HEINZ
HANDBOOK
of
Nutrition

9th EDITION

**Edited by David L. Yeung, Ph.D. and
Idamarie Laquatra, Ph.D., R.D.**

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HANDBOOK OF NUTRITION

NINTH EDITION

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Heinz Corporate Research Center

H.J. HEINZ COMPANY

Edited by

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General Manager - Global Nutrition
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This publication represents recent research on the subject matter contained herein. It is not
intended as a guide for those whose diets require accurate measurement of daily nutritive
intake. The information contained herein reports the averages of analysis of random samples
representing the products named.

FORWARD

During 2003, the H. J. Heinz Company adopted its "Global Operating Principles," which formally set forth the company's long-held commitment "to providing superior food products that enhance the lives and well-being of people everywhere." This ninth edition of the *Heinz Handbook of Nutrition* is testament to the company's constant dedication to superior nutrition and pure foods. In fact, company Founder Henry J. Heinz stood as the sole commercial food processor to back the 1906 Pure Food and Drug Act.

Today, our brands are among the most recognized in the world. They are sold on all inhabited continents and enjoyed in every time zone. As global purveyors, we are proud to share our more than 130 years of experience with health care professionals, scientists and researchers worldwide. Several generations of Heinz technologists, dietitians and nutritionists have made this handbook possible. We are proud to share it with you.

Bill Johnson

William R. Johnson
Chairman, President and Chief Executive Officer
April 2003

PREFACE

The Ninth Edition of **Heinz Nutritional Data** (now renamed Heinz Handbook of Nutrition) is a continuation of work started in 1934, when the H.J. Heinz Company published the first edition of "Nutritional Charts". This classic brochure of descriptive and tabular information relating to the nutritive value of foods was compiled for the guidance of physicians, nutritionists, dietitians, public health workers and home economists to aid them in devising diets for both the sick and healthy.

Through twelve editions, "Nutritional Charts" grew rapidly in scope and volume until it became necessary to change the format to book form. Thus, in 1949 **HEINZ NUTRITIONAL DATA** became the greatly expanded successor to the original "Nutritional Charts" incorporating much additional material from the fields of food, nutrition, biochemistry and medicine.

Nine editions and several revised printings attest to the wide and favorable reception given this publication. Most gratifying has been its adoption as a teaching aid by certain medical schools in this country. In this new edition, the RDAs have been replaced where available with the DRIs. Furthermore, a new chapter on Functional Foods has been added. This reflects the current interest in this area of nutrition. It is our hope that the **HEINZ HANDBOOK OF NUTRITION**, the title which better reflects the content of the book, will continue to be a useful reference manual for those concerned with the theory and practice of the science of nutrition.

ABOUT THE EDITORS

David Lawrence Yeung, Ph.D.

Dr. David Yeung is the General Manager-Global Nutrition Service, H.J. Heinz Company. He received his doctorate degree from the Department of Nutritional Sciences, Faculty of Medicine, University of Toronto, Toronto, Canada.

He was an associate professor in Applied Human Nutrition at the University of Guelph, Guelph Ontario, Canada before joining the H.J. Heinz Company of Canada. Dr. Yeung remains academically active. He is an adjunct professor in the Department of Nutritional Sciences, Faculty of Medicine, University of Toronto and has been an honorary professor in universities in Canada and the People's Republic of China.

Dr. Yeung has published over 70 articles in refereed scientific journals. He authored the book "Infant Nutrition- A Study of Feeding Practices and Growth From Birth to 18 Months"; and edited the monographs "Essays in Pediatrics Nutrition" and "Heinz Nutritional Data". The latter was revised to "Heinz Handbook of Human Nutrition" which was published in 1995. He has served on numerous scientific committees in Canada and the US.

Dr. Yeung has wide international experiences. He is the President of the Heinz Institute of Nutrition. He has, in this capacity, participated in programs in Australia, Canada, China, Czech Republic, Hungary, India, Indonesia, Malaysia, Poland, Portugal, Russia, Singapore, Spain, Thailand, The Philippines, USA and Venezuela. He has served as a consultant to the Food and Agriculture Organization (FAO), The Micronutrient Initiatives (MI), United States of America Agency for International Development (USAID).

In 1997, Dr. Yeung received the Earle Willard McHenry Award from the Canadian Society for Nutritional Sciences in recognition of distinguished service in nutrition.

ABOUT THE EDITORS

Idamarie Laquatra, Ph.D., R.D.

Idamarie Laquatra earned her graduate degrees in nutrition from The Pennsylvania State University. A licensed registered dietitian, Dr. Laquatra has experience in the clinical, academic and business fields. She has extensive training in nutrition counseling, has conducted research in this area, and authored and co-authored articles in peer-reviewed journals and chapters in texts.

Prior to earning her advanced degrees, Dr. Laquatra worked as a clinical dietitian in the hospital and nursing home settings. After completing her PhD, she became a postdoctoral fellow in Preventive Cardiology at the University of Medicine and Dentistry of New Jersey. In addition to her postdoctoral studies, she served as adjunct faculty at Montclair State University in Upper Montclair, New Jersey.

Her food industry experience includes eight years at the H.J. Heinz Company, first as Nutritionist for Heinz USA and then as Manager of Nutrition for Weight Watchers Food Company. She joined Diet Center, Inc. in 1992 as Vice President of Scientific Affairs and Training. In 1995, Dr. Laquatra began her nutrition consulting business, contracting with clients in both the non-profit and for-profit sectors.

Dr. Laquatra is also an active member of the American Dietetic Association (ADA). She served as President of the Pittsburgh Dietetic Association, was elected Pennsylvania Delegate, and was appointed Chair of the Advisory Committee of ADA's Food and Nutrition Conference and Exhibition in 2002. The Pennsylvania Dietetic Association presented her the Keystone Award in 1998 and the Outstanding Dietitian of Pennsylvania award in 2002.

ACKNOWLEDGMENTS

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CHAPTER 1

PROTEINS AND AMINO ACIDS

Proteins serve as indispensable constituents of every living cell. They participate in every aspect of cell metabolism. They function in growth, maintenance and repair of cells by their action as enzymes catalyzing metabolic reactions, as structural proteins maintaining the shape of the cell, as hormones regulating cell activities, as antibodies providing a defense mechanism, and as contractile proteins, transport proteins, toxins and components of intracellular structures. Proteins may form biologically significant compounds through conjugation with other molecules, such as, chromo-proteins, lipoproteins, nucleoproteins, glucoproteins and metalloproteins. Plasma proteins are also important in maintaining fluid and acid-base balance. Digestive processes depend upon enzymes from the stomach, intestinal glands and pancreas. Proteins may serve as an energy source providing 4 kcal (17 kJ) per gram. The protein content of some common foods is shown in Table 1-1. The total amount of body protein is approximately 19% of flesh weight; 45% of this protein is present in muscle (Table 1-2).

Proteins are composed of amino acids. These are low molecular weight compounds which are characterized by the presence of a terminal carboxyl group (-COOH) and an amino group ($-NH_2$) in the alpha-position. In proteins, amino acids are connected by peptide linkages which are formed between the carboxyl and amino group of two adjacent amino acids. In addition, disulfide bonds may form between the sulfur moieties of two sulfur-containing amino acids in the polypeptide chain.

The sequence of amino acids in the peptide chain is referred to as the primary structure of the protein. The secondary structure is determined by hydrogen bonding between different amino acids within the polypeptide chain. As a result of its secondary structure, some or all of the protein will take on an alpha-helix shape, a beta-configuration (pleated sheet structure) or a random configuration. The protein is further shaped and stabilized by hydrogen and disulfide bonds as well as electrostatic and hydrophobic attraction. Together these account for the protein's tertiary struc-

ture. The two basic shapes for protein, as determined by tertiary structure, are globular and fibrous. Some proteins also have a quaternary structure in which two or more similar polypeptide subunits are joined together as a result of electrostatic forces. Some proteins also have a metal ion or an organic cofactor which is essential for their biological activity.

Digestion of Protein

Most dietary proteins are ingested as large molecules. Only negligible amounts of free amino acids are naturally present in food. In order for absorption to occur, protein must be broken down to free amino acids and to di- and tripeptides. The first step in

Table 1-1

Protein Content of Some Common Foods

Food Items	Protein (g/100g)
Meat/Fish/Poultry	
pork, loinblade, roasted, lean and fat	23.7
beef, chuckblade, cooked, lean and fat	27.1
chicken, broiler or fryer, light and dark meat, with skin, roasted	27.3
salmon, chinook, smoked	18.3
tuna, canned white, in oil	26.5
sardines, Atlantic, canned in oil	24.6
Dairy and Egg Products	
cheese, cheddar	24.9
milk, whole (3.3%), fluid	3.3
milk, skim, fluid	3.4
milk, skim, dry, regular	36.2
egg, chicken, whole	12.5
Cereal and Grain Products	
flour, wheat, whole grain	13.7
rice, brown, medium grain, cooked	2.3
rice, white, medium grain, cooked	2.4
bread, white	8.2
bread, whole wheat	9.7
Vegetables	
peas, mature, sprouted, boiled	7.1
lentils, boiled	9.0
soybean, mature, boiled	16.6
potatoes, raw	2.1

Source: U.S. Department of Agriculture, USDA Nutrient Database for Standard References, Release 13, Agricultural Research Service, 1999.

Table 1-2

Distribution of Protein Stores in the Human Body
Male 168.5 cm, 53.8 kg.

	G	%
Total protein (N x 6.25)	10,000	100.00
Striated muscle	4,680	46.80
Skeleton	1,864	18.64
Skin	924	9.24
Adipose tissue	361	3.61
Estimate in Blood:		
Hemoglobin	750	7.50
Albumin	250	2.50

protein digestion involves the hydrolysis of protein into shorter chain peptides by the gastric proteolytic enzyme pepsin. Pancreatic enzymes released into the duodenum further hydrolyze the protein fragments to tripeptides, dipeptides and free amino acids. The pancreatic enzymes include carboxypeptidase A and B, elastase, trypsin and chymotrypsin. Each enzyme has a specific action as shown in Table1-3.

Intestinal Absorption of Amino Acids and Di- and Tripeptides

Transport of free amino acids into the intestinal mucosa is believed to occur by a sodium and energy-dependent carrier mediated process with some specificity for neutral, basic and acidic classes of amino acids. Absorption of di- and tripeptides, however, is the major route of amino acid uptake. The peptides are hydrolyzed to free amino acids by the action of dipeptidase and amino peptidase within the mucosal cell and then released into the portal circulation to be carried to the liver.

Plasma amino acid levels are less responsive to dietary influx than is the portal circulation as the liver plays an important role in monitoring and metabolizing absorbed amino acids. The liver is the main site of catabolism for all indispensable amino acids except the branched-chain amino acids which are mainly catabolized by muscle and kidney tissues. Plasma amino acid levels are also affected by dietary carbohydrate through the action of insulin. Insulin lowers plasma amino acid levels, particularly the branched-chain amino acids, by promoting their passage into

Table 1-3

Enzymes in Protein Digestion

SOURCE	PROENZYME	ACTIVATOR	ENZYME	ACTION
Stomach	Pepsinogen	HCl Pepsin	Pepsin	- not highly specific but preferentially splits bonds adjacent to aromatic amino acids and leucine.
Pancreas	Procarboxypeptidase A	Trypsin	Carboxypeptidase A	- cleaves bonds from the C-terminal end of the peptide when the carboxyl group is an aromatic or branched chain amino acid.
	Procarboxypeptidase B	Trypsin	Carboxypeptidase B	- cleaves bonds from the C-terminal end of the peptide when the carboxyl group is arginine or lysine.
	Trypsinogen	Enterokinase	Trypsin	- cleaves bonds within the peptide chain when the carboxyl group is arginine or lysine.
	Proelastase	Trypsin	Elastase	- cleaves bonds within the peptide chain when the carboxyl group is a neutral aliphatic amino acid.
	Chymotrypsinogen	Trypsin	Chymotrypsin	- cleaves bonds within the peptide chain when the carboxyl group is an aromatic amino acid.
Intestinal Cells			Aminopeptidase	- cleaves amino acids from the N-terminal end of the peptide chain.
			Dipeptidase	- hydrolyzes dipeptide fragments to free amino acids.

muscle. The plasma amino acid or extracellular amino acid pool represents the net effect of intestinal absorption versus hepatic and extrahepatic uptake and release.

There are no protein reserves as such. Because protein breakdown is a constant activity leading to catabolism of amino acids, there is a constant dietary requirement for amino acids even in adults. Body proteins are broken down to a greater extent when their dietary supply is inadequate. The proteins in the liver are most labile followed by muscle proteins.

Protein Requirements

The continuous breakdown of protein in tissues necessitates the requirement for protein in the diet, even in adults who have ceased growing. Dietary requirements for protein are based on the needs for both total amino nitrogen and indispensable amino acids and presently are set at 1 g/kg/day for normal adults. This requirement for protein is even greater for anabolic processes such as growth, pregnancy, lactation, rehabilitation after a debilitating disease, or malnutrition.

During pregnancy, protein requirements are increased for two reasons: first, to support growth of fetal and maternal tissues and second, to maintain these additional body stores. Estimates of protein requirements are based on growth and body composition at the end of each trimester due to rapid fetal growth, especially during the last trimester of pregnancy. The protein requirements for lactation are based on the average nitrogen content of breast-milk.

As well, growing children require an increased protein intake and thus have a proportionally greater protein requirement for growth. This increase is relatively small compared to the requirement needed to replace proteins which are turning over. See chapter 7 for a complete listing of protein requirements during different stages of pregnancy, lactation and growth.

Indispensable and Dispensable Amino Acids

There are twenty-two different naturally occurring amino acids. Some of the amino acids required for protein synthesis cannot be synthesized by the human body and must be supplied by the diet. These amino acids are referred to as indispensable. The structures

of the indispensable and dispensable amino acids are displayed in Figure 1-1.

The dispensable amino acids are not necessarily of less biological importance. They may be provided, per se, by ingested food, or, if missing in the diet, may be synthesized from metabolic precursors in the body.

Figure 1-1

Essential Amino Acids*

Histidine

H₂
+N
N
H CH₂
R

Isoleucine

CH₃
CH — CH₂ — CH₃
R

Leucine

CH₃
CH₂ — CH — CH₃
R

Lysine

NH₂
(CH₂)₄
R

Methionine

CH₂ — S — CH₃
CH₂
R

Phenylalanine

CH₂
R

Threonine

CH₃
CH — OH
R

Tryptophan

H — N
CH₂
R

Valine

CH₃
CH — CH₃
R

Non-Essential Amino Acids*

Alanine

CH₃
R

Arginine

+NH₂
HN — C — NH₂
(CH₂)₃
R

Asparagine

O
CH₂ — C — NH₂
R

Aspartic Acid

CH₂ — COO-
R

Cysteine

CH₂ — SH
R

Glycine

H
R

Cystine

CH₂ — S — S — CH₂
R   R

Glutamic Acid

CH₂ — CH₂- — COO-
R

Proline

COO+
N+

Hydroxyproline

HO   COO-
N+

Tyrosine

OH
CH₂
R

Serine

CH₂ — OH
R

Glutamine

O
CH₂ — CH₂ — C — NH₂
R

* (R represents H₃N+ — CH — COO-)

6

Their carbon skeletons are derived from intermediate products of carbohydrate or fat metabolism, usually in the form of ketoacids. An amino group becomes attached to this molecule converting it to the needed amino acid. The amino group is generally derived from another amino acid through transamination or from the ammonia available from deamination of dietary amino compounds or urea breakdown in the gut.

The estimated daily indispensable amino acid requirements for humans are shown in Table 1-4. Of the sulfur-containing amino acids, cysteine may be dispensable provided enough of its precursor, methionine, is present. Similarly, phenylalanine serves as a precursor for tyrosine; tyrosine requirements will be met provided there is sufficient phenylalanine. Histidine has been shown to be indispensable for the human infant but not for the human adult.

Table 1-4				
Estimated Amino Acid Requirements of *Humans*				
Requirements (per kg. of body weight) mg. per day				
Amino Acid	**Infants (3-4 mos.)**	**Children (~2 yrs.)**	**Children, (10-12 yrs.)**	**Adults**
Histidine	28	NK	NK	17
Isoleucine	70	28	12	42
Leucine	161	42	16	70
Lysine	103	44	12	51
Total S - containing amino acids [†]	58	22	10	26
Total aromatic amino acids [††]	125	22	16	73
Threonine	87	28	8	35
Tryptophan	17	4	3	11
Valine	93	25	14	48
Total without histidine	714	352	214	84

* Food and Nutrition Board. **Recommended Dietary Allowances**, 10th ed. National Academy of Sciences-National Research Council, Washington, D.C., 1989, p. 57.

† Methionine plus cystine

†† Phenylalanine plus tyrosine

NK not known

The quality of a food protein can be estimated by the Chemical Scoring method. This is a comparison of the amount of each indispensable amino acid per gram of protein with that in a reference high quality protein, usually egg or milk. The chemical score is determined by the limiting amino acid; that is, the amino acid which is present in the smallest proportion relative to its level in the reference protein. The higher the chemical score, the better the quality of the protein and the more effective it will be in meeting the body's needs.

If one or more of the indispensable amino acids is not present in sufficient quantities protein synthesis will be limited by the most limiting indispensable amino acid. Animal proteins such as meat, fish, milk and egg contain all the indispensable amino acids in amounts adequate to support human growth and normal physiological functions (Table 1-5). Generally, plant proteins are of lower quality than animal proteins as they tend to be low in one or more of lysine, threonine, tryptophan and methionine.

A protein of low quality can be improved by supplementation with another protein that contains high amounts of the indispensable amino acid lacking in the first protein. Complementary proteins, therefore, consist of a mixture of two or more proteins each having different limiting amino acids. When eaten together, complementary proteins supply all the indispensable amino acids in sufficient quantities. For example, grains, which are low in lysine, can be complemented by legumes or animal proteins which are high in lysine. Translated to actual foods, grain-legume combinations common in certain countries are beans and rice, and baked beans on toast.

Metabolism of Amino Acids

A general scheme of protein metabolism is shown in Figure 1-2. Absorbed amino acids may be used for tissue growth, maintenance and repair as well as synthesis of other nitrogen compounds and dispensable amino acids. After the anabolic needs of the body have been met, amino acids in excess are catabolized for the production of energy or if energy requirements have been met, are used in the synthesis of carbohydrate and fat.

The final products of protein catabolism include carbon dioxide, water, energy (as the high energy compound ATP), urea and

Table 1-5

Essential Amino Acid Content of Some Common Foods (mg/100 g of edible portion)

Food Items	Tryptophan	Threonine	Isoleucine	Leucine	Lysine	Methionine	Phenylalanine	Valine	Arginine	Histidine
Meat/Fish/Poultry										
beef, round, raw	205	697	713	1332	1388	388	630	806	1122	529
chicken, with skin, roasted	305	1128	1362	1986	2223	726	1061	1325	1711	802
liver, pork, cooked	366	1107	1320	2319	2007	645	1274	1607	1603	708
salmon, Atlantic, raw	222	870	914	1613	1822	587	775	1022	1187	584
Dairy and Egg Products										
cheese, cheddar	320	886	1546	2385	2072	652	1311	1663	941	874
milk, human	17	46	56	95	68	21	46	63	43	23
milk, cow's whole	46	149	199	322	261	83	159	220	119	89
egg, white only	130	478	594	887	716	362	615	670	573	237
egg, whole	152	600	682	1067	897	390	664	761	749	296
Cereal and Grain Products										
Bread, whole wheat	140	296	376	670	302	155	463	443	449	224
flour, wheat, all purpose, enriched	127	281	357	710	228	183	520	415	417	230
flour, rye, medium	106	325	362	649	325	140	475	470	425	209

Continued on next page

Table 1-5 *Continued*

Essential Amino Acid Content of Some Common Foods (mg/100 g of edible portion)

Food Items	Tryptophan	Threonine	Isoleucine	Leucine	Lysine	Methionine	Phenylalanine	Valine	Arginine	Histidine
Nuts and Seeds										
cashews, roasted	237	592	731	1285	817	274	791	1040	1741	399
sesame seeds dried	388	736	763	1358	569	586	940	990	2630	522
peanuts, roasted with skins	230	811	833	1535	850	291	1227	993	2832	599
Legumes										
soybeans, mature cooked	242	723	807	1355	1108	224	869	831	1291	449
lentils, cooked	81	323	390	654	630	77	445	448	697	254
peas, green, cooked	37	201	193	320	314	81	198	232	423	105

ammonia. Other nitrogenous waste products are creatinine and uric acid derived from creatine and purines respectively.

The main pathways of amino acid metabolism (Figure 1-3) include the following:

Transamination
The first step in the catabolism of amino acids is usually removal of the amino group by transamination. In this reaction the amino group of the amino acid is reversibly transferred to the carbon atom of a ketoacid. The reaction is catalyzed by an appropriate transaminase with pyridoxal phosphate serving as the co-enzyme. For a number of amino acids transamination results in the transfer of their amino group to form L-glutamate.

Oxidative Deamination
The L-glutamate synthesized from transamination undergoes

Figure 1-2

General Scheme of Protein Metabolism
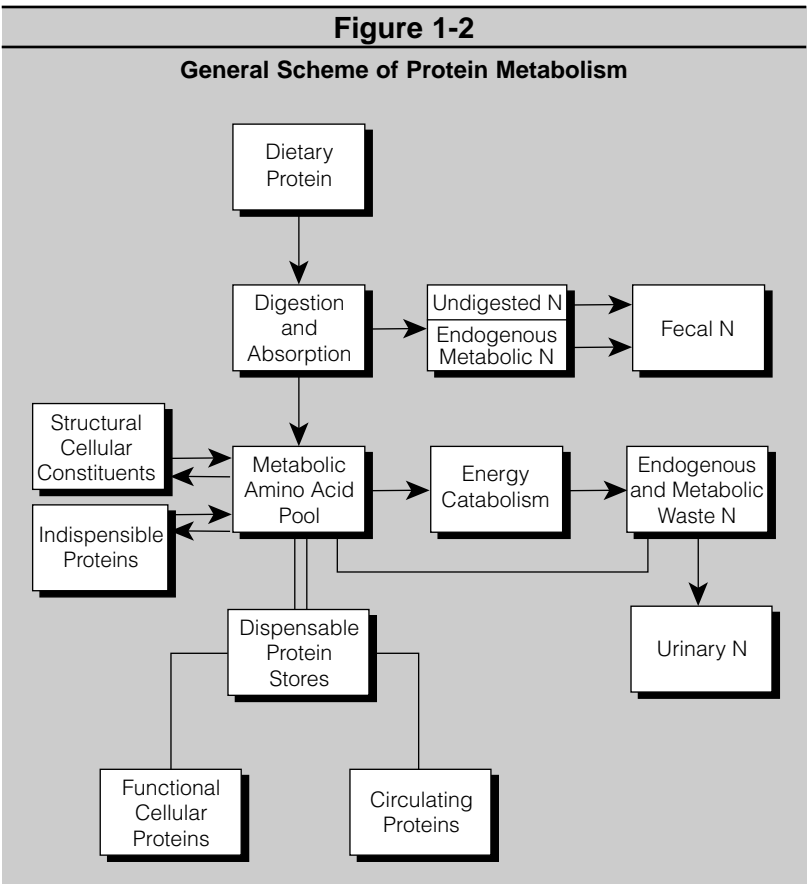
oxidative deamination to form ammonia. Ammonia, a potentially toxic by-product, may then be used for the synthesis of urea which is ultimately excreted. Oxidative deamination of other amino acids to form ammonia is catalyzed by L-amino acid oxidase.

Urea Cycle
The reactions of the urea cycle take place in the liver. The urea formed is derived from the amino groups donated by carbamoyl phosphate and arginine.

