism.

Lipoproteins are classified based on their density and composition. The main lipoproteins in blood are chylomicrons, very-low-density lipoproteins (VLDL), low-density lipoproteins (LDL), and high-density lipoproteins (HDL).

Main Lipoproteins

- Chylomicron
- VLDL
- LDL
- HDL

Chylomicrons are synthesized in the small intestine from dietary fat, and VLDL, LDL, and HDL are synthesized in the liver and small intestine. Chylomicrons enter the liver and are packaged into VLDL. VLDL is involved in the transport of triacylglycerol (TAG) from the liver to extrahepatic tissues. LDL transports cholesterol (so-called bad cholesterol) to tissues and HDL is responsible for “reverse transport” or removal of cholesterol (so-called good cholesterol) from tissues. The terms “bad” and “good” refer to the nature of

transport. For example, the “good” cholesterol refers to cholesterol being carried by HDL, which is meant to be routed to the liver for bile formation, or excretion, thus leaving the body and not deposited, like LDL, into blood vessels.

Fatty Acid Metabolism

The liver has a central role in lipid transport and metabolism as it is involved in the synthesis and catabolism of lipoproteins. Pathways of fatty acid degradation and biosynthesis are highly intertwined with pathways of carbohydrate metabolism due to the central role of acetyl CoA in both lipid and carbohydrate metabolism.

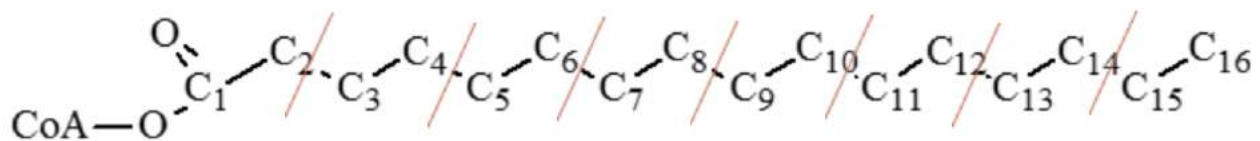
Fatty Acid Oxidation

Fatty acids released from the hydrolysis of TAG are transported via blood. Oxidation occurs in the mitochondria of tissues, such as skeletal muscle, cardiac tissue, liver tissue, adipose tissue, and other tissues by converting them to acetyl CoA, which is then catabolized in the citric acid cycle.

Fatty acids are oxidized in the mitochondria by converting them to acetyl CoA.

Before oxidation begins, fatty acids are activated (they react with CoA) to form fatty acyl CoA (ATP is needed for this step). Activated fatty acids are transported across the mitochondrial membrane (carnitine serves as a carrier). In β -oxidation, two carbons at a time are cleaved off from fatty acid CoA, starting from the carboxyl end. The hydrogen produced from fatty acids are taken up the chain by hydrogen acceptors (e.g., FAD, NAD⁺) and produces a high yield of ATP. Acetyl CoA released can enter the tricarboxylic acid (TCA) cycle for ATP production, can form ketone bodies, or can be used for resynthesis of fatty acids.

For example, during the β -oxidation of palmitic acid (C₁₆:0), through degradation of two carbon fragments, total ATP and net ATP generated is shown below.



- a. Carbon-carbon cleavage
 - i. 7 cleavage (shown by red lines) = 5 ATP per each cleavage (1 FADH + 1 NADH)
 - b. Oxidation of acetyl CoA
 - i. 8 acetyl CoA units entering the TCA cycle = 12 ATP per acetyl CoA unit
- Total ATP for 1 mole of palmitic acid (C₁₆:0) = 35 ATP + 96 ATP = 131 ATP
= 7 cleavage points (7 × 5 = 35 ATP) + 8 acetyl CoA units (8 × 12 = 96 ATP)

Net ATP generated by 1 mole of palmitic acid (C16:0) = $35 + 96 - 2 = 129$ ATP
(Two ATP used for initial activation and are then subtracted from 131 ATP)

Fatty acids produce higher levels of ATP than carbohydrates because they are less oxidized.

Fatty acids of 16 C and 18 C are oxidized through the β -oxidation pathway. Other longer-chain fatty acids are oxidized in the peroxisomes. β -oxidation of unsaturated fatty acids occurs until the first double bond is reached. The double bonds are isomerized from cis to trans and then hydrolyzed by isomerase, ultimately yielding acetyl CoA.

Lipogenesis and Fatty Acid Synthesis

Lipogenesis is the process of synthesizing lipids as a means of storing chemical energy. Fat cells, or adipocytes, are dispersed throughout the body and are considered a long-term energy depot. Lipogenesis encompasses fatty acid synthesis (cytosol of hepatocytes and adipocytes), adipocyte uptake, and storage of lipids as the body's "savings account."

What Causes Fatty Acid Synthesis? Glucose is the key signal for fat storage (feasting). Excess energy (ATP) generated from glycolysis and the TCA cycle is taken up by the body to convert it to fat. As ATP levels increase beyond the cells' requirements, the ATP begins to accumulate, which stimulates activity of the enzyme acetyl CoA carboxylase. Increased insulin concentrations are also required to stimulate acetyl CoA carboxylase activity.

Fatty acid biosynthesis begins with 2 C acetyl CoA (like β -oxidation). Acetyl CoA could come from fats, carbohydrates, or some amino acids. Fatty acid synthesis occurs in the cytosol (endoplasmic reticulum). The fatty acid chain is assembled in 2 C units (derived from acetyl CoA) by joining the carboxyl end of one fragment to the methyl tail of another, yielding palmitic acid (C16:0) as the end product.

Fatty acid synthesis occurs in the cytosol.

Briefly, acetyl CoA carboxylase uses one ATP to combine one mole of acetyl CoA to form malonyl CoA. Fatty acid synthesis begins with the addition of carbon dioxide to 2 C acetyl CoA to form 3 C malonyl CoA. Acetyl CoA carboxylase is the enzyme needed for this step. Malonyl CoA reacts with acetyl CoA to produce a 5 C intermediate compound, which is decarboxylated to form a 4 C butyryl CoA. This in turn then combines with another malonyl CoA and is decarboxylated to form 6 C caproyl CoA (Figure 8.1). The step is continued by adding malonyl CoA and losing CO₂ each time to produce 16 C palmitic acid as the eventual final product.

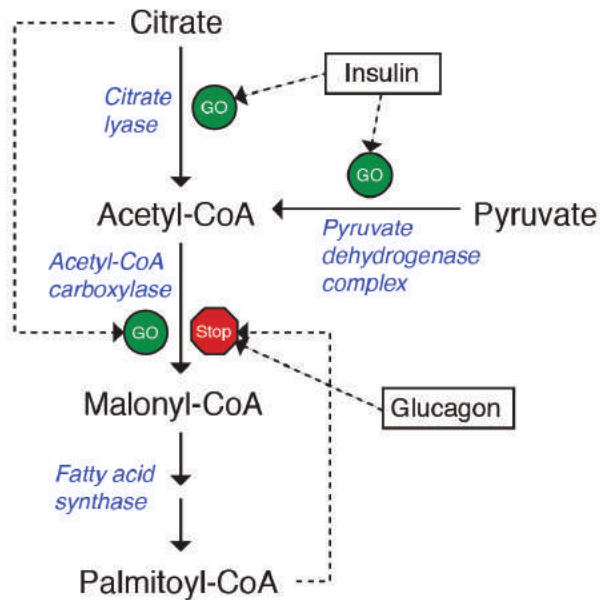


Figure 8.1. Fatty acid biosynthesis Source: <http://www.bioinfo.org.cn>

Most fatty acids are even numbered because each acetyl CoA, and subsequent malonyl CoA, is only two carbons in length. Each malonyl CoA adds two more carbons, and the resulting fatty acid is almost always evenly chained. Some very minor odd-numbered fatty acids (e.g., C17:0, C19:0) are present in animal tissues.

Fatty acid synthase is the enzyme involved in these steps. Palmitic acid (16 C) can be elongated to form 18 C stearic acid by elongases. Further formation of unsaturated fatty acid (oleic acid) C18:1 is by the introduction of double bonds by the desaturase enzyme. Mammals cannot insert double bonds beyond Δ -9 position. Therefore, linoleic (18:2 Δ -9,12) and linolenic (18:3 Δ -9,12,15) need to be provided in the diet and are called essential fatty acids. However, upon consumption, mammals can further desaturate and elongate the 18 C essential fatty acids to form longer chain 20 and 22 C fatty acids.

Storage and Mobilization of Lipids

Lipids are stored as TAG. The glycerol backbone of TAG is formed from dihydroxyacetone phosphate, produced from the preparatory phase of glycolysis (along with glyceraldehyde-3-phosphate). Glycerol is three carbons, and therefore, each carbon can bond together with a fatty acid. The bonding of the glycerol and fatty acids occurs mainly within the cytosol of hepatocytes or adipose tissue in ruminants (or the mammary gland in lactating animals). Once the TAGs have been formed, they are attached to lipoproteins and deposited in the blood for transport. The lipoprotein complexes are recognized by lipoprotein lipase, and the TAGs are removed and deposited as fat (e.g., fat pads in chickens, back fat in pigs).

Mobilization of lipids from storage sources for energy production is through the action of hormone-sensitive lipoprotein lipase, which releases free fatty acids and glycerol. Mobilization occurs during starvation (fasting), stress or increases in energy usage (e.g., disease). Hormones such as glucagon and epinephrine increase, and insulin is reduced under these conditions and stimulates the action of lipases (hormone sensitive).

In dairy cattle and in ewes during certain conditions such as negative energy balance, fat mobilization is at its peak and may exceed the rate at which acetyl CoA enters the TCA cycle. During such conditions, hepatic synthesis of ketone bodies (ketogenesis) occurs. The ketone bodies are acetoacetic acid, β -hydroxybutyric acid, and acetone. Ketosis is a metabolic disorder when excessive quantities of ketone bodies are produced and is also described in the chapter on bioenergetics (Chapter 17).

Ruminant Animals: Ruminant animals derive acetyl CoA directly from absorbed acetate (volatile fatty acid) rather than glucose. Citrate is permeable and is transported across the mitochondrial membrane and is converted to α -ketoglutarate with the production of nicotinamide adenine dinucleotide phosphate (NADPH). The α -ketoglutarate reenters mitochondria, while NADPH is used for fatty acid synthesis from acetate. It is a means for ruminants to conserve glucose and glucogenic precursors.

Key Points: Lipid Metabolism

Lipid Transport

- Chylomicrons enter the liver and are packaged into very-low-density lipoproteins (VLDL).
- VLDL delivers triacylglycerols (TAGs) from the liver to extrahepatic tissues. Once they unload the TAGs at the target tissues, their density increases and thus the LDL and HDL increase as well.
- LDL carries most of the cholesterol to tissues. Once they unload the cholesterol at the target tissues, their density increases, and thus HDL increases as well.
- HDL offloads all the remaining cholesterol and triglycerides to liver and is marked for excretion.

Lipid Storage

- For TAGs to enter the cells, lipoprotein lipase is required to hydrolyze them into fatty acids and glycerol again. The formation of TAGs requires a glycerol backbone, which can only come from glycolysis. Consequently, glucose is the key signal for fat storage.

Lipid Mobilization

- Mobilized lipids are used for energy production. Mobilization of fat out of adipose tissue requires another lipase. Hormone-sensitive lipase hydrolyzes TAGs and releases fatty acids and glycerol into blood circulation and provides other tissues with the substrate for energy.

β -Oxidation of Fatty Acids (Energy Production)

1. Activation of fatty acid
 1. is done by acetyl CoA synthase to yield fatty acyl CoA
 2. is ATP dependent, needs two ATP
2. Transportation into the mitochondria (carnitine needed)
3. Degradation of two carbon fragments (β -oxidation) provides one NADH and one FADH per each acetyl CoA.

Ketogenesis occurs under negative energy balance conditions in the liver.

- Excessive acetyl CoA accumulated from β -oxidation cannot be used in the TCA cycle; low incoming glucose due to starvation force the liver to make ketone bodies that can serve as an alternate energy source. However, continued synthesis leads to an accumulation of ketone bodies in the blood.

Fatty Acid Synthesis

- The location is cytosol.
- The starting material is acetyl CoA.
- The key enzymes are acetyl CoA carboxylase and fatty acid synthase complex.
- Starting with acetyl CoA (mainly from carbohydrates), two carbon units are added from the carboxyl to the methyl end.
- The cycle continues through the addition of malonyl CoA (building block for synthesis) and loss of CO₂ up to 16 C.
- Further elongation and desaturation take place to form long-chain fatty acids. However, mammals cannot insert double bonds beyond Δ -9 position.
- Two fatty acids, linoleic and α -linolenic, are essential and should be included in the diet.

Review Questions

1. Why do most plant and animal fats have even-numbered fatty acids?
2. What are essential fatty acids and why are they essential?
3. What are the sites of fatty acid synthesis and oxidation in the cell?
4. What is the starting material for fat synthesis?
5. What are ketone bodies, and what is ketosis?
6. This lipoprotein is commonly referred to as “good” cholesterol.
7. This lipoprotein is commonly referred to as “bad” cholesterol.

IX. Proteins

This chapter provides an introduction and discussion of proteins and amino acids that are important in the nutrition of food-producing animals.

New Terms

Amino acid
Dipeptide
Essential amino acid
Nonessential amino acid
Peptide bond
Polypeptide
Protein

Chapter Objectives

- To describe the chemical structure of proteins and amino acids
- To discuss the different classification of protein and amino acids
- To discuss essential and nonessential amino acids

Proteins

What Are Proteins?

The word *proteins* was coined by a Dutch chemist G. J. Mulder and originated from the Greek word “*proteios*”, meaning first or most important. Proteins are organic compounds made up of different building blocks (basic units) called amino acids joined together by peptide bonds (Figure 9.1). A dipeptide contains one peptide bond and two amino acids, whereas a tripeptide contains three amino acids and two peptide bonds. A peptide with more than ten amino acids is called a polypeptide. Proteins are essentially large polypeptides. The structure of a protein is determined first by the sequence of individual amino acids it has in the polypeptide chain. This is also called the primary structure of the protein.

Protein: Functions

- Body proteins (e.g., muscle, hair, hooves, skin)
- Blood proteins (e.g., albumin, globulin)
- Tissue proteins (e.g., collagen, keratin)
- Enzymes and hormones
- Immune system antibodies and other peptide growth factors

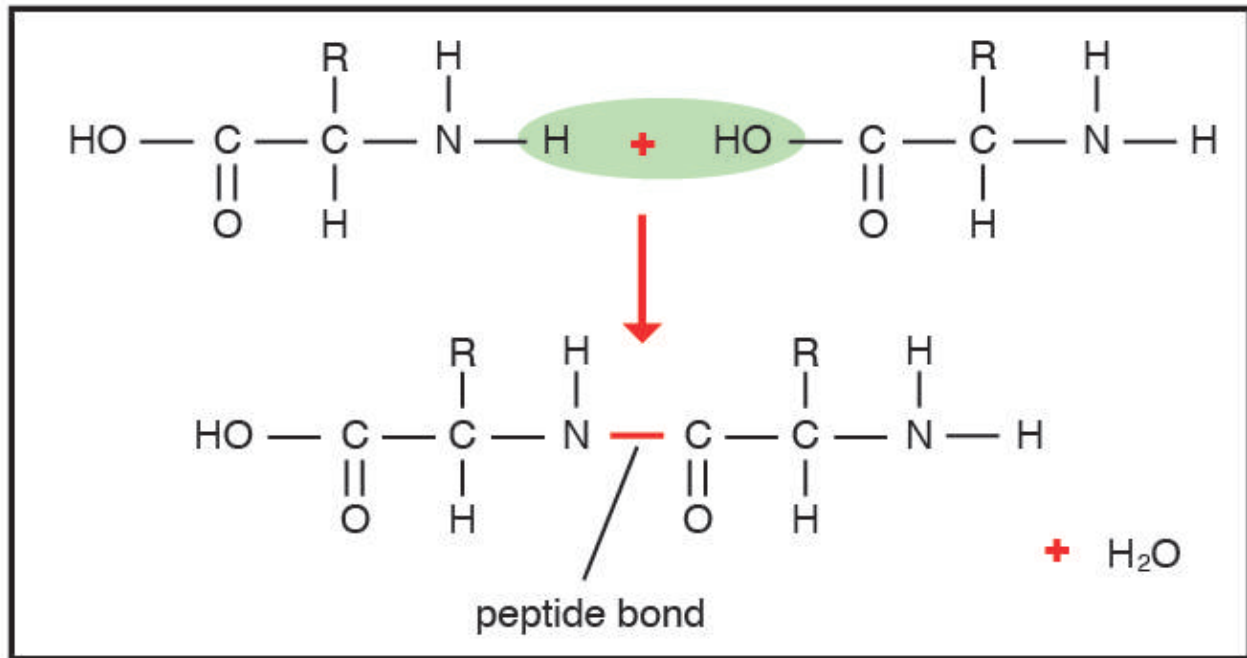


Figure 9.1. Peptide bond between amino and carboxyl group (Source: Google)

Protein Functions: Proteins are vital for life and are the major structural components of animal tissues (e.g., skin, muscles, wool, feather, tendons, eggs). In addition, proteins are also involved in biochemical (e.g., enzymes), immunological (e.g., immunoglobulins), transportational (e.g., lipoproteins), and other regulatory (e.g., hormones) activities. Proteins can also provide energy when needed.

Many of the structures in animal tissue (e.g., muscle) and metabolic reactions (e.g., enzymes, hormones) are catalyzed by proteins. Therefore, protein synthesis is essential for maintaining life process. Provision of adequate dietary protein and amino acids are essential for maintaining growth, health, and productivity in food-producing animals. Intestinal microflora can synthesize proteins from nonprotein sources in ruminant animals.

Protein requirements vary with life stages and are high during phases of fast growth in young animals and during pregnancy and lactation. Like other macronutrients, proteins contain carbon, oxygen, and hydrogen. In addition, proteins also contain nitrogen and sulfur (in some amino acids). It is the nitrogen that makes proteins very unique in animal nutrition with respect to its digestibility, metabolism, and disposal within the animal body.

Classification of Proteins

Proteins can be classified based on their shape; solubility in water, salt, acid, base, or alcohol; or according to the nature of the prosthetic group.

Classification Based on Solubility and Prosthetic Group

1. Globular proteins: soluble in water or dilute acids, bases, or alcohol
 - a. Albumin (water soluble; present as albumen in egg white; in blood circulation, it performs various functions [e.g., as a carrier of lipids])
 - b. Globulin (soluble in dilute neutral solutions; functions as part of the immune system in body defense [e.g., immunoglobulins])
2. Fibrous proteins: insoluble in water and are resistant to digestive enzymes
 - a. Keratins (e.g. wool, hair, feather, hooves, horn)
 - b. Collagen (can be converted to gelatin when heated; present in bone, teeth, tendons, and soft connective tissue)
3. Conjugated proteins: contain other nonprotein compounds in structure. Some examples follow:
 - a. Lipoproteins (lipid-carrying protein)
 - b. Hemoprotein (proteins with heme units)
 - c. Glycoproteins (proteins with sugar)
 - d. Nucleoprotein (proteins bound to nucleic acid)

These proteins have limited nutritional value but are important in biochemical, structural, and other metabolic functions. For example, feather meal is high in protein (keratin) but very low in digestibility and is of limited use in animal nutrition as a feed ingredient. Amino acids in the polypeptide chain in feather meal form disulfide bonds (-S-S-), which twist the polypeptide chain into a specific coiled structure such as helix or sheet. This is called a secondary structure. These bonds account for the tough physical properties of hooves and horns and their low digestibility.

The disruption of secondary structure by heat treatment causes denaturation of the proteins (e.g., egg white coagulation during cooking). Certain antinutritional factors in feed (e.g., trypsin inhibitor in soybean meal) are proteins. Heat processing denatures trypsin inhibitor in soybean meal and can enhance digestibility.

Amino Acids: Amino acids are the building blocks of proteins. There are more than 300 different amino acids known to exist in nature. Out of these, about 20 amino acids are important constituents of animal proteins and are associated with muscles, connective tissues, skin, feathers, horns, blood, enzymes, and hormones. There about 10 amino acids that should be present in the diet of animals because animal tissues cannot synthesize them or cannot make the adequate amount needed for metabolic functions; these are called essential amino acids. A few other amino acids such as citrulline and ornithine do not occur in animal tissues but are involved in cellular metabolic functions.

Amino acids are the building blocks of proteins.

Essential amino acids must be supplied through the diet.
Animals cannot synthesize them or cannot make the adequate amount needed.

All amino acids by definition contain at least one amino group (-NH₂) and one carboxyl group (-COOH) on the C atom adjacent to the carboxyl group (Figure 9.2). An exception to this is proline (imino acid), which is lacking a free amino group. The general structure of an amino acid is shown below by the amino acid glycine, the simplest of the amino acids

(Figure 9.2). The R group (shown in the red circle) in amino acids varies for different amino acids. The R group is the remainder of the molecule or any other group attached to the C atom. In the case of glycine, it is an H group. The amino group (NH₂) provides basic properties to the amino acid, and the carboxyl (COOH) group provides acidic properties. Amino acids important in animal nutrition are alpha (α) amino acids, which are carboxylic acids with an amino group on the α-carbon (or the first carbon attached to a functional group). A list of amino acids important in animal nutrition, their essentiality and classification are shown in Table 9.1.

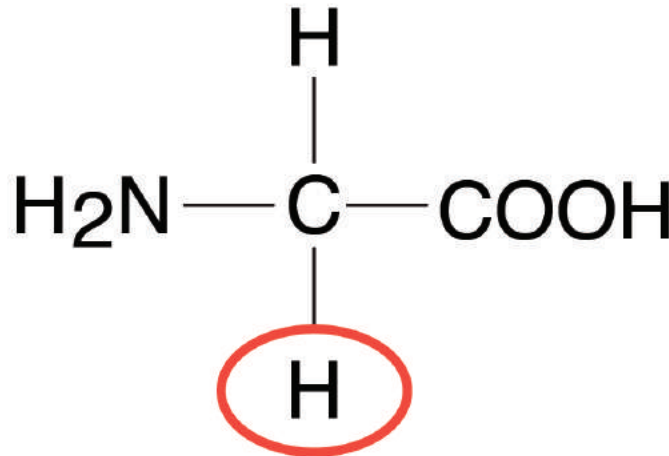


Figure 9.2. General structure of amino acid

Amino acids can exist in two isomeric forms, the D- and L-isomers. The D- and L-amino acids differ in their configuration of groups around the asymmetric α-carbon. Only L-amino acids are used in protein synthesis, except methionine, where both D- and L-amino acids can be used by the animal. DL methionine is commonly used as an amino acid supplement in animal feeds.

All amino acids except glycine contain an asymmetric α-carbon (with four different chemical groups attached to it). Compounds with asymmetric carbons can exist as isomers.

L-Alanine	D-Alanine
COOH	COOH
/	/
NH ₂ -C-H	H-C-NH ₂
\	\
CH ₃	CH ₃

Essential Amino Acids: Animal body can synthesize some amino acids in sufficient amounts. However, animals cannot synthesize some amino acids, or not in the amount that is needed for body requirements. Such amino acids need to be provided through diet in monogastric animals; these are called essential (indispensable) amino acids. Pigs, dogs, and humans need a total of 10, and chickens and cats need a total of 11 essential amino acids. A list of essential amino acids needed by monogastric animals is shown below.

It should be borne in mind that other nonessential amino acids are also physiologically important for metabolic functions in the body and are made from other precursors available through diet (e.g., carbohydrates, nonprotein

nitrogenous substances).

The need for essential amino acids varies in animals. For example, horses need essential amino acids, whereas ruminant

animals (e.g., cattle, sheep, goats) generally do not have a requirement of essential amino acids as they are synthesized by rumen microbes.

List of Essential Amino Acids and Their Common Abbreviations

1. Arginine (Arg)
2. Histidine (His)
3. Lysine (Lys)
4. Isoleucine (Ile)
5. Leucine (Leu)
6. Methionine (Met)
7. Phenylalanine (Phe)
8. Threonine (Thr)
9. Tryptophan (Try)
10. Valine (Val)

In addition to these 10 essential amino acids, cats and chickens need the following extra amino acids.

Cats need taurine (Tau), and chickens need glycine (Gly).

Table 9.1. Classification of amino acids according to structure, nature of side chain, and essentiality. E=essential; NE=nonessential.

Amino acid	Essentiality	Classification
Arginine (Arg)	E	Basic
Histidine (His)	E	Basic
Lysine (Lys)	E	Basic
Aspartic acid (Asp)	NE	Acidic
Glutamic acid (Glu)	NE	Acidic
Alanine (Ala)	NE	Aliphatic-Straight chain
Glycine (Gly)	E (chickens)	Neutral- Aliphatic-Straight chain
Isoleucine (Ilu)	E	Branched chain
Leucine (Leu)	E	Branched chain
Valine (Val)	E	Branched chain
Serine (Ser)	NE	Hydroxy
Threonine (Thr)	E	Branched chain
Cysteine (Cys)	NE	Sulfur-containing
Methionine (Met)	E	Sulfur-containing
Phenylalanine (Phe)	E	Aromatic
Tryptophan (Try)	E	Aromatic
Tyrosine (Tyr)	Ne	Aromatic
Hydroxyproline (Hydro)	Ne	Imino acid
Proline (Pro)	Ne	Imino acid

Key Points

1. Proteins can be found in structural components of the body and are needed for many metabolic functions.
2. The presence of Nitrogen makes protein unique.
3. More than 300 amino acids are identified. But only 20 amino acids are used to synthesize all proteins.
4. Three features of a typical amino acid include a carbon skeleton, a carboxyl group, and an amino group.
5. Acidic amino acids contain more carboxyl groups, and basic amino acids contain more amino groups. Neutral amino acids contain an equal number of carboxyl and amino groups.
6. Sulfur-containing amino acids are methionine and cysteine. Among these amino acids, methionine is essential because animals cannot synthesize it. Cysteine is not considered essential because if S is available, the body can make it.
7. Aromatic amino acids contain a ring structure.

8. Imino acids contain an imino instead of an amino group (e.g., proline).
9. Essential amino acids are those that cannot be synthesized by the animal body. There are 10 essential amino acids; cats need taurine, and chickens need glycine.
10. Amino acids are joined together by peptide bonds. A long chain of amino acids formed this way is called a polypeptide.
11. The primary structure of an amino acid is determined by the sequence of individual amino acids in the polypeptide chain. The amino acids in the polypeptide chain form disulfide bonds and hydrogen bonds, which twist the polypeptide chain into a specific coiled structure, such as a helix or a sheet. This is called a secondary structure. Proteins can be classified based on their shape; solubility in water, salt, acid, base, or alcohol; or according to the nature of prosthetic groups.

Review Questions

1. Name the linkage between two amino acids in a protein.
2. What are the essential amino acids? Why are they essential?
3. Compared to carbohydrates, why are proteins unique?
4. Which amino acid is essential to chickens but not humans? How about to cats?
5. Name one amino acid from the following groups: acidic, basic, aromatic, and sulfur-containing.
6. Differentiate between essential and nonessential amino acids.
7. Is proline an amino acid?
8. List the 10 essential amino acids for monogastric animals.
9. How many peptide bonds are there in a tripeptide?
10. Give an example of a globular, fibrous, and conjugated protein.

X. Proteins, Digestion and Absorption

This chapter discusses the process of digestion and absorption of proteins in monogastric and ruminant animals. The different enzymes involved in protein digestion and the mode of absorption of amino acids are also discussed.

New Terms

Aminopeptidase
Bypass proteins
Carboxypeptidase
Chymotrypsinogen
Endopeptidase
Enterokinase
Exopeptidase
Pepsin
Pepsinogen
Procarboxypeptidase
Trypsinogen
Trypsin
Urea

Chapter Objectives

- To introduce the sites of protein digestion or degradation in monogastric and ruminant animals
- To introduce different types of protein-digesting enzymes, their sites of release, and their mode of action
- To discuss the similarities and differences between monogastric and ruminant animals in protein digestion

Digestion is the process by which ingested feed is broken down physically and chemically to simple products for absorption from the digestive tract. In the case of proteins, it involves denaturing of proteins to expose the peptide bonds, followed by hydrolysis and release of free amino acids.

Protein digestion involves the denaturing of peptide bonds and the release of free amino acids.

Protein-Digesting Enzymes

Protein-digesting enzymes are either endopeptidase or exopeptidase. Endopeptidases break peptide bonds within the primary structure into smaller fragments. Exopeptidases cleave amino acids off the terminal end of the protein molecule. Carboxypeptidases remove an amino acid from the end with a free carboxyl group, and aminopeptidase act on the terminal amino acid with a free amino group.

Protein Digestion

Protein digestion begins in the stomach.

Gastrin, a hormone, initiates the breakdown of proteins in the stomach. The presence of food in the stomach leads to the secretion of pepsinogen by the chief cells of the gastric mucosa. Pepsinogen is activated to form pepsin (active form) through HCl produced by parietal cells of the gastric mucosa. Pepsin is an endopeptidase. In young animals, milk-coagulating rennin is secreted into the stomach for clot formation, which aids in transport into the small intestine.

Types of Protein-Digesting Enzymes

- Endopeptidase
- Exopeptidase
- Carboxypeptidase
- Aminopeptidase

Protein-Digesting Enzymes, Site of Production, and Active Forms

- Pepsin (Stomach)
- Enterokinase (Duodenum)
- Trypsinogen (Pancreas, inactive) to trypsin (small intestine)
- Chymotrypsinogen (Pancreas, inactive) to chymotrypsin (small intestine) by trypsin
- Procarboxypeptidase (Pancreas, inactive) to carboxypeptidase (chymotrypsin, small intestine) by trypsin

The next portion of digestion occurs in the small intestine, which plays a major role in protein digestion. The hormone secretin, in the duodenum, stimulates enzymatic secretions from the pancreas, which includes three inactive forms: trypsinogen, chymotrypsinogen, and procarboxypeptidase. Enterokinase, also secreted at the duodenum, converts trypsinogen into trypsin, which then converts chymotrypsinogen and procarboxypeptidase to their active forms—chymotrypsin and carboxypeptidase.

Trypsin plays a very crucial role in protein digestion in the small intestine.

Digestion is finished off by other enzymes including aminopeptidases and dipeptidases from mucosal membranes. The goal of this process is to bring polypeptides down to single free amino acids.

Just like carbohydrates and fats, absorption is facilitated by the villi within the small intestine into the bloodstream. Normal free proteins are transported via active transport, energy requiring, and use sodium as a kind of cotransported molecule. Whole proteins use a direct transport method that does not require energy. Free amino acids are the major form for absorption into the circulatory system. However, some di-, tri-, and oligopeptides are also absorbed. Specific carrier proteins based on the nature of the amino acid (e.g., neutral, basic, acid, large, small) are involved in amino acid

transport. The naturally occurring L-forms of amino acids are absorbed preferentially to D-forms. Some amino acids may compete with others for carrier proteins and transport. For example, arginine inhibits lysine transport and high concentrations of leucine increase the need for isoleucine. Some neutral amino acids inhibit basic amino acid transport.

The Fate of Amino Acids: Absorbed amino acids could be used for tissue protein, enzyme, and hormone synthesis and deamination or transamination, and the carbon skeleton can be used for energy. Undigested proteins in the hindgut are subjected to microbial fermentation leading to the production of ammonia and other polyamines.

Protein Digestion: Ruminants

Protein digestion in the ruminant animals can be divided into two phases: (1) digestion (degradation) in the reticulorumen and (2) digestion in the abomasum and small intestine. Therefore, in ruminant animals, dietary proteins are classified as rumen degradable and rumen undegradable proteins.

In ruminants, dietary proteins can be classified as degradable or undegradable proteins.

Like monogastric animals, the main goal for protein supplementation is to provide amino acids to the animal. However, in ruminants, proteins serve as a source of nitrogen for rumen microbes so they can make their own microbial protein from scratch. Microbes do not “care” where the nitrogen sources come from and can use nonprotein nitrogenous substances such as urea for microbial protein synthesis. Urea is 100% degradable in the rumen by microbial urease (can be toxic at higher levels).

Protein entering the rumen may be degraded by both bacteria and protozoa, which produce proteolytic enzymes. The rumen microbes provide proteases and peptidases to cleave peptide bonds in polypeptides to release the free amino acids from proteins. Several factors such as solubility and the physical structure of protein can affect rumen degradation. These rumen-degraded amino acids release NH_3 and the C skeleton by a process called deamination. Along with volatile fatty acids (from carbohydrates), rumen microbes synthesize their own microbial protein, which serves as a primary source of protein to the host ruminant animals.

Microbial protein is enough for maintenance and survival but not for high-producing animals. Ammonia absorbed from rumen is converted to urea and secreted into the blood as blood urea nitrogen (BUN). Urea can be filtered and recycled to the rumen via saliva or through the rumen wall. The concentration of BUN in ruminants reflects the efficiency of protein utilization.

Not all proteins are degraded in the rumen.

Proteins that are not degraded by rumen microbes are called escaped, “bypassed,” or “undegradable” (rumen undegradable protein, RUP), and have a low rumen degradation rates (e.g. proteins in corn).

RUP enters the abomasum and small intestine of the ruminant animal for digestion and absorption. Proteins reaching the small intestine could be RUP or those from microbial sources. The amino acid needs of the host animal are met by RUP and microbial proteins. Both ruminants and monogastrics require the essential amino acids in their diet, and

amino acids cannot be stored within the body, so a constant dietary supply is necessary. Some of the similarities and differences in monogastric and ruminant animals in protein digestion or degradation are shown in the table below.

Monogastrics	Differences (Ruminants)
Amino acid profile at small intestine reflects the diet	Amino acid profile at the small intestine is different from diet
No upgrading of low quality dietary protein	Up-grade low quality dietary protein
Protein quality not downgraded	Down-grade high quality dietary protein
Cannot use non protein nitrogen	Able to use non protein nitrogen (e.g. urea)
Constant supply of amino acids are required	Constant supply of amino acids are required

Research on “Bypass” Potential of Protein Supplements: Among the cereal grains, corn has the highest bypass potential. However, it should be noted that corn is deficient in essential amino acids such as lysine and methionine. Animal protein sources such as fish meal and meat meal have high bypass potential. Drying forages and heat treatment increases bypass potential. Feed processing methods, such as pelleting, steam rolling, or flaking, tend to denature the feed protein due to the generation of heat, thereby “protecting” the protein from lysis in the rumen. Rumen protected protein sources (through formaldehyde treatment) that remain intact in the rumen and dissolve in the abomasum are commercially available.

Key Points

1. Digestion of protein starts in the stomach with HCl. Acid denatures (unfolds) proteins.
2. Pepsinogen (inactive) is converted to pepsin (active form) by HCl. Pepsin cleaves proteins to form peptides.
3. The small intestine has several enzymes. Pancreas releases trypsinogen, chymotrypsinogen, and procarboxypeptidases.
4. Enterokinase secreted from duodenum converts trypsinogen to trypsin, which then converts chymotrypsinogen to chymotrypsin and procarboxypeptidases to carboxypeptidase.
5. Degradation by the pancreatic and small intestinal enzymes results in amino acids and di- and tripeptides.
6. Absorption by villi and microvilli occurs using carrier proteins and energy. Absorption is affected by the nature of amino acids. Some whole proteins and di- and tripeptides are also absorbed.
7. In ruminants, rumen microbes release enzymes (proteases and peptidases) that cleave peptide bonds and release amino acids.
8. The microbes then deaminate (remove amino group) the amino acid, releasing NH₃ and C skeleton.
9. Microbes use NH₃, C skeleton, and energy to synthesize their own amino acids.
10. Ruminant has no amino acid requirement. Instead, they have a nitrogen requirement. Ruminants break down dietary protein into ammonia and C skeleton through rumen microbes and synthesize their own microbial protein. Therefore, a portion of a ruminant’s protein requirement can be met with nonprotein nitrogen (NPN). Urea is an example of NPN. A readily available carbohydrate source to provide the C skeleton for protein synthesis is critical. Otherwise, the toxic ammonia builds up quickly in the rumen.

11. Proteins leaving the rumen are microbial proteins and those that escape rumen degradation (bypass proteins, proteins that are not extensively degraded in the rumen).
12. Feed processing can affect the bypass ability of proteins.

Review Questions

1. List the enzymes involved in protein digestion in the stomach and in the small intestine.
2. What animals can utilize nonprotein nitrogen (NPN) and why?
3. In monogastric animals, protein digestion starts in the ____.
 - a. Mouth
 - b. Stomach
 - c. Small intestine
 - d. Pancreas
4. The major digestive enzyme secreted by the stomach is ____.
 - a. Amylase
 - b. Lipase
 - c. Pepsin
 - d. Trypsin
5. Proteins that are not extensively degraded in the rumen are also called ____.
 - a. "Bypass proteins"
 - b. Rumen undegradable proteins
 - c. Rumen degradable proteins
 - d. Both a and b are correct
6. Trypsin is not responsible for activating the following proenzyme(s).
 - a. Enterokinase
 - b. Chymotrypsinogen
 - c. Procarboxypeptidase
 - d. All are true
7. What happens to amino acids in the rumen?

XI. Proteins, Metabolism

All proteins in the body are in a state of constant flux, the size of the amino acid pool depends on a balance between synthesis and degradation. In this chapter, amino acid metabolism involving protein and nonessential amino acid synthesis and disposal of toxic ammonia is discussed.

New Terms

Citrulline
Deamination
Deoxyribonucleic acid (DNA)
messenger RNA (mRNA)
Ornithine
Protein turnover
Ribosomes
Transamination
transfer RNA (tRNA)
Urea
Uric acid
Urea cycle

Chapter Objectives

- To introduce the fate of absorbed proteins and synthesis of nonessential amino acids
- To discuss the process of detoxification of ammonia produced through nitrogen metabolism

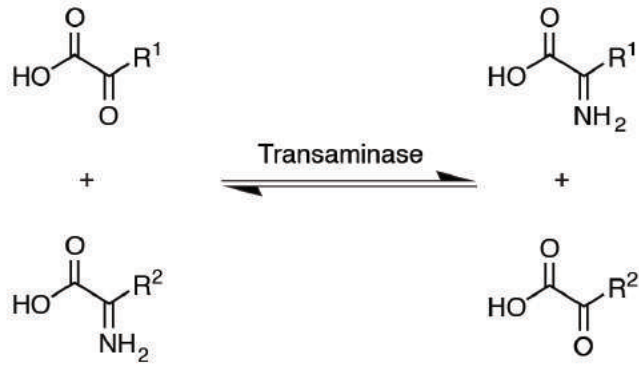
The Fate of Absorbed Proteins

Absorbed proteins are used for anabolic purposes such as synthesis of nonessential amino acids, tissue protein synthesis, enzyme or hormone synthesis, deamination, or transamination.

Amino acid synthesis and degradation are brought about by two reactions called transamination and deamination that occur in the liver.

Synthesis of Nonessential Amino Acids

The liver is the major site of amino acid metabolism. The liver has enzymes such as transaminases and is responsible for nonessential amino acid synthesis through a process called transamination. In this reaction, an amino group from one amino acid is transferred to an organic acid to form a new amino acid. Vitamin B6 (pyridoxine) is needed for transaminase activity.



Transamination also provides a link between protein and carbohydrate metabolism, where certain amino acids can use their C skeleton for glucose synthesis. Deamination is the removal of amino groups from amino acids to form ammonia. This process is needed for getting rid of nitrogen from the animal's body. After deamination or transamination, C skeletons are left and are used for making glucose, ketone bodies, or energy production.

Figure 11.1. Transamination reaction between an amino acid and an alpha-keto acid Source: Wikipedia

Transamination is a chemical reaction that transfers an amino group to a keto acid to form new amino acids.

All amino acids, except leucine and lysine, are glucogenic, meaning that they can use the C skeleton for glucose synthesis. Leucine and lysine are strictly ketogenic amino acids (forms ketone bodies) and can provide acetyl CoA as an energy source. Since both carbons of acetyl CoA are lost in the tricarboxylic acid (TCA) cycle, it cannot provide glucose. Some amino acids (isoleucine, phenylalanine, tyrosine, tryptophan) are both glucogenic and ketogenic.

The Fate of the Carbon Skeleton

- Oxidized for energy
- Used for glucose synthesis
- Used for ketone body formation
- Used for fat synthesis

All amino acids, except leucine and lysine, are glucogenic. Leucine and lysine are strictly ketogenic.

Some amino acids can be glucogenic or ketogenic.

Urea Cycle and Detoxification of Ammonia

The ammonia liberated from amino acid degradation is toxic to the central nervous and needs to be excreted or detoxified. Most mammals detoxify ammonia and excrete it as urea in the urine, while birds excrete it as uric acid (a white substance in the excreta). The detoxification of ammonia to form urea is brought about by the urea cycle through two tissues (liver and kidney; Figure 11.2).

Detoxification of ammonia to urea is through the urea cycle.

Two nonprotein amino acids (amino acids not used for protein synthesis) involved in the urea cycle are ornithine and citrulline. The first step in the urea cycle is the formation of carbamoyl phosphate through the condensation of ammonium ions with bicarbonate ions in the mitochondria of the liver.

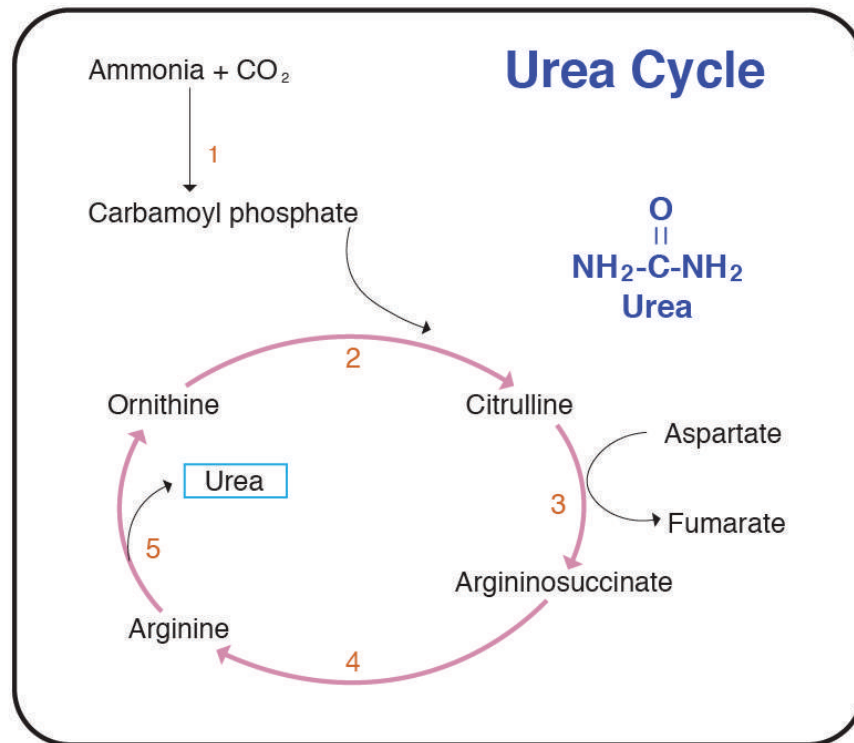


Figure 11.2. Urea cycle Source: <https://www.slideshare.net>

Ornithine reacts with a compound called carbamoyl phosphate and forms citrulline. Citrulline is easily permeable and gets into the cytosol and reacts with aspartate, forming argininosuccinate (2 ATP needed). The enzyme argininosuccinate lyase cleaves argininosuccinate into arginine and fumarate and fumarate enters the TCA cycle. Arginine is lysed into ornithine and splits urea off, producing ornithine, to start the cycle again. Hence arginine can be a nonessential amino acid but not available for protein synthesis.

The kidneys synthesize arginine from citrulline. The liver breaks down arginine into urea and ornithine.

Poultry cannot synthesize carbamoyl phosphate and hence they cannot make urea. Instead, glutamic acid, glycine, and methionine are used for uric acid synthesis. Hence poultry need high levels of methionine, arginine, and glycine in their diet for optimum production. Since the formation of urea through the urea cycle is ATP dependent, feeding animals poor quality or excess protein is energy demanding and can lead to environmental problems (e.g., air ammonia, groundwater pollution).

In the ruminant animals, rumen NH₃s are sent to the liver via the portal vein and undergo the urea cycle back to urea and enter the blood, where they are eventually secreted in the urine or brought back into the digestive tract as an N source for the rumen microbes.

Types of RNA

- Messenger RNA
- Transfer RNA
- Ribosomal RNA

Protein synthesis occurs in every tissue of the body. Protein synthesis lies ultimately in the genetic code. The details of protein synthesis are beyond the scope of the current chapter and are explained in other biochemistry books.

Briefly, nucleic acids such as deoxyribonucleic acid (DNA; molecule that stores genetic information) and ribonucleic acid (RNA) consists of nucleotides. DNA contains the genetic code of the animal and is the blueprint of protein synthesis. DNA controls the

formation of RNA (a template for transcription). There are three different types of RNA (ribosomal RNA, messenger RNA, and transfer RNA). All three are involved in protein synthesis. Ribosomal RNA is the part of the structure of ribosomes, which has three base codons that code for amino acids (site of protein formation). Transfer RNA picks up specific amino acids from cytoplasm and transports it to the ribosomes (workbench where proteins are made). Transfer RNA (tRNA) acts like an adapter during protein synthesis. Each tRNA carries a specific amino acid. Messenger RNA determines the sequence of amino acids (translation) in the protein formed. Both messenger RNA (mRNA) and tRNA are produced from the DNA template. The synthesis of each protein is controlled by a different mRNA. As the peptide chain is formed, an empty space cannot be formed, which limits the peptide chain formation and protein synthesis. All 20 amino acids are needed for protein synthesis. For example, lack of essential amino acid in the diet can stop peptide chain formation and protein synthesis and affect body weight gain and animal performance.

Protein synthesis needs nucleic acids such as DNA and RNA.

Protein turnover is a dynamic process involving continuous and simultaneous protein synthesis and protein degradation. The net rate of protein gain or loss is governed by the balance of synthesis and degenerative processes. Constant turnovers of proteins in the body and the loss of proteins, mainly in feces, are the basis for protein requirement. Even when an animal is not growing, it still has a protein requirement. The amount of protein needed in the diet depends on age, physiological (e.g., pregnancy, lactation) and pathological status, and quality of the protein supplied.

Key Points

1. The liver is the major site of amino acid metabolism.
2. Nonessential amino acids are synthesized through the process of transamination.
3. Degradation of amino acid involves two processes (deamination and transamination).
4. Deamination is the removal of amino groups from the C skeleton and the release of ammonia.
5. Toxic ammonia is disposed through the urea cycle for detoxification of NH_3 into urea (mammals) or uric acid (birds).
6. Two important functions of the urea cycle are detoxification of ammonia (liver) and provisioning of arginine (kidney) to form urea and ornithine.
7. Two nonprotein amino acids (ornithine and citrulline) are involved in the urea cycle.
8. The C skeleton then can be used for energy, glucose, other amino acids, or ketone body synthesis.
9. All amino acids except leucine and lysine are glucogenic.
10. Four amino acids are both ketogenic and glucogenic (e.g., tyrosine, tryptophan, isoleucine, phenylalanine).
11. Protein synthesis requires transcription and translation. The DNA template is transcribed into messenger RNA (mRNA), which in turn, serves as a template (translation) for protein synthesis.
12. Transfer RNA (tRNA) carries the individual amino acid to the ribosomes and links together to form a polypeptide chain. Twenty individual amino acids are required at the same time to complete this task.
13. There are constant turnovers of proteins in the body, and there is a loss of proteins in the body as well, mainly in feces.
14. Protein need depends on age, physiological status, and quality of the protein supplied in the diet.

Review Questions

1. What are the functions of ornithine and citrulline?
2. In what form is nitrogen excreted in (a) swine and (b) chickens?
3. Why do poultry have a greater need for arginine and methionine?
4. In the urea cycle, carbamoyl phosphate condenses with _____.
 - a. Citrulline and forms ornithine
 - b. Ornithine and forms citrulline
 - c. Arginine and forms citrulline
 - d. Ornithine and forms arginine
5. Proteins can be used for _____.
 - a. Gluconeogenesis

- b. Ketogenesis
 - c. Fat synthesis
 - d. All of the above
6. Two amino acids are strictly ketogenic. Which two?
7. Which of the following molecules is responsible for carrying the specific amino acids to the ribosome for protein synthesis?
- a. tRNA
 - b. mRNA
 - c. DNA
 - d. rRNA
8. Differentiate between deamination and transamination.

XII. Proteins and Amino Acids, Quality

The estimation of nutrient requirements for livestock and the determination of the extent to which different feedstuff sources supply essential nutrients are needed for efficient, economical, and sustainable feeding. In this context, estimation of protein requirements and the extent to which different protein sources supply essential and nonessential amino acids are researched in detail.

New Terms

Amino acid score
Antagonism
Bioavailability
Biological value
Digestibility
Ileal digestibility
Limiting amino acid
Net protein utilization
Protein efficiency ratio
Protein quality

Chapter Objectives

- To introduce and discuss the factors affecting the protein quality of animal feeds
- To introduce different methods for assessing protein digestibility
- To differentiate between fecal and ileal digestibility and its importance in ration formulation for monogastric animals

The term protein quality refers to the availability of amino acids that the protein supplies, and digestibility considers how the protein is best utilized. There are various methods that rank the quality of different types of protein, and this chapter describes some of the in vivo animal bioassays and in vitro laboratory analysis commonly used in animal nutrition.

Protein quality is a term used to describe how well a protein from feed matches animal requirements. With high-quality protein, less is needed in the diet.

Factors Affecting Protein Quality

- Amino acid profile
- Content and balance of essential and nonessential amino acids
- Content of limiting amino acids
- Protein digestibility and bioavailability

Several factors can affect protein quality. Animals do not have a protein requirement, rather they have an amino acid requirement. The basic function of dietary protein is to supply adequate amounts of required amino acids. Thus the quality of feed protein depends on the amino acid profile of the diet, the content of essential amino acids as well the content of limiting amino acids, and the digestibility and physiological utilization of amino acids after digestion. These factors are most important for nonruminants. Solubility and digestibility are also important for ruminant animals.

Limiting amino acids are the essential amino acids that interrupt protein synthesis due to its limited amount and the great demand for them.

The two most common limiting amino acids in animals fed corn- and soy-based commercial diets in the US are lysine and methionine.

Protein Digestibility and Bioavailability

What is meant by the digestibility of protein?

The digestibility of protein can be defined as the fraction of the protein ingested that is absorbed by the animal—that is, not excreted in feces. Digestibility can therefore be calculated by measuring dietary protein input and fecal output as shown in the following equation:

Digestibility of protein can be defined as the fraction of the protein ingested by the animal and not excreted in feces.

$$\text{amino acid digestibility (\%)} = \frac{\text{amino acid consumed} - \text{amino acid excreted}}{\text{amino acid consumed}} \times 100$$

Digestibility assays are a favored technique for measuring the availability of amino acids. Most common and published values on the digestibility of protein and amino acids in monogastric animals are based on fecal or excreta analysis because of its simplicity and sample size, as a large number of animals could be used without sacrificing the animals. Fecal-based digestibility measurements, however, suffer from the modifying and variable effects of the hindgut (microbes' dietary protein utilization and the contribution of microbial and endogenous proteins to amino acid excretion in feces contributing to fecal amino acid output).

Digestibility can be divided into fecal and ileal.

Fecal digestibility assays are done through fecal collection and analysis, while ileal digestibility assays are done through digesta collection at the ileal junction, either using cannulated animals or slaughter in small animals such as poultry.