Glucose-Alanine Cycle
The glucose-alanine cycle serves as the main pathway by which amino groups from amino acids in skeletal muscle are transported to the liver for conversion to glucose. In skeletal muscle, amino acids donate their amino group to pyruvate forming alanine by

Figure 1-3

Schematic Outline of Amino Acid, Carbohydrate and Fat Metabolism
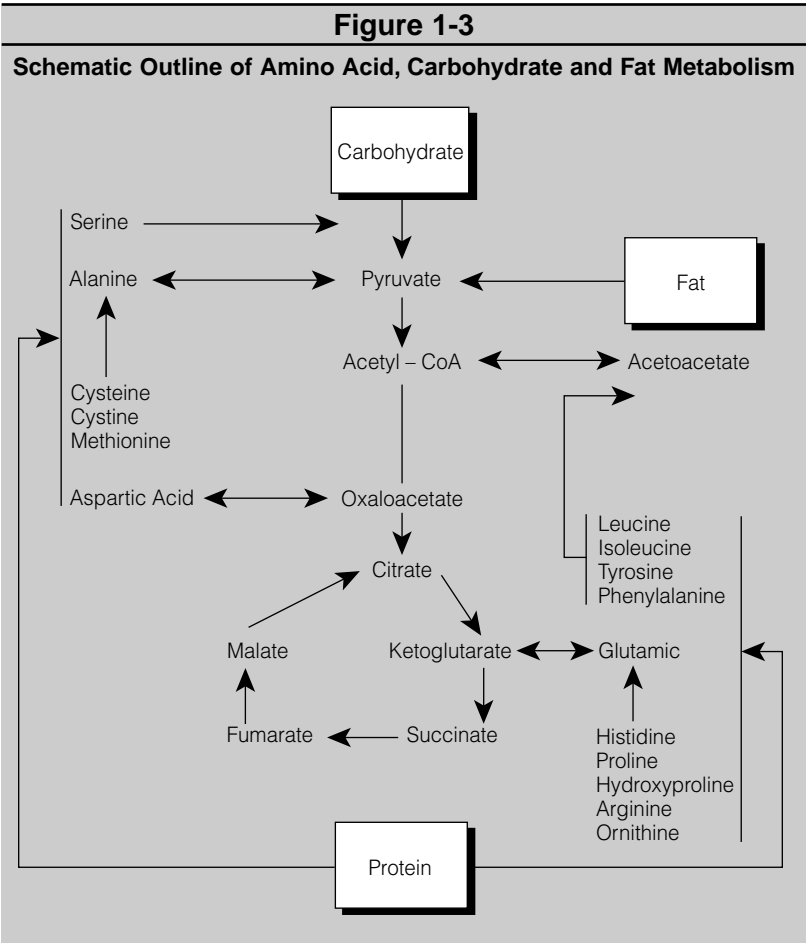
the action of alanine amino transferase. Alanine is released into the blood for uptake by the liver. There, transamination is reversed resulting in synthesis of pyruvate and ultimately glucose. Glucose may be released from the liver and returned to muscle where it is converted back to pyruvate, thus completing the cycle. Glucose derived from the alanine cycle may also be used as an energy source for the liver and other tissues.

Citric Acid Cycle

In the catabolism of amino acids, the deaminated carbon skeleton enters the citric acid (Krebs) cycle. The citric acid cycle and the oxidative phosphorylation reactions serve as the final common pathway of carbohydrate, fat and protein catabolism. Oxidation of citric acid cycle intermediates by the electron transport system is coupled with the formation of energy in the form of ATP. Carbon dioxide and water are by-products of the reactions.

Under conditions of energy deficit the deaminated carbon skeletons are catabolized to produce energy. When energy needs are met by carbohydrate and fat intake, the deaminated carbon skeletons may be converted to carbohydrate and fat. All amino acids except leucine are glycogenic, that is, they may contribute to the synthesis of glucose. Leucine is ketogenic as it may serve as a precursor for ketone bodies and fatty acids but not for glucose synthesis. Some amino acids function in both capacities. These are isoleucine, phenylalanine, and tyrosine.

Individual Characteristics of Amino Acids

The most important function of amino acids lies in the fact that they are the building blocks of tissue proteins. Several of them have additional and individual physiological functions which deserve brief mention.

Glycine is an important precursor for the synthesis of purine bases, porphyrins, creatinine, glutathione and conjugated bile salts. During periods of rapid growth the demand for glycine may be relatively very high. Glycine is utilized by the liver in the elimination of toxic phenols. For instance, benzoic acid, when ingested orally, is conjugated with glycine in the liver and excreted as hippuric acid in the urine.

Glutamic Acid plays an important role in the metabolism of ammonia and in the formation of the neurotransmitter gamma-

aminobutyric acid (GABA) in the nervous system. It is a component of the vitamin folate. Glutamic acid is the predominant amino acid in wheat protein (gliadin). As monosodium glutamate (MSG), it is a powerful flavor enhancer.

Phenylalanine and tyrosine serve as precursors for the hormones epinephrine, norepinephrine and thyroxine. They are also the origin of the pigment melanin found primarily in the skin. The body can convert phenylalanine to tyrosine, but not vice-versa. Hence, phenylalanine is an indispensable amino acid but tyrosine is not.

Histidine is indispensable for the human infant but its essentiality for the adult has not been shown except in certain disease states such as uremia. By losing its carboxyl group, histidine is converted to histamine which is a physiologically important substance found in many tissues. Histamine stimulates production of hydrochloric acid in the stomach. It is also a powerful vasodilator. The release of histamine in the skin accounts for urticaria which may result from food allergy. A metabolite of histidine, 3-methyl-histidine, is excreted in the urine and provides a measure of muscle protein breakdown.

Tryptophan serves as a precursor for the vitamin nicotinic acid (niacin) as well as for serotonin (5-hydroxytryptophan), a neurotransmitter, which is present in many tissues including the brain.

Proline and hydroxyproline consist of a pyrrole ring. This same ring structure is found in the porphyrins which form hemoglobin and the cytochromes. The porphyrin molecule and also that of cyanocobalamin (vitamin B_{12}), contain four pyrrole rings.

Arginine is essential for the formation of urea in the liver by the process known as the ornithine-arginine or urea cycle.

Cysteine, cystine and methionine are the principle sources of sulfur in the diet of man. Cysteine is present in the body partly in the form of cystine, which is formed by oxidation of the thiol groups of two cysteine molecules to form a disulfide bridge. Methionine is important in its function as a donor of methyl groups in various transmethylation reactions including synthesis of choline and creatine.

Taurine, can be synthesized from dietary cysteine or methionine and is therefore not considered an essential nutrient. However, there was concern that formula fed infants might be at risk in developing

taurine insufficiency because levels of taurine in formulas were much lower than those found in human milk. This distinction is especially important in the case of the preterm infant who may require taurine and cysteine at birth due to delayed enzyme maturation. Consequently, current infant formulas are all enriched with taurine.

Dietary Management of Phenylketonuria

There are a number of inborn errors of indispensable amino acid metabolism. For example, phenylketonuria (PKU) is an inherited disorder of phenylalanine metabolism. Its incidence is approximately one in every ten thousand births.

Classical PKU is characterized by the absence of the enzyme phenylalanine hydroxylase. Without this enzyme, phenylalanine cannot be converted to tyrosine, so tyrosine becomes an indispensable amino acid. Although the phenylalanine is catabolized by an alternate metabolic pathway leading to increased phenylpyruvic acid excretion in the urine, blood phenylalanine concentration is greatly increased often to greater than 20 mg/dl. These high phenylalanine levels produce irreversible mental retardation unless detected early. Treatment consists of a phenylalanine restricted diet. The purpose of the diet is to provide limited amounts of phenylalanine to allow for normal growth and development while maintaining blood phenylalanine levels in a reasonable range (5-9 mg/dl).

A low phenylalanine infant formula is available on the market and is used as the product of choice for the first two years of life. This formula has been treated to remove most of the phenylalanine and serves as a source of all other indispensable amino acids. However, it does not contain enough phenylalanine to meet requirements. A phenylalanine source such as cow's milk is usually added to the formula in measured amounts to provide the necessary dietary phenylalanine. As the infant grows older, other sources of phenylalanine are included in the diet and the amount of cow's milk may be decreased accordingly. Generally, low protein sources such as cereals, fruit and vegetables are used to provide phenylalanine requirements.

At two years of age, the low phenylalanine infant formula may be replaced by the use of special dietary products which are devoid of phenylalanine. The diet plan must be frequently modified in

view of the patient's blood phenylalanine levels and the changing requirements for energy, protein and phenylalanine. An artificial sweetener that is widely used in food products today, aspartame, consists of aspartic acid conjugated to the methyl ester of phenylalanine. Individuals with PKU must therefore pay attention to foods that contain aspartame.

Little information exists as to the duration necessary to continue the therapeutic diet, and its influence on mental function. A prudent approach would be to continue the diet on a lifetime basis. It is well known that a liberalized diet for women with PKU during pregnancy is associated with birth defects, particularly mental retardation.

Protein Restriction and Overloading

Several dietary guidelines have recommended the daily dietary protein levels required to maintain health in most populations. Adult humans in different cultures survive on a wide range of protein intake from marginal levels of only 0.6 g/kg/d to perhaps 6.0 g/kg/d. In developed countries average protein intake is in the range of 1-2 g/kg/d but athletes with very high rates of energy expenditure frequently consume more protein (2.8 g/kg/d). Although high protein diets have been advocated for maintaining or increasing muscle mass, little is known about the effects of sustained consumption of a very high protein diet. Increased consumption of protein increases the renal blood flow and glomerular filtration rate and induces stress on the kidney. Dietary protein restriction lowers the glomerular capillary pressure, prevents proteinuria and progressive sclerosis and gives symptomatic relief in patients with chronic renal failure. Dietary protein affects bone in a variety of ways. Bone is the most protein-dense tissue of the body and dietary protein and its essential amino acids are necessary for de novo synthesis of bone matrix. However, high protein intake is known to interfere with calcium homeostasis. A twofold increase in protein intake can cause up to a 50% increase in calcium excretion, which in the absence of increased calcium intake would lead to bone resorption and potentially to osteoporosis. However, for treatment of obesity replacement of some dietary carbohydrate by protein in an *ad libitum* low-fat diet, may assist with weight loss. Dietary protein per kJ energy intake exerts a more powerful effect on satiety than either carbohydrate or fat.

Suggested Readings

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CHAPTER 2

CARBOHYDRATES

Carbohydrates are a class of organic compounds composed of carbon, hydrogen and oxygen. In general, they contain two atoms of hydrogen for each atom of oxygen; the same ratio as water. They are a source of energy for the body and offer a wide range of rheological and other functional properties to foods. In addition, carbohydrates also have a wide range of physiological effects which may be important to health. These effects include (a) effects on satiety/gastric emptying, (b) control of blood glucose and insulin metabolism, (c) protein glycosylation, (d) cholesterol and triglyceride metabolism, and (e) bile acid dehydroxylation.

The principle carbohydrates, their dietary sources and characteristics are presented in Table 2-1. Carbohydrates are classified as mono-, di-, oligo- and polysaccharides according to the number of sugar units they contain. The mono- and disaccharides are referred to as simple carbohydrates as they contain one and two sugar units respectively. Oligo- and polysaccharides, composed of many sugar units linked together, are considered as complex carbohydrates. Alternatively, dietary carbohydrates are classified into three principal groups: (a) Sugars consisting of mono- and disaccharides with degree of polymerization = 1 - 2, (b) Oligosaccharides consisting of maltodextrins, raffinose, stachyose and fructo-oligosaccharides (FOS) with degree of polymerization = 3 - 9, and (c) Polysaccharides, starches, cellulose, hemicellulose, pectins and hydrocolloids with degree of polymerization > 9.

Monosaccharides

Monosaccharides represent the basic carbohydrate unit. They are classified according to the number of carbon atoms and whether or not they are aldehyde or ketone derivatives. Trioses, tetroses, pentoses, hexoses, and heptoses contain 3, 4, 5, 6, and 7 carbon atoms respectively. All monosaccharides are reducing sugars. The hexoses are the only monosaccharides to occur in appreciable amounts in foods. The hexoses, glucose, fructose, galactose and mannose, all have the same empirical formula $C_6H_{12}O_6$ but have different chemical structures resulting in different physical

Table 2-1

Classification of Carbohydrates

Carbohydrate	Occurrence	Remarks
SIMPLE		
Monosaccharides		
Hexoses		
Glucose	Honey, fruits, corn syrup, sweet grapes, and sweetcorn; hydrolysis of starch and of cane sugar.	Physiologically the most important sugar; the "sugar" carried by the blood and the principal one used by tissues.
Fructose	Honey, ripe fruits, and some vegetables; hydrolysis of sucrose and inulin.	Can be changed to glucose in the liver and intestine; and intermediate metabolite in glucogen breakdown.
Galactose	Not found free in nature; digestive end product of lactose hydrolysis.	Can be changed to glucose in the liver; synthesized in body (breast tissue) to make lactose; constituent of glycolipids.
Mannose	Legumes; hydrolysis of plant mannosans and gums.	Constituent of polysaccharide of albumins and globulins, and mucoids.
Pentoses		
Arabinose	Derived from gum arabic and plum and cherry gums; not found free in nature.	Has no known physiologic function in man; used in metabolism studies of bacteria.
Ribose	Derived from nucleic acid of . meats and seafoods	Structural elements of nucleic acids, ATP, and co-enzymes, e.g., NAD and FAD.
Ribulose	Formed in metabolic processes.	Intermediate in direct oxidative pathway of glucose breakdown.
Xylose	Woodgums, corncobs and peanut shells; not found free in nature.	Very poorly digested and has no known physiologic function; used medicinally as a diabetic food.
Disaccharides		
Sucrose	Cane and beet sugar, maple syrup, molasses and sorghum.	Hydrolyzed to glucose and fructose; a non-reducing sugar.
Maltose	Malted products and germinating cereals; formed from diastatic hydrolysis of starch; an intermediate product of starch digestion.	Hydrolyzed to two molecules of glucose; a reducing sugar; does not occur free in tissue.
Lactose	Milk and milk products; formed in body from glucose.	Hydrolyzed to glucose and galactose; a reducing sugar.
Trisaccharides		
Raffinose	Cottonseed meal, molasses, and sugar beets and stems.	Only partially digestible but can be hydrolyzed by enzymes of intestinal bacteria to glucose, fructose, and galactose.

Table 2-1 *Continued*

Classification of Carbohydrates

Carbohydrate	Occurrence	Remarks
COMPLEX		
Oligosaccharides		
α-galactosides	Soybean and other legumes	Non-digestible sugars
Fructo-oligosaccharides	Jerusalem artichoke, chicory, onion	Non-digestible sugars Bifidogenic effects
Polysaccharides		
Digestible		
Glycogen	Meat products and seafoods.	Polysaccharide of the animal body; often called animal starch; storage form of carbohydrate in carbo-hydrate in body, mainly in liver and muscles.
Starch	Cereal grains, unripe fruits, vegetables, legumes and tubers.	Most important food source of carbohydrate in plants; composed chiefly of two constituents amylose and amylopectin; completely hydrolyzable to glucose.
Partially Digestible		
Inulin	tubers and roots of dahlias, artichokes; dandelions, onions, and garlic.	Hydrolyzable to fructose; used in physiologic investigation for determination of rate of glomerular filtration.
Mannosan	Legumes and plant gums.	Hydrolyzable to mannose but digestion incomplete; further splitting by bacteria may occur in large bowel.
Indigestible (Fiber)		
Cellulose	Skins of fruits, outer coverings of seeds, and stalks and leaves of vegetables.	Not subject to attack of digestive enzyme in man, hence an important source of "bulk" in diet; may be partially split to glucose by bacterial action in large bowel.
Hemicellulose	Pectins, woody fibers, and leaves.	Less polymerized than cellulose; may be digested to some extent by microbial enzymes, yielding xylose.
Gum	Hydrocolloids secreted at site of plant injury.	Composed of a variety of sugar and sugar deriva-tives but galactose and glucuronic acid predomin-ate. Gum arabic is plant hydrocolloid most com-monly used as a food additive.

properties (Figure 2-1). Other monosaccharides are important in intermediary metabolism and in combination with other compounds.

Hexoses

Glucose (dextrose) is the only hexose sugar known to exist in the free state in the fasting human body. Normally, it is present in the blood in a concentration of about 90 mg/dl. Glucose is a white crystalline solid, freely soluble in water with a sweet taste. Because of the asymmetrical carbon atoms in the molecule, solutions of glucose rotate polarized light. The rotation is to the right, hence the alternative name "dextrose".

Only a few foods contain more than traces of free glucose. Glucose is mainly obtained by the hydrolysis of glucose-containing oligo- and polysaccharides in the gut.

Figure 2-1

Structure of the Hexoses:
Glucose, Fructose, Galactose and Mannose

ß-D-Glucose ß-D-Fructose

ß-D-Galactose ß-D-Mannose
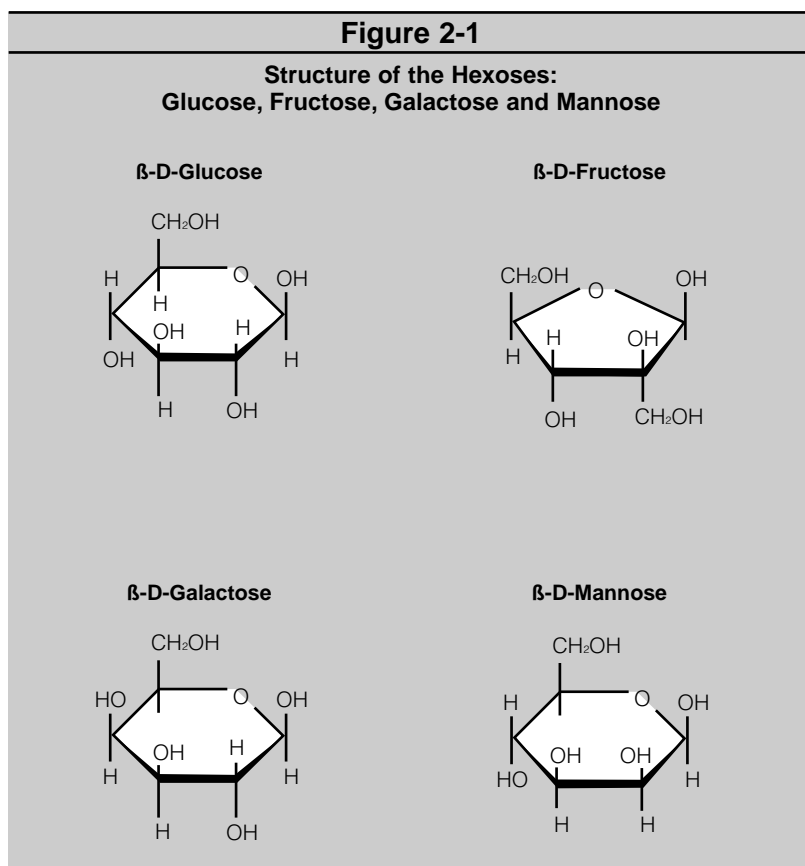
Fructose (levulose) is a highly soluble sugar and does not readily crystallize. It rotates polarized light to the left; hence its alternative name is levulose. Unlike glucose, galactose and aldohexoses (possessing an aldehyde group), fructose is a ketohexose (possessing a keto group). Fructose is found in free form in many fruits, vegetables, honey and in high fructose corn syrups. It is also obtained from hydrolysis of dietary sucrose.

Fructose in aqueous solution has a sweetness of 100-180 relative to 100 sucrose. However, the sweetness of fructose is affected by various factors in the carrier medium. For example, decreases in temperature and acidity increase the relative sweetness of fructose in solution. In complex foods such as cakes and puddings, sucrose is often perceived to be sweeter than fructose.

High fructose corn syrups came into commercial use in the early 1970's. They are referred to as corn syrups because they are produced chiefly from cornstarch. The term high fructose corn syrups is confusing as they may contain 42%, 55% or 90% fructose to 58%, 45% or 10% glucose respectively. High fructose corn syrups are used in the food industry because they provide greater solubility, enhanced flavor and preservation, decreased viscosity, improved storage capability and, because of the greater relative sweetness of fructose, the possibility of reduced calories when compared to sucrose.

Galactose is not found free in nature but combined with glucose to form lactose (milk sugar). It is also a constituent of many plant polysaccharides.

Mannose may be obtained from legumes in the diet. Small amounts of mannose are present in manna from which mannose takes its name.

Hexose Derivatives
Sorbitol (D-glucitol) is an alcohol which is made commercially from glucose by hydrogenation, by replacing the aldehyde group with an alcohol group. Although sorbitol is found in many fruits and vegetables, the synthetic product is used predominantly in dietetics. Its rate of absorption from the gut is so much slower than that of glucose that it has little effect on blood sugar levels. The slow intestinal absorption of sorbitol, combined with its mildly sweet flavor, has given it a wide use in the manufacturing

of special jams, marmalade, canned fruits, fruit drinks and chocolate meant for consumption by diabetic patients. Sorbitol has a lower caloric value (2.4 kcal/g) than glucose (3.8 kcal/g).

Mannitol is formed by the hydrogenation of mannose. It has a variety of industrial uses and may be used in food manufacturing, added as a drying agent in certain foods.

Inositol is an alcohol allied to the hexoses. It occurs in many foods and especially in the bran of cereal grains. Inositol, in combination with six phosphate groups forms the compound phytic acid which hinders intestinal absorption of calcium and iron. Free inositol, phosphatidyl inositol and polyphosphoinositides may be obtained from animal food sources. While not classified as essential for *humans*, inositol is an essential growth factor for human cells in tissue culture. Inositol acts as a lipotrophic factor for a number of animal species. Animals fed inositol-deficient diets develop a fatty liver and other lipid abnormalities. Animal studies also suggest that free inositol may have a role in spermatogenesis while inositol-pentaphosphate in the blood increases the affinity of hemoglobin for oxygen. Phosphatidyl inositol, as a component of cell membranes is believed to be involved in neural transmission, cellular response to external stimuli and regulation of enzyme activity.

Pentoses

The pentoses D-ribose and 2-Deoxy-D-ribose are essential components of nucleic acids, the high energy compound ATP and the coenzymes NAD and FAD. NAD and FAD participate in many intracellular redox reactions including the Krebs cycle. They are required as donors of hydrogen atoms for the formation of ATP by oxidative phosphorylation.

The pentoses most commonly present in human foods are L-arabinose and D-xylose which are widely distributed in fruits and root vegetables but in relatively small amounts compared to hexoses and disaccharides.

Disaccharides

The disaccharides are simple carbohydrates composed of two monosaccharide units. The most important disaccharides are sucrose, lactose and maltose (Figure 2-2).

Sucrose (cane sugar, beet sugar) is the familiar "sugar" in common domestic use. It is composed of one molecule of glucose and one molecule of fructose. Sucrose rotates polarized light to the right. However, since fructose is more levo-rotatory than glucose is dextro-rotatory, the hydrolysis of a sucrose solution into fructose and glucose favors the rotation to the left. Hence the hydrolysis of sucrose (which takes place very readily) has been described as "inversion". "Invert sugar" is the commercial name for the product, which is an equal mixture of glucose and fructose. Sugar cane and sugar beets are the major sources of sucrose.

Lactose is the principle sugar present in milk and is unique to mammals. It is made up of one molecule of glucose and one molecule of galactose.

Maltose is a disaccharide composed of two molecules of glucose. It is formed from the breakdown of starch in the malting of barley.