The term digestibility is often used synonymously with bioavailability. However, this is not true. Bioavailability of amino acid can be defined as an amino acid in a form suitable for digestion, absorption, and utilization. Bioavailability can only be estimated with animal growth bioassays using live animals.

Bioavailability of amino acids is simply the rate at which amino acids are absorbed and become available for protein synthesis.

Several animal and feed factors can affect both digestibility and bioavailability. For example, the source of protein, such as animal versus plant protein, and feed processing methods can affect protein digestibility. Animal proteins are more balanced with essential amino acids and nonessential amino acids. Plant proteins, such as soybean meal, can have antinutritional factors (e.g., trypsin inhibitor) that can affect protein digestibility and availability of amino acids.

Feed processing methods such as heating, roasting, and extrusion can disrupt antinutritional factors like trypsin inhibitor in soybean meal. However, overheating (browning, or Maillard reaction) makes amino acids unavailable due to a complex reaction with sugars. In addition, other interrelationships such as antagonism, excesses of one amino acid interfering with the metabolism of another amino acid, making them unavailable (e.g., lysine and arginine) can precipitate amino acid imbalance.

Methods for Assessing Protein Quality

Protein quality can be assessed by bioassays using live animals or chemical assay.

Biological Value (BV): This is a measure of the proportion of absorbed protein from a feed that becomes incorporated into the proteins of the animal's body. It determines how readily the digested protein can be used in protein synthesis in the animal. BV assumes protein is the only source of nitrogen and measures the proportion of nitrogen absorbed by the body that is then excreted (fecal and urine). The remainder must have been incorporated into the proteins of the animal's body. A ratio of nitrogen incorporated into the body over nitrogen absorbed gives a measure of protein "usability" or BV. Biological value (BV) measures the percent of absorbed protein retained in the body.

$$BV = \frac{N \text{ intake} - N \text{ output (fecal N + urinary N)}}{N \text{ intake} - N \text{ fecal}} \times 100$$

Net Protein Utilization (NPU): As the name indicates, NPU determines the ratio of amino acid converted to proteins in the body (or retained in the body) to the ratio of amino acids supplied in the diet.

NPU measures percent of dietary protein retained in the body.

As a value, NPU can range from 0 to 1 (or 100), with a value of 1 (or 100) indicating 100% utilization of dietary nitrogen as protein and a value of 0 an indication that none of the nitrogen supplied was converted to protein. Egg protein has a high value of 1 (or 100)

$$NPU = \frac{\text{body N content with test protein} - \text{body N content with protein-free diet} \times 100}{N \text{ intake}}$$

It turns out that $NPU = \text{digestibility} \times BV$. Since NPU considers digestibility, it is a better estimate for protein quality.

Protein Efficiency Ratio (PER): The PER was the first method adopted for routine assessment of the protein quality of food. The PER is based on the weight gain of a test animal divided by its intake of a particular food protein during the test period. PER assumes that all protein is used for growth.

PER measures body weight gain per unit of protein consumed.

$$\text{PER} = \frac{\text{Body weight gain (g)}}{\text{Protein consumed (g)}} * 100$$

Both BV and PER = 0 when nongrowing animals are used.

Other chemical (in vitro) methods of assessing protein quality include the amino acid score. This is done through laboratory analysis of amino acid profiles using high-pressure liquid chromatography, and the results are compared to a standard (or reference) protein such as egg protein (albumen) and given a score. Although no animals are involved, these scores can give some easy and quick information. However, they do not give any information on palatability, digestibility, or availability.

$$\text{Amino acid score} = \frac{\text{mg of Amino Acid per g of test protein}}{\text{mg of Amino Acid per g of reference protein}}$$

Key Points

1. The nutritional quality of a protein is determined by digestibility, essential amino acids, and limiting amino acid composition for nonruminants. The two most limiting amino acids are lysine and methionine.
2. Solubility and digestibility are important factors for ruminants. Ruminants have no amino acid requirement. Instead, they have a nitrogen requirement.

3. Protein quality can be assessed by bioassays or chemical assays. Biological value (BV) measures percent of absorbed protein retained in the body. High BV of a protein means less is needed.
4. Net protein utilization (NPU) measures percent of dietary protein retained in the body. Since NPU considers digestibility, it is a better estimate for protein quality. Another bioassay used is the protein efficiency ratio (PER), which measures body weight gain per unit of protein consumed.
5. Amino acid score or chemical score compares the amount of individual essential amino acids to that of an ideal or reference protein. The amino acid that has the lowest score is assigned to the test protein. The chemical score is easy to conduct but does not consider digestibility or palatability in live animals.
6. Digestibility and bioavailability of amino acids can be affected by disproportionate amounts of amino acids in the diet (excess or shortage), protein source, feed processing, and antagonism. Symptoms for both deficiency and toxicity of individual amino acids can affect animal productivity. Amino acid antagonism such as lysine-arginine is common in poultry.

Review Questions

1. What is protein quality? List the factors that affect protein quality?
2. What is a limiting amino acid? What are the two most common limiting amino acids, and why are they limiting in animal diets?
3. Differentiate between digestibility and bioavailability.
4. What is protein quality? List two in vivo tests for protein quality.
5. Which bioassay method would you use to assess protein quality? Why?
6. What is an amino acid score?

XIII. Vitamins

This chapter provides an introduction and discussion of vitamins that are important in the nutrition of food-producing animals.

New Terms

Antioxidant
Carotenoid
Cholecalciferol
Fat-soluble vitamins
Osteomalacia
Quinones
Retinal
Retinol
Rickets
Tocopherol
Water-soluble vitamins

Chapter Objective

- To introduce and discuss different vitamins of importance in animal health, nutrition, and food quality

What Are Vitamins?

Vitamins are a group of chemically unrelated organic molecules that are needed in minute amounts for different physiological functions. The name “vitamin” originated from the term vital amine and refers to a group of compounds having specific roles in metabolism. Vitamins,

although organic compounds, do not provide energy like other macronutrients and are not used for the synthesis of structural compounds. However, they function as enzyme precursors, or coenzymes, in different metabolic processes.

Most vitamins need to be provided to the animal through diet, while some of vitamins can be synthesized by the rumen and hindgut microbes or by exposure to sunlight. Deficiency of vitamins in a diet leads to disease conditions, reduced productivity and animal welfare, and reduced immunity in food-producing animals. The dietary requirements of vitamins are very low. In recent years, megadoses of some vitamins (e.g., vitamin E) have been used in animal diets as a means to enhance animal immunity and to improve food quality aspects.

A general classification of vitamins is based on their solubility, as fat- or water-soluble vitamins. The fat-soluble vitamins are vitamin A, vitamin D, vitamin E, and vitamin K. The water-soluble vitamins include members of the B-complex group and vitamin C.

Vitamins

- Do not provide energy like macronutrients
- Function as catalysts in energy-producing reactions
- Make fats more fluid or decreases their melting point when there is an increased amount

Vitamins are classified into fat- and water-soluble vitamins.

Vitamin Classifications

1. Fat soluble (e.g., vitamin A, vitamin D, vitamin E, vitamin K)
2. Water soluble (e.g., B complex group and vitamin C)

Fat-soluble vitamins

- Vitamins A, D, E, K
- Associated with fat during digestion and absorption
- Storage in liver, adipose tissue, and excess storage can be toxic for some vitamins (e.g., A and D)
- No daily need
- Deficiency is very slow

Water-soluble vitamins

- Total nine, all B vitamins and vitamin C
- Soluble in water and excess excreted through urine
- No storage and less toxic
- Daily requirement (except vitamin B12)
- Serve as cofactor in biochemical reactions
- Deficiency is fast

Fat-Soluble Vitamins

Vitamin A

This vitamin was discovered by M. Mori in 1922 as a “fat-soluble factor” present in butter and fish oil, and he named it A. The general term vitamin A includes several related compounds called retinol (alcohol), retinal (aldehyde), and retinoic acid (acid form; figure 13.1). Of these three molecules, retinol is the biologically active form of vitamin A.

Retinol is the biologically active form of vitamin A.

Vitamin A is required in the diet of all animals. Vitamin A in the diet can be provided as a vitamin or through its precursor carotenoids present in plants. In animal feeding, most vitamin A is supplied by synthetic sources, which can be produced economically.

Carotenoids are the plant form of or the precursor of vitamin A (figure 13.1). Carotenoids are pigments present in plant cells (> 600 types) that provide the deep orange/yellow color of plant foods such as carrots, sweet potatoes, and pumpkins. There are two forms of carotenoids: carotenes and xanthophylls. Among these, carotenes (especially β -carotenes) have vitamin A activity. The other carotenoids present in plants (xanthophylls) do not have vitamin activity and are involved in providing color pigments. These types of carotenoids are increasingly used in diets for plumage color enrichment (e.g., exotic birds kept in captivity), egg yolk pigmentation, and aquaculture feeds and in the diets of ornamental fish.

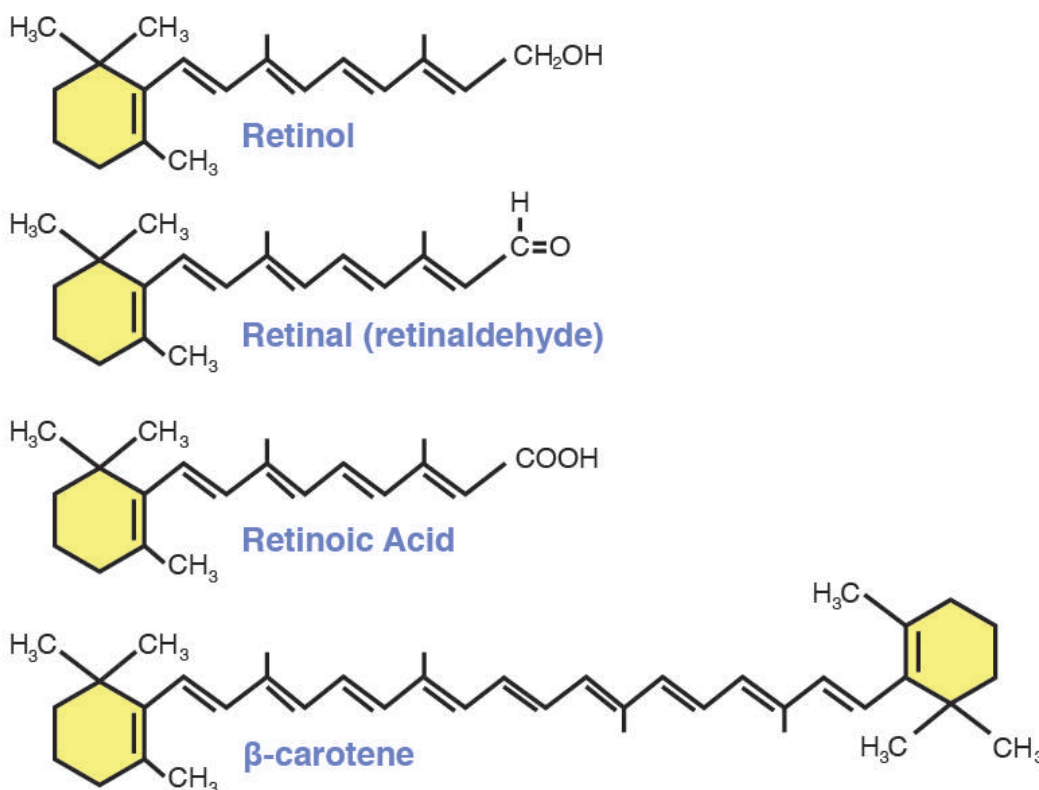


Figure 13.1. Different forms of vitamin A

Functions

In the body, vitamin A plays a role in several distinct functions, including vision, bone growth, reproduction, and maintenance of epithelial cells, which cover the body surface (e.g., skin) and mucous membranes of body cavities (e.g., respiratory, urogenital, digestive tract).

The role of vitamin A in night vision is well established. In the rods of the retina, retinal combines with a protein called opsin to form rhodopsin (also called visual purple). Rhodopsin is light sensitive and enables the eye to adapt to changes in light intensity. Upon exposure to light, rhodopsin splits into retinal and opsin. The energy that is released is

transmitted through the optic nerve leading to vision. However, lack of retinal leads to inefficient rhodopsin recycling making the rod cells insensitive to light changes, eventually leading to night blindness.

Vitamin A is needed for the proliferation and differentiation of cells. Vitamin A is also needed for mucoprotein production, which serves as a barrier and thus protects cells against bacterial invasions. Deficiency of vitamin A can lead to a failure in the differentiation of epithelial cells to mature, mucus-producing cells and to normal epithelial cells being replaced by dysfunctional, stratified, keratinized cells, increasing the susceptibility to infection. Xerophthalmia is a condition in humans and animals that is caused by vitamin A deficiency; it leads to dryness and irritation of the cornea and conjunctiva of the eye and results in cloudiness and infection.

Vitamin A is also needed for normal skeletal and tooth development and reproductive processes. Vitamin A's role in bone growth is related to its involvement in bone cell (osteoclast and osteoblast) division and maintenance of cell membranes. Vitamin A is also needed for reproductive functions such as spermatogenesis and estrus cycles.

Vitamin A and carotenoids can function as antioxidants thereby protecting cells from oxidative stress and are also involved in modulating cell-mediated and humoral immune responses in animals.

Metabolism

Vitamin A in the diet is digested and absorbed along with fat. In the diet, vitamin A is present as esters, is hydrolyzed by pancreatic lipase, and is incorporated into lipid micelles. Upon reaching the microvilli, they are transferred to mucosal cells, where they are reesterified and are incorporated into the chylomicrons and transported to the lymph for storage in the liver as retinyl esters. The hydrolyzed retinyl esters to free retinol and are complexed with retinol binding proteins and are transported through the blood to the needed tissues.

Carotenoids are split into two within the intestinal mucosal cells to form retinal and are reduced to form retinol. However, a wide variation exists among animals in the bioconversion of carotenoids to retinol. One IU of vitamin A = 0.6 µg of β-carotenes. Some animals, such as cats, cannot convert β-carotene to vitamin A due to the lack of the β-carotene splitting dioxygenase enzyme and need preformed vitamin A from animal sources.

Toxicity

As a fat-soluble vitamin, long-term consumption of vitamin A may lead to toxic symptoms. However, symptoms will vary with species, age, and physiological condition. Skeletal abnormalities and thickening of the skin are reported with hypervitaminosis.

Functions	Deficiency	Excess
Diverse functions such as production of vision pigments, resistance to infectious agents and maintenance of health in many epithelial cells	Impaired growth	Skeletal abnormalities
	Loss of epithelial integrity	Skin thickening
	Reproductive failure	Scaly dermatitis
		Swelling and crusting of eyelids

Vitamin D

Vitamin D includes a group sterol compound that regulates calcium and phosphorus metabolism in the body. Vitamin D is formed by the irradiation of sterols in plants and in the skin of animals and can be called a “sunshine” vitamin. The two major forms of vitamin D are ergocalciferol (vitamin D₂, activated plant form) and cholecalciferol (D₃, activated animal form; Figure 13.2).

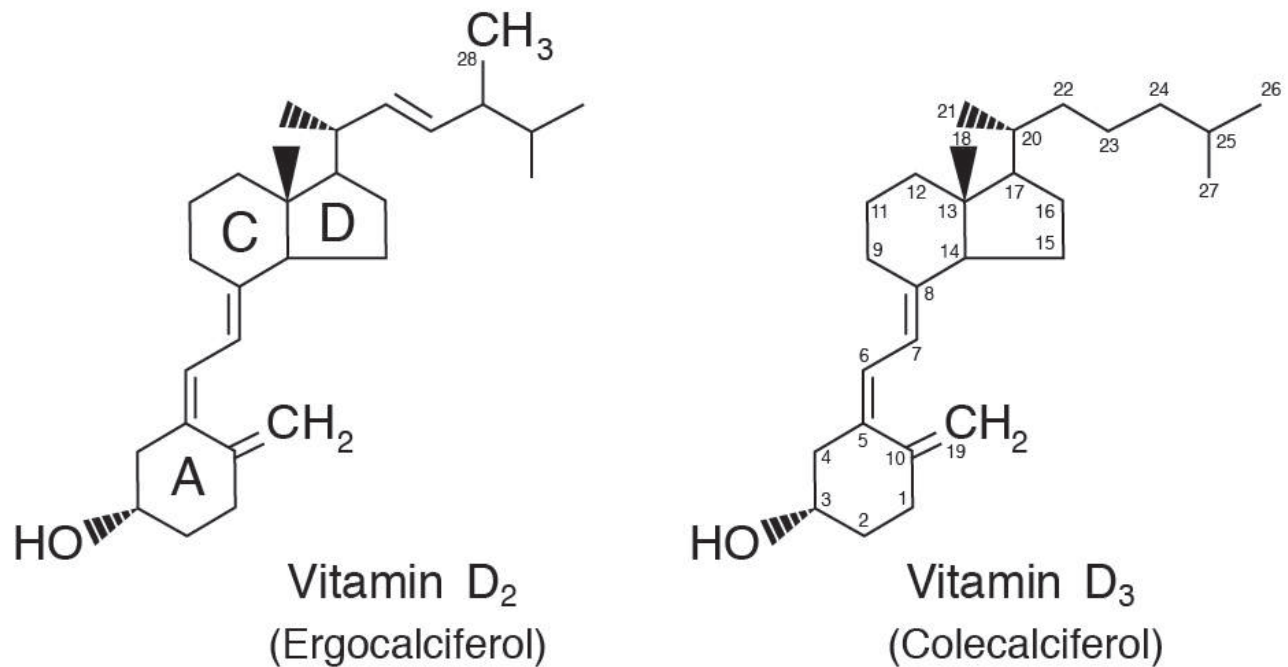


Figure 13.2. Plant and animal forms of vitamin D

Ergocalciferol (vitamin D₂) in plants is formed upon exposure to sunlight after harvest (or injury) and not in living plant cells. Sun-cured forages and hay are good sources of vitamin D in grazing ruminant animals. Animals kept in confinement, as in modern pig and poultry commercial operations, without exposure to sunlight will require vitamin D. The activated animal form of vitamin D₃ (cholecalciferol) is the form that is of importance in other omnivores and carnivores. In most animals, vitamin D₂ can be converted to vitamin D₃. The efficiency of conversion is very low in poultry.

Cholecalciferol (vitamin D₃) is the form of vitamin D that is of nutritional value to most animals.

In the body, vitamin D₃ is synthesized from cholesterol when it is converted to 7-dehydrocholesterol in the skin upon exposure to ultraviolet irradiation. To become active, it is transported from the skin to the liver, where it is hydroxylated to form 25-hydroxycholecalciferol. This compound is transported through the blood to the kidneys, where it is further

hydroxylated to form 1,25-hydroxycholecalciferol, also called calcitriol, which is the most metabolically active form of vitamin D.

Vitamin D is really a hormone. It can be synthesized in the animal body from cholesterol.

Functions

Because vitamin D is produced in the body and due to its regulatory functions in calcium and phosphorus homeostasis, it is also considered as a hormone. In addition to this, other parts of the body (gastrointestinal tract, kidneys, bones) and parathyroid hormones work in conjunction with vitamin D in blood calcium homeostasis and bone calcification. Normal blood calcium levels are achieved by adjusting the dietary absorption of calcium from the gastrointestinal tract and by the release of calcium from the bone. Calcium-binding proteins are needed for proper absorption of calcium and phosphorus from the gut. In the gastrointestinal tract, vitamin D stimulates the synthesis of calcium-binding proteins enabling calcium and phosphorus absorption from the diet. Under a condition of hypocalcemia (low blood Ca level), parathyroid hormones (PTH) stimulate Ca absorption (from gut) and resorption (from bone and kidney tubules) indirectly by stimulating the production of vitamin D.

Vitamin D works along with the gut, bones, and kidneys in maintaining blood Ca levels.

Vitamin D also affects normal bone growth and calcification by acting with PTH to mobilize Ca from bone and by causing an increase in P resorption in the kidneys. Altogether, vitamin D works along with the intestines, bones, and kidneys to maintain an optimal level of blood Ca and P that is needed for normal bone mineralization.

A deficiency of vitamin D leads to impaired bone mineralization and abnormal skeletal development and results in a condition called rickets in young animals and osteomalacia in growing animals. In each of these instances, inadequate bone calcification leads to lameness, crooked legs, and spontaneous fracture of long bones. Vitamin D deficiency can be prevented by exposure to sunlight for a few minutes, although skin pigmentation affects the amount of sunlight required. White-skinned animals require less sunlight than dark-skinned animals. Similarly, animals such as llamas have higher requirements due to the nature of their high-elevation habitat and exposure to solar radiation. Vitamin D is also used for treating milk fever (discussed in detail in chapter 15) in dairy cows.

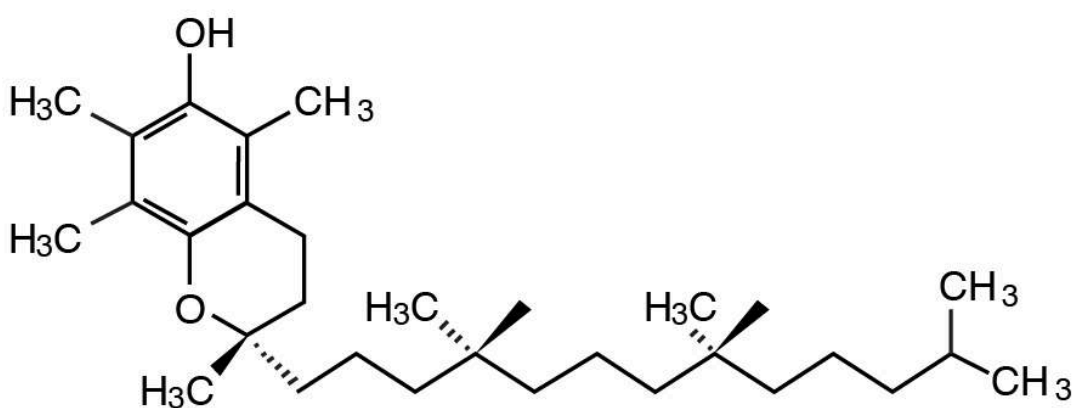
Among food sources, egg yolks and organ meats are good sources that could be used in pet animal feeding. Other concentrated sources include cod liver oil. In food animal feeding, vitamin D (cholecalciferol) is included as a Vitamin D supplement.

Functions	Deficiency	Excess
A steroid hormone.	Rickets (young animals)	Abnormal deposition of Ca in soft tissues (kidney, aorta, lungs).
Synthesized in the skin when exposed to sunlight	Osteomalacia (growing animals)	
Regulates blood Ca level		
Facilitate absorption of calcium from the intestine, and thereby assist in maintaining calcium homeostasis		

Vitamin E

Vitamin E is a term that is used to describe a group of chemically related compounds called tocopherols and tocotrienols. Among the different isomers, α -tocopherol is the most active biological form of vitamin E and is the one that is added to animal diets (Figure 13.3). Other isomers with less biological effects include β -, γ -, δ -tocopherol and α -, β -, γ -, δ tocotrienols. Most commercially available vitamin E is DL- α -tocopheryl acetate. One IU of vitamin E is defined as 1 mg of all-rac- α -tocopherol acetate. The only stereoisomer of α -tocopherol found in nature is RRR- α -tocopherol, which is the most biologically effective form of vitamin E in animals.

RRR- α -tocopherol is the most biologically effective form of vitamin E in animals.



Vitamin E (α -tocopherol)

Figure 13.3. Vitamin E structure

Functions

The function of vitamin E in the body is to serve as a biological chain-breaking antioxidant and to protect cells and tissues from oxidative damage induced by free radicals and other lipid oxidation products. Vitamin E prevents the oxidation of lipids by serving as a free radical scavenger and donates electrons from the hydroxyl group of the molecule (antioxidant effect; Figure 13.3). Lipid peroxidation causes damage to unsaturated lipids in cell membranes resulting in the disruption of the structural membrane and cell integrity.

In prepared feeds, the formation of such peroxidized compounds can cause a reduction in palatability, rancidity, and destruction of nutrients and can also affect animal health while reducing organoleptic and sensory quality of the food produced. In addition to lipids and oxidative stress, vitamin E can protect other nutrients such as proteins and vitamin A. Due to these roles, the level of vitamin E in a diet depends on the level of polyunsaturated fatty acids, degree of peroxidative damage, and other external stressors.

Vitamin E also has a sparing action on the mineral selenium, which is a cofactor for the enzyme glutathione peroxidase, which functions to reduce lipid peroxides. The inactivation of lipid peroxides protects the cell membrane from further damage. By preventing the oxidation of the cell membrane polyunsaturated fatty acids, vitamin E spares selenium.

Among food sources, plant oils and egg yolks (depending on hen diet) are good sources of vitamin E. Since vitamin E is highly prone to destruction, proper storage of prepared feed (away from heat and light) is necessary to prevent oxidative changes to fat and to maintain vitamin E levels.

Vitamin E is an antioxidant, and high levels of polyunsaturated fatty acids and vitamin A in the diet increase the requirement for vitamin E.

Deficiency

Vitamin E deficiency can produce white muscle disease, exudative diathesis, and encephalomalacia. White muscle disease is caused by the degeneration of skeletal and heart muscle fiber, which leads to rapid death due to heart failure. Exudative diathesis in chickens is caused by leaky capillaries in the breast muscle. Treatment with either vitamin E or selenium will be successful in both cases. However, encephalomalacia (crazy chick disease) can only respond to vitamin E treatment.

Toxicity

Vitamin E is the least toxic of the fat-soluble vitamins and high levels are added in the diets of animals (beef cow, poultry) to enhance food nutritional and aesthetic value and lipid stability.