Figure 2-2

Structure of the Disaccharides: Maltose, Sucrose and Lactose
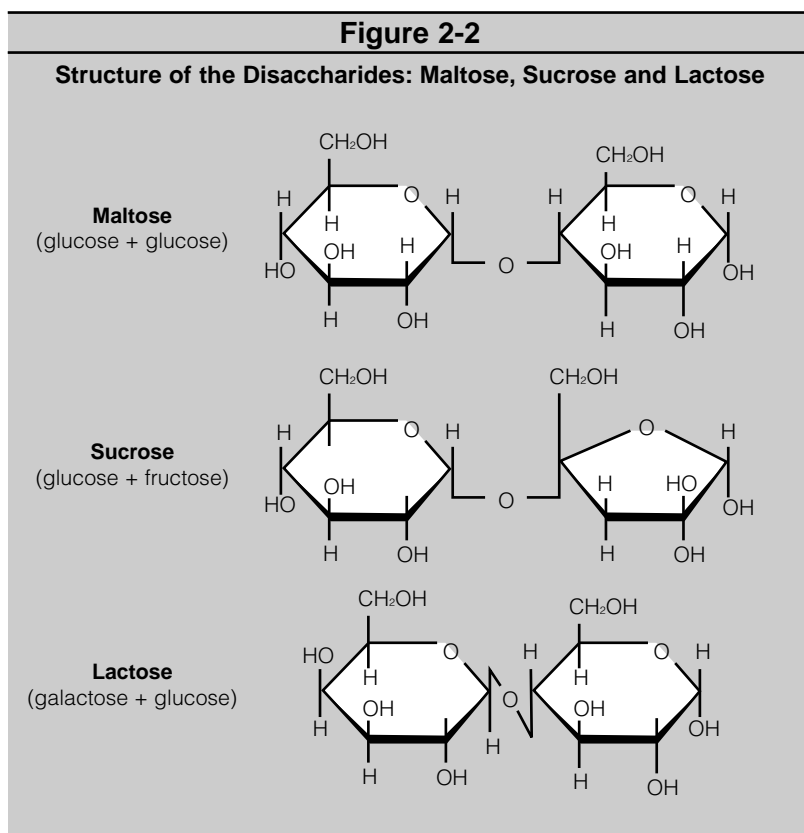
Oligosaccharides

Oligosaccharides are complex carbohydrates composed of three to ten monomeric units. The most important oligosaccharides are the α-galactosides and fructooligosaccharides.

α-Galactosides such as raffinose, stachyose and verbascose are soluble oligosaccharides and are present in high concentrations in soybean and other legumes. These sugars consist of a sucrose linked via α-galactoside bond to galactose. These sugars are not digested because the small intestine of humans lacks the α-galactosidase required for its hydrolysis. As a result, virtually 100% of ingested α-galactosides reach the colon and become potential substrates for colonic fermentation by intestinal bacteria leading to an increase in fecal output, production of short chain fatty acids and prebiotic effects.

Fructooligosaccharides are natural food ingredients commonly found in varying amounts in dietary plant foods. They are present as plant storage carbohydrates and constitute up to 60-70% of the dry matter in some plant tissues such as Jerusalem artichokes, chicory and onions. Inulin type fructooligosaccharides or fructans are composed of β-D-fructofuranoses attached by β-2-1 linkages with a degree of polymerization from 2 to 60, and of other oligofructoses which are produced by partial enzymatic hydrolysis of inulin (DP < 10).

The difference in chain length between inulin and oligofructose accounts for their functional attributes. Inulin forms microscopic crystals when sheared in water or milk and forms a smooth creamy texture providing a fat-like mouthfeel. Inulin has been used successfully to replace fat in table spreads, baked goods, fillings and dressings. Oligofructose has a sweet, pleasant flavor, is highly soluble and can be used to improve the flavor and sweetness of low calorie foods and the texture of fat-reduced foods.

Inulin and oligofructose are not digested in the upper gastrointestinal tract, therefore they have a reduced caloric value. They have been termed as "prebiotic" because they selectively stimulate the growth of intestinal bacteria (*Bifidobacteria*).

Polysaccharides

The three most important polysaccharides, glycogen, starch and cellulose, are all polymers of glucose, i.e., they are made up of many molecules of glucose linked together. In contrast to the sugars already mentioned they have a bad or very bland taste.

Starch is the form in which carbohydrates are stored within the seeds and roots of plants. Starch granules are encased within a cellulose wall. About 15-20% of the total starch consists of an unbranched chain (alpha-1, 4 glycosidic bonds) of several hundred glucose units and is known as amylose. Amylose is responsible for the intense blue color resulting on the addition of a trace of iodine to a solution of starch, a test used to identify the presence of starch. The main component of starch is amylopectin, which is a highly branched glucose polymer (points of branching are alpha-1, 6 glycosidic bonds). If free from amylose, amylopectin gives a brownish - violet color on the addition of iodine. Starch grains, as obtained from fresh plants, are completely insoluble in water but can be dispersed in cold water. Heat, however, promotes a solution which remains fairly stable, although it may form a jelly on cooling. The strength of the gel varies with the source such as corn, potato or rice.

The digestion of starch within the small intestine is incomplete and depends on its physical form, the nature of starch granules, and the effects of food processing. Moist heat causes starch grains to swell and rupture; thus, cooking renders the starch in vegetable foods more available for digestion.

During food manufacturing starches may be physically or chemically altered to confer more desirable properties, such as greater viscosity, to the finished product. The altered starches are referred to as modified starch. Some starches that are resistant to digestion are called resistant starch. These resistant starches have several positive physiological effects such as control of insulinemia, and colonic fermentation leading to production of short chain fatty acids.

Glycogen is the form in which the animal cell stores carbohydrate. It is more highly branched than amylopectin. Stored in the liver and muscle, glycogen is utilized for energy during fasting and continuous exercise. The amount of glycogen that can be stored by tissues is limited to approximately 500 g. Therefore it is quickly depleted during continuous exercise.

Cellulose is a major component of plant cell walls. The glucose units are joined by beta-1, 4 glucosidic bonds rather than alpha-1, 4 glycosidic bonds found in starches. The human digestive system contains the enzyme alpha-amylase, which can cleave the alpha-1, 4 glycosidic bonds but not the beta-1, 4 bonds. Consequently cellulose cannot be digested in *humans*.

Cellulose, as a dietary fiber, increases gastrointestinal mobility and therefore facilitates fecal elimination. Excessive intake of cellulose and other dietary fibers can interfere with nutrient absorption by binding to nutrients and by increasing their rate of elimination. Cellulose also serves as a medium for the growth of intestinal microflora thereby enhancing fermentation in the gut and resulting in gas formation.

Gums are complex heteropolysaccharides and are used as emulsifiers, stabilizers, thickeners, suspending agents and foam enhancers. Gum arabic and gum tragacanth are plant exudates from shrubs and trees and are used as emulsifying and texturing agents in soft drinks, candies, snacks and salad dressings and sauces. Guar gum and locust bean gums are seed gums composed of galactomannans that are water soluble and provide a highly viscous medium in ice creams and salad dressings. Xanthan gum is a microbial gum containing a cellulose backbone. It is used in pourable salad dressing, fruit juices and dry-mix products. These viscous polysaccharides also reduce intestinal absorption of glucose and therefore diminish postprandial hyperglycemia. Moreover, these are highly fermentable in the large intestine and promote short chain fatty acid (SCFA) production. The absorption of SCFA increases absorption of sodium and water and thereby decreases diarrhea.

Functions

Carbohydrates are the major source of food energy and provide 4 kcal/g (17 kJ/g). Glucose can be utilized for energy by all tissues of the body. The central nervous system, lungs, adrenal cortex and red blood cells normally require glucose as their sole source of energy.

Dietary carbohydrate is protein sparing i.e., when supplied in adequate amounts it prevents unnecessary protein breakdown for use as a source of energy.

If carbohydrate is absent from the diet, ketone bodies are formed from incomplete breakdown of fatty acids producing acidosis and dehydration. Excessive breakdown of tissue protein to serve as a source of glucose also occurs. Provision of approximately 100 g carbohydrate per day prevents the undesirable formation of ketone bodies; hence, carbohydrate may be referred to as antiketogenic.

When combined with amino groups, carbohydrates serve as a source of non-essential amino acids. A number of compounds derived from carbohydrates serve important biological functions. These compounds include glucuronic acid, hyaluronic acid, heparin, immunopolysaccharides, DNA and RNA. Dietary fiber provides the roughage and bulk necessary for optimal functioning of the gastrointestinal tract. Intestinal bacteria may breakdown and utilize a small part of the cellulose and thus indirectly contribute additional nutrients.

Carbohydrate foods serve as carriers of carotene (the precursor of vitamin A), water soluble vitamins, minerals and sources of dietary fibre.

Digestion and Absorption

The most important digestible dietary carbohydrates are glucose, fructose, maltose, lactose and starch. The digestion of carbohydrate is facilitated mainly by the disaccharidase and amylase enzymes found in the gastrointestinal tract. Salivary alpha-amylase and acid hydrolysis in the stomach have a limited role in carbohydrate digestion. Most of the digestion of starch takes place in the duodenal lumen by the action of pancreatic alpha-amylase. This enzyme is specific for the alpha-1, 4 glycosidic linkages.

Maltose and other straight chain oligosaccharides (up to nine glucose units) are products of amylose hydrolysis. Hydrolysis of amylopectin produces branched chain oligosaccharides called limit dextrins (unhydrolyzed alpha-1, 6 glycosidic linkage).

The remaining hydrolysis takes place by the action of enzymes located in the brush border of the intestinal mucosa. Only mono-saccharides can enter the mucosal cell. Glucoamylase (maltase) hydrolyzes maltose and the straight chain oligosaccharides to glucose. Sucrose is hydrolyzed by sucrase to fructose and glucose. Lactose is similarly hydrolyzed by lactase (beta-galactosidase) to glucose and galactose. The limit dextrins are hydrolyzed to glucose by alpha-(1,6)-glucosidase.

Glucose and galactose share a common sodium-linked, energy dependent carrier system which enables active transport into intestinal mucosal cells. A small portion of entry is believed to be by passive diffusion. The mechanism of fructose transport into the intestinal mucosal cells is thought to be by passive and/or facilitated diffusion.

The rate-limiting step in the absorption of glucose and galactose is hydrolysis of lactose by the enzyme lactase. In contrast, the other disaccharidases hydrolyze monosaccharides at a rate greater than the rate of uptake, i.e. uptake is the rate-limiting step in absorption.

Absorbed monosaccharides are transported from the intestinal mucosa via the portal vein to the liver. They may be utilized directly for energy by all tissues, temporarily stored as glycogen in liver or muscle, or converted into fat, amino acids and other biological compounds.

The entry of glucose into most tissues including heart, muscle and adipose tissue is governed by the presence of the hormone insulin. Uptake of glucose by the liver and central nervous system however, is not insulin-dependent.

Metabolism

The reactions involved in carbohydrate metabolism cannot take place without the presence of the B vitamins which function as coenzymes (Figure 2-3). Phosphorous, magnesium, iron, copper, manganese, zinc and chromium are also necessary as co-factors.

Figure 2-3

Vitamins and Minerals in Carbohydrate Metabolism

Glycogen

Phosphorous

Glucose – 1 – Phosphate

Magnesium

Glucose   →   Glucose – 6 – Phosphate

Nicotinic Acid

Pentose Phosphate

Hexose 1:6 Phosphate

3 Glyceraldehyde Phosphate

Phosphorus	Nicotinic Acid

1:3 Phosphoglyceric Acid

3 Phosphoglyceric Acid

Phosphopyruvic Acid

Magnesium

Phosphoglyceric Acid

Oxaloacetic Acid

Thiamin

Nicotinic Acid

Pyruvic Acid ⇄ Lactic Acid

Phosphorus
Magnesium

Thiamin,
Riboflavin.
Nicotinic Acid,
Pyridoxine,
Pantothenic Acid

$CO_2 + H_2O$

The main pathways of carbohydrate metabolism include the following:

Glycolysis
Glycolysis serves to release energy from glucose or glycogen through the formation of two molecules of pyruvate. Under anaerobic conditions lactate is formed from pyruvate. This reaction is important in muscle when energy demands exceed oxygen supply. The other monosaccharides, fructose, galactose and mannose also undergo glycolysis after conversion to glycolytic intermediates.

Citric Acid (Krebs) Cycle and Electron Transport System
Approximately 90 percent of the energy derived from carbohydrate, protein and fat is produced via the citric acid (Krebs) cycle and electron transport system (Figure 1-3). During specific steps of the electron transport chain sufficient energy is generated to phosphorylate ADP to ATP, which is the energy currency of the cell. The reactions of the electron transport chain are collectively referred to as oxidative phosphorylation.

Pentose Phosphate Shunt
An alternate pathway of glucose utilization accounting for up to 10 percent of the energy derived from carbohydrate is the pentose phosphate shunt. The reactions of the shunt occur mainly in liver, muscle and red blood cells. The pathway is also significant as it produces ribose (used in nucleic acid synthesis) and NADPH. NADPH is the reduced form of NADP which functions as a hydrogen carrier in many intracellular redox reactions. NADPH is required during fatty acid synthesis.

Glycogenesis
Excessive glucose may be converted to glycogen by glycogenesis. Glycogenesis is especially important in liver and muscle but can take place in almost all tissues. The human body has a limited capacity to store glycogen; the major portion of excess carbohydrate is converted to fat.

Glycogenolysis
Glycogenolysis refers to the breakdown of glycogen to glucose. From the liver, the glucose can be transported to other tissues or catabolized *in-situ* for energy. Since muscle does not possess glucose-6-phosphatase, glycogen is converted to lactate. Conversion of lactate to pyruvate is limited in muscle so most lactate is metabolized in the liver.

Metabolic Relationships of Carbohydrate to Protein and Fat

Gluconeogenesis

Gluconeogenesis refers to the formation of glucose from non-carbohydrate sources: namely certain amino acids and the glycerol fraction of fats. Normally the liver is the main site of gluconeogenesis, but under certain circumstances, such as starvation, the kidney becomes important. Gluconeogenesis is an important source of glucose in the body when carbohydrate intake is limited and body glycogen is depleted. It is also the primary pathway for the utilization of lactate and glycerol which accumulate in muscle tissues during strenuous exercise.

Most of the reactions of gluconeogenesis consist of a reversal of glycolytic reactions. Almost all amino acids are potentially glucogenic. The glucogenic amino acids are those which can be converted to pyruvate or into intermediates of the citric acid (Krebs) cycle (Figure 1-3). In higher animals, there is no metabolic pathway by which fatty acids or acetyl-CoA can be used to synthesize glucose. However, glucose can be synthesized from the glycerol moiety of hydrolyzed triglycerides and by the 3-carbon remnant resulting from beta-oxidation of odd-chain fatty acids.

Synthesis of Protein and Fat from Carbohydrate

Dispensable amino acids may be synthesized in the body from ketoacids derived from carbohydrate metabolism as discussed in Chapter 1. Excess carbohydrate may be converted to triglycerides by the formation of glycerol from glycerolphosphate and from fatty acids synthesized from acetyl-CoA.

Carbohydrate in the Diet

Sources

Sources of dietary carbohydrate are shown in Table 2-1. Simple carbohydrates are found in fruits, vegetables and milk. Refined sugar is often added to foods. Complex carbohydrates are present in grains, tubers and legumes.

Composition of the Diet

The proportion of dietary energy from carbohydrate sources in most diets fall in the range of 40% to 80% of energy. Generally, the overall proportion of carbohydrates decreases as that of fat increases. In affluent societies there has also been a decrease in

complex and an increase in simple carbohydrates in the diet attributed to the increased use of processed foods. It is still unclear what the shift in macronutrient consumption means; however, those individuals who obtain a low (<30%) proportion of energy from fat (and presumably have a lower risk of coronary heart disease) also tend to obtain a high proportion of energy from sugars.

Currently, the general consensus among nutritionists is that individuals should maintain 45-65 percent of dietary energy from carbohydrate, of which a substantial proportion should be in the form of complex carbohydrate and fiber.

Disorders of Carbohydrate Metabolism
There are a number of aberrations in carbohydrate metabolism that have clinical consequences. These include lactose intolerance, sucrose intolerance, galactosemia and diabetes mellitus.

Lactose Intolerance
Lactose intolerance is caused by a deficiency or absence of the intestinal enzyme lactase. Lactase is normally present in sufficient quantities in infants. Congenital lactase deficiency, in which levels of lactase are low or absent at birth, is very rare. For individuals susceptible to lactase deficiency the levels of this enzyme progressively decline after infancy. Thus, the individual becomes progressively less tolerant to lactose in milk and milk products (dietary sources of lactose). Primary lactose intolerance is particularly prevalent among adults of African, Asian and Mediterranean cultures. Symptoms of lactose intolerance include abdominal distention, cramps, flatus and diarrhea due to the presence of unhydrolyzed lactose in the gut. Secondary causes of lactose intolerance include gastrointestinal infections, malabsorption syndrome and gastrointestinal surgery.

Treatment of lactose intolerance centers around avoidance of lactose-containing foods such as milk and milk products. The degree of avoidance of these foods varies with the individual, depending on the amount of lactase activity. Most adults with primary lactose intolerance can tolerate small amounts of milk and milk products, but lactose-free milks are readily available. Cheese and yogurt may be better tolerated than fluid milk, as a large amount of the lactose present in these products is hydrolyzed or fermented to lactic acid.

Lactose-free formula is available for infants with congenital lactase deficiency. Commercial enzyme preparations that can be added to milk prior to ingestion in order to hydrolyze most of the lactose present are also available. For those individuals with secondary lactose intolerance the clinical problem must be treated first. The therapeutic diet usually limits the major sources of lactose. As the clinical problem is rectified and symptoms of lactose intolerance improve, increasing quantities of lactose containing foods may be introduced into the diet.

Sucrose Intolerance

Sucrose intolerance may exist when there is a deficiency of the intestinal enzyme sucrase alpha-dextrinase. The symptoms of sucrose intolerance are similar to lactose intolerance due to the presence of unhydrolyzed sucrose and, to a much lesser extent, alpha-limit dextrins (which have a lower osmotic activity than sucrose) in the gut. Avoidance of table sugar and highly sweetened foods is usually effective in preventing symptoms of sucrose intolerance.

Galactosemia

Galactosemia is a rare inherited disorder of galactose metabolism. There are two types of galactosemia. The first type involves the absence of the enzyme necessary for the transformation of galactose-1-phosphate to glucose-1-phosphate (transferase deficiency galactosemia). The second type of galactosemia is less common. Galactose-1-phosphate cannot be formed due to lack of the enzyme galactokinase. The major alternate pathway of galactose metabolism is the formation of the alcohol derivative galacitol.

The symptoms of galactose transferase deficiency are severe and include vomiting, liver damage, growth retardation, cataracts (due to deposition of galacitol in the lens) and mental retardation. Early detection and dietary treatment are essential. The main clinical feature of galactokinase deficiency is cataracts. Early detection and dietary treatment may prevent formation.

The dietary treatment of galactosemia consists of a lactose and galactose-free diet. All lactose containing foods (notably milk and milk products) are eliminated from the diet. Galactose containing foods, for example, liver, organ meats, certain fruits, legumes, are avoided. Infants are initially fed a lactose- (and

galactose) free formula and then progress to the solid foods allowed within their restricted dietary regimen.

Some liberalization of the diet may be permitted once the individual reaches early teens but careful monitoring of erythrocyte galactose-1-phosphate and galactosuria is necessary.

During pregnancy, the galactosemic woman must follow a strict lactose and galactose-free diet to prevent damage to the fetus.

Diabetes Mellitus
Diabetes mellitus affects close to 15.7 million or 5.9% of the American population. Nearly 0.8 million new cases are diagnosed every year. Diabetes is the seventh leading cause of death. Diabetes mellitus is a group of metabolic disorders characterized by abnormal glucose tolerance, specifically hyperglycemia, due either to a deficiency in insulin production and/or a decrease in insulin sensitivity in target cells. The hyperglycemia syndromes are frequently accompanied by altered carbohydrate, protein and lipid metabolism, and long term pathology that affects the blood vessels such as retinopathy with potential loss of vision, nephropathy leading to renal failure, peripheral neuropathy with risk of foot ulcers, amputation and Charcot joints, and autonomic neuropathy causing gastrointestinal, genitourinary, cardiovascular symptoms and sexual dysfunction.

The two major types are insulin-dependent diabetes mellitus (IDDM) or type 1, and non-insulin diabetes mellitus (NIDDM) or type 2. IDDM results from an abnormality of the beta-cells of the islets of Langerhans in the pancreas causing a deficiency in the synthesis of insulin. Formerly this type was referred to as a juvenile-onset, ketone prone or brittle diabetes. IDDM usually occurs in individuals prior to age 20 but can occur up to 40 years of age. The onset is usually sudden, the abnormality of glucose tolerance is severe and insulin synthesis is greatly reduced or lacking. Individuals at risk of developing IDDM can often be identified by serological evidence of an autoimmune pathologic process occurring in pancreatic β-cell and by genetic markers. IDDM individuals are usually of normal weight. Symptoms associated with IDDM include polyuria, polydipsia, polyphagia, weight loss and ketosis. These symptoms are caused by the sugar buildup in the blood and its loss in the urine through frequent urination. This excessive urination results in dehydration and

increased thirst. In addition to causing high blood glucose, the lack of insulin results in breakdown of stored fats and protein for energy. Ketosis is the result of incomplete combustion of fatty acids leading to an accumulation of ketone bodies in the plasma and urine. Ketonuria is accompanied by acidosis and dehydration. In severe cases coma and death will occur unless there is prompt insulin therapy.

NIDDM is the most common type of diabetes and is associated with obesity. The individual frequently has measurable or even excessive amounts of insulin in the blood but may have impaired insulin-mediated uptake of glucose by muscle. Hyperglycemia in NIDDM is sufficient to cause pathological and functional changes in various target tissues even before diabetes is detectable. The abnormality in carbohydrate metabolism during this asymptomatic period can be demonstrated from fasting plasma glucose levels or by oral glucose tolerance test. Ketosis is seldom present. NIDDM can be controlled by diet alone or by diet and/or oral hyperglycemic agents. Symptoms of NIDDM include degenerative changes in the vascular system including hypertension, heart disease, retinitis, and peripheral neuritis.

The mechanism for diabetes-induced damage of micro and macro vascular functions and organ injury is not very clear. Hyperglycemia is linked to pathogenesis of nephropathy, retinopathy and atherosclerosis in long term diabetes. Various mechanisms have been proposed to explain the hyperglycemia induced organ damage in diabetes. (a) Activation of the aldose-reductase pathway leading to accumulation of sorbitol in nerves, (b) accelerated non-enzymatic glycosylation and deposition of advanced glycosylated end products (AGEs) and (c) activation of protein kinase C (PKC) in vascular tissues. These reactions may initiate a cascade of events culminating in diabetic complications.

The nutrition goals for individuals with diabetes include the following:

❑ Maintenance of as near normal body glucose as possible by balancing food intake with insulin (either endogenous or exogenous) or oral glucose-lowering medications and activity levels

❑ Achievement of optimal lipid levels

❑ Provision of adequate calories for maintaining or attaining reasonable weights for adults, for normal growth and development rates for children and adolescents, to meet increased metabolic needs during pregnancy or lactation, or for recovery from catabolic illnesses

❑ Prevention, delay or treatment of nutrition-related risk factors and complications. Nutrition assessment and intervention are essential for risk factor reductions from obesity, dyslipidemias and hypertension.

❑ Improvement of overall health through optimal nutrition (Chapter 6 summarizes the nutritional guidelines and nutrients for individuals in various countries. These guidelines are suitable for individuals with diabetes)

For the IDDM, meals are planned so that the dietary energy and to a lesser degree carbohydrate content, correspond to the peak action and duration of insulin therapy. Between meal snacks, particularly during peak insulin action times, are included in the diet. For the NIDDM, meals are evenly spaced and of similar energy in carbohydrate content.

The overall composition of the diabetic diet recommended in the 1980s by the American Diabetes Association (ADA) was as follows:

❑ ≤60% of total energy from carbohydrate;

❑ 12-20% of total energy from protein;

❑ not greater than 30% of total energy from fat;

❑ less than 10% of total energy from saturated fat;

❑ up to 10% (preferably 6-8%) of total energy from polyunsaturated fatty acids;

❑ Cholesterol should be limited to 300 mg or less daily

Currently, optimal levels of macronutrients are not set; instead macronutrient intake is based on a combination of usual eating patterns and desired metabolic outcomes.

Achievement of the ADA diabetic diet is facilitated by the use of an exchange system. Foods are grouped according to their nutrient composition, i.e. carbohydrate, protein and fat content.
A fixed number of servings of each group may be permitted for each meal or snack according to the daily meal plan or pattern.

The ADA diabetic food groups or exchange system is based on the following:

I. milk exchange list
    - skim (one serving contains 8 g protein, trace amount of fat, 12 g carbohydrate and 90 Kcal)
    - low fat(one serving contains 8 g protein, 5 g fat, 12 g carbohydrate and 120 Kcal)
    - whole (one serving contains 8 g protein, 8 g fat, 12 g carbohydrate and 150 Kcal)
II. vegetable exchange list (one serving contains 5 g carbohydrate, 2 g protein and 25 Kcal);
III. fruit exchange list (one serving contains 15 g carbohydrate and 60 Kcal);
IV. starch/bread exchange list (includes breads, cereals, starchy vegetables and prepared foods, one serving contains 15 g carbohydrate, 3 g protein, trace amount of fat, 80 Kcal);
V. meat exchange list
    - lean meat (one serving contains 7 g protein, 3 g fat and 55 Kcal);
    - medium - fat meat (one serving contains 7 g protein, 5 g fat and 75 Kcal);
    - high fat meat (one serving contains 7 g protein, 8 g fat and 100 Kcal);
VI. fat exchange list (one serving contains 5 g fat and 45 Kcal);

The use of an exchange system serves to supply relatively fixed amounts of carbohydrate, fat, protein and energy at set meals and snacks each day.

In the past one of the main emphases was to avoid simple sugars in the diets of diabetics. This was based on the assumption that sugars are more rapidly digested and absorbed than starches and thus aggravates hyperglycemia to a greater extent. However, there is evidence to show that the use of sucrose as part of the meal plan does not adversely impact blood glucose in individuals with diabetes, type 1 or type 2. Furthermore, sucrose has been shown to produce a glycemic response similar to bread, rice and potatoes. Nevertheless, sucrose and sucrose-containing foods must be substituted for other carbohydrates and foods and not simply added to the meal plan. Today's emphasis is on the total carbohydrate in the diet rather than just the sugar.

There is also evidence to suggest that the fiber intake among diabetic individuals should be increased over and above that which is recommended for healthy eating by the population at large. Although certain soluble fibers are capable of delaying glucose absorption, the effect of dietary fiber on glycemic control is insignificant.

Exercise is an essential element in the therapy of IDDM. Physical activity has an insulin-like effect and enhances glucose utilization. The reason for this is not clear. Physical activity may alter insulin sensitivity of the target cells. Hence all individuals with IDDM require regular exercise. This is also recommended for NIDDM individuals as physical activity enhances weight loss and maintenance of normal body weight.

Dietary Fiber

Epidemiological studies have provided evidence that a high intake of dietary fiber exerts preventive effects against cancer, atherosclerosis and NIDDM. Cellulose, hemicellulose and pectin are structural polysaccharides of plant cell walls. They are indigestible by human enzymes in the small intestine and together with lignin and gum, are classified as dietary fiber. Dietary fibers are classified into two different types: **Soluble Dietary Fibers** such as pectins, gums, β-glucans, starches and other storage polysaccharides; **Insoluble Dietary Fibers** include cellulose and lignins. The soluble fibers are generally present in high levels in oats, barley, nuts fruits, beans and vegetables. Whole grains and cereals are a major food source of insoluble fiber. The soluble and insoluble fibers have very different influences on normal gut physiology and disease processes. Dietary fiber provides bulk, gentle laxation and ease of elimination. Cellulose has a high affinity for water and therefore increases stool weight, decreases transit time and intraluminal pressure. Pectin and gum enhance gel formation thereby slowing gastric emptying. They may also bind bile acids and hence decrease serum cholesterol levels. Lignin increases steroid excretion and may have antioxidant properties. Table 2-2 lists the dietary fiber content of some common foods.

The microflora in the colon have the capacity to ferment dietary fibers resulting in the production of short chain fatty acids (SCFA). SCFA are quickly absorbed and utilized by colonocytes thereby maintaining cell integrity in the colon.

Dietary fiber has been postulated to have beneficial effects on diabetes, atherosclerosis, cancer, appendicitis, prevention of duodenal ulcer formation, ischemic heart disease, cholecystitis, ulcerative colitis, Crohn's disease, varicose veins, deep vein thrombosis and hiatus hernia. However, the effectiveness of dietary fiber on these clinical problems is not definitive and requires further study. Over-enthusiastic use of dietary fiber is not suggested.

Table 2-2

Dietary Fiber Content of Some Commonly Consumed Foods

Food Item	Dietary Fiber (g/100g)
Cereals	
bran, wheat, crude	42.8
bran, oat, raw	15.4
bread, brown	4.7
bread, white	2.4
bread oatmeal	4.0
breakfast cereals, corn	2.8
breakfast cereals, rice	1.1
breakfast cereals, wheat	8.9
flour, white	2.7
flour, whole grain	12.2
rice, white, boiled	1.7
soya flour, full fat	11.2
soya flour, low fat	13.5
Fruits	
apple, raw	2.7
banana, raw	2.4
pear, no skin	2.4
raisins, seedless	4.0
Nuts	
almond, dried	10.9
peanut, raw	8.5
Vegetables	
baked beans, canned plain	5.0
brussel sprouts, raw	3.8
carrot, raw	3.0
carrot, boiled, drained	3.3
cucumber, raw	0.8
green peas, frozen	4.7
potato, raw	1.6
tomato, raw	1.1
tomato, sauce	1.4

Source: U.S. Department of Agriculture, USDA Nutrient Database for Standard References, Release 13, Agricultural Research Service, 1999.

Excessive fiber in the diet can reduce mineral absorption resulting in a negative balance of iron, zinc and other trace elements. It can also interfere with the consumption of a balanced diet.

Artificial Sweeteners

In natural environment, sweet taste is the cue for edibility and there is an innate tendency to prefer sweetness. The average adult consumes about 43 lb of sugar per year accounting for 10-12% of total energy intake. High sugar intake is associated with adverse effects on dental health and also aggravates hyperglycemia. Artificial sweeteners are available as an acceptable means of curtailing the excess refined sugar in the diet. Saccharin, aspartame, acesulfame-K and sucrose polymers are the common artificial sweeteners currently approved for use in USA and Canada.

Saccharin: is a petrolium byproduct and occurs as a white crystalline powder. It is 200 - 700 times sweeter than sucrose. It is a relatively safe substance, is not metabolized in the body and is excreted unchanged in the urine. It is considered safe at <1% dietary level but excessive intake has been associated with the potential risk of bladder cancer. Although a typical diet would normally not exceed a modest amount of saccharin, pregnant women are advised to avoid saccharin and children should not exceed more than 2 cans of saccharin sweetened soft drinks per day.

Aspartame: is a dipeptide of aspartic acid and phenylalanine. It is 180 times as sweet as sucrose. Since it is heat and acid labile and loses its taste, its use is limited. Aspartame is metabolized into several products including phenylalanine and aspartic acid and therefore carries a risk for individuals with phenylketonuria (pku) proportional to the consumption of equivalent amounts of phenylalanine.

Acesulfame-K is a derivative of acetoacetic acid and is 200 times sweeter than sucrose. It is heat stable and is approved for broad product applications.

Sucrose polymers, made by selective chlorination of sucrose, are 600 times sweeter than sucrose and are becoming very popular as alternate sweeteners.

Intake of sweeteners should however be limited to the established safe levels only.

Suggested Readings

British Nutrition Foundation: **Complex Carbohydrates in Foods.** London, Chapman and Hall, 1990.

Carbohydrates in Human Nutrition – a summary of joint FAO/WHO expert consultation, 1997.

Dobbing, J. (Ed.): **Dietary Starches and Sugars in Man: A Comparison.** London, Springer-Verlag, 1990.

Nutrition Recommendations and Principles for People with Diabetes Mellitus. **Diabetes Care** 21:S32-SS35, 1998

Kritchevsky, D., Bonfield, C., (Eds.): **Dietary Fiber in Health and Disease.** New York, Plenum Press, 1997.

Report of the Expert Committee on Diagnosis and Classification of Diabetes Mellitus. **Diabetes Care** 21:S5-S19, 1998.

CHAPTER 3

LIPIDS

Lipids constitute a heterogeneous group of compounds which are related more by their physical rather than by their chemical properties. They are insoluble in water and soluble in non-polar organic solvents (alcohol, ether, benzene, chloroform and acetone). Lipids are important dietary constituents because of their high energy value and also because they deliver fat soluble vitamins and essential fatty acids found in foods. The lipids include fatty acids, triglycerides, phospholipids, sphingolipids, sterols, waxes, glycolipids and lipoproteins.

Fatty Acids

Fatty acids vary with respect to their size, and the number and position of double bonds found in the molecule. They may be classified by the number of carbon atoms as short chain (C_{4-6}) medium chain (C_{8-12}) and long chain (C_{12+}).

Saturated fatty acids are devoid of double bonds and may be represented by the general formula $CH_3(CH_2)_n COOH$ where n can be any even number from 2 to 24. The most common ones are:

lauric acid	$CH_3 (CH_2)_{10} COOH$
myristic acid	$CH_3 (CH_2)_{12} COOH$
palmitic acid	$CH_3 (CH_2)_{14} COOH$
stearic acid	$CH_3 (CH_2)_{16} COOH$

Of the saturated fatty acids palmitic acid and stearic acid are the most widely distributed.

Monounsaturated fatty acids contain one double bond. The most common dietary monounsaturated fatty acid is oleic acid which has the formula:
$CH_3(CH_2)_7CH=CH(CH_2)_7COOH$.
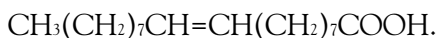
The polyunsaturated fatty acids contain two or more double bonds. Linoleic acid, linolenic acid and arachidonic acid are important examples of polyunsaturated fatty acids. Their formulas are shown:

linoleic acid:
$CH_3(CH_2)_4CH=CH. CH_2. CH=CH(CH_2)_7COOH$

α-linolenic acid:
$CH_3CH_2CH=CH. CH_2CH=CH. CH_2CH=CH(CH_2)_7COOH$

arachidonic acid:
$CH_3(CH_2)_4CH=CH.CH_2CH=CH.CH_2.CH=CH.CH_2CH=CH(CH_2)_7COOH$
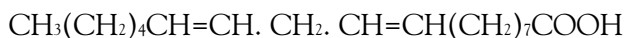
Linoleic and α-linolenic acid are **essential fatty** acids as they cannot be synthesized de novo and must be obtained from the diet. Arachidonic acid can be synthesized in the body from linoleic acid. If sufficient linoleic acid is provided in the diet, arachidonic acid requirements will be met. Of the unsaturated fatty acids, oleic acid and linoleic acid are the most widely distributed.

Triglycerides
Fats occur chiefly in foodstuffs and in the fat deposits of most animals in the form of triglycerides. Triglycerides are esters of glycerol and fatty acids and can be represented by the formula:

$$
\begin{array}{l}
CH_2\text{-}O\text{-}\underset{\underset{O}{\parallel}}{C}\text{-}R_1 \\[1em]
CH\text{-}O\text{-}\underset{\underset{O}{\parallel}}{C}\text{-}R_2 \\[1em]
CH_2\text{-}O\text{-}\underset{\underset{O}{\parallel}}{C}\text{-}R_3
\end{array}
$$
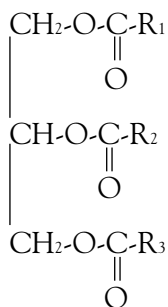
where R_1, R_2 and R_3 represent identical or different fatty acids with even numbers of carbon atoms. In naturally occurring fats, the component fatty acids of the various triglycerides are usually not identical.

In human and animal storage fat R_1 and R_3 are usually saturated fatty acids (palmitate and stearate) while R_2 is unsaturated. Human milk is an exception however: R_3 and R_3 are unsaturated. Pork fat is also an exception and follows a similar pattern to milk fat. Vegetable oils generally have an unsaturated fatty acid (usually linoleic acid) at the R_2 position with saturated fatty acids located at the R_1 and R_3 positions.

If the triglyceride is a solid at room temperature it is called a fat; if liquid it is an oil. The melting point of a fat depends on its fatty acid composition. Hard fats contain mostly saturated fatty acids while liquid oils are predominately composed of unsaturated fatty acids (Table 3-1). Saturation of the unsaturated fatty acids by hydrogenation will convert liquid oil into a hard fat (for example, solid white vegetable shortening and margarine). Isomerization of some of the remaining double bonds from the naturally occurring cis to trans form also occurs. Trans isomers have the same energy value as the cis form; however, their biological activities are altered. For example, trans isomers of linoleic acids do not have essential fatty acid activity.

Table 3-1		
Present Fatty Acid Content of Some Commonly Used Cooking and Salad Oils		
Type of Oil	**Saturated Fatty Acids %**	**Monounsaturated Fatty Acids %**	**Polyunsaturated Fatty Acids %**
canola	7	59	30
coconut	88	6	2
corn	13	25	59
cottonseed	26	18	52
olive	17	71	10
palm	52	38	10
peanut	17	48	32
safflower	10	15	75
sesame	14	40	42
soybean, partially hydrogenated	15	43	38
soybean, not hydrogenated	15	24	61
sunflower	12	24	65
walnut	10	15	70

Sources: ISEO: "Food, Fats and Oils," 6th ed. Washington, D.C.,
          Institute of Shortening and Edible Oils, 1988.

Phospholipids

After the triglycerides, the next largest lipid component in the body is phospholipid. A great variety of phospholipids is present in the body.

Phosphatidyl esters (phosphoglycerides) are an important class of phospholipids. They are found predominantly in animal tissues as a structural component of cell walls and mitochondria. In the blood, as a component of lipoproteins, phospholipids serve in the transport of fat between tissues. Only a very small amount of phospholipid is present in fat stores.

Phosphoglycerol has the formula:

$$
\begin{array}{l}
CH_2OH \\
| \\
CHOH \quad OH \\
| \qquad\quad | \\
CH_2 - O - P = O \\
\qquad\qquad | \\
\qquad\qquad OH
\end{array}
$$
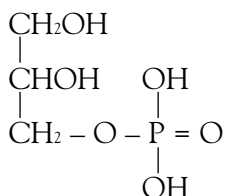
Phosphoglycerides are derived from phosphoglycerol by esterification of two hydroxyl groups with fatty acids and attachment of an alcohol moiety to the phospholipid radical. The phosphoglycerides may be represented by the general formula:

$$
\begin{array}{l}
CH_2 - O - C - R_1 \\
| \qquad\qquad || \\
| \qquad\qquad O \\
| \\
CH - O - C - R_2 \\
| \qquad\quad || \\
| \qquad\quad O \\
| \\
| \qquad\qquad O \\
| \qquad\qquad || \\
CH_2 - O - P - O - R_3 \\
\qquad\qquad OH
\end{array}
$$
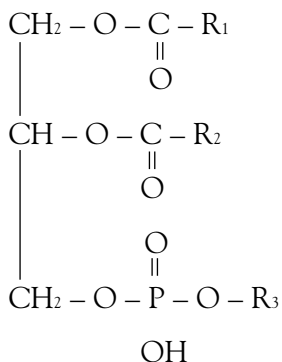
where R_1 is a fatty acid, usually saturated, R_2 is a fatty acid, usually unsaturated, and R_3 is an alcohol base (usually nitrogen containing). The constituent fatty acids vary widely. Like the triglycerides, phospholipids are not individual compounds but mixtures of different compounds. Ethanolamine and choline are the most common bases forming phosphatidylethanolamine and phosphatidylcholine (lecithin) respectively.

Sphingolipids
The sphingolipids are a class of lipids in which the glycerol is replaced by sphingosine or its derivative. Sphingosine is both an alcohol, and a nitrogen containing base as shown below:

$CH_3(CH_2)_{12}$. CH=CH.CH(OH). CH(NH$_2$). CH$_2$OH
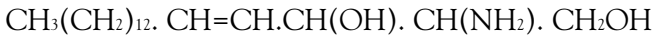
The three classes of sphingolipids are sphingomyelin, cerebrosides and gangliosides.

Sphingomyelin is an important constituent of the myelin sheath of nerves. It is present in most animal cell membranes. Sphingomyelin contains the base sphingosine, linked through a phosphate molecule to choline. In the cerebrosides, sphingosine is linked with one or more sugar units and a fatty acid is joined to the amino radical of the base. The cerebrosides are widely distributed in nervous tissues.

The gangliosides are similar to the cerebrosides except that the polar head group contains several sugar units. They are found in neural tissue membranes and to a lesser extent in other tissue membranes. Gangliosides are important as components of certain receptor sites on cell membranes.

Sterols
Sterols are alcohols containing the steroid structure of four fused carbon rings as shown in Figure 3-1. Like all alcohols, they can form esters with fatty acids and other organic acids.

Figure 3-1

The General Formula of Cholesterol
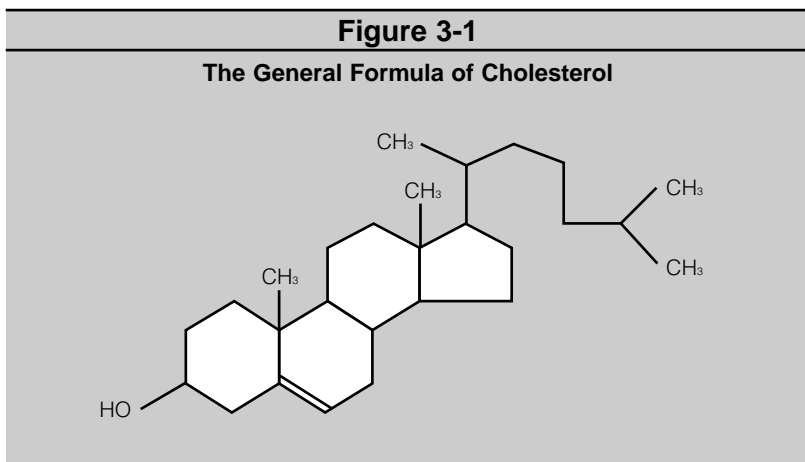
Table 3-2

Fatty Acid and Cholesterol Content of Some Common Foods

Food Items	Total Fat (g/100g)	Saturated Fatty Acid (g/100g)	Monounsaturated Fatty Acid (g/100g)	Polyunsaturated Fatty Acid (g/100g)	P/S	Cholesterol (mg/100g)
Meat/Fish/Poultry						
beef, 5% fat, cooked	4.9	1.7	1.9	0.2	0.1	84
lamb, 9% fat, cooked	9.2	3.3	4.0	0.6	0.2	92
veal, 6% fat, cooked	5.8	2.3	2.2	0.4	0.2	109
pork, 25% fat, cooked	25.1	9.1	11.5	2.8	0.3	82
chicken, light meat, no skin	3.9	1.2	1.1	0.9	0.8	77
salmon, pink, canned	6.1	1.5	1.8	2.1	1.3	55
lobster, cooked	0.6	0.1	0.2	0.1	0.8	72
shrimp, cooked	1.1	0.3	0.2	0.4	1.5	195
Dairy and Egg products						
egg, whole	10.0	3.1	3.8	1.4	0.4	425
egg, yolk	30.9	9.6	11.7	4.2	0.4	1281
milk, 3.5-4% fat	3.3	2.1	1.0	0.1	0.1	13
cheese, cheddar	33.1	21.1	9.4	1.0	0.04	104
Nuts						
almonds	56.5	5.3	36.7	11.9	12.3	0
peanuts	50.0	9.6	23.6	14.4	1.5	0
walnuts	61.9	5.6	14.2	39.1	7.0	0

Source: Shils M.E., Olson J.A., Shike M and Ross, A.C. (eds). Modern Nutrition in Health and Disease. 9th ed. Williams and Wilkins, Philadelphia, 1999.

Cholesterol is the predominant sterol in higher mammals. It is present in all animal tissues but not in plants. Egg yolk, dairy products, shellfish and meats contain fairly large amounts of cholesterol (Table 3-2).

Cholesterol is not an essential nutrient. All animals are capable of synthesizing cholesterol. The bile salts (sodium glycocholate and sodium taurocholate) are formed by the combination of the amino acids glycine and taurine with cholic acid which is a derivative of cholesterol. The bile salts play an essential role in emulsification, digestion and absorption of dietary fats. Estrogen, androgens, progesterone, and most of the adrenocortical hormones are derived from cholesterol.

Ergosterol is one of a group of sterols found in many plants, notably yeasts and fungi. Under the influence of ultraviolet light, it is converted into vitamin D as is 7-dehydrocholesterol which is the natural precursor of vitamin D in animal tissues.

Rather than cholesterol, plants contain a sterol compound known as phytosterol (plant sterol). Several different phytosterols occur naturally in plant foods and plant-derived oils.

Waxes
Waxes are fatty acid esters of higher alcohols. They occur widely in the cuticle of leaves and fruit and in the secretions of insects. Waxes are not an important constituent of any of the higher land animals nor do they contribute extensively to normal human diets.

Functions
Dietary fat is the most concentrated source of energy, supplying 9 kcal/g, (37 kJ/g). Like carbohydrate, dietary fat is protein sparing. Fats lend palatability to food as many of the substances responsible for the flavor and aroma of food are fat-soluble and are associated with fat in the diet.

Dietary fat acts as the source of fat-soluble vitamins and the essential fatty acids. The essential fatty acids are required for growth, reproduction, skin integrity, utilization of body fat and maintenance of cell membranes. They are also precursors of prostaglandins and a number of other compounds including thromboxanes, endoperoxides and leukotrienes.

Stored fat in the form of adipose tissue insulates and protects internal organs while serving as a reserve source of energy. Fats are important as structural components of cell and mitochondrial membranes as well as being constituents of specific membrane receptor sites.

Digestion and Absorption

The majority of dietary fat is ingested in the form of triglycerides (90-95 percent). Small amounts of cholesterol, cholesteryl ester, phospholipid (chiefly lecithin), mono- and diglycerides are also present in food.

Digestion of fat occurs mainly in the duodenum by the action of enzymes secreted by the pancreas. These enzymes include pancreatic lipase, colipase, cholesteryl esterase and phospholipase-A. Both the colipase and phospholipase-A are secreted in proenzyme form and activated by trypsin. For effective hydrolysis of triglycerides to occur, pancreatic lipase requires the coenzyme colipase, bile salts (secreted by the liver) and a basic pH. A basic pH is achieved in the duodenum by the release of bicarbonate from the pancreas. In concert with the peristaltic contractions of the intestine, the bile salts break up the fat into a fine emulsion exposing a large surface area to lipase action. Bile is also responsible for solubilizing the fat-soluble vitamins, which is essential for their normal absorption. The bile salt concentration tends to be low in newborn infants. Human breast milk contains its own lipase which hydrolyzes triglycerides independent of bile salt concentration.