Functions	Deficiency	Excess
Free radical scavenger	White muscle disease	Non toxic
Antioxidant function	Crazy chick disease	High levels are added in animal diets to enhance lipid stability in omega-3 fatty-acid rich foods, increase visual aspects in red meat, and to reduce heat stress and enhance immune function in poultry.
Affects immune response	Reduction in feed and food lipid quality and rancidity	
	Reproductive failure	

Vitamin K

Vitamin K includes a group of compounds called the quinones. Vitamin K1 is found in green plants (phylloquinones) and vitamin K2 (menaquinones) is synthesized by hindgut bacteria. Vitamin K's are absorbed readily with fat in the gastrointestinal (GI) tract. The liver converts vitamin K1 and K3 to K2 before it is used. The metabolically active form of vitamin K is menaquinones. Menadione (vitamin K3, synthetic form) is the most common version of vitamin K that is included in animal diets.

Functions

Vitamin K is needed for the synthesis of prothrombin, a blood-clotting protein. The blood-clotting process needs several proteins such as thromboplastin, prothrombin, fibrinogen, and fibrin. The enzymes needed for these processes are vitamin K dependent, and hence deficiency of vitamin K leads to failure in fibrin clot formation, hemorrhages, and/or prolonged bleeding time. Often, subcutaneous hemorrhages appear over the body surface, giving a blotchy, bluish appearance to the skin. This can also be of economic importance in food-producing animals leading to a reduction in carcass quality or condemnation.

Gastrointestinal bacterial can provide the needed vitamin K to most animals either through absorption from the hindgut or through coprophagy (eating feces). However, animal husbandry practices, such as confinement housing or raising animals in cages or pens with wire floors, or antibiotic therapy can limit the availability of vitamin K in the animal diet. Certain coccidiostats containing sulfa drugs can cause vitamin K deficiency as sulfa drugs are an antagonist of vitamin K. Mold growing on weather-damaged sweet clover hay or silage contains dicoumarol, which is very similar to vitamin K in structure. Dicoumarol is a competitive inhibitor of vitamin K. Animals that consume moldy sweet clover hay or silage develop a vitamin K deficiency, leading to internal hemorrhaging and death in calves. Another antagonist of vitamin K is Warfarin, a rat poison causing anticoagulation. It is also a competitive inhibitor of vitamin K. Vitamin K is routinely administered in rodenticide poisoning in pets because the active ingredient (Warfarin) in these rodenticides are anticoagulants, causing bleeding and hemorrhaging.

Vitamin K is needed for the blood-clotting process in the animal body.

Phylloquinone and menaquinone derivatives are nontoxic even at higher levels. However, menadione given in prolonged high doses produces anemia and other abnormalities in animals.

Function	Deficiency	Excess
Serves as cofactor in carboxylation reactions of the activation of proteins necessary for blood clotting.	Vitamin K deficiency leads to prolonged blood clotting time and hemorrhaging. Deficiency is rare because vitamin K is synthesized by microbes in the hindgut. Antibiotics and other vitamin K inhibitors in the diet can cause deficiency	Non toxic

Key Points

1. Fat-soluble vitamins include vitamins A, D, E, and K.
2. They are digested and absorbed along with dietary fat and can be found in the micelle in the gastrointestinal (GI) tract after digestion, and once absorbed into the body, they are transported by chylomicron to various tissues. They are stored in the liver and adipose tissue.
3. Vitamin A was discovered by M. Mori in 1922 as a fat-soluble factor.
4. Retinol is the chemical name of vitamin A. Retinal and retinoic acid are related compounds. β -carotene, a plant pigment, is the precursor of vitamin A. Vitamin A has two distinct functions: participation in the visual purple cycle in the retina and maintenance of epithelial cells. The former requires retinal, and the latter requires retinoic acid.
5. β -carotene can split into two units of retinol in the intestinal mucosa cells; however, this conversion is not efficient. Some animals, such as cats, cannot absorb β -carotene and process it in the liver. Vitamin A activity depends on its double bonds, which are quite labile. An antioxidant is required to protect vitamin A.
6. Night blindness, dryness of eyes, diarrhea, kidney stones, and abortion are some typical vitamin A deficiency symptoms. Excessive vitamin A intake will eventually lead to toxicity. Symptoms include dermatitis, skin thickening, and weight loss. Each international unit represents 0.3 μg retinol. The requirement is a 2,000 IU/kg diet.
7. Vitamin D is really a hormone. It can be synthesized in the animal body from cholesterol. The whole process involves three different tissues: skin, liver, and kidney; UV light is required.
8. Vitamin D, a fat-soluble factor in cod liver oil, is known to cure rickets in humans. Its function is to modulate calcium metabolism in the animal body. Vitamin D increases blood Ca^{++} concentration by increasing Ca^{++} absorption from the GI tract and resorption from bone.
9. Vitamin D deficiency leads to calcium deficiency. Therefore, deficiency symptoms are the same as those of calcium deficiency, such as abnormal skeletal development.
10. Overdose of vitamin D will cause toxicity, which can lead to calcification of soft tissues such as the lungs, aorta, and heart.
11. Vitamin E comprises a group of compounds called tocopherols and tocotrienols.
12. Tocopherols contain a group of compounds with varying amounts of vitamin E activity; α -tocopherol is the most active one.
13. Vitamin E has two functions: avoiding oxidation and promoting normal reproduction in rats. All cell membranes are made of lipid bilayers containing many polyunsaturated fatty acids (PUFAs). Vitamin E serves as an antioxidant to protect the integrity of cell membranes.
14. Vitamin E deficiency problems include white muscle disease, exudative diathesis, and encephalomalacia. All these problems occur in young, growing animals. White muscle disease is caused by degeneration of skeletal and heart muscle fiber, which leads to rapid death due to heart failure. Exudative diathesis in chickens is caused by leaky capillaries in the breast muscle. Treatment with either vitamin E or selenium will be successful in both cases. However, encephalomalacia (crazy chick disease) can only respond to vitamin E treatment.
15. High levels of PUFA and vitamin A in a diet increase the requirement for vitamin E. It turns out that vitamin E prevents the formation of lipid peroxide from PUFAs, and Se, a cofactor in the enzyme of glutathione

peroxidase, is required to remove lipid peroxide.

16. Phylloquinone (K1), menaquinone (K2), and menadione (K3) are the three forms of vitamin K. The only known function of vitamin K is as a cofactor in carboxylation reactions of the activation proteins necessary for blood clotting.
17. Vitamin K is absorbed readily with fat in the GI tract. The liver converts vitamin K1 and K3 to K2 before it is used. Mold growing on sweet clover contains dicoumarol, which is very similar to vitamin K in structure. Animals that consume moldy sweet clovers develop a vitamin K deficiency. Dicoumarol is a competitive inhibitor of vitamin K.
18. Another antagonist to vitamin K is Warfarin, a rat poison. It is also a competitive inhibitor of vitamin K. Vitamin K deficiency leads to prolonged blood-clotting time and hemorrhaging. Deficiency is rare because vitamin K is synthesized by microbes in the hindgut. No toxicity is observed. The requirement of vitamin K (menadione) is a 0.5 mg/kg diet.

Review Questions

1. Why can an animal grow well with little vitamin A in its diet but needs to have vitamin B in its diet everyday?
2. Name one major deficiency problem resulting from a dietary lack of vitamin A and vitamin K.
3. Cows eating sun-cured hay or forage will get this form of vitamin D.
 - a. Ergocalciferol
 - b. Cholecalciferol
 - c. 7-dehydrocholesterol
 - d. Cholesterol
4. Exposing your lovebirds to sunshine will help in getting this vitamin.
 - a. Vitamin A
 - b. Vitamin D
 - c. Vitamin B
 - d. Vitamin C
5. What are the functions of vitamin E?
6. What is coprophagy? Why is it important to animals in terms of vitamin K availability?
7. Why is moldy sweet clover toxic to animals?
8. Vitamin D is _____.
 - a. An organic compound
 - b. A hormone
 - c. A cholesterol-derived compound
 - d. All of the above
9. Drinking milk will give this form of vitamin D.
 - a. Ergocalciferol
 - b. Cholecalciferol

- c. 7-dehydrocholesterol
- d. Cholesterol

10. The back fat of pigs will have these vitamins in stored form

- a. Vitamin A
- b. Vitamin D
- c. Vitamin B
- d. Both a and b

XIV. Water-Soluble Vitamins (B and C)

This chapter provides an introduction and discussion of water-soluble vitamins that are important in the nutrition of food-producing animals.

New Terms

Biotin
Choline
Cobalamin
Folic acid
Niacin
Pyridoxine
Pantothenic acid
Riboflavin
Thiamine
Vitamin C

Chapter Objective

- To introduce and discuss different water-soluble vitamins of importance in animal growth, health, and nutrition

What Are B Vitamins?

The B vitamins (also called B complex vitamins) are originally grouped together because of their similar metabolic functions. The nine chemically unrelated organic molecules function as metabolic catalysts (coenzymes) for energy metabolism pathways, for cellular maintenance, or for blood cell formation in the animal body. A list of water-soluble vitamins, coenzymes, and functions are shown in table 14.1. The B vitamins discussed in this section include thiamine, riboflavin, niacin, pyridoxine, pantothenic acid, biotin, folic acid, cobalamin, and choline. In ruminant animals and in herbivores, microbial synthesis meets the requirements, while in monogastric animals, such as pigs and poultry, daily supplementation is essential. B complex vitamins are also prone to loss during feed processing.

Water-Soluble Vitamins

B vitamins are water soluble and are needed in the daily diet of monogastric animals.

Thiamine

Thiamine consists of one molecule of pyrimidine joined with one of thiazole. Thiamine is also referred to as vitamin B1, as it is the first vitamin identified. Thiamine is a component of the enzyme thiamin pyrophosphate (TPP), which is involved in several key reactions in energy-producing pathways.

Thiamine functions as a coenzyme in enzymatic decarboxylation of pyruvate.

Dietary thiamine is converted into TPP inside cells to participate in the energy-producing pathway. Oxidation decarboxylation reactions, such as pyruvate dehydrogenase and α -ketoglutarate dehydrogenase, require TPP. It also is used in transketolase reactions for nicotinamide adenine dinucleotide phosphate (NADPH) production in the pentose cycle. Due to its role in carbohydrate metabolism, an animals' thiamine requirement is influenced by the level of

carbohydrates in their diet. Cereal grains are good sources of thiamine. However, since thiamine is heat labile, feed processing can destroy thiamine content. Thiamin requirement is linked to the energy content of the diet (0.5 mg/1,000 kcal diet)

Deficiency: Thiamin also plays a specific role in neurophysiology because a typical thiamin deficiency is beriberi, a dysfunction in the nervous system. Polyneuritis is another typical symptom of thiamin deficiency in chicks. Several compounds resemble thiamine in chemical structure and can function as antagonists, causing a thiamine deficiency. Raw fish and bracken ferns (a perennial) contain an enzyme, thiaminase, which destroys thiamin, causing a deficiency that causes a neurological disorder called Chastek paralysis, named after a farmer who observed similar condition in silver foxes. Heat treatment denatures thiaminase and prevents the problem. Amprolium (coccidiostat) blocks activation of TPP and can cause a thiamine deficiency.

B Vitamins as Coenzymes in Metabolic Functions

- Thiamine
- Riboflavin
- Niacin
- Pyridoxine
- Pantothenic acid
- Biotin
- Riboflavin

Riboflavin

Riboflavin is named for its yellow color (flavin) and sugar (ribose). Riboflavin (vitamin B2) is relatively heat stable but easily destroyed by light. Riboflavin functions in the body as a component of two different coenzymes: flavin mononucleotide (FMN) and flavin adenine dinucleotide (FAD). Both of these enzymes are involved in dehydrogenation/oxidation reactions that function in the release of energy from carbohydrates, fats, and proteins (the tricarboxylic acid [TCA] cycle, oxidation, electron transport chain).

Deficiency: As with most B vitamins, deficiency leads to a reduction in growth in young animals. Diets low in riboflavin can cause lesions in the corners of the mouth and anorexia and can cause loss of hair and diarrhea in young animals.

Niacin

Niacin is the accepted generic description for pyridine 3-carboxylic acid and its derivatives showing the nutritional activity of nicotinic acid. Niacin functions as a constituent of two important coenzymes nicotinamide adenine dinucleotide (NAD) and NADPH. These coenzymes serve as hydrogen carriers in several important metabolic processes involving carbohydrate metabolism (glycolysis) and other energy deriving pathways involving carbohydrates, fats, and proteins, such as the TCA cycle and oxidative phosphorylation.

In animal diets, niacin present in cereal grains is in a bound form and is not biologically available to the animal. For example, one of the bound forms of niacin in wheat is called niacytin and is not biologically available. Corn contains niacinogen, which binds niacin tightly and makes it unavailable for absorption. Niacin from animal sources is highly available.

In addition to dietary sources, most animals (except cats) are capable of synthesizing niacin from the essential amino

acid tryptophan. As a result, levels of tryptophan can affect niacin requirements. However, feeds low in niacin are usually also usually low in tryptophan. Cats should receive the entire supply of niacin from their diet.

Deficiency: This causes a condition called black tongue disease in dogs, and in chickens, it causes poor feathering around the eyes, also called spectacled eyes. The requirement is a 10–90 mg/kg diet. Pellagra (thick skin, dermatitis) is a typical deficiency symptom of niacin in humans associated with poor diet (high grain, no meat) and poverty.

Pyridoxine

Pyridoxine comprises three different forms: pyridoxine (plant), pyridoxal (animal), and pyridoxamine (animal). Pyridoxal, which is a component of the coenzyme pyridoxal 5-phosphate is the biologically active form. Pyridoxal 5-phosphate participates in a wide variety of biochemical reactions, most of them involving amino acid metabolism, such as transamination, deamination reactions, and decarboxylation reactions. Pyridoxal phosphate is also required for the synthesis of hemoglobin and the conversion of tryptophan to niacin. Vitamin B6 deficiency can precipitate niacin deficiency. Therefore, deficiency symptoms are similar for these two vitamins.

Deficiency: Symptoms include convulsions and reduced immune response. The requirement is a 1–3 mg/kg diet and is linked to the protein level in the diet.

Pantothenic Acid

Pantothenic acid occurs in all tissues of the body. The vitamin name is derived from the Greek term pan meaning “all,” or “everywhere.” Pantothenic acid was identified as a constituent of coenzyme A, the coenzyme required for acetylation of numerous compounds in energy metabolism. CoA is required in the formation of two-C fragments from fats, amino acids, and carbohydrates for entry into the citric acid cycle and for the synthesis of steroids. Deficiency of this vitamin is extremely rare, and in extreme cases, in addition to the reduced growth rate, in pigs, it leads to a condition called goose-stepping, an abnormal gait, due to nerve degeneration. Other signs of deficiency include a rough coat, anorexia, and impaired productivity. The Ca salt is the most common form in which the vitamin is added to diets.

Biotin

Feeding raw egg whites to rats causes skin lesions and loss of hair and were cured by a protective factor found in the liver. The original name given to this compound was vitamin H because it protected haut, a German word for “skin.” Biotin was isolated from egg yolk in 1936, a growth factor for yeast.

Biotin is a prosthetic group that binds to the lysine of the enzyme via a peptide bond to form biocytin, which serves as a cofactor in carboxylase reactions such as acetyl CoA carboxylase carboxylase (the first step in lipogenesis) and pyruvate carboxylase (the first step in gluconeogenesis). These important metabolic pathways make biotin very important in lipid

and carbohydrate metabolism. Biotin acts as a carbon dioxide carrier (carbon fixation) in reactions in which carbon chains are lengthened.

Deficiency: Biotin deficiency is rare. It causes dermatitis and hair loss. This is usually caused not by lack of biotin in the diet but instead, the antivitamin avidin binds biotin and makes it unavailable for digestion and absorption. Eggs are a rich source of biotin. But egg whites contain avidin. However, cooking denatures avidin, making the biotin available for absorption. Deficiency symptoms may be found in swine kept in pens with slotted floors with limited or no access to fecal matter as hindgut bacteria produce biotin. Lack of biotin has been shown to cause a condition called footpad dermatitis in chickens fed wheat-based diets. Requirements of biotin are a 0.1–0.3 mg/kg diet (dry basis). Animals subjected to antibiotic therapy that causes a decrease in bacterial population may need an extra supply of biotin.

Folic Acid

Folacin is a generic term used to describe folic acid and related compounds. The active form of folacin in the body is called tetrahydrofolic acid. Dietary sources of folacin are converted mainly in the liver to tetrahydrofolic acid. Vitamin B12 enhances the conversion of folacin to tetrahydrofolic acid. The function of tetrahydrofolic acid is as a transport vehicle for single carbon units. Tetrahydrofolic acid is required for purine, pyrimidine, glycine, serine, and creatine synthesis. Both purine and pyrimidine synthesis is required for DNA synthesis and thus cell replication.

Deficiency: Lack of folic acid leads to less DNA and cell multiplication and affects all mitotically active cells. These include hematopoietic cells and all epithelial cells. Since rapidly dividing cells are most affected, it causes a condition called megaloblastic anemia. Folic acid deficiency is the most prominent human vitamin deficiency. Up to one-third of all pregnant women in the world may experience a folic acid deficiency during pregnancy. Folic acid and vitamin B12 have a close relationship—vitamin B12 deficiency will precipitate folic acid deficiency. The inclusion of antimicrobials increases the possibility of folate deficiency. Requirements of folic acid are a 0.25 mg/kg diet.

B Vitamins in Cell Maintenance and Blood Cell Formation

- Folacin
- Cobalamin
- Choline
- Cobalamin (Vitamin B12)

Cobalamin (Vitamin B12), the last B vitamin, was discovered in 1948. Cyanocobalamin is the vitamin and deoxyadenosyl cobalamin is the coenzyme form. Vitamin B12 is unique in that it has a trace element mineral (cobalt) as its active site. It is also the only vitamin that is synthesized only by microorganisms. Similar to folic acid, cobalamin is involved in the transfer of single carbon units during various biochemical reactions. Folic acid serves as a coenzyme for several enzyme systems involving methyl transfer in fat and carbohydrate metabolism and for myelin synthesis. Cobalamin is required for the oxidation of propionic acid in ruminant animals.

The stomach plays an important role in the absorption of vitamin B12. The stomach provides the acidity and pepsin to release the tightly bound vitamin B12 from the dietary source. The stomach also secretes an intrinsic factor, a specific binding glycoprotein. The vitamin B12–intrinsic factor complex travels to the ileum and is absorbed into a portal vein. Calcium is required for B12 absorption in the ileum. The absence of glycoprotein can lead to vitamin B12 deficiency. Deficiency symptoms are very similar to folic acid deficiency. The requirement is extremely low: 5–50 µg/kg diet for nonruminants. Cobalt is required only for ruminants; the rumen microbes will synthesize cobalamin.

Deficiency: This is very similar to folic acid deficiency, causing anemia and neural disorders. Lack of vitamin B12 or folacin interferes with the absorption of nutrients. Changes in the epithelial cells of the intestine, along with shortened villi, are observed. In livestock species, loss of appetite and reduced growth are observed. In ruminants, rumen microbes can synthesize all B vitamins; therefore, there is no requirement. However, they must have cobalt to synthesize vitamin B12. Requirements of cobalamin are 5–50 µg/kg diet (nonruminants). In ruminants, only cobalt is needed.

Vitamin C

It was discovered in 1747 that scurvy can be prevented by the ingestion of lemon juice. Ascorbic acid (Vitamin C) was recognized as a vitamin in 1933. Ascorbic acid has a structure closely related to monosaccharide sugars. It is synthesized from glucose by plants and most animal species. No coenzyme form is identified. Ascorbic acid is required for hydroxylation reactions of the amino acids proline and lysine in the formation of collagen, elastin synthesis, and neurotransmitter (norepinephrine, epinephrine) synthesis. Collagen is important for normal bone formation. It also functions as an antioxidant, reducing oxidative stress. Ascorbic acid can be synthesized from glucose by all mammals except primates and guinea pigs. Therefore, there is no requirement for livestock species. Proline and OH-proline are required for collagen synthesis.

Deficiency: This results in scurvy, a disease affecting humans with impaired wound healing, capillary bleeding, faulty bone formation, and anemia; it was first reported in sailors at sea. Normally, no deficiency symptom can be detected in all mammals except primates and guinea pigs. No daily requirement is established for livestock animals.

Table 14.1. Water-soluble vitamins, coenzymes/cofactors, and functions

Vitamin	Coenzyme/cofactor	Function
Thiamin	Thiamin pyrophosphate	Coenzyme in energy-producing pathways (pyruvate dehydrogenase, α -ketoglutarate dehydrogenase)
Riboflavin	FAD/FMN	Coenzyme in energy-producing pathways (TCA, β -oxidation, electron transport chain)
Niacin	NAD/NADP	Coenzyme in energy-producing pathways (Glycolysis, TCA, β -oxidation, electron transport chain)
B6	Pyridoxal phosphate	Coenzyme in protein (amino acid) metabolism (transamination reactions)
Pantothenic acid	Coenzyme A / acyl carrier protein	Coenzyme in energy-producing pathways (pyruvate dehydrogenase, α -ketoglutarate dehydrogenase)
Biotin	Biocytin	Cofactor in carboxylase reactions (lipogenesis, gluconeogenesis)
Folic acid	Tetrahydrofolic acid	Transporter for carbon units, involved in cell replication (DNA synthesis)
B12	Deoxyadenosyl cobalamin	Involved closely with folic acid
C	Not identified	Involved in collagen and neurotransmitter synthesis and acts as an antioxidant

Key Points

1. Water-soluble vitamins include nine B vitamins and vitamin C. All function as cofactors in biochemical reactions. They are either a coenzyme when bound to the enzyme with noncovalent bonding or a prosthetic group when a covalent bond is involved (biotin is an example).
2. Thiamin (vitamin B1) is converted into thiamin pyrophosphate (TPP) inside cells to participate in the energy-producing pathway. Oxidation decarboxylation reactions, such as pyruvate dehydrogenase and α -ketoglutarate dehydrogenase, require TPP. It also is used in transketolase reactions for nicotinamide adenine dinucleotide phosphate (NADPH) production in the pentose cycle. Raw fish and bracken ferns contain an enzyme, thiaminase, which destroys thiamin causing deficiency. Heat treatment denatures thiaminase and prevents the problem.
3. Riboflavin (vitamin B2) functions as a coenzyme for FMN and FAD in energy metabolism pathways. There is no storage of riboflavin or any other B vitamins with the exception of B12. Reduced growth and skin lesions are common deficiency symptoms. The requirement is 1–4 mg/kg diet.
4. Niacin consists of two different compounds: nicotinic acid and nicotinamide. It is chemically the simplest vitamin. Coenzyme forms for niacin are NAD and NADP. They are involved in hydrogen transfer.
5. Niacin can be synthesized from tryptophan (limited conversion in cats). However, feeds low in niacin are usually also low in tryptophan. Corn contains niacinogen, which binds niacin tightly and makes it unavailable for absorption. Deficiency in dogs leads to black tongue disease; in chickens, it leads to spectacled eyes. Pellagra is a typical deficiency symptom of niacin. It is associated with poor diet and poverty. The requirement is 10–90 mg/kg diet.
6. Pyridoxine (Vitamin B6) is one of several vitamins still maintaining its original name. It contains three different forms: pyridoxine (plant), pyridoxal (animal), and pyridoxamine (animal). The coenzyme form is pyridoxal phosphate (PALP). PALP participates in a wide variety of biochemical reactions, most of them involving amino acid metabolism and heme synthesis.
7. The synthesis of tryptophan from niacin requires B6. Vitamin B6 deficiency can precipitate niacin deficiency. Therefore, deficiency symptoms are similar for these two vitamins. Deficiency symptoms include convulsions and reduced immune response. The requirement is 1–3 mg/kg diet and is linked to the protein level in the diet.
8. Pantothenic acid was identified as a constituent of coenzyme A in 1950. It also functions as a component in acyl carrier protein. It is important in both energy-producing pathways and fatty acid metabolism. The vitamin itself does not have the active site. The requirements of pantothenic acid are 5–15 mg/kg diet.
9. Toxic effects of feeding raw egg whites to animals were first observed in 1916. Later, biotin was identified as the missing vitamin that caused the problem. Biotin is a prosthetic group that binds to the lysine of the enzyme via a peptide bond. It serves as a cofactor in carboxylase reactions such as acetyl CoA carboxylase and pyruvate carboxylase.
10. Biotin deficiency causes dermatitis and hair loss. This is usually not caused by a lack of biotin in a diet. Instead, the antivitamin avidin binds biotin and makes it unavailable for digestion and absorption. Raw egg whites contain a lot of avidin. The requirement is very low, 0.1–0.3 mg/kg diet.
11. Folic acid is the most prominent human vitamin deficiency. The active form is called tetrahydrofolic acid (THFA). The function of THFA is as a transport vehicle for single carbon units. This is important for purine synthesis and thymidylate synthesis, both are required for DNA synthesis and thus cell replication.
12. Folic acid and vitamin B12 have a close relationship—vitamin B12 deficiency will precipitate folic acid deficiency. Folic acid deficiency will affect all mitotically active cells. These include hematopoietic cells and

all epithelial cells. It is a potent antimicrobial in feeds. The requirement is small, 0.25 mg/kg diet.

13. Vitamin B12, the last B vitamin, was discovered in 1948. Cyanocobalamin is the vitamin and deoxyadenosyl cobalamin is the coenzyme form. Cobalt is the active site. It serves as a coenzyme for several enzyme systems involving methyl transfer.
14. The stomach plays an important role in the absorption of B12. It provides the acidity and pepsin to release B12 from the dietary source. The stomach also secretes an intrinsic factor (IF), a specific binding protein for B12. Calcium is required for B12 absorption in the ileum. Deficiency symptoms are very similar to folic acid deficiency. The requirement is extremely low, 5–50 µg/kg diet for nonruminants. Cobalt is required only for ruminants.
15. It was discovered in 1747 that scurvy can be prevented by the ingestion of lemon juice. Ascorbic acid (Vitamin C) was recognized as a vitamin in 1933. No coenzyme form is identified. It is required for hydroxylation reactions in collagen synthesis and neurotransmitter synthesis. It is also functioning as an antioxidant. Ascorbic acid can be synthesized from glucose by all mammals except primates and guinea pigs. Therefore, there is no requirement for livestock species.

Review Questions

1. The first vitamin that was identified due to a disorder in humans, causing beriberi, is _____.
 - a. Pantothenic acid
 - b. Thiamin
 - c. Riboflavin
 - d. Niacin
2. Which amino acid can form the vitamin niacin?
 - a. Threonine
 - b. Methionine
 - c. Tryptophan
 - d. Tyrosine
3. What vitamins are involved in the transfer of carbon units and cell replication (DNA synthesis)?
4. Dogs, mink, foxes, or cats fed raw fish can become deficient in this B vitamin.
 - a. Thiamin
 - b. Riboflavin
 - c. Biotin
 - d. Niacin
5. The vitamin that functions as a coenzyme in protein and N metabolism is _____.
 - a. Vitamin E
 - b. Pyridoxine (vitamin B6)
 - c. Vitamin C
 - d. Vitamin K

6. Which vitamin is involved in collagen and neurotransmitter synthesis and is also an antioxidant?
7. A lack of cobalt in the diet of a ruminant animal will lead to a deficiency of this vitamin.
 - a. Vitamin K
 - b. Vitamin B12
 - c. Vitamin B6
 - d. Niacin
8. What are the coenzymes/cofactors for riboflavin, niacin, and pantothenic acid?
9. Describe the role of the stomach in the absorption of vitamin B12.
10. The vitamins that function as coenzymes in energy metabolism are ____.
 - a. Thiamin, riboflavin
 - b. Pyridoxal phosphate (vitamin B6), vitamin A
 - c. Vitamin D, vitamin A
 - d. Vitamin E, vitamin A
11. A vitamin that functions in the metabolism of volatile fatty acids in ruminants.
 - a. Vitamin B6
 - b. Vitamin B12
 - c. Vitamin C
 - d. Niacin
12. Which enzyme reactions require biotin? Name the antivitamin that causes biotin deficiency.
13. An experimental drug added to animal diets has been shown to inhibit the functions of coenzyme A and make it unavailable to facilitate energy metabolism in the animal. Which one of these vitamins is an integral part of coenzyme A?
 - a. Pantothenic acid
 - b. Niacin
 - c. Riboflavin
 - d. Thiamin
14. The rat chow you got at Walmart if fed to guinea pigs, could cause deficiency of which of the B vitamins?
15. Niacin is a vitamin that was discovered in the search for the cause of pellagra, a skin dermatitis found in humans. Which amino acid can form niacin?
 - a. Threonine
 - b. Taurine
 - c. Tryptophan
 - d. Tyrosine

XV. Minerals

This chapter provides an introduction and discussion of different minerals that are important in the nutrition of food-producing animals.

New Terms

Calcium
Calcium homeostasis
Dietary cation-anion balance
Electrolytes
Grass tetany
Hypocalcemia
Macrominerals
Magnesium
Microminerals
Milk fever
Osteomalacia
Parathyroid hormone
Phosphorus
Rickets
Sulfur

Chapter Objective

- To introduce and discuss different inorganic elements of importance in animal health, welfare, and nutrition

What Are Minerals?

Minerals are inorganic elements that are essential for the animal body's physiological functions and metabolic processes. The mineral matter constitutes about 4% of the animal body's weight, and their presence is essential for maintaining life and animal health. Minerals are more integrally a part of all biological functions in the body than any other single class of nutrient. The functions include expression and regulation of genes and enzyme systems that regulate cellular function, activity and functionality of vitamins, osmotic balance,

detoxification, immunity, cell membrane function, acid-base balance and regulation, and structural support and growth (i.e., bone). Scientific literature lists 21 essential minerals. The current chapter will discuss only those definitely implicated in different animal nutrition problems in practical situations.

Minerals are classified into two groups—macro and micro (trace) minerals—based on the amounts needed in diet and not based on their importance for physiological functions. Macrominerals are those minerals that occur in appreciable amounts in the animal body and are required in large quantities in the diet (> 0.01%). Macrominerals include calcium, phosphorus, magnesium, sulfur, and electrolytes (sodium, potassium, chloride). The functions and deficiencies of macrominerals and electrolytes (Na, K, Cl) discussed in this section are shown in table 15.1.

Microminerals are required in trace amounts (< 0.01%), in milligrams, micrograms, or parts per million. Microminerals discussed include manganese, zinc, iron, copper, selenium, iodine, cobalt molybdenum, and chromium. Minerals cannot be added to a diet in their elemental forms but rather need to be added as salts that are combined with other minerals (NaCl, CaCO₃, MnSO₄, etc.).

Minerals

- Minerals are inorganic elements present in animal tissue.
- Minerals do not provide energy.
- Minerals are needed in minute quantities in the diet.

Minerals are classified into macro and micro minerals based on their presence and need in the animal diet.

Calcium and Phosphorus

Functions

Both calcium (Ca) and phosphorus (P) function as structural components in the animal body. Approximately 99% of the Ca and 80% of the P in the animal body occur in bones and teeth as a compound called hydroxyapatite. The other 1% of Ca is distributed in cellular fluids, where they are involved in different metabolic and physiologic activities such as blood coagulation, nerve impulse and cell permeability maintenance, activation of certain enzymes, muscle contraction, or serving as activators of ion channels.

The phosphorus that is found in the soft tissues of the body is involved in important phosphorylation reactions that are part of cellular oxidative pathways for energy metabolism. For example, phosphorus is a component of the central compound in energy metabolism, adenosine triphosphate (ATP), which is a phosphorylated compound. Similarly, deoxyribonucleic acid (DNA) and ribonucleic acid (RNA) contains phosphorylated pentose sugars. Phosphorus is also part of cell membrane phospholipids that are involved in maintaining cellular fluidity and transport of nutrients into cells. Thus calcium and phosphorus are crucial to different metabolic processes that sustain animal life. Cereal grains are rich in phosphorus. However, P in cereal grains are present in the bound form as phytate or phytic acid. The availability of P from such bound sources varies (20%–60%). Monogastric animals lack the enzyme phytase to release them from the bound form and the term available P is commonly used to designate unbound forms of P in the diet of monogastric animals. Ruminant animals produce microbial phytase enzyme that can split and liberate P.

Regulation of Blood Calcium Levels: The body has a strictly controlled physiological regulation called *homeostasis*—that is, maintenance of a steady state of circulating blood plasma calcium. This is brought about by the action of parathyroid hormone (PTH), calcitonin, and active forms of vitamin D as shown below. When blood Ca is low (hypocalcemia), PTH is released from the parathyroid gland, which leads to increased Ca and P resorption from bone, increased P excretion into urine, and increased synthesis of active forms of vitamin D in the kidneys; this in turn is due to an increase in absorption of dietary Ca from the gastrointestinal (GI) tract.

Stimulus	Hormone	Action	Result
↓ Low Blood Ca (Hypocalcemia)	↑ Para Thyroid Hormone (PTH)	↑ Bone Ca Mobilization, kidney resorption, gut Ca absorption	↑ Blood Ca ↓ Excretion in urine
↑ High Blood Ca (Hypercalcemia)	↑ Calcitonin	↓ Bone mineralization, gut Ca absorption	↓ Blood Ca

In conjunction with PTH, vitamin D also enhances the mobilization of Ca from bone by increasing the activity of osteoclasts. Overall, the net result is an increase in blood Ca level to normal levels. When blood Ca is high (hypercalcemia), another hormone called calcitonin is released by the parafollicular cells of the thyroid gland. Calcitonin reverses PTH functions to lower blood Ca level to normal by decreasing calcium mobilization from bones.

Parathyroid hormones and active-form vitamin D are the most important regulators of blood calcium homeostasis.

Calcium and phosphorus also have an important relationship to each other within the diet. The ratio of calcium to phosphorus is also important. Excess dietary Ca forms insoluble complexes with phosphorus, resulting in decreased P absorption. High P or phytate P in the diet can inhibit Ca absorption. Cereal grains are rich in P, but most of it is in the bound form as phytate P (> 30%–60%) and limits the absorption of other nutrients by forming complexes that are resistant to the action of digestive enzymes. The recommended ratio of Ca:P is 1:1 (small animals) to 2:1 (large animals). Feeding diets with improper ratio of Ca:P or supplementing feeds with high levels of one of these minerals can lead to calcium phosphorus imbalance. Such problems affect skeletal health, productivity, and animal welfare and may lead to economic loss.

Disorders Associated with Calcium Phosphorus Deficiency or Imbalance in Animals

Bone serves as the storehouse of minerals, especially calcium and phosphorus. Thus imbalance in calcium and phosphorus leads to structural deformities in animals as well as eggshell quality in egg-laying hens. Several bone growth disorders are associated with calcium phosphorus deficiency, imbalance, or excess in food-producing animals.

Rickets is a condition occurring in young growing animals due to normal growth in the organic matrix but insufficient mineralization. Osteomalacia occurs in adult animals with a Ca-deficient diet. Excessive loss of Ca from bone causes brittle, demineralized bones. Osteoporosis is the result of a loss of both mineralization and the organic matrix of bone. In both rickets and osteomalacia, bones become soft and often deformed due to improper calcification. In fast-growing animals, such as chickens and pigs, where skeletal mineral turnover is rapid, Ca deficiency may produce profound changes. In large animals, such as cows and sheep, it takes a longer time to show deficiency symptoms. Lameness, leg weakness, abnormal gait, and spontaneous fractures may accompany osteomalacia. A reduction in bone ash content occurs in all cases of Ca deficiency or Ca-P imbalance.

Rickets in young animals and osteomalacia in older animals occur due to Ca and P deficiency.

Severe Ca deficiency may produce hypocalcemia, which causes tetany and convulsions. Milk fever, or parturient paresis, in dairy cows is a classic example of hypocalcemia and Ca tetany. The animal's body temperature drops, it shows signs of tetany, and it eventually collapses with head bent over the flank. This is attributed to the lowered blood Ca levels. Treatment is aimed at increasing blood Ca through an intravenous supply of Ca salts such as CaCl₂, Ca-lactate, or Ca-gluconate. A high-dose vitamin D injection should be given five days before calving to enhance Ca absorption.

Milk fever always happens in high-producing dairy cows within the first 24 hours after calving because of the high Ca demand of lactation coupled with hormonal insufficiency. Under normal conditions, bone Ca minerals are utilized to meet the high demand for milk Ca. However, mobilization of bone minerals is under hormonal control, especially by PTH. A good management practice is providing a low Ca diet at least 14 days before calving to “prime” or stimulate endocrine activity so that when lactation begins, Ca mobilization from bones increases due to increased PTH secretion. Electrolyte balance is also important to prevent milk fever and is discussed under electrolytes. Cows with milk fever usually recover rapidly following intravenous administration of Ca.

Providing a low Ca diet during the dry period in cows is recommended to minimize the incidence of milk fever in dairy cows.

Similar to milk fever, cage layer fatigue often happens to high-producing young hens during the peak egg production phase (>35 week of age). Egg laying demands a high supply of Ca for eggshell formation. Lack of enough Ca leads to increase mobilization from bones leading to leg weakness. Affected birds may show reluctance to move, may move to a corner of the cage, or may produce deformed or soft-shelled eggs. The Ca requirements of egg laying hens are much higher than other animals, and the hens should be provided a minimum of 3.3 g of Ca/day for egg production. Use of Ca sources and larger particle size that enhances retention in the gut are highly recommended in hen diets. Hen osteoporosis is also one of the major welfare issues in older hens after the laying cycle; this leads to broken bones and leg weakness.

The ratio of Ca:P is important in bone growth and development. Excess P and low Ca is the common situation in animals fed grain-based diets and low-quality hay or in pets fed homemade meat-based diets. Developmental bone-related disorders occur in young horses fed high-energy diets and in large breeds of dogs fed extra Ca-supplemented diets. Diets that provide Ca:P ratio of 1:1 to 2:1 with slow bone growth is recommended. Similar cases related to Ca-P imbalance has been reported in large cats (tigers, cheetahs) kept in a zoo when fed meat-only diets compared with the meat and bone diets they consume in the wild. A low ratio of Ca-P leads to high level of P and low Ca in the blood. Such a situation causes PTH to increase its secretion, stimulating urinary P excretion and mobilization of Ca from bone. In chronic cases, prolonged dietary imbalance leads to nutritional secondary hyperparathyroidism. Affected animals have demineralized bones, loss of bone mass, joint pain, swelling, lameness, and reluctance to move. In many tropical areas of the world, soil is deficient in P, and animals grazing in such places often develop a depraved appetite and abnormal chewing and eating behaviors, which is termed pica. High fluoride interferes with P digestion and absorption. Pastures contaminated with fluorine gas from industrial sources will precipitate P deficiency in animals.

Dietary Ca:P ratio should be 1:1 to 2:1 for optimum bone health.

Calcium and phosphorus	Deficiency	Excess
Component of the skeleton	Skeletal abnormalities	Nutritional secondary hyperparathyroidism
Provides structural support	Rickets	Ca calcification in tissues upon excess and urinary calculi formation
P involved in metabolism and functions as part of cell membrane phospholipids	Osteomalacia	Excess Ca interferes with absorption of other minerals like Zn

Magnesium

Magnesium is the third most abundant element in the body, is present in the body as phosphates, and carbonates in bone and in liver and skeletal muscle cells. In the skeletal system, Mg is involved providing structural roles, while in the cells, Mg is required to activate several enzymes that split and transfer phosphatases. As a cation in the intracellular fluid, Mg is involved in the metabolism of carbohydrates and proteins. Along with Ca, sodium, and potassium, Mg plays an important role in muscle contraction and transmission of nerve impulses.

Dietary Mg is absorbed mostly from the ileum. No carrier is needed for Mg absorption. Vitamin D does not affect Mg absorption. Homeostatic control of blood and tissue Mg is not well understood, and PTH increases the release of Mg from bone. Nutritional secondary hyperparathyroidism is associated with increases in urinary excretion and reduced serum Mg.

Deficiency: Magnesium is widespread in food sources. A common problem of grazing livestock is called grass tetany. It is also known as “wheat grass poisoning.” It occurs most frequently in livestock that feeds on lush green pastures of cereal forages or native pastures in the spring season. It most frequently occurs on wheat grass pastures. It has been linked to increased trans aconitate in the green pasture in the spring. Trans aconitate binds Mg and leads to Mg deficiency. The symptoms include muscle tetany, head retraction, staggering, convulsion, and extreme sensitivity to noise or touch. Both nitrogen and potassium inhibit Mg absorption. High levels of N and K are usually present in lush fertilized pastures; livestock grazed on fertilized pastures are more susceptible to grass tetany. Treatments include intravenous injection of an Mg solution, feeding Mg from different sources, pasture rotation, and providing dry forages.

Grass tetany is the most common Mg deficiency in grazing animals.

Magnesium functions	Deficiency	Excess/toxicity
Skeletal structure/neuromuscular	Grass tetany or wheat grass poisoning	Depressed feed intake
Activation of enzymes	Nervous behavior	Loss of reflexes
	Muscle tetany	Diarrhea
		Cardiorespiratory depression

Sulfur

Sulfur (S) serves as a structural component of skin, hair, wool, feather, cartilage, and connective tissue. Sulfur is required by the body mainly as a component of S-containing organic compounds. These include chondroitin sulfate; mucopolysaccharide, found in cartilages; the hormone insulin; and the anticoagulant heparin. Sulfur is also an integral part of three amino acids: methionine, cysteine, and cystine. The largest portion of S in the body is found within S-containing amino acids. A high-S-containing amino acid is generally recommended in the diets of birds during rapid feather growth as well as in the diets of sheep for wool growth. Sulfur is also found in enzymes such as glutathione peroxidase, which functions as an antioxidant. In addition, S is a component of two B vitamins, thiamin and biotin, involved in carbohydrate and lipid metabolism. As a component of coenzyme A, S is important in energy metabolism too.

Sulfur as a part of S-containing amino acids is in high need during feather and wool growth.

Deficiency: Reduced feather and wool growth and weight gain can occur due to S deficiency. Inorganic S is very poorly absorbed from a diet. S requirement can be met with organic S found in S-containing amino acids. In sheep, S supplementation may help in microbial protein synthesis and weight gain when nonprotein nitrogen is included in the diet.

Toxicity: Because intestinal absorption is very low, S toxicity is not a practical problem.

Electrolytes

Sodium, Potassium, and Chlorine

Electrolytes are electrically charged, dissolved substances; the animal body is kept electrically neutral. Acid-base balance is determined by the difference between total anion and cation intake and excretion. In this section, sodium (Na), potassium (K), and chlorine (Cl) are discussed together because these three minerals are electrolytes and help in creating an ionic balance and in keeping cells alive. The electrolytes play a vital role in maintaining the acid-base balance (pH maintenance in the blood and tissue), cell membrane signal transductions, and osmotic pressure in intra- and extracellular fluids.

Normal ratios among electrolytes are remarkably constant among species. The animal body has regulatory systems to control the concentrations of these minerals. However, they cannot be stored and need to be supplied in the diet daily. Common salt (NaCl) is added to the diets of all animals and is given free choice to grazing animals. Salt is also used as a vehicle to deliver other trace elements such as iodized salt or trace-mineralized salt. In pigs and poultry diets, the addition of 0.3% to 0.5% salt is standard practice.

Sodium (Na⁺) is the main extracellular cation found outside the cells (extracellular) and blood. Sodium functions in conjunction with other ions to maintain cell permeability in the active transport of nutrients across membranes. The sodium pump (Na-pump) controls electrolyte balance and is a major part of the basal metabolic rate in the body. Sodium is also required for muscle contraction and nerve impulse transmission. Sodium is included in animal diets as sodium chloride (NaCl)

Potassium (K) is the major cation found in greater concentrations within the cells (intracellular fluid). Ionized K within the cells provides osmotic force, which maintains fluid volume. Cellular potassium is also involved in several enzymatic reactions. Maintaining potassium balance is important for the normal functioning of the heart muscle.

Chloride is the negatively charged anion that counterbalance the role of positively charged cations (K and Na). Chlorine (Cl) accounts for two-thirds of anion present in extracellular fluid involved in regulating osmotic pressure. Chlorine is also necessary for the formation of hydrochloric acid, which is needed for the activation of gastric enzymes and initiation of protein digestion in the stomach. Chloride is supplied through NaCl in the animal diet.

Deficiency: Usually, these three elements are fairly abundant in normal diets and deficiency is rare.

Toxicity: The kidneys normally regulate the excretion of electrolytes. Therefore, toxicity is very rare; unless, it is due to

renal diseases, restricted water intake, or high salinity in water. High Na intake although associated with hypertension in humans is not reported in animals.

Electrolytes (Na, K, Cl-)	Deficiency/imbalance	Excess/toxicity
Na: Major extracellular cation, maintains osmotic pressure and acid-base balance	Deficiency not common	Salt toxicity in nonruminants, staggering gait, and high K can inhibit Mg absorption.
Cl: Major extracellular anion, maintains osmotic pressure, acid-base balance, and HCl in digestion	Imbalance can cause leg abnormalities in poultry, acidosis, alkalosis, reduced growth, and milk fever in dairy cows	
K: Major intracellular cation, maintains osmotic pressure, acid-base balance, and muscle activity.		

Dietary Electrolyte Balance in Food Animal Health and Production

There is an increased interest in the balance of electrolytes in food animal production to maintain animal health, welfare, and productivity. Alterations in acid-base balance can lead to acidosis or alkalosis in animals affecting animal health and productivity. Under most circumstances, dietary electrolyte balance is expressed as Na+K-Cl (meq/kg). For poultry, the optimal balance is 250 meq/kg, and for pigs, it should be in the range of 100–200 meq/kg dry matter (DM). Dietary electrolyte imbalance has been associated with leg abnormalities, such as tibial dyschondroplasia (slipped tendon); reduced growth, affecting appetite; and poor poultry growth, productivity, and welfare.

Electrolyte balance is important in maintaining skeletal health and growth in pigs and poultry.

In ruminant animals, electrolyte balance is important in preventing acidosis and alkalosis. Dietary cation-anion difference is usually adopted in dairy cattle feeding to reduce the incidence of milk fever. Prepartum alkalosis may increase the incidence of milk fever in dairy cattle, whereas acidosis may prevent it. Prepartum diets high in forages are also rich in K and could reduce the ability of the cow to maintain Ca homeostasis and could cause milk fever. Diets that reduce blood pH can cause blood Ca to increase and reduce the milk fever.

Alkaline diets increase the incidence of milk fever, and acidic diets prevent milk fever.

Table 15.1. Macrominerals, functions, and deficiencies

Mineral	Function	Deficiency
Ca	Structural = bone, teeth (99%) Metabolic = muscle contraction, blood coagulation (1%)	Rickets, Osteomalacia, Osteoporosis
P	Structural = bone, teeth (80%) Metabolic = intracellular anion, ADP /ATP (20%)	Rickets, Osteomalacia, Pica
Mg	Structural = bone, teeth (50%) Metabolic = phosphatase cofactor (ADP/ATP), oxidative phosphorylation	Grass tetany
S	Structural = skin, hair, feathers, collagen, cartilage Metabolic = S-containing molecules	Reduced wool growth, Reduced weight gain
Na	Main extracellular cation, maintains osmotic pressure, membrane potentials, acid-base balance	
Cl	Main extracellular anion, maintains pressure, acid-base balance, HCl	
K	Main intracellular cation, maintains osmotic pressure, membrane potentials, acid-base balance	

Key Points

1. Scientific literature lists 21 essential minerals. Only those of practical importance in animal nutrition were discussed.
2. Depending on their quantitative abundance in the diet, minerals are divided into major, or macrominerals (> .01% in diet), and trace, or microminerals (< .01% in diet).
3. Calcium (Ca) has two functions: structural components (99%) and metabolic activity (1%). Bones are made of an organic matrix and mineral salts. The former includes collagen and mucopolysaccharides. Mineral salts include hydroxyapatite, which contains 36% Ca and 18% P.
4. When an animal's blood Ca is low (hypocalcemia), parathyroid hormone (PTH) is released from the parathyroid gland. PTH will increase Ca and P resorption from bone, increase P excretion into urine, and increase the synthesis of active vitamin D. The net result is to elevate blood Ca level to normal. When blood Ca is high (hypercalcemia), calcitonin is released from the thyroid gland. Calcitonin reverses PTH functions, which results in lower blood Ca level.
5. Rickets occurs only in young growing animals due to normal growth in the organic matrix but insufficient mineralization.
6. Osteomalacia occurs in adult animals on a Ca-deficient diet. Excessive loss of Ca from bone causes brittle, demineralized bones. Osteoporosis is the result of loss both in mineralization and in the organic matrix of

bones. Milk fever is a metabolic disorder with high-producing dairy cows that occurs within the first 24 hours after calving. Their body temperature drops, they show signs of tetany, and they eventually collapse. This is attributed to the lowered blood Ca. Good management practice can prevent this problem.

7. Phosphorus (P) has two functions: structural components (80%) and metabolic activity (20%). Its metabolism is tightly coupled with Ca. Three key factors affecting their metabolism are (1) adequate amount of both Ca and P, (2) suitable ratio of Ca:P in the diet (1.1:1 to 2:1), and (3) adequate amount of vitamin D.
8. Grains are high in P, in the form of phytate, or phytic acid. Only microbial phytase is capable of liberating P. Deficiency of P includes rickets and osteomalacia. Animals deficient in P often develop abnormal chewing and eating behavior, which is termed pica. High fluoride interferes with P digestion and absorption.
9. Magnesium (Mg) is the third most abundant element in the body. Half of Mg can be found in bone and teeth, and the other half is used as a cofactor for various phosphatases.
10. Grass tetany is the most common Mg deficiency in grazing animals. Nitrogen and potassium both inhibit Mg absorption. In ruminants, a high-protein diet inhibits Mg absorption.
11. Sulfur (S) is an element in methionine and cysteine. It is required for hair, fur, and feather growth. Cartilage and connective tissue also require S. It is also a component of several B vitamins (thiamin, biotin, and coenzyme A) and the hormone insulin. S requirements can be met with organic S found in S-containing amino acids.
12. Sodium (Na), potassium (K), and chlorine (Cl) are important in maintaining osmotic pressure, membrane potentials, and acid-base balance in tissues of animals. Na is the major extracellular cation, Cl is the major extracellular anion, and K is the major intracellular cation.
13. Electrolyte balance is important in maintaining animal growth, health, and welfare, and any imbalance can cause leg disorders in poultry and milk fever in dairy cows.

Review Questions

1. What are the differences between osteomalacia and rickets?
2. What minerals are associated with bone and teeth formation?
3. What minerals are considered electrolytes?
4. Name the major extracellular cation and anion.
5. Which mineral can be supplied by dietary protein (amino acids)?
6. Calcium levels in the blood are tightly regulated by a complex series of events. Outline what happens in a dairy cow with low blood calcium during early lactation (hypocalcemia). Include what hormones are secreted, nutrients are involved, and organs are affected.
7. You are visiting a poultry farm and the hens are laying soft-shelled eggs and are showing reluctance to move. These hens may have this condition.
 - a. Rickets
 - b. Hen osteoporosis
 - c. Cage layer fatigue

- d. B and C
8. During hypercalcemia (high blood Ca), this hormone decreases bone mineralization.
- a. Parathyroid hormone
 - b. Calcitonin
 - c. Gastrin
 - d. Insulin
9. Grass tetany, or “wheat grass poisoning,” is a condition that may occur in cattle grazing lush, rapidly growing grass in the spring of the year. What is the cause of grass tetany?
- a. Low blood potassium (K)
 - b. Low blood magnesium (Mg)
 - c. Low blood phosphorus (P)
 - d. Low blood calcium (Ca)
10. Which nutrient enhances Ca absorption from the gut?
- a. Vitamin A
 - b. Phosphorus
 - c. Vitamin D
 - d. Vitamin E
11. The unit for dietary electrolyte balance in an animal ration is ____.
- a. Mg/g
 - b. Ppm
 - c. Meq/kg
 - d. $\mu\text{g/g}$
12. The dietary cation-anion difference in animal ration is calculated as follows:
- a. $(\text{Na} + \text{K}) - (\text{Cl})$
 - b. $(\text{Na} + \text{Cl}) - \text{K}$
 - c. $(\text{K} - \text{Na}) + (\text{Cl} + \text{S})$
 - d. $(\text{Cl} + \text{Na}) - (\text{K} + \text{S})$
13. This electrolyte is a component of gastric secretion.
- a. Na^+
 - b. K
 - c. Cl^-
 - d. S

XVI. Microminerals

This chapter provides an introduction and discussion of different microminerals that are important in the nutrition of food-producing animals.