The absorption of fat into the intestinal cells from the lumen is dependent upon micellar formation (Figure 3-2). Micelles are molecular aggregates largely composed of B-monoglyceride, free fatty acids and bile salts. Small amounts of cholesterol, lyzophospholipid and the fat-soluble vitamins are also present. Micelles are water soluble; their lipid constituents are not. Micelles can render lipids soluble in the aqueous medium of the intestine thereby enabling their entry into the brush border of the intestinal cells. Uptake of micellar lipid is believed to occur by passive diffusion. The bile salts do not enter the cells but are released and move down the lumen to be reabsorbed in the ileum whereupon they return to the liver. A small proportion of bile salts is excreted in the feces.

Within the mucosal cells, resynthesis of triglyceride and phospholipid occurs. Some of the absorbed cholesterol is re-esterified to cholesteryl ester.

Since lipids are insoluble in water they must be complexed with protein (lipoprotein) for transport in the lymph and blood. Most absorbed dietary fat is transported in the form of lipoproteins or chylomicrons. The chylomicrons are released into the lymphatic system and ultimately enter the blood.

Most of the dietary triglycerides are composed of long chain triglycerides (LCT) as their fatty acid moieties contain more than 12 carbons. Their digestion, absorption and transport occur as described above.

Figure 3-2

Absorption and Transport of Fat
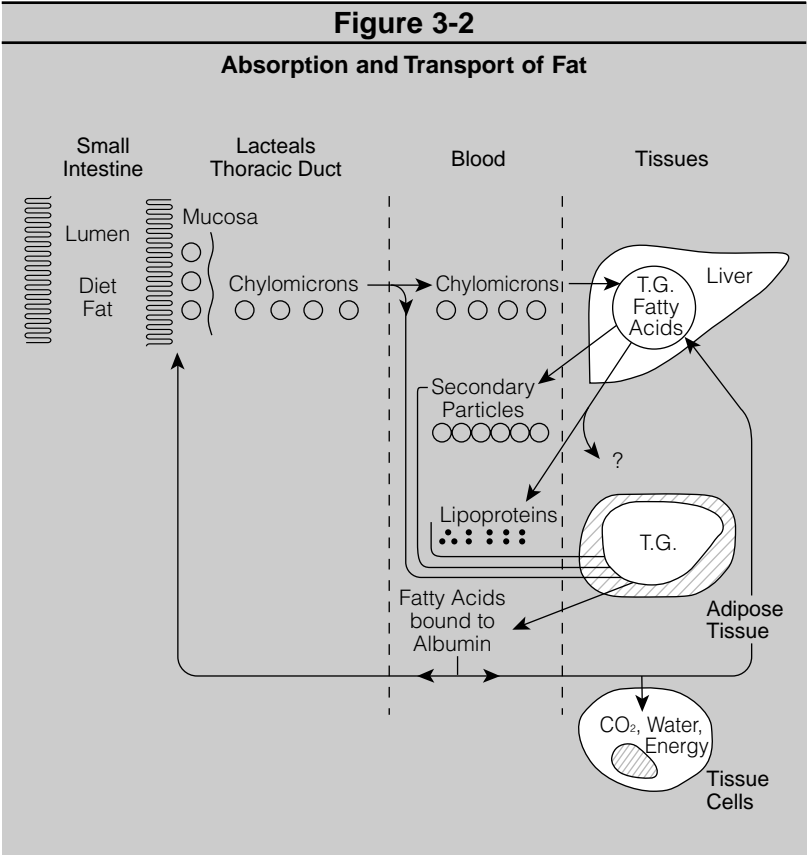
Short and medium chain triglycerides (SCT and MCT) are metabolized differently. Although the bile salts aid in the digestion and absorption of MCT and SCT, they are not required since both MCT and SCT are sufficiently soluble in an aqueous medium. Many of the B-monoglycerides of the MCT and SCT undergo further hydrolysis to glycerol and free fatty acids. However, intact MCT and SCT can be directly absorbed. The intact triglycerides are then hydrolyzed in the mucosal cells.

Unlike the LCT which leave the mucosal cells as chylomicrons via the lymphatics, the bulk of the MCT and SCT leave the intestine by the portal vein complexed with albumin. MCT and SCT then enter the liver where they are predominantly oxidized. Lauric acid, which consists of 12 carbon atoms, enters the circulatory system by both routes.

Transport of Lipids

Lipids are rendered soluble in plasma by their association with proteins. The majority of plasma lipids are transported as lipoproteins. The lipoproteins are composed of a non-polar core of triglyceride and cholesteryl ester surrounded by cholesterol with an outer layer of hydrophilic protein and phospholipid. The four main classifications of the lipoproteins are the chylomicrons, very low density lipoproteins (VLDL), low density lipoproteins (LDL) and high density lipoproteins (HDL). Their lipid composition is shown in Table 3-3. The proteins associated with lipoproteins are termed apoproteins.

Table 3-3				
The Composition of Plasma Lipoproteins				
Lipoproteins	Protein	Phospholipids	Cholesterol	Triglycerides
Chylomicrons S_f 400* and above	0.5-2.5	3-15	2-12	79-95
Very-low-density (VLDL) S_f 20-400 (prebeta-)	2-13	10-25	9-24	50-80
Low-density (LDL) S_f 0-20 (beta-)	20-25	22	43	10
High-density (HDL) (alpha-)	45-55	30	18	5-8

* S_f refers to flotation rate-Svedberg units. Density of lipoproteins varies inversely with S_f.

Free fatty acids are transported in the bloodstream bound to plasma albumin. Fat soluble vitamins and carotenoids, sterols and hormones are also transported in the blood in combination with specific plasma proteins.

Chylomicrons

Chylomicrons are synthesized in the intestinal mucosal cells. They are largely composed of long chain triglycerides obtained from the diet. Chylomicrons transport most of the dietary triglyceride from the intestine. Upon entering the bloodstream via the thoracic duct, chylomicron triglycerides are subject to hydrolysis by lipoprotein lipase present in the capillary endothelium. The liberated free fatty acids and glycerol then enter the tissues. The chylomicron remnant is taken up by the liver and catabolized. By this process most of the dietary cholesterol and some of the dietary triglyceride is transported to the liver. Portions of the chylomicron remnants do not enter the liver and are believed to form nascent HDL particles instead.

VLDL

VLDL serve to transport endogenous triglycerides to extrahepatic tissues. The majority of VLDL is synthesized in the liver but a small portion is formed in the gut.

The VLDL triglycerides are hydrolyzed by lipoprotein lipase in a similar fashion to the chylomicrons. However, most VLDL remnants do not undergo hepatic uptake. Instead, they undergo a series of reactions forming intermediate density lipoproteins and ultimately low density lipoproteins (LDL). Some VLDL remnants are believed to be catabolized to form nascent HDL.

LDL

LDL serve to deliver cholesterol from the liver (via VLDL) to the extrahepatic tissues. Over two-thirds of the total plasma cholesterol is carried by LDL. Cellular uptake is receptor-mediated. LDL particles bind to a cell-surface receptor and enter the cell by endocytosis. LDL is subsequently catabolized in the cell liberating cholesterol. The increase in intracellular cholesterol activates a series of reactions resulting in decreased intracellular cholesterol synthesis, increased esterification of cholesterol as cholesteryl ester for storage and decreased synthesis of cell-surface LDL receptors. By this mechanism, levels of intracellular free cholesterol are controlled.

HDL

Nascent HDL particles are believed to be synthesized in the liver and gut. Some nascent HDL is thought to be derived from chylomicron and VLDL catabolism. Nascent HDL particles are discoid in appearance and are composed of phospholipid, cholesterol and protein. HDL cholesterol accounts for approximately one-fifth of the total plasma cholesterol.

The HDL are believed to deliver cholesterol from the peripheral tissues to the liver where it is catabolized and HDL cholesterol is eliminated via the biliary system.

Metabolism

Excess dietary triglycerides are stored mainly in adipose tissue. They may be transported there by chylomicrons or by VLDL. Under conditions of energy deficit triglycerides are mobilized and oxidized to form ATP. The main pathways of lipid metabolism include the following:

Lipolysis and Beta-oxidation

The hydrolysis and release of triglycerides from adipose stores are controlled by a hormone sensitive tissue lipase. This enzyme is much more active in fat cells of fasted individuals than in fed individuals. It is activated by epinephrine, norepinephrine, glucagon, ACTH and several other hormones, but deactivated by insulin and prostaglandin E_1. Lipolysis (fat breakdown) is a stepwise reaction which removes fatty acids from the glycerol moiety one at a time.

These fatty acids are then catabolized by a series of reactions known as beta-oxidation. The term beta-oxidation arises from the fact that oxidation occurs at the carbon that is in the beta-position relative to the carboxyl group. Beta-oxidation consists of a cyclic series of reactions in which two carbons are removed from the fatty acid per cycle in the form of acetyl CoA. The acetyl CoA then proceeds through the citric acid (Krebs) cycle and oxidative phosphorylation to produce ATP, CO_2 and water.

Ketosis

Ketosis occurs when the rate of formation of ketones by the liver is greater than the ability of extrahepatic tissues to oxidize them. The accumulation of ketones lowers blood pH resulting in acidosis. Ketosis results whenever large amounts of fat are ingested in the absence of carbohydrates or during prolonged starvation when glycogen stores are depleted and fat alone is catabolized for energy.

The ketone bodies include acetoacetate, beta-hydroxybutyrate and acetone. The liver is incapable of oxidizing ketone bodies. The ketone bodies enter the bloodstream to be oxidized in the extrahepatic tissues. Acetoacetate and beta-hydroxybutyrate are converted back to acetyl CoA and enter the citric acid (Krebs) cycle. Acetone is not readily oxidized and is excreted via the lungs (where it contributes a characteristic acetone odor) and urine.

Lipogenesis

Fat synthesis (lipogenesis) and breakdown (lipolysis) take place continuously. If dietary intake of carbohydrate, protein and fat exceeds the body's energy requirements, they are stored as triglycerides in the liver and adipose tissue. The main sites of triglyceride synthesis are the liver, adipose tissue and the intestinal mucosa. The fatty acids for lipogenesis are derived from the hydrolysis of fats and also from the synthesis of acetyl CoA through the oxidation of fats, glucose and some amino acids.

Lipogenesis from acetyl CoA is accomplished essentially by building up the carbon chain by the successive addition of 2-carbon fragments. NADPH generated by the Pentose Phosphate Shunt is required for this process. The fatty acids as acetyl CoA are then attached stepwise to glycerophosphate to form the triglyceride.

Cholesterol Metabolism

Due to the association between elevated plasma cholesterol levels and increased risk of coronary heart disease, the metabolism of cholesterol in the body deserves mention.

Cholesterol may be obtained from the diet or synthesized endogenously in a variety of tissues including the liver, adrenal cortex, skin, intestine, testes and aorta. Increasing dietary cholesterol suppresses cholesterol synthesis in the liver but not in other

tissues. Nevertheless, this results in an increase in total body cholesterol. Synthesis of cholesterol may be affected by levels of saturated fat in the diet. Most of the dietary cholesterol is transported to the liver as a component of the chylomicron remnants. Cholesterol is delivered from the liver to tissues by LDL derived from VLDL and is transported from the tissues to the liver by LDL as well as HDL.

In the liver, cholesterol may be converted into bile acids and enter the enterohepatic circulation. Ninety to 95 percent of the bile salts are reabsorbed; the remainder are lost in the feces. Excretion of cholesterol from the body is associated with this excretion of bile salts in the feces. Some of the cholesterol is converted by intestinal bacteria to neutral steroids and is also lost by this route.

Relationship between Fat and Carbohydrate and Protein

Both carbohydrate and protein, when present in excess quantities can be converted to fat. Carbohydrate may be converted to triglyceride utilizing glycerol phosphate and acetyl CoA obtained from glycolysis (Figure 1-3). Ketogenic amino acids that are metabolized to acetyl CoA may be used for synthesis of triglycerides.

Hydrolysis of triglycerides can contribute to gluconeogenesis by the release of glycerol which in turn is used in the synthesis of glucose. However, the hydrolyzed free fatty acids cannot contribute to glucose synthesis nor to protein synthesis.

Levels and Sources of Fats in the Diet

Approximately 38% of the energy content of diets of affluent societies is contributed by dietary fat. This is generally considered too high for optimal health. The general consensus among nutritionists is that calories from fat should be maintained at approximately 30% of energy intake, equally divided among saturated, monounsaturated and polyunsaturated fats. The aim is to moderate dietary fat intake while providing an adequate intake of the essential fatty acids. An adequate intake of essential fatty acids is believed to be five percent of dietary energy.

The chief sources of dietary fat are vegetable oils, butter, margarine, shortening, lard, milk, cream, cheese, eggs, fish and meat. Examples of the fat content in some commonly consumed foods

is shown in Table 3-2. Sources of the essential fatty acid linoleic acid include the polyunsaturated oils, soybean, safflower, cottonseed, canola and corn oil. Arachidonic acid, while found in small amounts in animal tissues, is the major polyunsaturated fatty acid of animal fats excluding fish. Linolenic acid rich foods include soybean oil and linseed oil. Sources of the longer chain derivatives of linolenic acid include fish and shellfish.

Dietary Fat and Blood Cholesterol

Elevated plasma LDL cholesterol levels increase the risk of cardiovascular diseases (CVD), such as coronary vascular disease and atherosclerosis, which are major causes of death in affluent societies. Although evidence for the cause and effect relationship between elevated blood cholesterol and CVD is not yet established, epidemiological studies do suggest an increased risk of the development of CVD as plasma cholesterol rises above 220 mg/dL (5.7 mmol/L) (Table 3-4 shows normal lipid levels in the blood).

A high fat diet, specifically if one high in **saturated fat**, is one of the risk factors associated with elevated blood cholesterol. Evidence for an effect of dietary cholesterol on blood cholesterol is inconclusive. Foods that have a high cholesterol content include liver, egg yolk, kidney, brains, sweetbreads, shellfish and fish roe (Table 3-2). The effect of saturated fats is much more pronounced than that of increasing dietary cholesterol. All animal fats contain a high proportion of saturated fats with the exception of those from fish and shellfish. Vegetable oils are usually composed of high levels of **unsaturated fatty acids**. Palm and coconut oils are the exception. Over 80 percent of the fatty acids in coconut oil are saturated.

Table 3-4	
Normal Blood Lipid Levels in Humans	
Lipid	**Normal Range in mg/dL (mmol/L)**
Total Cholesterol	50-220 (3.9-5.7)
HDL	> 50 (> 1.3)
LDL	< 130 (< 3.4)
VLDL	< 40 (<1.0)

Source: Murray, R.K. et al., **Harper's Biochemistry**.
    24th ed. Appleton and Lange, Stamford, 1996.

Increasing the levels of **polyunsaturated fat** (particularly n-6 polyunsaturates) in the diet has a hypocholesterolemic effect (LDL and HDL-cholesterol). However, saturated fats are twice as effective in raising cholesterol as polyunsaturated fats are in lowering it. Most vegetable oils for example, soybean oil, safflower oil, cottonseed oil and corn oil are rich in polyunsaturated fatty acids.

Monounsaturated fatty acids in the diet have a lowering effect on LDL cholesterol but not HDL cholesterol. Peanut oil and olive oil are examples of fats which are rich in the monounsaturated fatty acid, oleic acid.

Stearic acid, a saturated fatty acid of 18 carbons, does not share the hypercholesterolemic effect of other saturated fatty acids. Beef is a common food rich in stearic acid. Unfortunately, beef also contains other saturated fatty acids which raise plasma cholesterol levels.

Trans Fatty Acids

Trans polyunsaturated fatty acids are formed during hydrogenation of polyunsaturated fatty acids. Trans unsaturated fatty acids are produced commercially in large amounts by heating vegetable oils in the presence of a metal catalyst and hydrogen to form shortening and margarines. The extent of hydrogenation is dependant on the PUFA content of the starting oil and the physical properties of the end product. Partial hydrogenation is a technique used to stabilize PUFAs and to create solid fats, but it removes important PUFAs such as linoleic and linolenic acids from the diet.

 Trans fatty acids do not exhibit the cholesterol lowering properties of the naturally occurring cis form of polyunsaturated fats. In fact, trans fatty acids behave like saturated fats. Metabolic and epidemiological studies have shown that the effects of trans fatty acids on cardiovascular diseases appear to be stronger than those of saturated fatty acids. Replacing cis fatty acids with trans in the diet increases LDL cholesterol and Lp(a) and lowers HDL cholesterol. Trans fatty acids have also been found to increase the requirements for EFA.

Trans fatty acids are found mainly in hydrogenated vegetable oil products such as margarines and shortenings and may constitute up to 35% of the fat in these foods. They also occur naturally in small quantities in milk and butter. Some margarines are made from the addition of a polyunsaturated oil to a more solid hydrogenated fat. This process can produce a margarine with a significantly higher content of cis-form PUFA. Soft margarines have lower levels of hydrogenated fats than the hard ones. Non-hydrogenated margarines are available commercially.

Omega - 3 Fatty Acids

The essentiality of the omega-3 (n-3) fatty acids, in particular α-linolenic acid, has been an unresolved issue in human nutrition for several decades. Now there is evidence that the omega-3 fatty acids have a distinct role in the structure and function of biological membranes, particularly in the retina in the eye and the central nervous system. Besides their role as structural components of biological membranes, the long chain n-3 and n-6 fatty acids are precursors of prostaglandins, prostacyclins, thromboxanes and leuko-trienes.

Animals cannot synthesize fatty acids with double bonds at either the n-3 or n-6 positions and are therefore dependent on dietary sources for these fatty acids. They can, however, further desaturate and elongate these fatty acids to arachidonic acid and docosahexanoic acid (DHA) which are important components of membrane lipids. Besides their role as structural components of biological membranes, the long chain n-3 and n-6 fatty acids are precursors of prostaglandins, prostacyclins, thromboxanes and leuko-trienes.

Omega-3 fatty acids are present in mammalian tissues primarily in the form of DHA and are generally most concentrated in phospholipids. Very high levels are found in the retina, cerebral cortex, testes and sperm. The levels of DHA in brain and retinal phospholipids are similar in mammals despite wide variations in dietary lipids. This suggests that DHA plays an important role in brain and retinal functions.

DHA is critical nutrient for growth and functional development of the infant brain. Since DHA is found in sufficient quantities in human milk but not in conventional formulas, breast-fed

children have been shown to perform better on tests of development or cognition, verbal ability or school performance. The inclusion of DHA in the diet improves learning ability as well as visual acuity. Availability of dietary DHA is critical for the development of preterm infants, since DHA uptake by brain and retina increases substantially during the last trimester of pregnancy. DHA deficiency may be associated with fetal alcohol syndrome, attention deficit hyperactivity disorder, cystic fibrosis, phenylketonuria, unipolar depression, aggressive hostility and adrenoleukodystrophy. A decrease in DHA in the brain may be associated with cognitive decline during aging and with onset of sporadic Alzheimer's disease.

In recent years, fish and fish oils containing high levels of eicosapentanoic acid (EPA) and DHA were found to have beneficial effects on hypertension, hyperlipidemia, thrombosis, coronary heart disease and immunological disorders. Fish oils are found to have a universal and pronounced hypotriglyceridemic effect in both normal and hypertriglyceridemic patients. This appears to function through the suppression of very low density lipoprotein synthesis and stimulation of high density lipoprotein production. Additionally, the Omega-3 fatty acids tend to increase bleeding time by inhibiting platelet aggregation, but the exact mechanism for the platelet inhibition has not been elucidated.

The quantitative requirement for Omega-3 fatty acids in human nutrition is not clear. It appears that consumption of 4 to 6 ounces (120-170 g) of fish several times a week may be beneficial to health. Fish, such as salmon, mackerel, mullet, trout, canned sardines, smelt and tuna, shellfish and marine mammals contain large quantities of DHA and therefore provide the human diet with a rich source of Omega-3 fatty acids (Table 3-5).

Table 3-5

Omega-3 Fatty Acid Content of Some Common Fish Products

Food Items   **Omega-3 Fatty Acids**

	ALA (alphalinolenic acid) 18:3[1] (g/100g)	EPA (eicosapentanoic acid) 20:5 (g/100g)	DHA (docosahexanoic acid) 22:6 (g/100g)
Finfish			
anchovy, European	-3	0.5	0.9
bass, stripped, raw	tr[2]	0.2	0.6
carp, raw	0.3	0.2	0.1
cod, Atlantic	tr	0.1	0.2
flounder, unspecified	tr	0.1	0.1
haddock	tr	0.1	0.1
halibut, raw	tr	0.5	0.4
herring, Atlantic	0.1	0.7	0.9
mackerel, Atlantic	0.2	0.9	1.4
perch, all varieties	0.1	0.9	1.6
pike, walleye	tr	0.1	0.2
salmon, chinook	0.1	0.8	0.6
salmon, coho	0.2	0.4	0.7
smelt, rainbow	0.1	0.3	0.4
snapper, red	tr	tr	0.2
sole, European	tr	tr	0.1
trout, rainbow	0.1	0.2	0.4
tuna, bluefin	—	0.4	1.2
whitefish, lake	0.2	0.3	0.9
Crustaceans			
crab, Alaska, king	tr	0.2	0.2
lobster, cooked	—	0.1	tr
shrimp, unspecified	tr	0.2	0.1

1 The first number (18) indicates the number of carbon atoms, and the second number (3) denotes the number of double bonds in the fatty acid.

2 tr=trace amounts

3 dashes denote lack of reliable data for nutrient known to be present

Sources: Provisional Table on the Content of Omega-3 Fatty Acids and Other Fat Components in Selected Foods, U.S. Department of Agriculture, Human Nutrition Information Service, HNIS/PT-103, 1988.

U.S. Department of Agriculture, USDA Nutrient Database for Standard References, Release 13, Agricultural Research Service, 1999.