New Terms

Anemia
Cobalt
Copper
Ferritin
Goiter
Iodine
Iron
Manganese
Metalloenzyme
Microminerals
Molybdenum
Parakeratosis
Perosis
Selenium
Transferrin
White muscle disease
Zinc

Chapter Objective

- To introduce and discuss different microelements of importance in animal health and nutrition

The difference between macro- and microminerals is based on their requirements in the diet. Microminerals are required in trace amounts (< 0.01%, milligrams or micrograms) and function as activators of enzymes or as components of organic compounds. The following microminerals will be discussed in this chapter: manganese, zinc, iron, copper, selenium, molybdenum, iodine, and cobalt.

Manganese (Mn)

Functions

Manganese (Mn) is a trace mineral that is a dietary essential for animals. In the animal body, Mn is widespread but is concentrated in bone and liver. Manganese is essential for the maintenance and production of the mucopolysaccharide of the organic matrix of the bone. Thus Mn is essential for bone formation and health. Consequently, Mn-deficient animals have normal tendon growth but slow or abnormal bone growth. This leads to symptoms such as perosis (slipped tendon) in chicks and crooked calf in young ruminants. Manganese also serves as an important cofactor for many enzymes that catalyze carbohydrate, fat, and protein metabolism. A large portion of Mn is located within the mitochondria, where it activates a number of metal-enzyme complexes, such as pyruvate carboxylase, that regulate carbohydrate metabolism. Manganese also functions as a cofactor in lipid metabolism through its role in cholesterol and fatty acid synthesis. The absorption of manganese from the diet is very poor and is less than 10% of intake. Excessive dietary Ca or P inhibits Mn absorption. Manganese is absorbed from the gastrointestinal tract as Mn²⁺, oxidized to form Mn³⁺, and transported to tissues using transferrin as a carrier. Excessive Mn in diet can induce iron deficiency.

Deficiency: Many skeletal abnormalities are associated with manganese deficiency and are related to default in mucopolysaccharide synthesis. Lameness, shortening and bowing of legs, and enlarged joints in pigs, sheep, goats,

and cattle are reported. In poultry, perosis (slipped tendon) occurs with Mn deficiency. Affected birds will have a malformation of the tibiotarsal joint, bending of long bones, and gastrocnemius tendon slipping from its condyle. In cattle eating range lupine, bone-related disorders are reported. This is because lupine contains substances that interfere with Mn absorption causing deficiency. Reproductive problems such as delayed estrus, poor conception, decrease in litter size and livability in large animals, and reduction in hatchability in birds are reported due to Mn deficiency. Toxicity: Manganese toxicity is very rare.

Manganese	Deficiency	Excess
Component of organic matrix of bone.	Skeletal abnormalities and crooked legs in large animals	Toxicity very rare. Excess Ca and P interfere with absorption of Mn.
Involved in energy metabolism and lipid synthesis.	Perosis or slipped tendon, parrot beak in birds	Excess Mn reduce Fe absorption
	Reproductive problems	

Zinc (Zn)

Zinc (Zn) is widely distributed in the animal body. High concentrations of Zn can be found in the liver, bones, and animal body coverings, such as hair, wool, skin, and feathers.

Zinc is a cofactor or constituent (metalloenzyme) for more than 100 enzyme systems in the animal body. These include nucleic acid and protein synthesis and metabolizing enzymes (e.g., as DNA and RNA polymerases). Zinc concentration in tissue is highly related to the tissue distribution of enzymes to which it is related. Zn is a component of insulin and in this way functions with carbohydrate metabolism. Zn is also required for retinol-binding protein synthesis and is important for T-cell function in immunity and reproductive functioning. Absorption of Zn is about 5% to 40% of the intake and is affected by several factors.

Metallothionein, a low molecular weight binding protein, has a high affinity for binding to Zn and is involved in the transfer of Zn from intestinal mucosa cells to plasma and metabolism of Zn. High levels of Zn stimulates synthesis of metallothionein, which binds and traps Zn inside the mucosal cells. The absorption of Zn is affected adversely by high dietary Ca, and the presence of phytate aggravates it. Dietary phytate chelates with Zn, limiting its availability (and the availability of other minerals such as P too) to the animals. Zn absorption requires a common carrier shared by iron, copper, and zinc. Therefore, excessive iron impairs zinc absorption. When mucosal cells are sloughed off, Zn is lost in feces.

Cell differentiation and replication are impaired with Zn deficiency. Therefore, rapidly growing tissues such as the skin, gastrointestinal tract, and reproductive tract are most affected. As Zn is mainly distributed through body coverings such as skin, hair, wool, skin, and feathers, deficiency is associated with skin- or feather-related conditions. Zinc deficiency causes a condition called parakeratosis, or severe dermatitis, with dry, scaly, and cracked skin and poor feathering in poultry. Due to the role of Zn in immunity and T-cell functions, impaired or delayed wound healing occurs with Zn deficiency. Both high Ca and phytate decrease Zn absorption and thus precipitate Zn deficiency. Animal diets containing cereal grains and soybean meal increase Zn requirement due to the high content of phytic acid in these products.

Zinc	Deficiency	Excess
Cofactor or metalloenzyme for more than 100 enzymes involved in protein synthesis and metabolism	Skin, feather, wool related problems.	Least toxic of trace elements
	Parakeratosis: scaly, cracking of skin.	
	Impaired wound healing	
	Deficiency can be induced by diets high in Ca and phytate	

Iron (Fe)

Iron is present in all cells of the animal body, but the largest proportion of the body's iron is present as a component of the protein molecule hemoglobin (> 65%) and myoglobin (> 4%). Hemoglobin is a complex protein present in red blood cells consisting of a haem group (porphyrin) containing ferrous (Fe²⁺) iron and a protein (globin). The metabolic requirement for iron is for the synthesis of respiratory pigments (hemoglobin) that is needed for transporting oxygen from lungs to tissues.

Iron is also a cofactor for several metalloenzymes such as cytochromes, respiratory pigments (hemoglobin, myoglobin), peroxidases, and catalases. Dietary iron is supplied either as inorganic ions (ferric or ferrous iron) or as organically bound iron as a part of the hemoglobin molecule. Nonhaem iron is absorbed primarily in the ferrous (Fe²⁺) state. Ferric iron is reduced to ferrous iron in the intestine. Fe⁺⁺ (ferrous iron) is the form that is being absorbed.

Absorption of Fe in the duodenum is poor and is regulated according to the body's need for the mineral, type of food consumed, and intestinal environment. Acidic conditions in the intestine enhance iron absorption because inorganic iron in the ferrous form is more readily absorbed than iron in the ferric state. Organic haem iron originating from hemoglobin and myoglobin animal tissue, such as meat, is better absorbed than nonhaem iron from plant sources. Low body stores and an increase in metabolic need during periods of active growth and gestation lead to increased absorption. Dietary factors like phytates and tannins and other divalent elements, such as Zn, Mn, and Cu, can inhibit Fe absorption due to their competition for the same binding protein.

The ferrous iron must convert into ferric iron (Fe⁺⁺⁺) before they can be transported. This requires a Cu-containing enzyme, ceruloplasmin. Transferrin is a ferric iron-containing protein, which is the major iron transporting protein found in blood. Once inside the enterocyte, iron can be stored as ferritin (an iron-containing protein) or transferred into the plasma, where it binds to transport protein transferrin, the form of which is transported through the plasma. Iron can be stored in tissues bound to two other proteins, a soluble form (ferritin) or an insoluble form (hemosiderin). Chief storage sites in the body are bone marrow, the liver, and the spleen. Most animals are efficient in conserving iron, so loss is minimal, unless it is due to blood loss such as in parasitic infections, injury, parturition, or surgery.

Iron deficiency leads to hypochromic (less hemoglobin) and microcytic (smaller cell) anemia and reduced growth. These can be attributed to simple iron deficiency and are common among baby pigs (piglet anemia) or to induced iron deficiency, such as cotton pelt in mink. Sow milk is low in iron, and competition among littermates, rapid growth of piglets, and low placental maternal transfer aggravate iron deficiency and cause anemia in baby pigs. Cotton pelt in mink is caused by formaldehyde in the pacific hake binding to iron makes it unavailable for absorption. These deficiencies can be treated by injecting animals with organic iron—that is, iron dextran.

Iron	Deficiency	Excess
Constituent of several metalloenzymes, respiratory pigments (hemoglobin), and various enzymes.	Anemia (hypochromic, microcytic) leading to fatigue	Iron overload can be toxic and cause diarrhea, reduced growth, metabolic acidosis and death
	Growth retardation	

Copper (Cu)

Copper (Cu) is required for hematopoiesis (red blood cell formation). As such, the metabolism of Cu and iron are very much related. Copper serves as a component of different enzyme systems. This includes lysyl oxidase needed for collagen and elastin crosslinking. Inadequate crosslinking can lead to rupture of major vessels and defective bone matrices. Copper is a component of cytochrome C oxidase, which is involved in electron transport and ATP generation. Most of the Cu found in the blood is bound to the plasma protein ceruloplasmin. This Cu-dependent protein functions as a carrier of Cu and is necessary for plasma iron for binding to transferrin. Copper is also a component in the antioxidant enzyme superoxide dismutase, responsible for destroying free radicals and preventing membrane damage and cell death. Copper is needed for the enzyme (tyrosinase) conversion of amino acid tyrosine to the pigment melanin. Lack of Cu can lead to inefficient melanin formation and lack of pigmentation causing changes in coat color and loss of crimp in wool (steely wool). Supplementing Cu has been shown to enhance immunity in ruminant animals.

Absorption: Like iron, Cu is absorbed according to the need in the animal. Metallothionein, a cysteine-rich protein, is involved in the absorption. After absorption, mainly in duodenum, Cu is complexed with plasma protein albumin and mainly stored in the liver, where it is used for ceruloplasmin and other proteins needed by the body. Zinc inhibits copper absorption, whereas phytate increases copper absorption by binding to zinc. In ruminants, there is an interaction between Cu and molybdenum. Excess molybdenum causes Cu deficiency by binding to Cu and forming an insoluble complex in blood. Ascorbic acid inhibits the absorption of Cu. Sheep are sensitive to Cu toxicity due to their low ability to excrete Cu in bile. Copper toxicity causes red blood cell hemolysis. Copper accumulates in the liver cells until they are saturated causing oxidative damage. The breakdown of liver cells releases large amount of Cu into the blood causing RBC damage. Hemolysis causes metallic-green-colored kidneys, chocolate-colored blood, and reddish urine. An inherited disorder of Cu metabolism causing Cu toxicosis occurs in certain breeds of dogs.

Copper	Deficiency	Excess
Constituent of several metalloenzymes, lysyl oxidase, cytochromes, superoxide dismutase.	Anemia (hypochromic, microcytic). Scouring or diarrhea	Red blood cell hemolysis, reddish urine and liver damage causing death. Sheep is sensitive to Cu toxicity.
	Changes in coat color, hair pigmentation	
	Loss of crimp in wool	
	Skeletal deformity	
	Nervous disorders: swayback	
	Lesion of circulatory system: aortic rupture	

Selenium (Se)

Selenium is a component of glutathione peroxidase, an enzyme that deactivates lipid peroxides that are formed during lipid oxidation. Se shares this property with vitamin E in preventing peroxidation of polyunsaturated fatty acids in cell membranes and thus protecting cell integrity. Thus Se and vitamin E have a sparing effect on the requirements of each other micronutrients. Se is also a component of other selenoproteins in blood and muscle. The midpiece of sperm requires selenoprotein. Se is also involved in thyroid gland functions as deiodinase that converts the thyroid hormone thyroxine to its metabolically active form, triiodothyronine. Sulfur-containing amino acids are important in the metabolism of Se. Microbes in the rumen replace Se with S in their S-containing amino acid synthesis and are absorbed in the duodenum as amino acids. Se is stored as selenomethionine and selenocystine.

Se-deficient or toxic soils occur in different parts of the US and the world, affecting Se content of forages and grains produced from such places. Se deficiency causes nutritional muscular dystrophy in all species (white muscle disease, exudative diathesis). Affected animals have white streaks on both skeletal and heart muscles, stiffness, and difficulty in locomotion. The white color of skeletal and heart muscles are due to the deposition of Ca salts in the degenerating muscle tissue. Exudative diathesis occurs in chickens due to cell membrane damage; cellular fluid exudes into body cavities under the skin, causing ascitic-like conditions. Se deficiency symptoms can be treated with both vitamin E and Se. Subclinical deficiency of Se may cause the incidence of retained placenta in dairy cattle. The addition of Se to animal diets has been shown to enhance antioxidant status and lipid stability through reduced lipid peroxidation.

Se is required in very small quantities. The range between deficiency and toxicity is very narrow. A 0.02 ppm of Se is required and 5 ppm is considered toxic. Se toxicity causes alkali disease. Animals affected by alkali disease show abnormal hoof and hair growth, loss of hair, and cracking and breaking of hooves. In addition, Se toxicity causes acute blind staggers, which are caused by central nervous system damage. The mode of action of Se toxicity is not known at this time. Toxicity can be prevented by providing animals with a high-protein diet or inorganic sulfate in their diets. Se can be provided as pellets placed in rumen. Recently, Se fertilization of forages has been attempted to enhance organic Se in the diet of animals foraging in Se deficient soils.

Selenium	Deficiency	Excess
Constituent of glutathione peroxidase, deiodinase and selenoproteins.	Nutritional muscular dystrophy	Alkali disease
Antioxidant protection	White muscle disease	Blind staggers
	Exudative diathesis	

Cobalt (Co)

Cobalt (Co) is a constituent of vitamin B12. Cobalt is widely distributed in tissues such as in the liver, kidneys, and bones. The forms in which it appears in tissues other than as a part of vitamin B12 are not clearly known. Due to its close association as a chelated mineral with B12, the deficiency symptoms of cobalt align with vitamin B12 deficiency symptoms. Lack of cobalt in a diet leads to reduced ruminal synthesis of vitamin B12. Ruminant animals have high cobalt requirements. This is due to their inefficient vitamin B12 synthesis and low ability to absorb vitamin B12. Cobalt-deficient (and vitamin-B12-deficient) ruminant animals are unable to metabolize volatile fatty acids (propionic acid) for

energy production, and thus affected animals will have high propionate in their blood and reduced appetite leading to emaciation. Because propionate is the precursor of blood glucose, affected animals will have hypoglycemia. Cobalt deficiency occurs in the soil in different parts of the world, thus leading to low levels of Co in the forages consumed by grazing ruminants. Dense pellets of cobalt are given orally to cobalt-deficient ruminants. These pellets lodge in the rumen and supply cobalt for rumen microbes for vitamin B12 synthesis. Inorganic Co is absorbed very poorly from the gastrointestinal (GI) tract, and due to the low absorption rate, toxicity is unlikely.

Cobalt	Deficiency	Excess
Constituent of vitamin B12. Important for volatile fatty acid metabolism in ruminant animals.	Emaciation, low appetite, anemia, reduced growth in ruminant animals.	Toxicity unlikely

Iodine (I)

The only known function of Iodine is as a constituent of thyroxine (tetra iodothyronine) and triiodothyronine, thyroid gland hormones. Tetra iodothyronine is synthesized by the thyroid gland and is released into the tissues and is converted to the active form, triiodothyronine. An iodine-containing protein, thyroglobulin, is the precursor of thyroxine. Thyroxine stimulates cellular oxidative processes and regulates the basal metabolic rate. The thyroid gland contains the highest concentration of I and is followed by other organs such as the stomach, intestine, mammary glands, and skin. The key organ for I metabolism is the thyroid gland. More than 80% of total body iodine can be found in the thyroid gland. The uptake of I by the thyroid is enhanced by thyroid-stimulating hormone (TSH) secreted by the anterior pituitary gland. I is stored in the thyroid gland mainly as a glycoprotein called thyroglobulin.

Deficiency of I leads to reduced regulation of the basal metabolic rate (BMR). Tissues of I-deficient animals consume less oxygen, and a reduction in the basal metabolic rate is associated with reduced growth rates and gonadal activity. In these animals, the skin becomes dry and the hair becomes brittle. Reproductive problems are associated with abortion, stillbirths, or irregular estrus in females and deterioration of semen quality in males. I deficiency in young animals is called cretinism, a syndrome characterized by failure to grow, multiple skeletal deformities, and skin lesions. Thyroid enlargement leads to a condition called goiter. The enlargement is due to an attempt of the thyroid gland to secrete more thyroxine in response to TSH stimulation. TSH is released in response to reduced thyroxine production. In the absence of adequate thyroxine for inhibiting TSH release, the thyroid gland becomes hyperactive and increases in size (hypertrophy).

Goiter could occur in animals eating I-deficient forages or those feeds containing goitrogens (substances that interfere with the iodination process in thyroxine synthesis). Such feeds can cause induced I deficiency in animals. Numerous plants contain thyroid inhibitors or goitrogens. Plants in the cabbage family (Brassica forages, kale, turnip, rapeseed) are noteworthy for their goitrogenic activity. The requirement of I is about 0.2 to 0.3 ppm.

Long-term chronic intake of large amounts of I reduces thyroid uptake of I and leads to toxic symptoms called hyperthyroidism. Excess I disturbs all thyroid functions leading to increased BMR, increased pulse rate, and increased nervousness and excitability.

Iodine	Deficiency	Excess
Constituent of thyroxin, a thyroid gland hormone which are major integrators of maintaining basal metabolic rate.	Hypothyroidism (reduced growth rate, gonadal activity, reproductive problems, hair loss, drying of the skin). Cretinism in young animals Goiter (enlargement of thyroid gland)	Hyperthyroidism causing goiter-like conditions

Molybdenum (Mo)

In addition to the microminerals discussed, there are several other elements that have been shown to have positive effects on animal growth, immunity, and health. These include molybdenum and chromium.

Molybdenum (Mo) is a cofactor of the enzyme xanthine oxidase and nitrogenase. Mo is used as fertilizer on pasture. It is rare to see Mo deficiency; however, it is common to see Mo toxicity. Excessive Mo inhibits Cu absorption and binds Cu in blood to form an insoluble complex and thus cause Cu deficiency.

Chromium (Cr) has been identified as an essential nutrient in animals. The role of Cr in glucose metabolism and in the ability of cells to take glucose has been identified. In swine nutrition, the addition of Cr is used as a feed additive to reduce carcass fat, and supplementation of Cr has been shown to enhance immunity and reduce respiratory disease in cattle.

Key Points

1. Manganese is concentrated in the animal bones. It is an important cofactor for many enzymes involved in energy and protein metabolism. Mn is also required for mucopolysaccharide synthesis. This is a major component in the organic matrix of bones. Consequently, deficient animals have normal tendon growth but slow bone growth. This leads to symptoms such as perosis in chicks and crooked calf in other animals. The latter is usually associated with the ingestion of range lupine by cows. Lupine contains substances that interfere with Mn absorption.
2. Zinc can be found in animal body coverings, such as hair, wool, skin, and feathers. Zn is a cofactor for more than 100 enzyme systems in the animal body. Zn absorption requires a common carrier shared by iron, copper, and zinc. Therefore, excessive iron impairs zinc absorption. High levels of Zn stimulate the synthesis of metallothionein, which binds and traps Zn inside the mucosal cells.
3. Skin- or feather-related problems, parakeratosis, and impaired wound healing are associated with Zn deficiency. Both high Ca and phytate decrease Zn absorption and thus precipitate Zn deficiency.
4. In addition to the cofactor role in the cytochrome system, Fe is a component of heme. The absorption of Fe in the duodenum is poor. Fe⁺⁺ (ferrous iron) is the form that is being absorbed. Divalent elements such as Zn, Mn, Cu, phytate, and tannins inhibit Fe absorption. Ferrous iron must be converted into ferric iron

- (Fe⁺⁺⁺) before it can be transported. This requires a Cu containing the enzyme ceruloplasmin.
5. Transferrin is a ferric-iron-containing protein, which is the major iron transporting protein found in blood. Iron can be stored in either a soluble form as ferritin or an insoluble form as hemosiderin. Iron deficiency leads to hypochromic and microcytic anemia and reduced growth. It can be attributed to a simple iron deficiency, such as in baby pigs, or an induced iron deficiency, such as cotton pelt in mink. The latter is caused by formaldehyde in the pacific hake binding to iron makes it unavailable for absorption.
 6. Copper is required for hematopoiesis (red blood cell formation). It also serves as a cofactor for many different enzyme systems. Zn inhibits Cu absorption. Since phytate binds to Zn, phytate increases Cu absorption. Ascorbic acid inhibits Cu absorption.
 7. Cu is transported into the liver from the gastrointestinal (GI) tract by albumin. The liver incorporates Cu into ceruloplasmin, which is the major transport vehicle of Cu. Deficiency symptoms include scouring, changes in coat color, loss of crimp in wool, anemia, aortic rupture, and swayback. Sheep are sensitive to Cu toxicosis.
 8. Selenium is a component of glutathione peroxidase, an enzyme for the removal of lipid peroxides. Se is also a component of two other selenoproteins. The midpiece of sperm requires selenoprotein. Microbes in the rumen replace S with Se in their S-containing amino acid synthesis. They are absorbed in the duodenum as amino acids.
 9. White muscle disease and exudative diathesis are two Se deficiency symptoms, which can be treated with both vitamin E and Se. Deficient animals also show liver necrosis. The range between deficiency and toxicity is very narrow: .02 ppm is required and 5 ppm is considered toxic. Toxicity symptoms include acute blind staggers, caused by central nervous system damage, and chronic alkali disease, in which animals show loss of hair and cracking and breaking hooves. The mode of action of Se toxicity is not known at this time. Toxicity can be prevented by providing animals a high-protein diet or adding inorganic sulfate to the diet.
 10. Cobalt (Co) has only one known function, which is a constituent of vitamin B12. It can be provided in pellet form deposited in the rumen. Injection of Co has no effect. This is because Co is required for rumen microbes to synthesize vitamin B12. Deficiency symptoms are easily confused with gross malnutrition or starvation.
 11. Iodine's only function is as a constituent of thyroxin, a thyroid gland hormone that regulates the basal metabolic rate. Thyroid-stimulating hormone (TSH), secreted by the anterior pituitary gland, enhances the iodine uptake by the thyroid gland.
 12. Short-term deficiency leads to hypothyroidism, with reduced growth rate and reproductive problems, hair loss, and dry skin. Long-term deficiency leads to goiter. Without iodine, thyroxin cannot be synthesized. This causes the release of TSH, which in turn causes the hypertrophy of the thyroid gland.
 13. Induced I deficiency can be caused by goitrogen, can be found in plants from the Brassica species. Goitrogens block the iodination process in thyroxin synthesis, which triggers I deficiency symptoms. Excessive I also leads to goiter.
 14. Molybdenum (Mo) is a cofactor of xanthine oxidase and nitrogenase. Mo is used as fertilizer on pasture. It is rare to see Mo deficiency; however, it is common to see Mo toxicity. Excessive Mo inhibits Cu absorption and binds Cu in blood to form an insoluble complex and thus causes Cu deficiency. Mo toxicity can turn to Cu deficiency.
 15. Chromium is shown to have effects on glucose metabolism and fat synthesis. It is used as a feed additive to reduce carcass fat in swine and to enhance immunity and reduce respiratory disease in cattle.

Review Questions

1. What is hypochromic anemia? Give an example of when it occurs? And why?
2. What is ceruloplasmin? What is transferrin? Why are they important?
3. Scourin, changes in coat color, loss of crimp in wool, anemia, aortic rupture, and swayback are typical deficiency symptoms for one particular mineral. Name that mineral. What functions does this mineral play to have such a diverse effect on animals?
4. Why are Cu deficiency symptoms the same as in Mo toxicity?
5. Most mineral deficiency problems can be treated with an injection of the specific mineral involved. However, Co deficiency can only be prevented or treated when treatment is delivered orally. Why?
6. The transport form of iron in the blood is _____.
 - a. Hemosiderin
 - b. Ceruloplasmin
 - c. Ferritin
 - d. Transferrin
7. The mineral that is a part of glutathione peroxidase enzyme is _____.
 - a. Ca
 - b. Mn
 - c. P
 - d. Se
8. Goiter is a condition caused by the deficiency of _____.
 - a. Iron
 - b. Iodine
 - c. Manganese
 - d. Magnesium
9. Generic dry dog food disease is a skin disorder in dogs fed poor quality plant-based diets with low digestibility. This skin disorder is due to the lack of this mineral.
 - a. Zinc
 - b. Iodine
 - c. Selenium
 - d. Calcium
10. Piglet anemia is the most common form of anemia in baby pigs and is due to the lack of this mineral.
 - a. Iron (Fe)
 - b. Copper (Cu)
 - c. Zinc (Zn)
 - d. Iodine (I)

XVII. Bioenergetics

This chapter discusses energy metabolism in the animal body and the movement of energy from one form to another. As energy is the most important commodity in the animal diet, this section discusses units of measurements, distribution of energy in the whole animal, and disorders related to energy metabolism.