Table 3-6

Characteristics of Hyperlipoproteinemias

Type	Other Names	Genetic Form	Plasma Cholesterol Level	Plasma Triglyceride Level	Risk Factor in Athero-sclerosis	Major Secondary Causes	Clinical Presentation	Treatment
I	Exogenous hypertriglyceridemia Familial hyperglyceridimia Familial chylomicronemia Fat-induced hyperlipidemia Hyperchylomicronemia	Autosomal recessive; rare	Normal or slightly increased	Very greatly increased	Risk not apparently increased	SLE; dysgamma globulinemia; insulinopenic diabetes mellitus	Pancreatitis Eruptive xanthomas Hepatosphenomegaly Lipemia retinalis	*Dietary:* low intake of fat; no alcohol; weight reduction
II	Familial hypercholesterolemia Familial hyperbetalipoproteinemia Familial hypercholesterlemic xanthomatosis	Autosomal dominant; common	Greatly increased	(a) Normal (b) Slightly increased	Very strong risk factor, especially for coronary atherosclerosis	Excess dietary cholesterol; hypothyroidism; nephrosis; multiple myeloma; porphyria; obstructive liver disease	Accelerated atherosclerosis Xanthelasma Tendon and tuberous xanthomas Juvenile corneal arcus	*Dietary:* low cholesterol, low fat diet consisting mainly of polyun-saturated fats. *Drugs:* cholestyramine; colestipol; niacin; probucol. *Possible Surgery*
III	Broad beta disease Familial dysbetalipoproteinemia Floating betalipoproteinemia	Mode of inheritance unclear; uncommon but not rare	Greatly increased	Greatly increased	Very strong risk factor for atherosclerosis, especially in peripheral circulation	Dysgammaglobulinemia; hypothyroidism	Accelerated atherosclerosis of coronary and peripheral vessels Planar xanthomas Tuboeruptive and tendon xanthomas	*Dietary:* reduction to ideal weight; mainten-ance of low cholesterol, balanced diet *Drugs:* clofibrate; niacin
IV	Endogenous hypertriglyceridemia Familial hyperprebetalipo-proteinemia Carbohydrate-induced	Common, often sporadic when familial; genetically heterogeneous	Normal or slightly increased	Greatly increased	Probable risk factor, especially for coronary atherosclerosis	Excess alcohol consumption; oral contraceptives; diabetes mellitus; glycogen storage disease; pregnancy; nephrotic syndrome; stress	Possible accelerated atherosclerosis Glucose intolerance Hyperuricemia	*Dietary:* Weight reduc-tion; low carbohydrate diet; no alcohol *Drugs:* niacin
V	Mixed hypertriglyceridemia Combined exogenous and endogenous hypertriglyceridemia Mixed hyperlipemia	Uncommon but not rare; genetically heterogeneous	Normal or slightly increased	Very greatly increased	Risk of atherosclerosis not clearly increased	Alcoholism; insulin dependent diabetes mellitus; nephrosis; dysgammaglobulinemia	Pancreatitis; Eruption xan-thomas; Hepatospheno-megaly; Sensory neuropathy; Lipemia retinalis; Hyperurice-mia; Glucose intolerance	*Dietary:* Weight reduc-tion; low fat diet; no alcohol *Drugs:* niacin

Hyperlipoproteinemias

Elevated plasma lipids and cholesterol levels have been classified into 5 types of hyperlipoproteinemias (Table 3-6). They are differentiated by their appearance in serum, the levels of cholesterol and triglyceride and the classes of lipoproteins that serve to transport the lipids. Treatment of these conditions consists of the diet modification and may involve:

- ❑ decreased total fat
- ❑ decreased saturated fat
- ❑ decreased energy intake
- ❑ limited cholesterol intake
- ❑ limited simple sugar intake
- ❑ increased complex carbohydrates
- ❑ moderate alcohol consumption

Dietary Fat and Cancer

High levels of dietary fat are thought to increase the risk of many types of cancer, especially colon, lung, ovary and prostate cancers. Furthermore, epidemiological as well as animal studies have demonstrated that not only the amount but also the type of fat consumed is important. Not all fats increase the risk of cancer; preliminary evidence suggests that omega-3 fatty acids may protect against certain cancers. At present, the mechanism by which dietary fat increases cancer risk is not clear; however, modulation of prostaglandin and leukotriene synthesis appears to be a possible mechanism. The dietary recommendations made by AICR/WCRF to reduce cancer risk include limiting the total fat intake and using vegetable oil instead of animal fat. The overall amount of fat in a diet should not be more than 30% of total energy intake, with a predominance of mono- and polyunsaturated forms. Use of monounsaturated fats, such as olive oil, especially with minimum hydrogenation, may be beneficial.

Fat Substitutes

These are substances that provide similar viscosities and organoleptic properties as fat. Since these are not metabolized in the gut, they have therefore been recommended for use in low fat

foods and diets for fat management. At present two types of fat substitutes are approved by the FDA for human use.

Microencapsulated Proteins: Fat substitutes containing microencapsulated dietary proteins from egg and/or milk and which provide a creamy smooth sensation very much like that of fat are available. Since it is a protein, it contains only 3.8 kcal/g instead of 9 kcal/g energy and it is digested similarly to dietary protein. Since the encapsulation process does not ensure heat stability, this may only be used in low fat foods and diets not involving cooking or frying. A microencapsulated protein product has been approved by the FDA as **GRAS** for use as a fat substitute in low temperature products such as ice cream. A product made from oat bran and has been shown to be useful in glycemic load and cholesterol management. Because these products are made from dietary proteins, there are important safety concerns regarding their possible antigenicity and allergenicity.

Sucrose polyesters made from sucrose and vegetable oils are another type fat substitute. They are mixtures of 6 to 8 molecules of dietary fatty acids esterified to the hydroxyl groups of sucrose. The physical characteristics are dependent upon the type of fatty acids used. It is heat stable and is not metabolized in the body. The organoleptic properties are similar to those of common dietary fats. Because of these properties they behave like a dietary fat in food but their nutritional effects are different from dietary fat. Because they are not absorbed or metabolized in the gut, important safety concerns related to the absorption of fat-soluble vitamins, have been raised. The FDA has approved the use of such sucrose polyesters as a replacement for fats and oils in certain types of foods and is currently available in potato chips and similar snack foods in the Unites States. However, FDA approval also required addition of 1.9 mg α-tocopherol, 51 mg retinyl acetate/palmitate, 12 IU vitamin D and 8 mg vitamin K_1 per gram of sucrose polyester to compensate for the antinutritive effects on fat soluble vitamins.

Suggested Readings

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CHAPTER 4

VITAMINS

The vitamins constitute a group of organic compounds which are essential in small quantities for the normal metabolism of other nutrients and maintenance of physiological well-being. These compounds cannot be synthesized by the body and must be obtained from the diet. Vitamins are found in varying quantities in different foods. Most foods contain a variety of vitamins, but no one food contains all of them in sufficient quantities to satisfy human requirements. Absence or relative deficiency of vitamins in the diet can lead to a characteristic deficiency state and disease. These deficiencies can be avoided by consuming a wide variety of foods in adequate amounts.

The vitamins are classified according to their solubility in water and fat solvents. The fat soluble vitamins include: vitamin A (retinol), vitamin D (calciferol), vitamin E (tocopherol), and vitamin K (menadione). The water soluble vitamins include: vitamin B_1 (thiamin), vitamin B_2 (riboflavin), vitamin B_3 (niacin), biotin, vitamin B_6 (pyridoxine), pantothenic acid, folate, vitamin B_{12} (cobalamin) and vitamin C (ascorbic acid).

The vitamins may also be differentiated by their biological activities. In general, fat soluble vitamins are stored in appreciable amounts in body tissues and, do not have to be supplied daily in the diet. The water soluble vitamins are not stored to any great extent and therefore need to be included in the diet every day. Habitual intake of excessive amounts of fat soluble vitamins is toxic. Excessive amounts of water soluble vitamins are usually excreted in the urine. However, harmful effects of extremely high doses of some of the water soluble vitamins have been reported.

FAT SOLUBLE VITAMINS
VITAMIN A
Vitamin A refers to any compound or mixture of compounds having vitamin A activity. In animals, vitamin A exists largely in the preformed state as retinol or as one of its related compounds: 3-dehydro-retinol, retinal, retinyl ester or retinoic acid. In plants,

vitamin A occurs in the precursor or provitamin forms as carotenoids which may be converted into vitamin A. Vitamin A cannot be synthesized *de novo* by plants or animals and carotenoids are the only source of vitamin A for the entire animal kingdom.

Carotenoids are a class of closely related natural pigments synthesized by plants. Their main function is to absorb light during photosynthesis and provide protection against photosensitization. Over 600 different carotenoids have been identified and approximately 40 of these occur in common food sources. Beta-carotene, alpha-carotene, lutein, alpha-cryptoxanthin and lycopene are the most common carotenoids found in human plasma. Some of these carotenoids, such as beta-carotene, alpha-carotene, and alpha-cryptoxanthin, are metabolized in the small intestine and function as precursors of vitamin A. However, other carotenoids, such as lycopene and lutein, are devoid of provitamin A activity. Carotenoids are also potent antioxidants and important physiological modulators. In recent studies, lycopene has been suggested to have potential anticarcinogenic properties.

Retinol is a high molecular weight alcohol attached to a beta-ionone ring (Figure 4-1). The beta-ionone ring is essential for vitamin A activity. Retinol is a pale, viscous, fat soluble compound which is fairly heat stable but easily destroyed by oxidation

Figure 4-1

Structure of Retinol and Beta-Carotene

Retinol
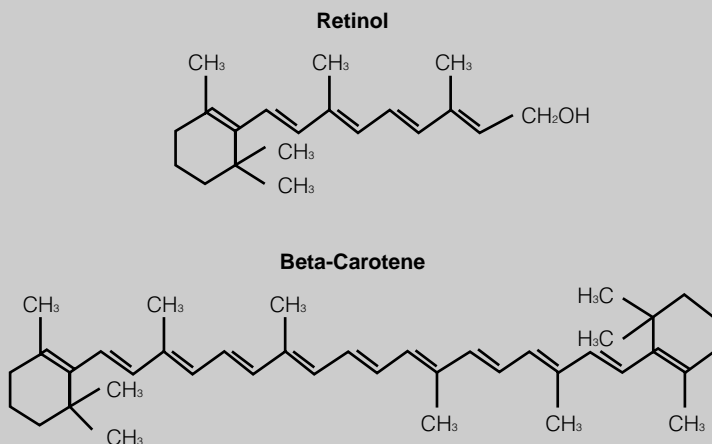
Beta-Carotene

and ultraviolet light. Beta-carotene is a symmetrical molecule consisting of two beta-ionone rings conjugated by a double bond in the center (Figure 4-1). Theoretically, hydrolysis of beta-carotene in the gut should yield two molecules of retinol. However, because of physiologic inefficiency in conversion of beta-carotene to retinol, the overall utilization of dietary beta-carotene as vitamin A from food is taken as one-sixth that of retinol. The efficiency of utilization of the other provitamin A carotenoids is about half that of beta-carotene.

Table 4-1

Vitamin A Content of Some Common Foods

Food Items	Vitamin A (IU/100g)[d]
Dairy and Egg Products[a]	
butter, regular	3,058
eggs, raw, whole	635
cheese, cheddar	1,059
milk, cow's, fluid, (whole)	126
Vegetables[b]	
carrot, raw	28,129
chives, raw	4,353
collards, boiled	3,129
lettuce, romaine, raw	2,600
cabbage, savoy, boiled	889
tomato, red, ripe, raw	623
Fruits[b]	
apricots, raw	2,612
papaya, raw	284
oranges, raw	205
Liver[c]	
beef, cooked, fried	36,105
lamb, cooked, fried	25,998
chicken, cooked	16 375
Fish[c]	
halibut, cooked, dry heat	179
sardines, canned in oil	224

[a] Vitamin A values based on chemically determined preformed vitamin A and β-carotene.

[b] Vitamin A values based on provitamin A carotenoids only.

[c] Vitamin A values based on chemically defined preformed vitamin A.

[d] 10,000 IU = 3000 RE

See Tables 6-4 and 6-5 for recommended intakes for Canadians and Americans.

Source: U.S. Department of Agriculture, USDA Nutrient Database for Standard References, Release 13, Agricultural Research Service, 1999.

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Sources

The average North American diet provides about half of its vitamin A activity as carotenes from plant sources. Beta-carotene is the most significant carotenoid in the diet; gamma-and alpha-carotene and cryptoxanthine are also present in the diet. Sources of the provitamin A carotenoids include dark green leafy vegetables (chlorophyll masks the yellow carotene color), deep yellow vegetables, tomatoes and deep yellow fruit. Table 4-1 shows examples of the vitamin A content in some commonly consumed foods.

The remainder of dietary vitamin A is obtained from preformed vitamin A from animal sources. Sources of preformed vitamin A include liver, fish liver oil extracts, egg yolks, enriched milk products such as margarine and skim milk and evaporated milk. Some animal products contain both preformed vitamin A and provitamin A; the latter being obtained from the animal's diet.

Absorption and Storage

Free retinol obtained from the diet or hydrolyzed from retinyl esters in the gut is absorbed into the intestinal mucosal cells, re-esterified and incorporated into chylomicrons, which ultimately enter the circulation. Chylomicron remnants containing retinyl esters are taken up by the liver. Conversion of the ingested carotene to vitamin A takes place primarily in the cells in the intestinal mucosa but also occurs in the liver and possibly the kidney.

Figure 4-2

Interrelationships Among Retinol, Retinal and Retinoic Acid
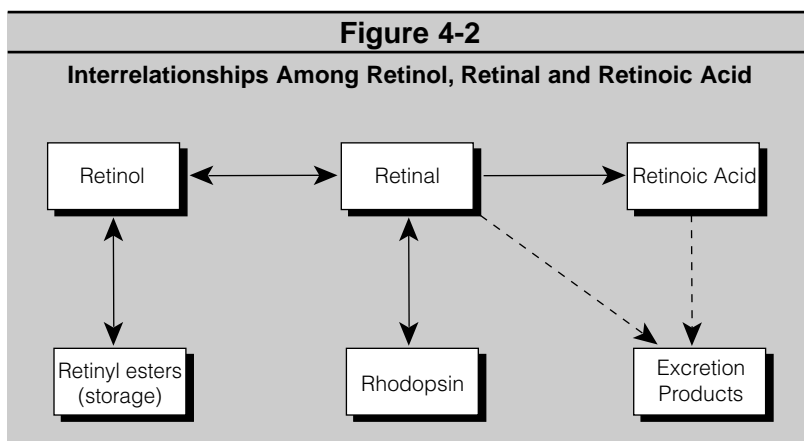
Absorption of vitamin A and the carotenoids requires the presence of bile in the intestinal tract and other conditions favorable for fat absorption. Retinoic acid, however, is absorbed directly into the intestinal mucosa and released into the portal circulation complexed with albumin.

Storage of vitamin A occurs primarily in the liver as retinyl ester. When needed, retinol is mobilized from the liver and transported in the circulation to tissues complexed with retinol binding protein (RBP).

The metabolic relationship among retinol, retinal, retinoic acid and retinyl ester is shown in Figure 4-2. The conversion of retinal to retinoic acid is irreversible.

Function

Vitamin A plays an important role in normal vision. The photoreceptors of the eye in the retina, which are sensitive to dim light, are the rods that contain 11-cis retinal that combines with the protein opsin to form rhodopsin in the dark. Rhodopsin when bleached by light is converted to all trans retinal and opsin. Conversion of the trans 11-cis retinal completes the cycle (Figure 4-3). However, when insufficient retinal is available to regenerate rhodopsin, the conversion is incomplete and night blindness (inability to see in dim light) results.

Retinal is also involved in daytime vision as a component of iodopsin. This pigment, contained in the cones of the retina, is sensitive to bright light.

Vitamin A is essential for the integrity and normal growth of epithelial cells. It is also involved in cell differentiation. In the presence of sufficient vitamin A, mucus secreting goblet cells are formed from epithelial basal cells. When there is a lack of vitamin A, the basal cells keratinize becoming hard, dry and irregular in shape.

Vitamin A is required for proper growth and development of bones and teeth. It is also necessary for normal reproduction in animals. Vitamin A is important for the maintenance of membrane integrity and functions of membranes such as those in the skin, respiratory and genito-urinary tract.

Retinoic acid supports normal growth but cannot replace retinol and retinal for night vision.

Deficiency

Vitamin A deficiency is one of the most prevalent forms of malnutrition in the world. Pregnant women, infants and young children are most susceptible.

Primary vitamin A deficiency is due to inadequate intakes of vitamin A or its precursors, the carotenoids. Causes of secondary vitamin A deficiency include malabsorption of fat and the fat-soluble vitamins, failure to convert dietary carotene to preformed vitamin A and depletion of body reserves. Parasitic infections associated with fever and systemic acute phase response also cause secondary vitamin A deficiency. This is probably induced by inflammatory cytokine-related mechanisms which include decrease in the hepatic secretion of retinol-RBP complex, the loss of the complex to the extravascular space or an increased loss in the urine.

The basic pathology of vitamin A deficiency is hyperkeratinization of skin and keratinizing metaplasia of the lining of the respiratory, gastrointestinal and genitourinary tracts and the endocrine,

Figure 4-3

Vitamin A and the Visual Cycle
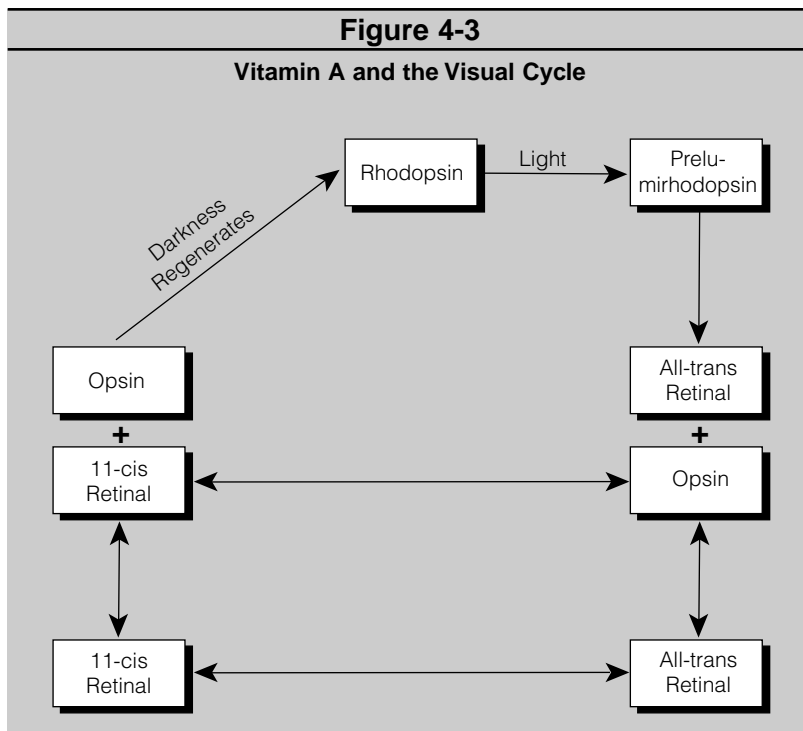
salivary, sebaceous and lacrimal glands. Vitamin A deficiency is associated with increased childhood morbidity and mortality. This is due to an increased risk of infectious diseases particularly in developing countries. Vitamin A supplements can help to achieve a rapid reduction in early childhood mortality and a lower level of childhood mortality can be sustained as long as adequate vitamin A supplementation in maintained.

The first symptoms of vitamin A deficiency are night blindness and drying of the conjunctiva of the eye. Bitot's spots may be present in the cornea of the eye. With continued vitamin A deficiency, progressive damage to the eye results from drying of the cornea and irreversible corneal damage resulting in xerophthalmia, keratomalacia, and blindness. In children, retarded growth may occur as a result of vitamin A deficiency.

Toxicity

Excessive ingestion of carotenoids, while not toxic to man, results in carotenemia and yellow discoloration of the skin. In large doses preformed vitamin A is toxic to man. Chronic toxicity of vitamin A produces variable symptoms. These may include: anorexia, nausea, vomiting, abdominal pain, dry skin, rashes, headaches, loss of hair, abnormal skin pigmentation, increased fragility and pain in the long bones, menstrual irregularities and enlargement of the liver and spleen.

Due to the danger of chronic toxicity, regular consumption of supplemental doses of vitamin A above 3000 RE (10,000 IU) for children or 7500 RE (25,000 IU) for adults is contraindicated.

Health Benefits

Low intakes of vitamin A and carotene are associated with an increased risk of developing certain cancers, such as breast cancer. However, the role of vitamin A in lung cancer is still unclear. The belief that vitamin A reduces cancer risk is based on the following observations: (1) the requirement of vitamin A for the maintenance of epithelial tissues, a common location where many cancers are located, (2) tumor surveillance by the immune system is dependent on adequate levels of vitamin A and (3) gene expression may be directly influenced by vitamin A and retinoids.

Biologically β-carotene acts as an antioxidant that may have a protective effect against free-radical damage of cellular membranes. For this reason there is much interest in increasing its intake. Ingestion of a large amount of carotenoids is nontoxic but may result in a benign condition characterized by yellow pigmentation of the skin.

VITAMIN D

Vitamin D is essential for the maintenance of calcium homeostasis. It represents a group of sterol compounds that possess anti-rachitic properties that prevent the development of rickets i.e. bone disease of infancy and early childhood. It is produced by irradiating a precursor or provitamin D with ultraviolet light. The two main provitamins that are of nutritional interest are ergosterol and 7-dehydrocholesterol. Ergosterol is found in plants while 7-dehydro cholesterol is found primarily in the skin of humans and animals. These two compounds are converted by ultraviolet light, either from sun or artificial sources, to vitamin D_2 (ergocalciferol) and vitamin D_3 (cholecalciferol) respectively. The two compounds possess equal vitamin D activity. They are stable to heat, alkalis and oxidation. Their structures are shown in Figure 4-4.

Vitamin D is both a hormone and prehormone. Although suffi-cient vitamin D_3 could theoretically be synthesized in the body

Figure 4-4

Structure of Vitamin D_2 and Vitamin D_3

Vitamin D_2
Ergocalciferol

Vitamin D_3
Cholecalciferol
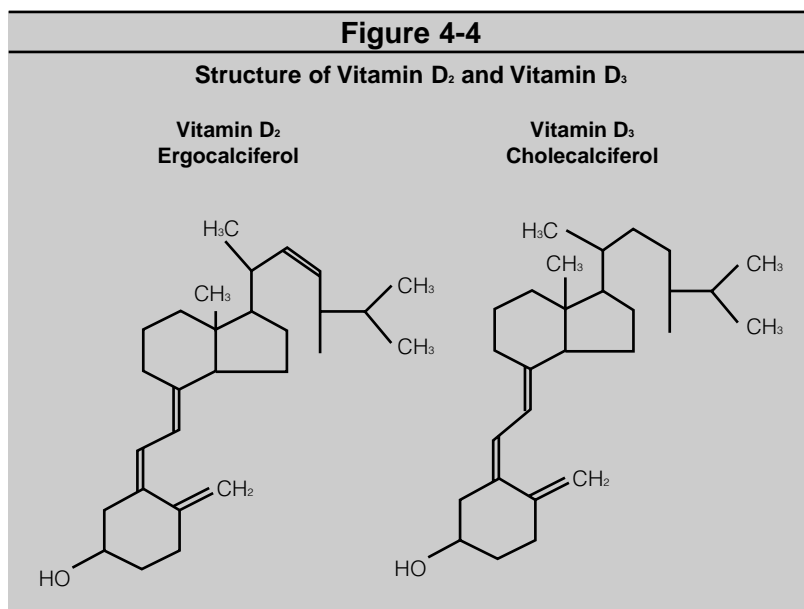
by the action of sunlight, the majority of the world's population must rely on additional dietary sources to meet requirements. Vitamin D_3 is converted to its hormonal form 1.25-dihydroxy D_3 which then acts upon its target tissues (intestine, bone and kidney) to regulate calcium and phosphorus absorption and metabolism.

Sources

The amount of vitamin D_3 formed from the endogenously synthesized 7-dehydrocholesterol is dependent upon the amount and intensity of sunlight as well as the degree of skin pigmentation. Dark-skinned people are protected from ultraviolet radiation by melanin pigments so that less vitamin D_3 is synthesized in them than in pale-skinned people.

Table 4-2

Vitamin D Content of Some Common Unfortified Foods

Food Items	Vitamin D (IU/100g)
Dairy and Egg Products	
butter	56
egg, whole, raw	52
cheese, cheddar	12
milk, human (100ml)	4
milk, cow's, unfortified (100ml)	40
Fish, Shellfish	
sardines, canned in oil	272
salmon, pink, canned	624
herring, pickled	680
shrimp, mixed, cooked	152
mackerel, canned	228
Meat	
liver, chicken, raw	50-65
liver, pork, raw	40
liver, beef, raw	8-40
liver, lamb, raw	20
liver, calves, raw	0-15
beef, steak	13
Oils	
cod liver oil	16,700
corn oil	0

See Tables 6-4 and 6-5 for recommended intakes for Canadians and Americans.

Source: Machlin, L.J. (ed). **Handbook of Vitamins, 2nd Edition.** Marcel Dekker, Inc., New York, 1991

Combs, G.F.Jr. **The Vitamins: Fundamental Aspect of Nutrition**, 2nd Edition. Academic Press, San Diego, CA, 1998.

Dietary sources of vitamin D include fatty fish such as tuna, salmon and sardines, eggs, liver, butter and cream (Table 4-2). However, the amounts of vitamin D naturally occurring in commonly consumed foods is small. The main source of dietary vitamin D is from fortified foods such as milk and margarine. Fish liver oil extracts also contain vitamin D and are used in the preparation of vitamin D concentrates. Other sources of vitamin D include yeast and fungi but are of minor dietary importance.