New Terms

Calorimetry
Comparative slaughter technique
Digestible energy
Energy
Gross energy
Heat increment
Ketosis
Metabolizable energy
Net energy
Obesity
Total digestible nutrients

Chapter Objectives

- To introduce different energy terminology and discuss energy flow through an animal
- To discuss the measurement of energy retention and disorders related to energy metabolism in livestock

Energy is not a NUTRIENT, but a property of some nutrients such as carbohydrates, fats, and proteins.

Why Study Bioenergetics in Animal Nutrition?

Bioenergetics is the study of the balance between energy intake and utilization by the animal for different life-sustaining processes (e.g., osmoregulation, digestion, locomotion, tissue synthesis). Energy intake in the animal is through feed and energy losses are through different sources such as heat, feces, urine, and other gaseous losses. Bioenergetics enables the nutritionist to formulate the ration per the energy need of the animal and helps in evaluating different feedstuffs accurately. As feed represents the major cost of raising livestock (>65%), formulating the right diet will cut down on feed costs and enhance animal productivity and health while minimizing nutrient loss to the environment.

Studying energy measurements and partitioning in animals is important for ration formulation and optimizing animal production.

In the US, the calorie is the unit usually used to express feed energy. In other countries and scientific journals, the joule is used as the unit to express energy. One calorie is the amount of heat required to raise the temperature of one gram of water by 1° C from 15.5° C to 16.5° C. One thousand (1,000) calorie is one kilocalorie (1 kcal) and 1 kcal is 4.184 joules (J). For practical purposes, kcal is commonly used in ration formulation and in expressing caloric value of feeds.

Energy Measurements: Different energy measurements and flows of energy in the animal are shown in Figure 17.1.

Gross energy (GE) is the total amount of chemical energy in the diet consumed. It is also known as the heat of combustion. GE content of feed is measured as heat liberated during complete burning (oxidation) of the feed sample and is determined by an apparatus called a bomb calorimeter. Briefly, the feed sample is burned in a combustion chamber (bomb) inserted in another chamber containing a known weight of water. The heat liberated during burning of the feed raises the temperature of the water. From the weight of the sample, the weight of the water, and the increase in temperature, the GE of the feed can be calculated. This measurement is easy, precise, and accurate. However, GE does not have much practical value, as it does not provide much information on the nutritional value of the feed and does not account for palatability, digestibility, or other animal physiological factors. High-protein and high-fat feeds will have more energy than high carbohydrate feeds, and feeds with high ash will have less energy than lower ash feeds.

Gross energy is the amount of heat liberated when a feed sample is completely burned into carbon dioxide and water and is determined by a bomb calorimeter.

To determine the fraction of the GE that animals utilize for different metabolic processes, animal bioassay studies or digestion trials should be conducted to assess different losses through feces, urine, gas, and heat.

Digestible energy (DE) is the energy remaining in the diet after fecal energy is subtracted. DE represents the indigestible components of the feed that will be excreted in the feces; however, they still contain energy that was not utilized by the animal. Fecal loss of energy is the major source of energy loss to the animal and depends on the nature of the feed. For example, diets containing high fiber may have less digestibility, and fecal loss will be higher than starch-based diets.

- Energy Terms**
- Gross energy
 - Digestible energy
 - Metabolizable energy
 - Net energy

Because DE measurements need fecal energy loss, animal feeding trials need to be conducted. Digestibility trials are easy to conduct. Total feed intake and total fecal output are recorded, and GE is determined based on the diet and fecal matter using a bomb calorimeter. DE is calculated as GE - fecal energy (FE). Most DE values in feed tables are experimentally determined using live animal feeding trials. The carbohydrate fraction of digestibility is highly variable due to the presence of fiber (less digestible) and nonfiber (starch; highly digestible) components. DE is not a true value and is an

“apparent” value as the gastrointestinal tract contributes to extra energy voided in feces. This “extra energy” originates from endogenous sources such as sloughed-off cells, unused enzymes, or other microbial contributions.

$$\text{Digestible Energy} = \text{Gross Energy (GE)} - \text{Fecal Energy (FE)}$$

Metabolizable Energy

Metabolizable energy (ME) is defined as the energy remaining after urinary loss and gaseous losses arising from the gastrointestinal tract are subtracted from DE. Values obtained reflect losses due to digestion, fermentation, and metabolism of the feed by the animal. Urinary loss is the major one and is the total energy lost in urine. Urinary losses are usually stable but can increase when high protein is included in the diet. Urine is the end product of metabolism, which contains energy in different compounds, such as urea. Gaseous products of digestion include combustible gases produced by the digestive tract during fermentation of food by microbes (methane, carbon monoxide, hydrogen). In ruminant, 4% to 8% of feed energy is lost from the rumen as methane.

Losses from gaseous loss are minor and are ignored in monogastric species. On average, combined energy losses in gases and urine are about 18% of DE in ruminant animals.

$$\text{ME} = \text{DE} - \text{Urinary Energy (UE)} - \text{Gaseous Energy}$$

To determine metabolizable energy, metabolic trials are conducted using live animals. Daily intake (feed) and losses (fecal, urinary, gaseous) of energy are documented. Breathing masks (or chambers) are used to assess gaseous losses. Bomb calorimetry is done on all the samples. The amount of methane expelled is documented and is multiplied by its energy concentration (13.2 Mcal /kg). Collection of urine and measuring gaseous losses are prone to errors, and thus ME is less accurate. Diets with high protein content can increase urea loss, and diets with fiber can increase methane and acetate loss. ME is usually conducted in poultry because urine and feces are voided together and gaseous loss is negligible in these species. ME is more difficult to determine than DE, and most of the tabular values for ME are calculated. ME can be used for two purposes: (a) maintenance and (b) production.

Net Energy

Net energy (NE) is ME minus the heat generated by the inefficiency of transforming energy from one form to another. Simply speaking, heat generated is heat lost during the energy transformation process and is called the heat increment (HI). No matter what purpose ME is used for, this is not at 100% efficiency. This inefficiency of the biological system is represented by HI.

$$\text{NE} = \text{ME} - \text{Heat Increment (HI)}$$

HI is a difficult concept, and it is very difficult to determine accurately. Live animals continually produce heat, and HI depends on analyzing fasting versus fed animals. Heat increment is therefore all the heat produced by the act of eating, chewing, and digesting the feed and absorbing the nutrients from the gut. When an animal is fasted, stored nutrients are used instead of absorbed nutrients. Heat increment represents the difference in the efficiency of using absorbed nutrients (fed animals) versus stored nutrients (fasted animal). The type of feed (e.g., fiber vs. starch) also can affect HI.

HI = heat loss of the eating animal – heat loss of the fasting animal.

NE is the remainder of the “useful” energy after all the losses “available” to the animal and could be used for both animal maintenance and production purposes (e.g., milk, eggs, meat, etc.)

NE = NE (maintenance) + NE (production)

NE represents the fraction of the total energy consumed that is utilized for production purposes.

NE represents the best scientifically designed energy system because NE is the actual amount of energy that is useful to the animals; it should be the best way to describe feed energy. Nonetheless, we seldom directly measure NE systems due to the cost and difficulty of determining the NE values. The NE values in feed tables are derived from total digestible nutrients (TDN), DE, body weights, and regression equations based on experiments depending on the species.

Energy Flow Diagram

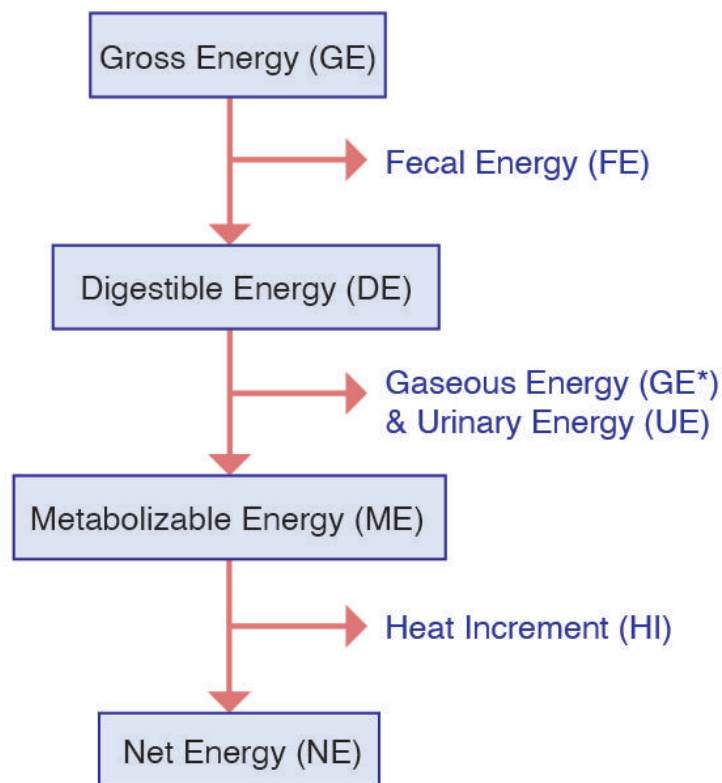


Figure 17.1. Energy flow chart

*This is negligible in monogastric animals.

Methods for Measuring Heat Production and Net Energy

To determine NE, HI has to be measured, and it is not an easy task. Measuring HI requires whole animal calorimeter (respiration chambers). This equipment is very expensive and is limited. Therefore, NE values are limited especially for large animals. Total heat production measured by direct or indirect calorimetry is often employed in NE calculation. Alternatively, NE can be determined by measuring the energy retention of the animal using the comparative slaughter technique. The different methods are briefly discussed below.

Calorimetry: Animals lose heat to the environment through conduction, convection, radiation, or evaporation. The latter loss is through the skin, respiratory tract, or excreta. Heat loss is measured directly using direct calorimetry or indirect calorimetry.

In direct calorimetry, sensible heat loss is measured as a rise in temperature of the medium (e.g., water) circulating outside the walls of the chamber. Evaporative loss can be determined by the increase in humidity of the ventilating air. The equipment used for these types of trials is costly and very limited.

Indirect calorimetry is based on the principle that metabolic heat production is the result of the oxidation of organic compounds. Thus heat production can be calculated from the amount of oxygen consumed and the amount of carbon dioxide produced. However, this measurement is not 100% accurate as nitrogenous compounds of protein oxidation, such as urea, and other anaerobic fermentation products, such as methane, are not accounted for.

Direct calorimetry measures heat production directly. Indirect calorimetry measures gas exchange as it is related to heat production from the oxidation of organic compounds.

Indirect calorimetry can be open or closed. In the open type, which is the most common, a mask or hood or animal chamber may be used. Air intake and carbon dioxide and methane output are precisely measured. Automated gas analysis and computer control make it much easier to handle air intake and CO₂ output. These types of machines are common in use. But errors in measurement can affect the results obtained.

In the closed type, the animal is kept in a temperature-controlled chamber. Air in the chamber is continuously circulated through absorbent silica gel or KOH, which removes water and carbon dioxide. Air pressure is maintained using a constant supply of oxygen, and methane is allowed to accumulate within the chamber. Oxygen use is determined as the amount of oxygen supplied to maintain the pressure and carbon dioxide production is determined from the amount collected by the absorbent.

Comparative Slaughter Technique: In this test, live animal feeding trials are conducted by providing a common ration of known energy for a two week adaptation period. At the end of the adaptation period, a group of animals is slaughtered and body composition and gross energy are determined to get baseline information. The remaining are fed the same ration for a certain period of time and are then slaughtered and body composition is determined. Energy retention is then determined as the difference between body energy content in the initial baseline animals versus those from the animals at the end of the trial. Information derived simulates live animal feeding trials under normal conditions but requires a large number of animals and is time consuming, expensive, laborious, and destructive (animals can only be used once).

The comparative slaughter technique is a better estimate than calorimetry but needs a long time and is costly and the animals can be used only once.

Feeding Systems in Diet Formulation

Energy is the most important “commodity” of a diet and all feeding systems seek to match the nutrient need of the animals. As explained, GE is of no value to the animal as it does not give any information on digestibility or palatability. In the US, among the different energy systems, DE is commonly used in swine, and ME is commonly used in poultry. In ruminants, in addition to NE, TDN analysis is also used.

TDN is an old system of estimating the energy content of feeds. TDN is commonly used in ruminant animals and is carried out by conducting digestion trials. TDN is the summation of the digestible crude protein, digestible fiber, digestible nitrogen-free extract, and digestible ether extract and is expressed as a percentage of the total amount of feed. The additional energy value of fat is compared to carbohydrates by the inclusion of 2.25 as a multiplier. The digestible value of protein is given the same value as carbohydrates and thus indirectly corrects for urinary N loss.

In practice, TDN is in between DE and ME. The major weakness of TDN is that in ruminants, TDN overestimates the energy supply from high-fiber feed ingredients, such as straw and hay, compared to highly digestible low-fiber-containing cereal grains. TDN still is the most popular system used on farms as it is easy to understand and a large database is also readily available.

$$\text{TDN} = \text{digestible crude protein} + \text{digestible crude fiber} + \text{digestible nitrogen-free extract} + (\text{digestible ether extract} \times 2.25)$$

Several factors can influence energy need, such as activity, body size, environment, physiological state (pregnancy, lactation), breed/strain, hide thickness, and coat condition.

Disorders Associated with Energy Intake

Excess or inadequate energy intake can lead to several disorders in food-producing animals.

Obesity: Obesity is considered a disorder associated with excess dietary energy intake and is more commonly diagnosed in companion animals (e.g., dogs) and equines. Obesity can decrease the quality and length of the animal's life. The greater the deviation from optimum body weight and body condition score (BCS), the greater the incidence and severity of orthopedic disorders and cardiovascular diseases.

Obesity occurs when dietary energy exceeds energy expenditure by the body. Influenced by daily energy expenditure and other factors (genetics, diet composition [fat, fiber], neutering). Treatment of obesity is mainly through diet management (high fiber, low fat), and exercise.

Disorders such as bovine ketosis, ovine/caprine pregnancy toxemia, and fat cow syndrome are associated with

inadequate energy intake. Ketosis is common a few days after calving. Low dry matter intake leads to negative energy balance. Due to the energy demands of early lactation and pregnancy (e.g., twins or triplets in ovine) and glucose shortages, depot fats are oxidized to acetyl CoA via β -oxidation. The tricarboxylic acid (TCA) cycle intermediates, particularly oxaloacetate, are limited due to less energy intake, resulting in ketone body formation and increased concentrations of ketone bodies (acetoacetic acid, β -hydroxybutyric acid, acetone) in body fluids. In affected animals, ketone bodies are excreted in urine and acetone is excreted through the lungs as “sweet breath.” Diet management (providing high-quality forages) is recommended at least two to three weeks before calving. Dextrose (50%) IV, propylene glycol (glucose precursor), and glucocorticoids are recommended as therapy.

Fatty liver syndromes occur in cattle and horses during negative energy balance. Depot fats are broken down, resulting in an increase in free fatty acids in the blood. Excessive free fatty acids are presented to the liver and result in fat accumulation in the liver, also called hepatic lipidosis.

Bioenergetics: Summary

1. Bioenergetics is the topic of energy and its metabolism, or biochemical thermodynamics.
2. Energy is a concept and not a nutrient. Energy is the property of some nutrients.
3. The unit of energy is the calorie or kilocalorie. It is the amount of heat required to raise the temperature of one gram of water by 1° C.
4. Physiological oxidation happens inside an animal's body through various metabolic pathways. Physical oxidation takes place inside a bomb calorimeter, which converts feed energy to heat.
5. Gross energy is determined in a bomb calorimeter. This provides a measurement of total energy in feed.
6. Digestible energy (DE) is determined by subtracting energy loss in feces from the gross energy (GE) of feed. Digestion trials are needed to get this value. Not all DE is retained by the animal.
7. Metabolizable energy (ME) represents retained energy.
8. ME supports two different functions: tissue maintenance and production. Maintenance functions include all organ work (e.g., heart, lungs, kidneys) and ion balance and production include products (e.g., milk, meat, eggs).
9. ME is most commonly used in poultry, as feces and urine are voided together and easy to measure, while DE is more commonly used in swine.
10. Net energy (NE) accounts for all the losses and is theoretically more accurate.
11. NE accounts for heat increment (HI), energy loss as heat. HI is heat production associated with nutrient digestion, absorption, and metabolism.
12. Methods to measure energy retention and heat production include direct and indirect calorimetry and the comparative slaughter technique.
13. Total digestible nutrients analysis uses digestibility and proximate analysis to provide an estimate of the energy content of a feed.
14. Disorders of energy metabolism include ketosis and obesity.

Review Questions

1. What is a calorie? How are the total calories of a feed determined?
2. Draw the energy diagram from gross energy to net energy.
3. What is digestible energy?
4. What is metabolizable energy (ME)? List two factors affecting the ME content of a feed.
5. What is heat increment (HI)? List the factors affecting HI?
6. How do you define net energy (NE)?
7. What are the two methods to measure energy retention?
8. List the advantages and disadvantages of the different methods used for measuring energy retention?
9. What is TDN?
10. List two disorders associated with energy metabolism.

XVIII. Water in Animal Nutrition

This chapter discusses the role and importance of water in food-producing animals.

New Terms

Metabolic water
Polioencephalomalacia
Total dissolved solids
Water quality

Chapter Objective

- To introduce and discuss the physiological role, requirement, and quality of water for maintaining animal health and productivity

Water is essential for sustaining life and ranks second to oxygen in importance. Water is needed in greater quantity than any other orally ingested substance and is classified as a macronutrient. Sources of water include drinking water, metabolic water (produced during catabolism of carbohydrates, fats, and proteins to carbon dioxide and water), and the water that presents as moisture in different feed ingredients. Metabolic water serves as the sole source of water in desert and hibernating animals, and feed water is the major water source for marine animals.

Where Animals Obtain Water

- Feed
- Drinking water
- Metabolic water

Why Is Water Important for Livestock?

Water is important for all organisms. Water makes up one-half to two-thirds of the body mass of adult animals and more than 90% of the body mass of newborn animals. Water is an essential constituent of almost all secretions of the body. Within the body, water is a universal solvent that facilitates cellular biochemical reactions involving digestion, absorption, and transportation of nutrients. The aqueous medium of water helps different digestive juices and food components interact, enhancing digestion, and helps in the excretion of waste products in the form of urine, feces, and perspiration, sweat, from the animal body. Because of the high specific heat of water, it helps in regulating body temperature, by absorbing the heat generated through different metabolic reactions. Water also regulates body temperature through evaporation as sweat or transports heat away from organs through blood. Water provides shape to body cells. Water helps in maintaining the acid-base balance of the body. Water acts as a cushion for tissue cells and the nervous system and protects the various vital organs against shocks and injuries.

Water is the single most important nutrient in the animal body. It is essential for all metabolic processes, chemical reactions, temperature regulation, eliminating waste from the body, and ultimately, health and survival.

Water Sources and Losses

The animal body derives water from different sources. These include drinking water, water present as part of feeds (moisture), or those liberated during several metabolic reactions. The importance of these different sources varies among species, habitat, and diet. For example, hibernating animals and desert rodents depend on metabolic water to keep them alive, whereas marine animals depend on their diet to derive their water requirements. Metabolic water also depends on the type of nutrient catabolized. Oxidation of fat produces the greatest amount of metabolic water. However, overall contribution of metabolic water to daily water needs is less than 5% to 10% in most animals. The water content of feed consumed by ruminant and nonruminant animals vary highly. Forages consumed by ruminant animals vary from 5% to 7% for mature plant products, such as hay, to more than 90% for young lush green vegetation. Animals such as sheep depend a lot on water derived from such green forages for their need. Most commercial diets fed to nonruminant animals such as pigs and poultry may contain 7% to 10% moisture. Some of the canned foods fed to pet animals such as dogs and cats may contain more than 75% moisture.

All animals experience daily water loss through different venues such as urine, feces, sweat, saliva, evaporation from the lungs through respiration, and milk in lactating animals. Among these, urinary loss accounts for the major loss. Water lost through urine serves as a tool to dispose of the toxic products of metabolism. Some animals such as birds are capable of concentrating urine and excreting it as uric acid instead of urea and thus conserving water. Urinary water loss depends on weather and on the type of food consumed. Consumption of excess water during heat stress can increase urinary volume. Animals that consume high-fibrous diets excrete more water in their feces. Fecal water excretion is higher in cows (30%–32%) compared to sheep (13%–24%) that void pellet-type dry feces to minimize water loss. Sweating is a means to dissipate body heat. In animals such as horses, loss of water through sweating is high. Animals such as chickens and dogs have very poorly developed sweat glands and compensate for heat loss by panting and increasing water intake. Daily clean water consumption is needed to make up for all the losses and is extremely important during periods of heat stress, especially in animals such as poultry.

Animal	L/day	Gal/day
Beef cattle	26-66	6.5-17
Dairy cattle	38-110	9.5-28
Horses	30-45	7-12
Sheep and goats	4-15	1-4

Water Requirements

An animal's water requirement depends on several factors such as ambient temperature, diet (energy level, fiber content, salt), physiological state (age, growth, pregnancy, ability to conserve water), level of exercise, and health. Environmental temperature (and associated humidity) is a major factor contributing to water intake. Water consumption, when expressed by unit of body weight for non-heat-stressed, nonlactating cattle, is around 5% to 6% of the body weight per day (or 2–5 kg of water for every kg of dry feed consumed) and can go up to 12% or more under heat stress. Water intake increases with higher environmental temperatures and increasing physical activity because of water lost through evaporative loss.

Dietary dry matter intake and feed water content are highly correlated with water intake at moderate temperatures. High-energy, high-fat, and high-protein diets increase water intake because of increases in metabolic waste and urinary excretion of urea as well as increases in heat produced by metabolism. The salt content of a diet increases water consumption. Diets high in fiber (bran, dry forages) increase water intake as well. Young animals have higher water requirements per body size as compared to large animals. Similarly, animals that conserve water, such as sheep and poultry, need lower levels than cattle. Pregnancy and milk production increases water intake too. Dairy cattle may require 38–110 L/d compared to beef cattle at 22–66 L/d.

Ambient temperature is the major factor affecting an animal's water intake. Other factors include age, type of diet, level of exercise, stage of growth, or pregnancy.

Water Restriction and Toxicity

Water shortage affects both domestic and wild animals. To compensate for the losses and to maintain all related physiological functions, animals should have access to a clean supply of water. Lack of enough water could lead to a reduction in feed intake and productivity. Dehydration of the body leads to a reduction in body weight and the consequences are worse in high environmental temperatures. Dehydration is accompanied by a loss of electrolytes, an increase in body temperature, and an increase in respiratory rate. Animals become highly irritable, and prostration and death follow after severe water deprivation. Water intoxication may occur as a result of a sudden ingestion of large amounts of water after a short period of deprivation and is due to the slow adaptation of the kidneys to the high water load.

Water restriction reduces feed consumption and is very stressful for animals.

Water Quality

Water per se is almost nontoxic, but problems with water arise from contamination with microbes, parasites, minerals, and various other toxic substances, such as pesticides. Water quality affects consumption, productivity, and animal health. Substances such as salts, pathogenic organisms, algae, and pesticides pollute water supplies and can affect palatability.

Mineral salts include carbonate and bicarbonates, sulfates, and chlorides of Ca, Mg, Na, and K. Other toxic substances in water include nitrate, iron salts, and hydrocarbons. Contamination with nitrate is common in farming intensive areas. Concentrations above 1,500 ppm may cause toxicity causing death from anoxia. Iron salts in groundwater cause rust deposits on pipes and may cause bacterial contamination by iron-utilizing bacteria. Pesticides such as malathion and organophosphates may get into water systems and can be toxic. Certain blue-green algae (cyanogenic) in lakes can produce toxic substances. Toxicity in livestock causing vomiting, frothing, muscle tremors, liver damage, and death are reported due to blue-green algae toxicosis.

Water quality can influence the development of polioencephalomalacia, a noninfectious disease affecting the brain in feedlot cattle. Most affected animals show aimless wandering, disorientation, blindness, recumbency, star-gazing posture, and edema in the brain, causing a “softness” in the brain. Water high in sulfates promotes polioencephalomalacia, apparently via a complex interaction with other minerals and B vitamins. Most domestic animals can tolerate a total dissolved solid concentration of 15,000 to 17,000 mg/L. Water containing less than 1,000 mg/L of total soluble salt is safe for all classes of livestock. At higher (> 5,000–7,000 mg/L) levels, it may cause mild diarrhea and an increase in mortality in poultry, but it could be acceptable to other livestock. A guideline for the interpretation of total dissolved solids in water is shown in table 18.1.

Table 18.1. Guidelines for interpretation of total dissolved solids content in water

Total dissolved solids, mg/L	Interpretation
<1000	Suitable for all classes of livestock
1000-1999	Satisfactory for all classes of livestock; may produce transient diarrhea in animals
2000-4999	Temporary water refusal and diarrhea may be seen when animals are exposed to such water sources. May reduce productivity in dairy cattle.
5000-6999	Likely to reduce productivity in dairy cattle. May reduce growth rates. May result in water refusal and diarrhea. Avoid if possible.
7000-10000	Unfit for swine, very risky in all other species. Avoid.
> 10000	Dangerous, avoid.

Key Points

1. Water is one of the most important nutrients, yet it is almost always neglected. Water serves as the fluid matrix of the animal body. Water gives form and structure and provides protection from environmental stress. The high solvent power of water permits the formation of solutions within which metabolic reactions occur.
2. There are different sources of water (e.g., diet, drinking, metabolic).
3. Metabolic water is produced during catabolism of carbohydrates, fats, and proteins to carbon dioxide and water and is important for hibernating animals.

4. Species difference, type of metabolism, and digestive tract type affect water requirements. For example, birds and fish have low requirements, while ruminants need a large quantity of water to suspend ingesta in the rumen.
5. An animal's water requirement is highly influenced by environmental temperature, humidity, diet (energy level, fiber content, salt), physiological state (age, growth, pregnancy), and level of exercise and health.
6. Water restriction affects animal health, growth, and productivity and is very stressful for animals.
7. Water containing less than 1,000 mg/L of total soluble salt is safe for all classes of livestock.

Review Questions

1. Water is one of the most important essential nutrients. Functions of water include:
 - a. Can act as a diluent
 - b. Carrier of waste from the body
 - c. Transport of nutrients
 - d. All of the above
2. List the sources of water in the animal body.
3. Major loss of water in a beef cattle is through which medium?
4. Water quality can affect cattle health. Name a non infectious disease in feedlot cattle associated with water quality.

XIX. Feed Additives

This chapter discusses the role, need, and use of different feed additives used in the diet of food-producing animals.

New Terms

Antibiotic growth promoters
Antioxidants
Carbohydases
Enzymes
Feed additives
Nonstarch polysaccharides
Phytase
Pro/prebiotics
Tocopherols

Chapter Objectives

- To introduce different feed additives that are used in the diet of food-producing animals
- To discuss the role of feed additives in increasing nutrient digestibility, gut health, animal productivity, and food product quality

Why Use Feed Additives in Animal Diets?

Feed additives are minor components of the animal ration and are used for improving the quality/digestibility of feed and the nutritive and aesthetic quality of food or improving animal performance and health.

Some of the most commonly used feed additives in animal rations include enzymes, pro- and prebiotics, antioxidants, antibiotic growth promoters, and coloring agents. Overall, these different ingredients are aimed at enhancing digestibility or availability of bound nutrients (e.g., enzymes), improving animal gut health (e.g., pro/prebiotics) and food product quality (e.g., antioxidants), reducing nutrient loss (e.g., phosphorus, nitrogen), and promoting environmental protection.

Feed Additives

- Enzymes
- Pro/Prebiotics
- Antioxidants
- Antibiotic growth promoters

Enzymes

Exogenous enzymes are commonly added to the diet of monogastric animals as a means to reduce the antinutritional effects of the feed. Cereal grains and plant-based byproducts are the major components of the ration. Several nutrients present in these products are in a “bound” form, meaning that monogastric animals do not have the enzymes to digest and release the nutrients to make them available to the animal. Phytate phosphorous and nonstarch polysaccharides (NSP) are the two major antinutrients present in feed.

Phosphorus (P) in cereal grains are present as phytic acid (myo-inositol hexakisphosphate), the primary phosphate storage compound in seeds. Typically, phytic acid contributes 50% to 80% of the total phosphate in plant seeds.

More than 50% to 80% of phosphorus (P) in the feed is present as phytic acid and is not available to the monogastric animals.

The salt form of phytic acid is called phytate, and almost all phytic acid (Figure 19.1) is present as a mixed salt (phytin). Phytate P is poorly available to monogastric animals due to the lack of endogenous enzymes and can reduce the digestibility of other nutrients, affecting growth and performance.

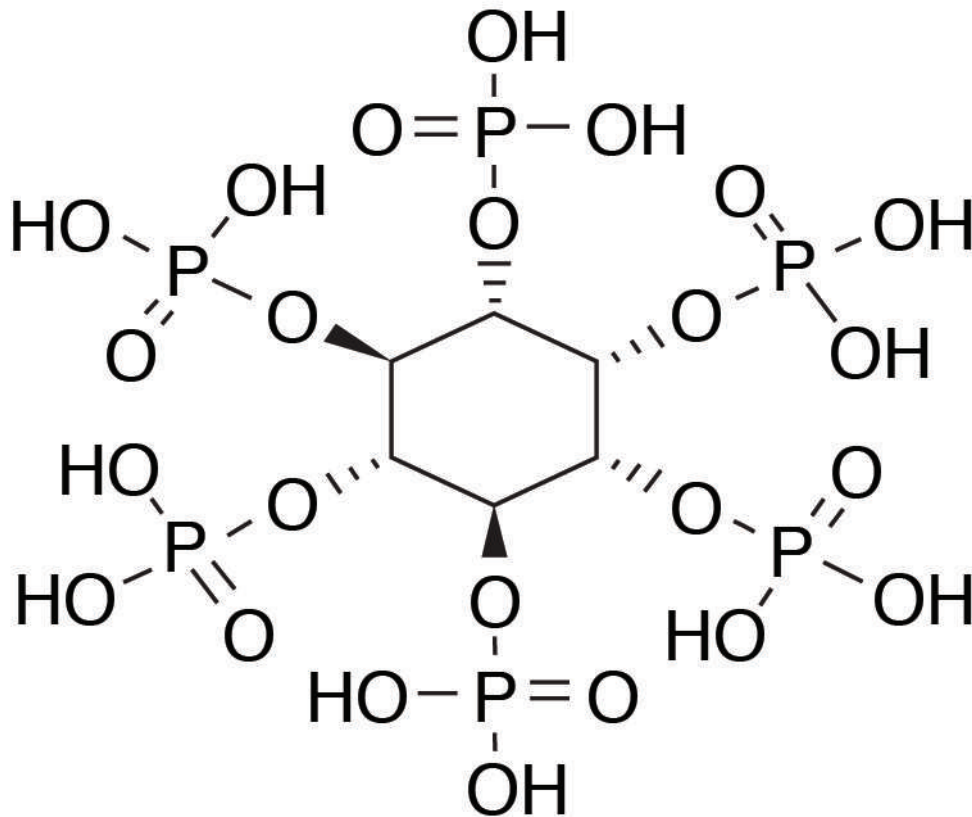


Figure 19.1. Phytic acid structure Source: Google

Microbial phytase is the most commonly used exogenous enzyme in the feed for monogastric animals. Phytase (myo-inositol hexakisphosphate phosphohydrolase) catalyzes the stepwise removal of phosphate from phytic acid or its salt phytate. Phytase can reduce the antinutritional effect of phytate and improve the digestibility of phosphorous (P), calcium, amino acids, and energy, as well as reduce the negative impact of inorganic P excretion to the environment. Phytase efficacy varies among feedstuffs due to phytate solubility. Overall, the efficacy of exogenous phytase enzymes is affected by both feed (source of enzymes, solubility, particle size) and animal (gut pH, retention time) factors.

NSPs are plant cell wall components and are the other major antinutrients present in cereal grains and oil seeds.

NSPs are a major part of fiber (fiber is the sum of NSP and lignin) and are regarded as feed components that are either poorly digested or have antinutritive properties. NSPs include water-soluble and insoluble components such as cellulose, lignin, arabinoxylan, glucan, and oligo- and polysaccharides containing different sugars such as arabinose, galactose, rhamnose, and mananose. The common NSP in rye and wheat are arabinoxylans, while barley and oats contain a high level of mixed-linked β -glucan (3%–4%; Figure 19.2).

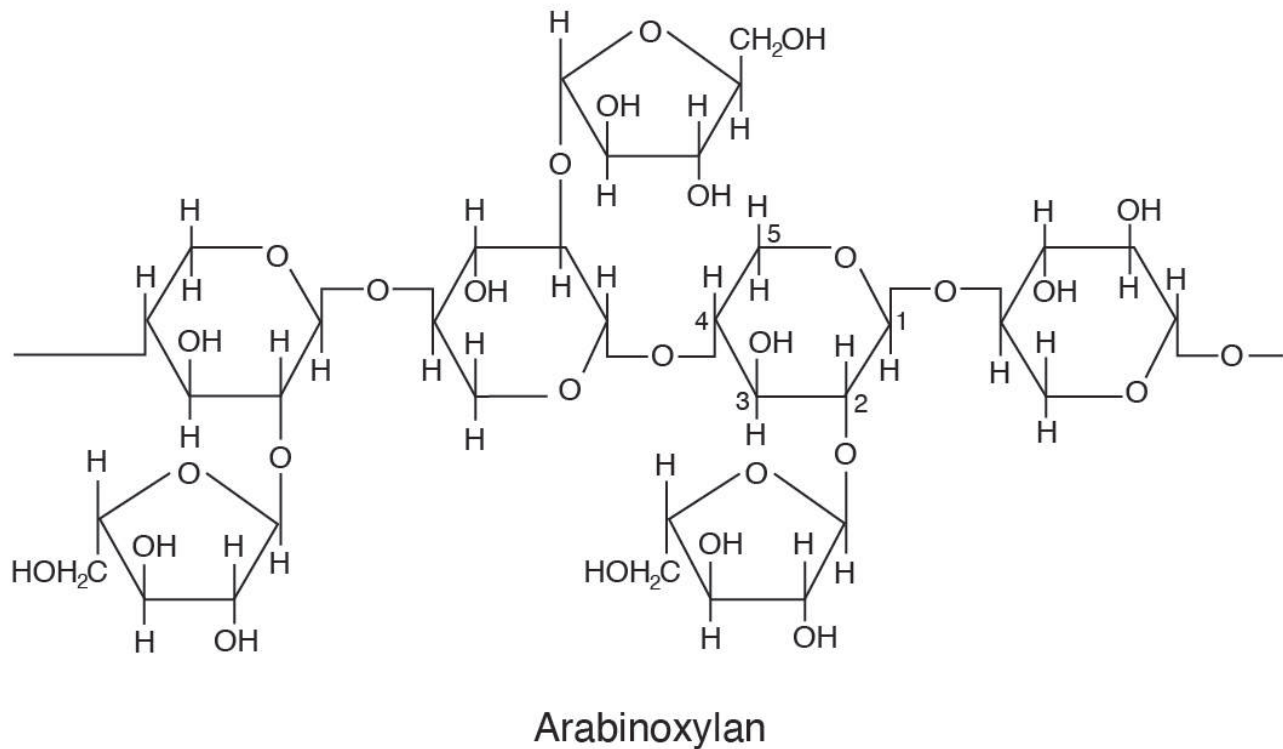


Figure 19.2. Nonstarch polysaccharides in wheat Source: Google

High levels of the NSP in the diet of monogastric animals are associated with reduced growth and performance. Specifically, high-NSP diets increase digesta viscosity, decrease digesta mixing and digestibility, and delay transit of digesta through the intestinal tract and thus are a greater opportunity for pathogenic bacterial overgrowth.

Carbohydrases are enzymes targeted to act on complex carbohydrates (NSPs) and sugars. Various enzymes, including xylanase, β -glucanase, and cellulase, have been reported to enhance the nutritive value of cereal byproducts for pigs and poultry. However, the efficacy of the enzymes depends on the solubility of the NSP and the complex nature of the carbohydrate. The future challenge for the feed industry is to produce highly efficacious enzymes that will lead to the utilization of both soluble and insoluble NSPs as an energy source for monogastric animals.

Carbohydrate-degrading enzymes act on complex carbohydrates and degrade them to smaller polymers and are used in monogastric animal diets high in NSP (e.g., wheat, barley, rye).

Pro/prebiotics

The term pro/prebiotics is used to designate organisms and or substances that contribute to healthy microbial balance in the intestine and include both living organisms and nonliving substances. Promoting favorable gut microflora is important especially when antibiotic growth promoters (AGPs) are phased out from animal feeds. Probiotics help prevent and control gastrointestinal pathogens and/or improve the performance and productivity of production animals through various mechanisms. Healthy microbial populations in the gastrointestinal (GI) tract are often associated with enhanced animal performance, reflecting more efficient digestion and improved immunity.

Pro/prebiotics function through enhancing the healthy microbial population of the gut.

Probiotics are live microorganisms and are of bacterial and nonbacterial origin. With the exception of certain yeast and fungal probiotics, most of the microorganisms used are of bacterial origin. Examples of bacterial probiotics are several species of *Lactobacillus*, *Bifidobacterium*, *Bacillus*, and *Enterococcus*. Nonbacterial (yeast or fungal) probiotics include *Aspergillus oryzae*, *Saccharomyces* and *Candida pintolopesii*.

Supplementation of probiotics has been shown to increase the population of beneficial microorganisms, such as *Lactobacilli* and *Bifidobacteria*. These healthy bacterial colonies then inhibit the growth of harmful microorganisms by producing substances (e.g., bacteriocins and/or organic acids) and by competitive exclusion. Thus probiotics serve as an alternative to antibiotic feed additives to manage and reduce pathogen load by reducing intestinal colonization and spread of other enteric pathogens. Probiotics have been shown to be effective in reducing postweaning diarrhea in piglets and morbidity and mortality in pigs.

Probiotics are live cultures of microorganisms that provide a balance of bacteria in the gut.
Lactobacillus and *Bifidobacterium* species are common choice as probiotics.

Prebiotics are nondigestible functional ingredients that selectively stimulate the growth of favorable bacteria in the gut of the host (food for the good microbes). Prebiotics are classified as disaccharides, oligosaccharides, and polysaccharides, and most commonly used prebiotics include lactulose, mannan-oligosaccharide (MOS), inulin, and fructooligosaccharides (FOS).

Prebiotics are nondigestible ingredients that serve as “food for the good microbes.”

Symbiotics may be defined as a mixture of probiotics and prebiotics that beneficially affects the host by improving the survival and implantation of health-promoting microbes in the gastrointestinal tract.

Antioxidants

With the advent of rations containing a high level of animal and vegetable fats, the requirement for antioxidant protection has become very apparent. Oxidation occurs in feeds and feedstuffs, resulting in the rancidity of fats and destruction of vitamins A, D, and E and amino acids, resulting in lowered energy values for the diet. Antioxidants are added in animal diets to improve the quality of feed and to increase shelf life. Antioxidants are of two types, natural and synthetic products. These products have the ability to prevent or terminate free radical production thereby providing protection against reactive oxygen species. Through these processes, antioxidants minimize rancidity of oils while enhancing shelf life along with feed quality.

Adding antioxidants to feed is an effective way of enhancing freshness and increasing shelf life.

Natural antioxidants include tocopherols, followed by other aromatic plant extracts (e.g., rosemary extract) and essential oils. The natural form of vitamin E is RRR- α -tocopherol. Synthetic vitamin E consists of a mixture of different stereoisomers and is called all-racemic- α -tocopherol or all-rac-tocopherol. One IU of vitamin E is equal to 1 mg of all-rac- α -tocopherol acetate. In the feed industry, vitamin E is available commercially as all-rac- α -tocopherol acetate and RRR- α -tocopherol acetate.

Other synthetic compounds with antioxidant properties include ethoxyquin (generic term for 1,2-dihydro-6-ethoxy-2,2,4-trimethylquinoline), butylhydroxytoluene (BHT), and butylhydroxyanisole (BHA). Ethoxyquin, however, has been demonstrated to be the most efficacious, followed by BHT and BHA.

Antibiotic Growth Promoters

Antibiotic growth promoters (AGPs) are substances administered at a low subtherapeutic dosage in the feed to destroy or inhibit bacterial growth and enhance performance.

The growth-promoting effects of antibiotics were discovered in the 1940s when chickens were fed feed containing byproducts of tetracycline fermentation. Since then, the use of growth promoters has been expanded to include a wide range of antibiotics that are applied to several species.

Antibiotic growth promoters are substances administered at a low subtherapeutic dosage in the feed for disease control and to enhance animal growth and performance.

The use of AGPs has risen with the intensification of livestock farming due to increased consumer demand and improvements in feed conversion to animal-derived foods. A wide range of veterinary antibiotic (natural, synthetic, or semisynthetic) compounds with antimicrobial activity are used as feed additives for disease control (prophylactic) or as growth promoters in livestock farming. These products offer proven benefits in animal health and food production, as well as a reduction of foodborne pathogens.

Several other biological and chemical substances are used to improve animal production performance.

Antibiotics are not the only substance added to animal feeds. Anabolic steroids and β -agonists have been largely used in intensive meat production to improve nutrient availability and lean meat deposition in food animals. The recombinant bovine somatotropin (rBST) has been used to enhance milk production in the dairy industry. β -agonists (cimaterol) are a class of substances that act on adrenergic receptors with anabolic effects in animals. Anabolic steroids enhance lean meat deposition and muscle protein accretion and metabolize fat stores, resulting in increased lean growth rates and are used in animal production systems to increase growth rate, feed efficiency, and carcass leanness. Anabolic feed additives have been banned in the European Union; however, in the United States, two hormones are approved as feed additives: melengestrol acetate for feedlot heifers and ractopamine for swine.

The excessive use of antibiotics, particularly at a subtherapeutic dosage to promote growth, can fuel the development of antibiotic-resistant bacteria, which puts the health of both animals and humans at risk. Antibiotics also enter the environment through animal excreta and wastewater, as do anabolic growth enhancers, which can be particularly detrimental to aquatic animals.

Several manufacturers of antibiotics have voluntarily withdrawn AGPs from the market for nontherapeutic use in the US. Many countries regulate, but do not prohibit, their use for growth promotion in animals.

Feeding AGP is prohibited in the European Union. Many countries regulate, but do not prohibit, their use for growth promotion in animals.

Others

In addition to the major additives listed previously, there are several other minor ingredients included in the feed depending on species, types of feed, and market. These include natural or synthetic pigmenters to enhance egg yolk and broiler skin color (e.g., carophyll, lutein, zeaxanthin), mold inhibitors to prevent mold growth (e.g., organic acids), mycotoxin binders that bind to the mycotoxins and prevent them from being absorbed, coccidiostats to prevent coccidiosis (protozoan infection in poultry), and pellet binders to reduce feed dust and help pellets adhere better.

Key Points

Enzymes

1. Phytases

Enzyme used to release P from phytic acid and increase P retention

2. Carbohydrases

Enzymes used to degrade complex carbohydrates

Probiotics

- Mixed cultures of live protective microbes
- Lactobacillus and Bifidobacterium

Prebiotics

- Nondigestible functional ingredients and “food for the good microbes”
- Oligosaccharides and polysaccharides
- Most commonly used

Antioxidants

- Prevent oxidation of lipids by scavenging free radicals (e.g., tocopherols)
- Provide protection from reactive oxygen species and increase shelf life.

Antibiotic Growth Promoters

- Prophylactic use, prevent the growth of harmful organisms
 - For example, Rumensin, Amprolium, ionophores
- Growth promotion
 - For example, Bacitracin, virginiamycin

Review Questions

1. List the benefits of including feed additives in animal diets.
2. Differentiate between probiotics and prebiotics.
3. A researcher added phytase enzyme to the diets of broiler birds and measured fecal and bone phosphorus (P) content and it turned out to be as follows:
 - a. Fecal P increase and bone P decrease
 - b. Fecal P decrease and bone P increase
 - c. Both fecal and bone P increase
 - d. Both fecal and bone P decrease
4. List any two feed additives that could be used to modulate gastrointestinal microbes and could be used as alternatives to antibiotic growth promoters in food animal production.
5. What are non starch polysaccharides (NSP) and how do they influence monogastric animals' growth and health?
6. What are antibiotic growth promoters (AGP) and list any two AGPs used in animal production.

XX. Measurement of Feed and Nutrient Utilization in Food-Producing Animals

This chapter discusses the importance of determining feed nutritional value, and different methods for assessing nutrient utilization in food-producing animals are emphasized.

New Terms

Apparent vs. true digestibility
Cannulation
Digestibility
Digestion trials
Fecal or total tract digestibility
Feed efficiency
Growth trials
Ileal digestibility
Rumen fistula

Chapter Objective

- To introduce and discuss some of the common methods used to measure feed and nutrient utilization in food animals

To develop feeding standards for food-producing animals, a knowledge and understanding of nutrients in the feed and its utilization by the animal is needed. Several animal and feed factors can affect nutrient utilization. These include species (ruminant vs. monogastrics), age (young vs. old), physiological (young vs. pregnant

or lactating) state of the animal, disease conditions, type of feed or processing (pellet vs. ground), and presence of antinutritional factors in feed.

What Affects Nutrient Utilization?

- Species
- Age of the animal
- Type of digestive tract
- Level and balance of nutrients
- Physical form of feed

Methods used for nutrient evaluation of feed are in general similar for all classes of livestock. In this chapter, some of the common methods used for assessing nutrient utilization are addressed.

Growth trials are often conducted to compare weight gain upon feeding different feeds. Usually growth trials involve ad libitum feeding of the experimental diets and are compared with a standard (basal) diet of known nutritive quality for a period of time.

Total feed consumption and weight gain are monitored and feed efficiency (weight gain per unit of feed; total weight gain/total feed consumed) is calculated. Although easy and simple to conduct and calculate, results obtained can be affected by several factors. Feed palatability and other physical characteristics and nutrient content can affect voluntary feed intake. Similarly, weight gain includes

Types of Feeding Trials

- Growth trials
- Digestion trials

body tissue mass, including ingesta and water, and can lead to errors in calculation, and thus the results obtained may not be a true reflection of the test diet.

Feed efficiency is weight gain per unit of feed consumed.

Digestion Trials

Evaluation of the feed is more precise if expressed in terms of the digestibility of each of the nutrients rather than its total content in the feed. Therefore, knowledge and understanding of nutrient digestibility is a necessary step in evaluating feedstuffs for ration formulation. Digestion trials are conducted to determine the proportion of the nutrients in the feed that are digested and absorbed from the gastrointestinal (GI) tract.

Digestibility is defined as the fraction of a nutrient ingested that is digested and absorbed by the animal.

Animals are fed the test diets for a period of time for several days and fecal samples are collected and are analyzed for excreted nutrient loss. Total nutrient intake from feed and nutrient disappearance at the end of the digestive tract (fecal loss) is calculated. This conventional method of feeding test diets and fecal collection methods are called fecal digestibility, or total tract digestibility test. However, results obtained through this study are not a true reflection of the digestibility of the test diet. A fraction of the nitrogen, fats, carbohydrates, and inorganic elements appearing in the feces is from endogenous sources. Thus fecal samples may contain undigested feed along with sloughed-off intestinal mucosal cells and digestive enzymes. Fecal nutrients originating from sloughed off intestinal cells and unused digestive enzymes of the animal are called endogenous nutrients. Since not all the nutrients in the feces were derived from the test diet, fecal digestibility is termed apparent digestibility, which represents the difference between the amount ingested and the amount appearing in feces.

Apparent digestibility (%) = $\frac{\text{Nutrient intake} - \text{Nutrient loss in feces}}{\text{Nutrient intake}} \times 100$

Indicator Methods

Although not a true measurement, apparent digestibility is used widely. Monitoring feed intake and fecal collection can be time consuming, laborious, and difficult to conduct in grazing animals. To overcome this, nonabsorbed indicators or markers (acid insoluble ash, chromic oxide) are added to the feed employed, and apparent digestibility is calculated as follows. By using this method, by grabbing a sample of feces from the pen (or the rectum), digestibility can be calculated in animals kept in confinement or on grazing ruminants.

$$\text{Apparent digestibility (\%)} = 1 - (\% \text{ marker in feed} / \% \text{ marker in feces}) \times (\% \text{ nutrient feces} / \% \text{ nutrient feed})$$

Apparent versus True Digestibility

The true digestibility of a nutrient is the proportion of the dietary intake that is absorbed from the GI tract, excluding endogenous contribution. Assessing endogenous contribution is not that easy.

Digestibility calculated after removing the endogenous nutrients contribution is called true digestibility.

In monogastric animals, in the hindgut, nutrients that are not digested and absorbed in the small intestine may be fermented by bacteria in the caeca and colon. For example, amino acids produced during protein fermentation in the hindgut are not utilized by the host animal as a source of amino acids. Therefore, ileal digestible amino acids provide a better representation of amino acids that become available to the animal than amino acids digested over the total digestive tract. Therefore, the digestibility of protein and amino acids at the ileal level is a valuable characteristic of the nutritional value of feed ingredients.

Digestibility of protein and amino acids at the end of the ileum is considered a better indicator of the availability of protein and amino acids than fecal digestibility.

Ileal digestibility can be determined with the use of animals fitted with an ileal cannula (special tubes fitted surgically at the end of ileocecal junction) or by collecting ileal digesta at slaughter (done in poultry). Because a quantitative collection of ileal digesta is not possible, inert digestibility markers (e.g., chromic oxide) are also used to estimate nutrient digestibility. Such markers should not interfere with digestive processes, should not be absorbed, and should behave similarly to the nutrient of interest in the GI tract. Although better estimates exist, expertise in surgery and other facilities are needed for these types of measurements.

Due to the high cost of conducting digestion trials, in vitro techniques simulating rumen fermentation are conducted in ruminant animals fitted with rumen fistula. These methods are used for screening feedstuffs or studying rumen function and metabolism.

Key Points

1. Both animal and diet factors can affect nutrient utilization.
2. Feeding trials are commonly done to measure digestibility (ability to break down nutrients and absorb them into the blood). Feeding trials require large numbers of animals. Feed intake records and body weight gain are required to calculate feed efficiency and digestibility. Digestibility is defined as the proportion of the nutrient that is absorbed by the animals. However, it does not provide any information on why certain diets are better than others.
3. Calculation of digestibility requires a total collection of feed intake and fecal output, which is difficult to do, especially with grazing animals. An indirect method was developed. This method utilizes either an internal (compound exists in feed) or an external indicator (compound is added to feed) as the basis to calculate the proportion of nutrients that are absorbed by animals. This dictates that an indicator must be able to pass through the gastrointestinal (GI) tract at the same flow rate as other nutrients, and it must not be digested and absorbed by animals. Chromic oxide and some rare earth elements (acid insoluble ash) are commonly used external indicators.
4. Since not all the nutrients in the feces were derived from a dietary source, this digestibility is termed apparent digestibility. Fecal nutrients from sloughed-off intestinal cells and digestive enzymes of the animal are called endogenous nutrients. Digestibility calculated after removing the endogenous nutrients is called true digestibility.
5. Cannulated animals are also used to determine digestibility of nutrients. Cannulations (special tubes fitted surgically into the gastrointestinal tract) are done to assess nutrient (e.g., protein, amino acid) digestibility (apparent vs. true) of feed.
6. Other in vitro studies simulating rumen fermentation are conducted in ruminant animals fitted with a rumen fistula. Animal digestibility trials can be time consuming and costly but give a better estimate of the digestibility and availability of nutrients and help in ration formulation.

Review Questions

1. List the factors that can affect nutrient utilization in animals.
2. Differentiate between:
 - a. Apparent vs. true digestibility
 - b. Fecal vs. ileal digestibility
 - c. Growth trial vs. digestion trials

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