Breastmilk is low in vitamin D. For this reason, it is generally recommended that exclusively breast-fed infants be given a vitamin D supplement.

Absorption

Intestinal absorption of vitamin D is analogous to that of vitamin A, in that, bile salts are required. Absorbed vitamin D is incorporated into the chylomicron and ultimately taken up by the liver. In the liver, vitamin D_3 is converted to 25-hydroxy D_3 which in turn is converted by the kidneys to 1,25-dihydroxy D_3 and 24, 25-dihydroxy D_3. The various vitamin D metabolites are transported in the blood to target tissues as a complex bound with γ_1-globulin. The metabolic pathway of vitamin D_2 is identical to that of vitamin D_3.

Functions

Vitamin D is required for normal bone mineralization. 1,25-dihydroxy D_3 regulates absorption of calcium and phosphorus in the intestine, increases renal calcium reabsorption and mobilizes calcium from bone to maintain blood levels and to facilitate the normal remodeling of bone structure in both growth and adult bone. In the gut 1,25-dihydroxy D_3 is believed to stimulate synthesis of the calcium and phosphorus transport proteins in the intestinal cells.

Synthesis of 1,25-dihydroxy D_3 in the kidney is influenced by circulating levels and parathyroid hormone (PTH). When serum calcium is low, PTH rises. PTH acts directly on the kidney stimulated conversion of 25-dihydroxy D_3 to 1,25-dihydroxy D_3.

Deficiency

Fundamentally, vitamin D deficiency results from inadequate exposure to ultraviolet light. There has been increasing concern regarding possible vitamin D deficiency as a result of an increased

use of sunscreens and UV-protective clothing. Moreover, vitamin D is also dependent on dietary sources. Essentially vitamin D deficiency is the combined result of inadequate exposure and deficient dietary intake. Secondary vitamin D deficiency may be due to poor absorption of vitamin D in the intestine.

Vitamin D deficiency produces rickets in infants and children which is characterized by insufficient mineralization of the growing bone. Weight bearing and mechanical stress on the growing individual result in bowing of the legs, knock knee, lateral thoracic depressions at the sites of attachment of the diaphragm (Harrison's grooves) and a characteristic pigeon chest deformity of the rib cage. Vitamin D deficiency also affects the calcification of teeth and dental development in children.

Vitamin D deficiency in adults results in osteomalacia. Demineralization is pronounced in the spine, pelvis and legs. Gravitational stress of the softer bones causes compression of the affected vertebrae, bowing of the long bones and a compression deformity of the pelvis.

The hypocalcemia of osteomalacia and rickets predisposes both adults and children to restlessness, irritability and tetany.

Toxicity
Habitual intake of excessive vitamin D is toxic. The symptoms include nausea, anorexia, polyuria, pruritis and calcification of soft tissues such as the kidney and heart. In infants, hypervitaminosis D can result in bossing of the skull, mental retardation and death. Mild vitamin D toxicity is manifested in elevated serum calcium levels.

Because of the toxicity of vitamin D, it is recommended that dietary intakes remain close to the recommended daily intake of 2.5 μg (400 IU) cholecalciferol. It has been suggested that intakes above 20 μg (3200 IU) cholecalciferol be avoided unless there is clinical supervision and frequent monitoring of serum calcium and phosphorus.

Health Benefits
Epidemiological evidence indicates that adequate vitamin D status can reduce the risk of osteoporosis. Through calcium metabolism, vitamin D may be important in regulating blood pressure and

improving some forms of hypertension. There is also preliminary evidence that vitamin D may have an anticarcinogenic effect in colon carcinogenesis. However, these health benefits need to be confirmed by well controlled studies.

VITAMIN E

Vitamin E refers to a group of eight naturally occurring high molecular weight alcohols each derived from Chroman-6-ol. They are divided into two groups: the tocopherols have a saturated side chain and tocotrienols possessing an unsaturated side chain (Figure 4-5). The tocopherols are classified as alpha, beta, gamma and delta-tocopherol; the tocotrienols are classified as alpha, beta, gamma and delta-tocotrienol. Of the vitamin E compounds, alpha-tocopherol has the greatest biological activity.

The vitamin E compounds are viscous oils stable to heat and acids and unstable to alkalis, ultraviolet light and oxygen. All compounds are powerful antioxidants protecting the oils with which they are associated in nature against oxidation and the attendant rancidity. Commercially, tocopherols may be esterified to acetate as this renders the compound less susceptible to oxidation.

Figure 4-5

Chemical Structures of Alpha-tocopherol and Alpha-tocotrienol

Alpha-Tocopherol

Alpha-Tocotrienol
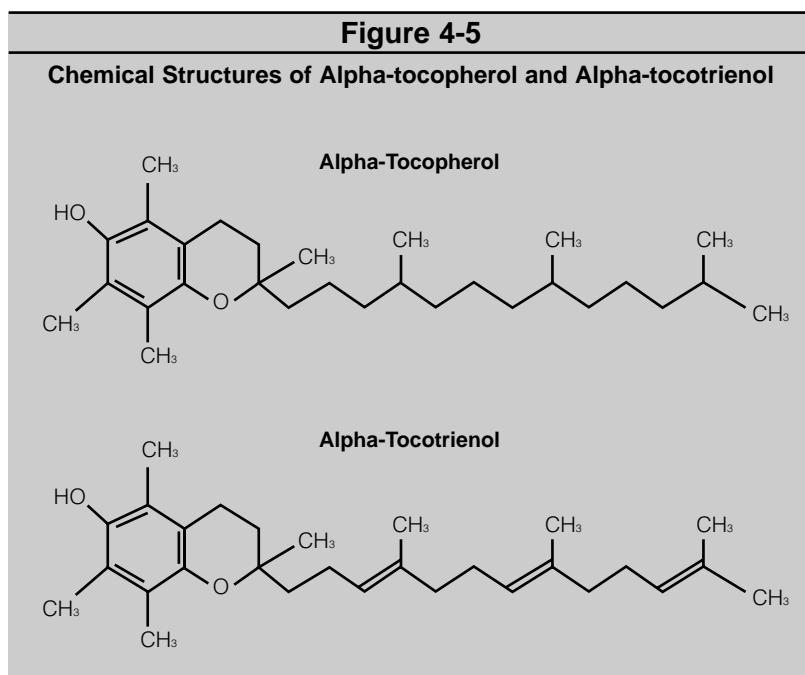
Sources

The vitamin E content in some commonly consumed foods is shown in Table 4-3. The richest dietary sources of the tocopherols are the vegetable and cereal seed oils. Wheat germ is particularly rich in alpha- and beta-tocopherol. Other excellent sources are soybean oil (alpha, beta and gamma forms), cottonseed oil (alpha and gamma), corn oil (mostly gamma), sunflower seed oil and margarine. Overall, gamma-tocopherol has a greater distribution than alpha-tocopherol in the vegetable oils, margarines and shortenings.

Virtually all vitamin E in foods of animal origin is in the form of alpha-tocopherol. Eggs, fish, all meats (especially liver) and butter contain small amounts of gamma-tocopherol.

The tocotrienols are found in certain cereal grains but their overall importance in the diet is probably low. Foods low in vitamin E include fruits, vegetables, nuts, cereals and the following oils: olive, coconut and peanut.

Table 4-3

Alpha-Tocopherol Content of Some Common Foods

Food Items	Alpha-Tocopherol (mg/100g)
Vegetable Oils (refined)	
wheat germ	192.4
cottonseed	38.3
safflower	43.1
palm	21.76
peanut	12.9
olive	12.4
soybean	18.2
coconut	0.3
Nuts	
almonds	5.6
filbert	23.9
peanut	7.4
brazil	7.6
pecan	3.1
walnut	2.6

Continued on next page

Table 4-3 Continued

Alpha-Tocopherol Content of Some Common Foods

Food Items	Alpha-Tocopherol (mg/100g)
Seeds and Grains	
oatmeal	0.7
wheat flour	0.1
rye flour	1.3
corn meal	0.7
rice, brown	0.7
bread, wheat	0.5
rice, white	0.1
Vegetables	
spinach	1.9
turnip, greens, cooked	1.7
broccoli	1.7
asparagus, cooked	0.4
brussel sprouts	0.9
carrot	0.5
Fruits	
peach	0.7
strawberry	0.1
pear	0.5
apple	0.3
grapefruit	0.3
banana	0.3
Dairy and Egg Products	
butter	1.6
eggs	1.1
milk, whole	0.1
Animal and Fish Products	
lard	1.2
halibut, cooked	1.1
shrimp	0.5
beef, tenderloin	0.2
pork	0.3
chicken	1.3
cod	0.6

See Tables 6-4 and 6-5 for recommended intakes for Canadians and Americans.

The vitamin E content in foodstuffs is quite variable as many factors will influence its presence in foods. Processing and storage will destroy some vitamin E in most foods. Losses of vitamin E from vegetable and cereal oils during storage is usually low; significant losses may occur in cooking.

Absorption
Only 20-40 percent of the dietary tocopherol (in free form or hydrolyzed from tocopherol ester in the gut) is absorbed. As is the case for the other fat soluble vitamins, the presence of bile is necessary for vitamin E absorption. The absorbed vitamin is incorporated into chylomicrons and transported via the thoracic duct to the liver. Vitamin E is transported to tissues by plasma lipoproteins. High concentrations of vitamin E are present in cellular and subcellular membranes of the adrenal and pituitary glands, testes and platelets. Vitamin E is stored in the liver, adipose and muscle tissues. Vitamin E metabolites include alpha-tocopheryl-quinone, hydroquinone and toco-copheronalactone. However, knowledge of all the vitamin E metabolites and their function is incomplete.

Functions
The mechanism of the physiological action of vitamin E is not fully known. It exerts a protective role in maintaining the integrity and function of cellular and subcellular membranes. It is theorized that vitamin E acts by protecting the polyunsaturated fatty acids (PUFA) of cell membranes from oxidative damage. Vitamin E is believed to be located in the cell membrane next to enzymes involved in reactions that produce oxygen-containing free radicals. In this regard, it may function as an antioxidant by reducing the free radicals formed in the body. When there is insufficient vitamin E, peroxidation of PUFA occurs producing membrane damage.

Vitamin E is thought to be one of several biological substances in the cell that act to remove oxygen-containing free radicals. Others include the enzymes glutathione peroxidase, catalase and superoxide dismutase.

Deficiency

Premature infants are at risk of vitamin E deficiency due to poor placental transfer of this vitamin and poor absorption from the immature gut. A vitamin E deficiency syndrome has been demonstrated in premature infants given a formula which is relatively high in PUFA and containing insufficient vitamin E. The deficiency syndrome consists of hemolytic anemia, edema, elevated platelet count, red blood cell structural changes and skin lesions.

In humans, vitamin E deficiency is rare. Experimentally, prolonged vitamin E deficiency results in increased hemolysis of red blood cells. In animals, vitamin E deficiency has been observed to result in reproductive failure, muscular dystrophy, macrocytic anemia, lactation failure, cardiovascular disease and a host of other diseases. Several studies have found the risk of cardiovascular disease and stroke to be inversely associated with vitamin E intake or plasma vitamin E concentrations. Randomized clinical trials using supplementation of vitamin E in high doses decrease the risk of myocardial infarction. More clinical trials of the efficacy of vitamin E in reducing cardiovascular disease risk are presently ongoing.

Toxicity

Vitamin E is relatively non-toxic compared to vitamins A and D. A metabolite of alpha-tocopherol, alpha-tocopherylquinone, is a vitamin K inhibitor and can prolong clotting time.

Altered coagulation for an individual on anticoagulation therapy taking 800 IU vitamin E has been described. There is no evidence that megadoses of vitamin E supplements have beneficial effects on body functions in healthy humans despite numerous claims to the contrary.

Health Benefits

Vitamin E helps protect membranes against free radicals because of its antioxidant effect. It therefore plays a role in inhibition of mutagen formation and repair of membranes and DNA and thus may be useful in cancer prevention. It is also suggested that vitamin E plays a role in cardiovascular disease through its ability to inhibit platelet prostaglandin release. Vitamin E may also play a role in reducing the risk of developing cataracts, in protecting against exercise-induced muscle injury and in reducing anemia in G6PD deficient subjects.

VITAMIN K

Vitamin K refers to a group of related compounds derived from menadione. Vitamin K_1 (phylloquinone) is found in plants. Vitamin K_2 (menaquinone-7) consists of a series of homologues having a variable number of isoprenyl units in its side chain (Figure 4-6). Vitamin K_2 is synthesized by microorganisms and is found in animal tissues and bacteria.

Vitamin K is stable to heat and exposure to air, but is destroyed by light, strong acids, alkalis and oxidizing agents.

Figure 4-6

Structure of Menadione, Vitamin K_1 and Vitamin K_2

Menadione
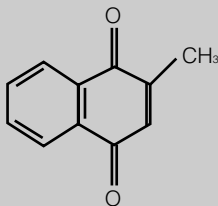
Vitamin K_1 (phylloquinone)
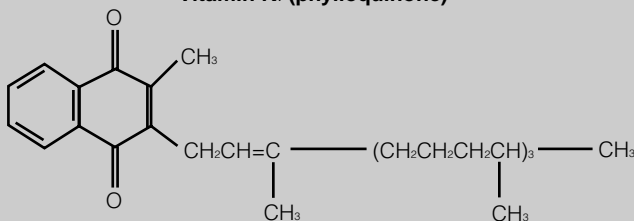
Vitamin K_2 (menaquinone)
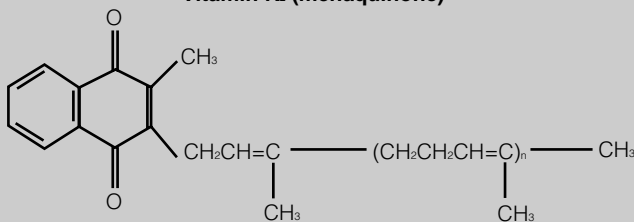
Sources

The most important dietary sources of vitamin K are green leafy vegetables such as cabbage and spinach. Liver and soybeans are also good sources. Lesser amounts of this vitamin are present in fruit, milk, meat, cereals and eggs (Table 4-4). However, up to half of the human supply of vitamin K is derived from bacterial synthesis in the intestine.

Absorption

Absorption of vitamin K, like the other fat soluble vitamins, is dependent upon the presence of bile and pancreatic secretions. Dietary vitamin K, synthesized by the intestinal microflora, is absorbed from the lower intestine and colon. Vitamin K is incorporated into chylomicrons and is ultimately transferred to beta-lipoproteins. The vitamin is not stored in tissues to any great extent.

Table 4-4

Vitamin K Content of Some Common Foods

Food Items	Vitamin K (mg/100g)
Vegetables	
Broccoli	270
Spinach	400
Lettuce	122
Cabbage	145
Cauliflower	10
peas, green	36
Dairy and Egg Products	
egg, chicken	2
butter	7
cheese, cheddar	3
milk, fluid	0.3
Other	
tea, brewed	0.05
coffee, brewed	10

See Tables 6-4 and 6-5 for recommended intakes for Canadians and Americans.

Source: U.S. Department of Agriculture, USDA Nutrient Database for Standard References, Release 13, Agricultural Research Service, 1999.

Combs, G.F.Jr. The Vitamins: Fundamental Aspect of Nutrition, 2nd Edition. Academic Press, San Diego, CA, 1998.

Functions

Vitamin K is necessary for normal blood coagulation. Synthesis of the four blood clotting factors, II, VII, IX and X is dependent on vitamin K. The vitamin is required for the carboxylation of certain glutamate residues of the factor II precursor. It is believed that similar vitamin K dependent carboxylations are involved in activation of the other blood coagulation factors.

Several vitamin K dependent proteins have been identified. They are located in the bone, kidney and plasma. Vitamin K is involved in the synthesis of X-carboxyglutamic acid in these proteins. The function and significance of these proteins is not clear.

Deficiency

Due to the widespread availability in the diet and bacterial synthesis of vitamin K in the gut, primary vitamin K deficiency is rare. Neonates are at risk for hemorrhage due to poor placental transfer of vitamin K and the absence of intestinal flora that are capable of synthesizing the vitamin. Vitamin K is commonly administered to mothers prior to delivery or to newborns immediately after birth. This is especially important for preterm infants, low birth weight infants, those on antibiotics and newborns who are exclusively breastfed.

Toxicity

Large doses of menadione may be toxic. Symptoms of vitamin K toxicity include liver damage, hypoprothrombinemia, petechial hemorrhages, renal tubule degeneration, and, in the premature infants, hemolytic anemia.

WATER SOLUBLE VITAMINS

THIAMIN

Thiamin is composed of a pyrimidine and a thiazole ring (Figure 4-7). It performs important biochemical functions as a coenzyme thiamin pyrophosphate (TPP) which is involved in energy metabolism. Thiamin is also present in tissues, as thiamin monophosphate and thiamin triphosphate. Thiamin is readily soluble in water and alcohols. It is stable in acid but is unstable to heat, oxidation and an alkaline pH.

Sources

The food sources for thiamin are numerous, but comparatively few commonly used foods supply it in concentrated amounts (Table 4-5). The richest sources include dry yeast, wheat germ, pork, organ meats (liver, heart and kidney), liver sausage, lean meats, eggs, green leafy vegetables, whole or enriched cereals, berries, nuts and legumes. Thiamin is also found in small amounts in many other foods.

The milling of grains removes those portions which are the richest in thiamin (the endosperm, the aleurone layer and the bran). As a result, white flour and polished white rice may be practically

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Figure 4-7

Structure of Thiamin and Thiamin Pyrophosphate (TPP)

Thiamin
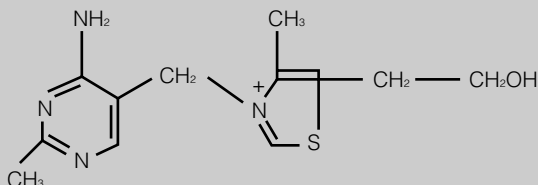
Thiamin pyrophosphate (TPP)

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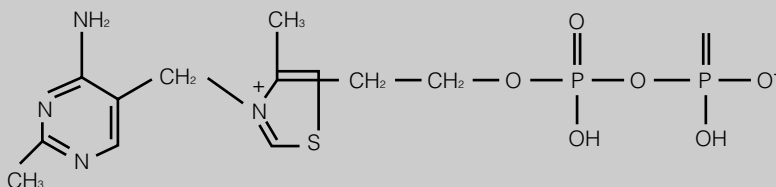
devoid of thiamin. This explains the prevalence of thiamin deficiency in populations where white rice constitutes the backbone of the diet. Among population groups who depend to a large degree on refined cereal products, the thiamin requirement is rarely met unless other good sources of thiamin are included in the diet. Large scale enrichment of flour and cereal products with thiamin has proved very beneficial in eliminating the risk of thiamin deficiency.

Table 4-5

Thiamin Content of Some Common Foods

Food Items	Thiamin (mg/100g)
Cereal and Grain Products	
wheat germ	1.88
bran flakes	1.30
flour, all-purpose, enriched	0.78
flour, whole wheat	0.45
bread, wheat	0.42
bread, rye	0.43
wheat, shredded	0.28
Meat	
pork chops, loin, lean, grilled	0.92
liver, lamb, fried	0.35
beef, roast	0.07
Beans	
beans, blackeye, boiled	0.24
beans, lima, boiled	0.16
beans, kidney, boiled	0.16
Dairy and Egg Products	
milk, whole, fluid	0.04
milk, skim, fluid	0.04
milk, dried, skimmed	0.42
cheese, cheddar	0.03
egg, chicken, whole	0.06

See Tables 6-4 and 6-5 for recommended intakes for Canadians and Americans.

Source: U.S. Department of Agriculture, USDA Nutrient Database for Standard References, Release 13, Agricultural Research Service, 1999.

Combs, G.F.Jr. The Vitamins: Fundamental Aspect of Nutrition, 2nd Edition. Academic Press, San Diego, CA, 1998.

Thiamin may be synthesized by the bacterial flora of the gastrointestinal tract, but the significance of this source of thiamin to the human is probably minimal.

Loss of thiamin during cooking occurs through extraction of the water soluble vitamin by the cooking water and through oxidation, especially in an alkaline pH. Such potential losses are held down to some extent by the nature of many thiamin containing foods which are consumed without excessive cooking (enriched bread, breakfast cereals). Parboiling of rice facilitates retention of some of the thiamin by forcing it into the endosperm before husking. Loss of thiamin in the cooking of meat tends to be high, with losses as high as 25-50 percent of the raw value.

Absorption, Storage and Excretion
The main site of thiamin absorption is the jejunum. Absorption takes place by active transport when the ingested concentration is low and by passive diffusion when the ingested concentration is high. The absorbed thiamin is phosphorylated to its esters in the intestinal cells. Thiamin absorption is decreased by folate deficiency and alcoholism. Thiamin deficiency may also be related to folate deficiency which is common among alcoholics.

Thiamin is not stored to any appreciable extent and the total amount in the body suffices for only a few weeks of normal functioning. The total amount of thiamin in the body is approximately 30 mg, with about 50 percent in the muscle. Being readily soluble in water, thiamin and its metabolites are excreted in the urine. Thiamin is nontoxic as excessive amounts are readily excreted.

Function
Thiamin functions as a coenzyme in energy metabolism and in the synthesis of pentoses and NADPH. It is also involved in nerve impulse transmission. As the coenzyme TPP, thiamin is required for the oxidative decarboxylation of alpha-ketoacids to carboxylic acids. TPP is necessary for entry of pyruvate into the citric acid (Krebs) cycle by converting pyruvate to acetyl CoA. TPP is also required for the citric acid (Krebs) cycle in the conversion of alpha-ketoglutarate to succinyl CoA. The presence of pantothenic acid (as CoA), riboflavin (as FAD) and niacin (as NAD) are required for oxidative decarboxylation. Lipoic acid is

required as well. The thiamin requirement is thus roughly proportional to the caloric content of the diet and is increased as the amount of carbohydrate metabolized increases.

TPP is required in the transketolase reaction of the pentose phosphate shunt for the transfer of an alpha-keto group from xylulose-5-phosphate to ribose-5-phosphate. The reactions of the pentose shunt are significant as they serve to provide the cell with pentose (used in nucleic acid synthesis) and NADPH (used in fatty acid synthesis).

Thiamin triphosphate is required for the transmission of nerve impulses in nervous tissue; the mechanism is not fully understood.

Deficiency
Primary thiamin deficiency as a result of inadequate supply is commonly seen in societies where the population subsists on polished rice. In these societies, breast-fed infants may display signs of thiamin deficiency since the milk of their mother is low in thiamin. In developed countries, thiamin deficiency syndromes occur almost exclusively in chronic alcoholics due to poor dietary habits and the effect of alcohol on thiamin absorption.

Thiamin deficiency affects the cardiovascular, muscular, nervous and gastrointestinal systems. The earliest manifestation of thiamin deprivation includes anorexia, fatigue, depression, irritability, poor memory, inability to concentrate and vague abdominal and cardiac complaints. With advancing deficiency neurologic symptoms appear in the form of a bilateral, symmetrical peripheral neuropathy which involves at first the most distal parts of the lower extremities. Once the neuropathy in the lower extremities is advanced, the hands and arms may also become involved. Thiamin deficiency may also cause defects in the myocardium.

Thiamin deficiency is known as beriberi. It is classified into dry, wet and infantile beriberi. Dry beriberi is characterized by peripheral polyneuritis, paralysis and muscle atrophy. In wet beriberi the clinical picture is, to a large degree, one of congestive heart failure with cardiac dilatation secondary to damage of the cardiac musculature, serous effusions and edema. A mild subacute form of beriberi has both neurological and cardiac manifestations. In infants, an acute form of the disease, infantile beriberi, runs a fulminating course and terminates in cardiac failure.

Several disorders of the central nervous system seen in chronic alcoholics may be attributed to lack of thiamin. The disorders include (a) polyneuropathy (factors other than thiamin deficiency may also be involved), (b) Wernicke's disease (signs are opthalmoplegia, nystagmus and ataxia), (c) Korsakoff's psychosis (signs are memory defect and confabulation) and (d) amblyopsia (dim vision).

RIBOFLAVIN

Riboflavin is a yellow isoalloxazine compound (Figure 4-8). It is important biologically as a component of the flavoprotein enzymes riboflavin 5' phosphate (FMN) and flavin adenine dinucleotide (FAD).

Riboflavin is relatively stable to heat, acids and oxidation but very unstable to light and ultraviolet radiation.

Sources

Common foods which supply significant amounts of riboflavin are organ meats (liver, heart and kidney), milk, liver sausage, green leafy vegetables, eggs, enriched cereal products, meat and cheese. Riboflavin is also found in smaller quantities in a wide variety of other foods (Table 4-6).

Figure 4-8

Structure of Riboflavin
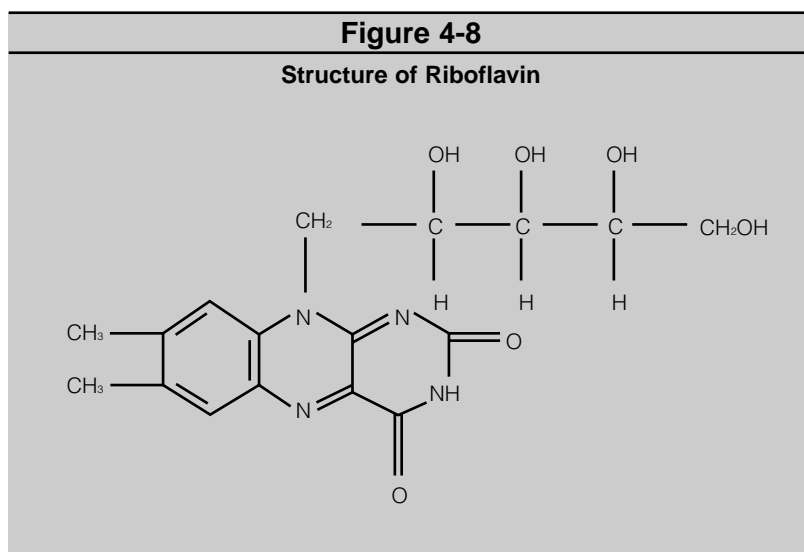
The relative heat stability of riboflavin, coupled with its slight solubility, serves to minimize losses during cooking. However significant losses of riboflavin can occur by exposure to light. For this reason milk, which is a good source of riboflavin, should be purchased and stored in containers that protect it from light.

Table 4-6

Riboflavin Content of Some Common Foods

Food Items	Riboflavin (mg/100g)
Dairy Products	
cheese, cheddar	0.38
cheese, cottage, creamed	0.16
ice cream	0.21
milk, whole	0.16
yogurt	0.14
Cereal and Grain Products	
bread, white, enriched	0.34
bread, whole wheat	0.21
bread, rye	0.34
oatmeal, cooked	0.02
Vegetables	
turnip greens, boiled	0.07
broccoli, cooked	0.11
collards, cooked	0.11
asparagus, cooked	0.13
beet greens, cooked	0.29
spinach, canned, drained	0.19
beans, lima, cooked	0.10
Meat/Poultry/Fish	
liver beef	2.8
chicken, cooked, flesh only	0.20
pork, cooked	0.11
sardines (canned)	0.23
salmon (canned)	0.19
tuna (canned)	0.07

See Tables 6-4 and 6-5 for recommended intakes for Canadians and Americans.

Source: U.S. Department of Agriculture, USDA Nutrient Database for Standard References, Release 13, Agricultural Research Service, 1999.

Combs, G.F.Jr. The Vitamins: Fundamental Aspect of Nutrition, 2nd Edition. Academic Press, San Diego, CA, 1998.

Absorption and Storage

FMN and FAD, the major forms in which riboflavin occurs in foods, require hydrolysis before absorption. Riboflavin is absorbed in the upper gastrointestinal tract by active transport. Riboflavin is present in tissues as FMN, FAD and free riboflavin. There is a limited ability to store riboflavin; its excretion is via the urine.

Function

As part of the coenzymes FMN and FAD, riboflavin may be reduced to form $FMNH_2$ and $FADH_2$ respectively. These coenzymes function as hydrogen carriers in many cellular reactions. For example, FAD is the hydrogen acceptor in the conversion of succinate to fumarate in the citric acid (Krebs) cycle. The hydrogen is then passed on to the electron transport system. FAD functions in the oxidative decarboxylic reactions noted in the section on thiamin. FAD is a coenzyme for glutathione reductase. This enzyme is used to determine riboflavin status. FMN is part of the amino acid oxidase enzyme that converts alpha-amino acids and alpha-hydroxy acids to alpha-keto acids. Riboflavin is required for normal growth and tissue maintenance, particularly the eyes.

Deficiency

Riboflavin deficiency is characterized by cheilosis, angular stomatitis, glossitis, seborrheic dermatitis and ocular manifestations: photophobia, itching, burning and circumcorneal capillary engorgement. The seborrheic dermatitis is usually found in the nasolabial region, near the inner and outer canthi of the eyes, behind the ears and on the posterior surface of the scrotum. Any one of these symptoms alone is not indicative of riboflavin deficiency. Riboflavin deficiency can only be diagnosed when the symptoms are coupled with a poor dietary history, and/or anomalies in metabolism requiring riboflavin as a co-enzyme. The symptoms, while undesirable, are rarely fatal.

In developed countries biochemical riboflavin deficiency has been noted in newborn infants treated with phototherapy for jaundice. Marginal biochemical riboflavin deficiency has been associated with the use of oral contraceptives in women of low socio-economic status.

NIACIN

Niacin, also known as nicotinic acid, is the generic description of pyridine-3-carboxylic acid (Figure 4-9). In the human, ingested niacin, is readily converted to niacinamide (nicotinamide). Niacinamide is important biologically as a component of the coenzymes nicotinamide adenine dinucleotide (NAD) and nicotinamide adenine dinucleotide phosphate (NADP).

Niacin is stable to air, heat, light, oxidation, acids and alkalis and is not destroyed in ordinary cooking processes.

Strictly speaking niacin is not a vitamin as it may be synthesized endogenously from the essential amino acid, tryptophan. This reaction requires the presence of vitamin B6 as pyridoxal phosphate. Sixty milligrams of tryptophan provide 1 mg of niacin. Niacin intake is therefore expressed in terms of niacin equivalents (NE) where:1 NE = 1 mg niacin (nicotinic acid) = 60 mg tryptophan.

Sources

The following foods are good sources of niacin: liver, meats, fish, whole grain and enriched breads and cereals, dried peas and beans, nuts and peanut butter (Table 4-7). Niacin may occur in untreated cereals such as wheat, corn and rye in a bound form niacytin which is virtually unavailable to humans. Alkali treatment serves to liberate the bound niacin. In addition to the niacin content of foods, the content of its precursor tryptophan must be considered. For example, milk and eggs are low in niacin but are good sources of tryptophan.

Figure 4-9

Structure of Niacin and Niacinamide

Niacinamide (Nicotinamide)
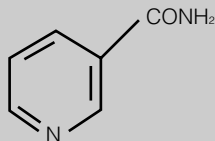
Niacin (Nicotinic Acid)
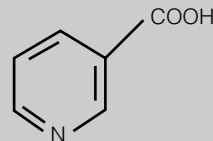
Absorption, Synthesis and Excretion

Niacin and niacinamide are readily absorbed by diffusion in the stomach and upper intestine. The conversion of tryptophan to niacin may be altered under certain conditions. The conversion is less efficient when intake of niacin and tryptophan is low and is more efficient during pregnancy or oral contraceptive use. In vitamin B6 deficiency, formation of niacin from tryptophan is reduced due to lack of the coenzyme pyridoxal phosphate. The main metabolites of niacin are excreted via the urine along with some free niacin.

Function

Niacin functions in energy metabolism in the amide form as part of the coenzymes NAD and NADP. Both compounds may be reduced to form $NADH + H^+$ and $NADPH + H^+$ respectively.

Table 4-7

Niacin Content of Some Common Foods

Food Items	Niacin (mg/100g)
Meat	
chicken, roast	9.2
beef, blade, roast	2.4
pork, leg, roast	4.6
lamb, leg, roast	6.6
sardine, canned	5.2
salmon, canned	6.5
rabbit, stewed, wild	6.4
turkey, roast, meat only	5.4
veal, leg, roast	9.9
Beans and Nuts	
peanuts, roasted	13.5
beans, green, boiled	0.6
beans, red kidney, boiled	0.6
beans, lima, boiled	0.4
peas, boiled	0.1
Grains	
flour, all purpose, enriched	5.9
flour, whole wheat,	6.4

See Tables 6-4 and 6-5 for recommended intakes for Canadians and Americans.

Source: U.S. Department of Agriculture, USDA Nutrient Database for Standard References, Release 13, Agricultural Research Service, 1999.

Combs, G.F.Jr. The Vitamins: Fundamental Aspect of Nutrition, 2nd Edition. Academic Press, San Diego, CA, 1998.

The coenzymes serve as hydrogen carriers in numerous cellular reactions including certain reactions in glycolysis, the Krebs cycle, electron transport system and β-oxidation. NADPH is required in the hexose monophosphate shunt.

Niacin is also required for the maintenance and function of the skin, gastrointestinal tract and nervous system as evidenced by symptoms occurring in niacin deficiency.

Deficiency

Primary niacin deficiency is usually associated with diets based chiefly on corn and proteins containing inadequate amounts of tryptophan. Corn is low in tryptophan and contains niacin in an unavailable bound form. Unless the corn is treated, for example with lime, to release the bound niacin, deficiency may result. Niacin deficiency may also be due to secondary causes such as a malabsorption syndrome.

Niacin deficiency is known as pellagra which presents as the 4 D's-*dermatitis, diarrhea, dementia* and *death*. Early signs of niacin deficiency include fatigue, anorexia, weakness, mild gastro-intestinal disturbance, anxiety irritability and depression. A scaly, bilateral, pigmented dermatitis appears generally in areas exposed to sunlight. The mucous membranes of the mouth are usually involved: glossitis, stomatitis, soreness, a burning sensation and ulceration of the buccal cavity may be present. Severe, extensive diarrhea is another common feature of the disease. In advanced cases of pellagra dementia may manifest. Untreated pellagra will result in death.

Toxicity

Both niacin and niacinamide are harmful when taken in large doses. Megadoses of niacin (3-6 g/day) produce a flushing and itching reaction. Pharmacological doses of niacin, but not niacinamide lower total serum cholesterol, LDL and triglycerides. However, they also produce increased incidence of arrhythmias, biochemical alterations and gastrointestinal disturbances. Thus, use of niacin as a pharmacological drug should only be done under strict medical supervision.

Large doses of niacinamide do not produce the itching and flushing reaction seen with niacin. Doses of 50-250 mg niacinamide have been used therapeutically in deficient subjects. Doses of 3-9 g have been reported to cause liver toxicity.

Health Benefits
Niacin, in the form of nicotinic acid (12.3 to 24.6 mmol or 1.5 to 3 g per day), reduces hyperlipidemia, since it reduces LDL cholesterol ("bad" cholesterol) and increases HDL cholesterol ("good" cholesterol).

BIOTIN
The structure of biotin is shown in Figure 4-10. It is stable to heat, light and reducing agents, but is labile to oxidizing agents, strong acids and alkali.

Sources
Biotin is widely distributed in foods (Table 4-8). Yeast and organ meats such as liver and kidney are excellent sources of biotin. Good sources are soybeans, rice bran, peanuts, chocolate, egg yolk, cauliflower and mushrooms. The bioavailability of biotin varies depending on whether it is in a bound (unavailable) or free form. As well, biotin is synthesized by the intestinal microflora. The extent of the microbiological synthesis in the intestine is not known nor is the biological significance of this source of biotin.

Function
Biotin is required for carbohydrate and fat metabolism. Biotin functions as a coenzyme in various carboxylases. It is required by pyruvate carboxylase for the conversion of pyruvate to

Figure 4-10
Structure of Biotin
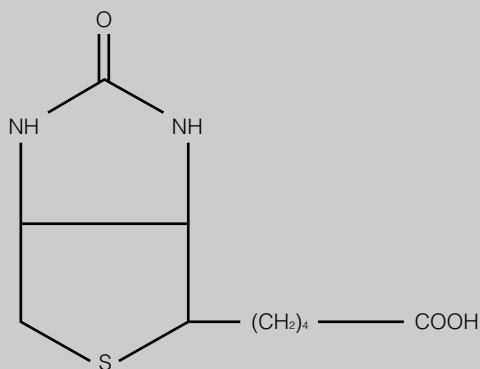
oxaloacetic acid in gluconeogenesis. Oxaloacetic acid is an important intermediate of the citric acid (Krebs) cycle and in fatty acid biosynthesis. Biotin is needed for the conversion of acetyl CoA to malonyl CoA as a coenzyme for acetyl CoA carboxylase. This reaction occurs in the initial stages of fatty acid synthesis.

Table 4-8

Biotin Content of Some Common Foods

Food Items	Biotin (mg/100g)
Meat/Poultry/Fish	
liver, chicken, fried	170
beef, roast	tr
pork, chops, loin. lean, grilled	3
chicken, roast, meat only	3
sardines, canned	5
Dairy Products	
milk, skim	2
cheese, cottage	3
Fruits	
Banana	2.6
strawberries, raw	1.1
orange	1.0
Vegetables	
peas, frozen, boiled	0.4
corn	N
onions, raw	0.9
beans, green	1.0
carrot, raw	0.6
tomato, fresh	1.5
Cereal and Grain Products	
bread, wholemeal	6
bread, rye	N
oatmeal	21

tr = traces

N = The nutrient is present in significant quantities but there is no reliable information on the amount.

See Tables 6-4 and 6-5 for recommended intakes for Canadians and Americans.

Source: Holland, B. et al., **The Composition of Foods, 5th Edition.** The Royal Society of Chemistry, Cambridge, UK., 1992

Although less well elucidated, biotin is known to be required for normal protein and cholesterol metabolism as evidenced by the signs of biotin deficiency; namely decreased protein synthesis and hypercholesterolemia.

Deficiency
Experimental biotin deficiency has been induced in humans by feeding a biotin deficient diet and large quantities of raw egg white. Raw egg white contains avidin, a heat labile protein which binds biotin rendering it unavailable for absorption. Symptoms of biotin deficiency include scaly dermatitis, grayish pallor, extreme lassitude, anorexia, nausea, anemia, depression, muscle pain, parethesia, hypercholesterolemia and electrocardiogram changes. There is also decreased protein synthesis.

Biotin deficiencies in humans have been reported in association with bizarre food habits, i.e. consumption of large quantities of raw egg whites and little else. Seborrheic dermatitis in infants under six months may be responsive to biotin administration. Certain biotin responsive genetic conditions involving defective carboxylase enzymes have been identified. Low biotin levels have been reported in pregnant women, alcoholics and persons with achlorhydria.

PANTOTHENIC ACID
Pantothenic acid is composed of hydroxyl and methyl substituted butyric acid (pantoic acid) joined by a peptide link to beta-alanine (Figure 4-11). Pantothenic acid is important biologically as a component of Coenzyme A (CoA) and as a prosthetic group of the acyl carrying protein (ACP); both of which participate in a number of cellular reactions.

Pantothenic acid is stable to oxidizing and reducing agents but is readily destroyed by heat, acids and alkali.

Sources
Pantothenic acid is widely distributed in plant and animal tissues. Good sources of pantothenic acid are organ meats, egg yolk, peanuts, legumes and whole grains (Table 4-9).

Figure 4-11

Structure of Pantothenic Acid

$$CH_2 - C - CH - C - NH - CH_2 - CH_2 - COOH$$

with:
- CH_3 above the first C
- HO below CH_2
- H_3C below the first C
- OH below CH
- O (double bond) above the second C
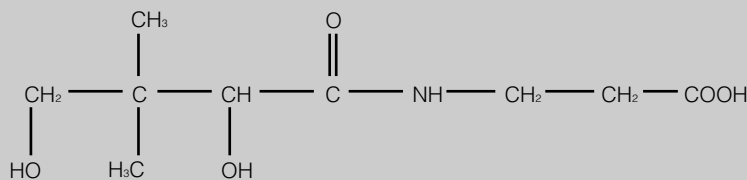
Functions

Pantothenic acid is required for the metabolism of carbohydrate, fat and protein as well as a number of other compounds. Pantothenic acid is a component of CoA which functions in acyl transfer reactions of the citric acid (Krebs) cycle. Acetyl CoA is required in many other processes including synthesis of fatty acids, triglycerides, cholesterol, sphingosine porphyrin (for heme synthesis) and acetylcholine (a neurotransmitter). Pantothenic acid is the prosthetic group of acyl carrier protein (ACP). ACP is required in the binding and transfer of acyl groups in fatty acid biosynthesis. More than 70 enzymes are known to use CoA or ACP.

Deficiency

Symptoms of pantothenic acid deficiency have been induced in humans by using the metabolic antagonist, omega methyl pan-tothenic acid and a pantothenic acid deficient diet. The resulting syndrome is characterized by abdominal pain and soreness, nausea, personality changes, insomnia, impaired adrenal function, weak-ness and cramps in the legs, paresthesia of the hands and feet and impaired antibody production.

Primary pantothenic acid deficiency in conjunction with multiple vitamin deficiency is believed to have occurred in Japanese prisoners-of-war in World War II. The "burning foot" syndrome (burning pain in the soles of the feet) was improved by pan-tothenic acid administration in some of the prisoners.

Table 4-9

Pantothenic Acid Content of Some Common Foods

Food Items	Pantothenic Acid (mg/100g)
Meat/Poultry/Fish	
beef, liver, raw	7.62
beef, kidney, raw	3.64
pork, loin, lean, roast	0.78
chicken, roast, meat only	0.97
salmon, canned	0.55
Legumes and Nuts	
peanuts, roast	1.39
soybean, roast	0.45
lentils, boiled	0.64
Dairy and Egg Products	
milk, nonfat, regular, dry	3.57
cheese, cheddar	0.41
milk, skim, fluid	0.33
milk, whole, fluid	0.31
egg, chicken, whole, raw	1.26
egg, chicken, yolk, raw	3.81
Cereal and Grain Products	
bread, whole wheat	0.55
bread, white	0.39
Fruits	
bananas	0.26
oranges	0.25
apples	0.06
Vegetables	
mushroom, boiled	2.16
potatoes, baked, flesh and skin	0.56
broccoli, raw	0.54
pea, green, cooked, boiled, drained	0.15

See Tables 6-4 and 6-5 for recommended intakes for Canadians and Americans.

Source: U.S. Department of Agriculture, USDA Nutrient Database for Standard References, Release 13, Agricultural Research Service, 1999.

Combs, G.F.Jr. The Vitamins: Fundamental Aspect of Nutrition, 2ⁿᵈ Edition. Academic Press, San Diego, CA, 1998.

VITAMIN B₆

Vitamin B₆ refers to the pyridines: pyridoxine, pyridoxal and pyridoxamine (Figure 4-12). The compounds are closely related and are converted to the biologically active coenzyme form of pyridoxal phosphate (PLP). However, pyridoxamine can also activate a number of vitamin B₆ dependent enzymes. Pyridoxine is stable to heat and acid but is rapidly destroyed by light, particularly in neutral or alkali solution. Pyridoxamine and pyridoxal are labile compounds. They are rapidly destroyed by exposure to air, light and heat.

Sources

Sources of vitamin B₆ are widespread (Table 4-10). Good sources of vitamin B₆ include meat, fish, poultry, organ meats, legumes, grains, certain fruits and vegetables. In animal products, vitamin B₆ is found largely as pyridoxal and pyridoxamine. In plants the main form of vitamin B₆ is pyridoxine.

Function

Vitamin B₆ is required for normal protein metabolism. Pyridoxal

Figure 4-12

Structure of Pyridoxine, Pyridoxal, Pyridoxamine and Pyridoxal 5-phosphate
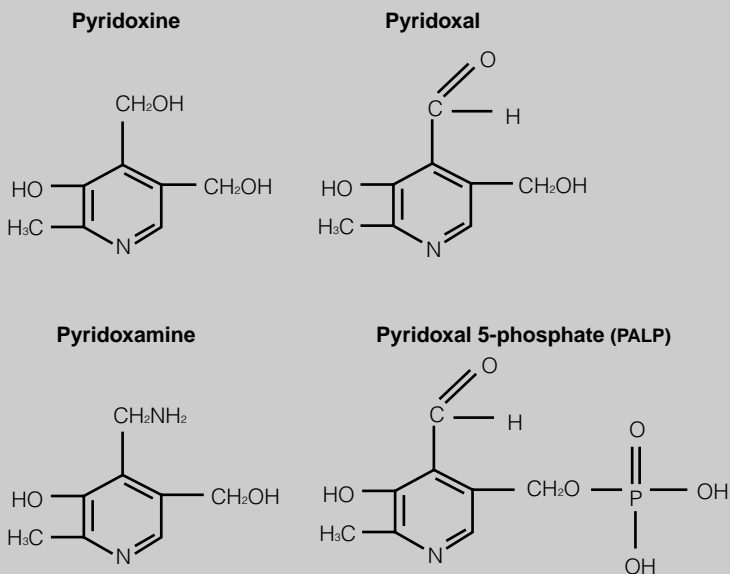
Table 4-10

Vitamin B₆ Content of Some Common Foods

Food Items	Vitamin B₆ (mg/100g)
Cereal and Grain Products	
rice, brown, boiled	0.14
wheat flour, whole	0.34
bread, whole wheat	0.18
bread, white	0.06
Meat/Poultry/Fish	
beef, liver	0.94
beef, kidney	0.51
pork, leg, roasted	0.40
pork, loin, blade, roast	0.38
lamb, leg, roast	0.14
chicken, leg, roast	0.33
salmon, canned	0.30
Vegetables	
potato, raw	0.26
spinach, raw	0.20
broccoli, raw	0.16
peas, green, boiled	0.22
cauliflower, raw	0.22
soybeans, roasted	0.21
tomatoes, raw	0.08
carrot, raw	0.15
beans, lima, boiled	0.16
cabbage, boiled	0.11
corn, sweet	0.06
Fruits	
banana	0.58
raisin, seedless	0.25
avocados, raw	0.28
apricots	0.05
oranges	0.06
apples	0.05
grapefruit	0.04
peaches	0.02
Dairy and Egg Products	
cheese, cheddar	0.07
milk, whole, fluid	0.04
milk, skim, fluid	0.04
egg, chicken, whole, raw	0.14

See Tables 6-4 and 6-5 for recommended intakes for Canadians and Americans.

Source: U.S. Department of Agriculture, USDA Nutrient Database for Standard References, Release 13, Agricultural Research Service, 1999.

Combs, G.F.Jr. The Vitamins: Fundamental Aspect of Nutrition, 2ⁿᵈ Edition. Academic Press, San Diego, CA, 1998.

phosphate functions as a coenzyme in transamination, decarboxylation and transulfuration reactions of amino acids. Vitamin B$_6$ dependent decarboxylases are involved in the synthesis of the neurotransmitters serotonin and norepinephrine. The conversion of methionine to cysteine, and cysteine to taurine also involve vitamin B$_6$-dependent enzymes. Conversion of tryptophan to niacin is dependent upon pyridoxal phosphate as a coenzyme. Vitamin B$_6$ is required for proper functioning of the phosphorylase enzyme in glycogenolysis and in the formation of delta-amino-levulinic acid in the synthesis of heme and in folate metabolism. Table 4-11 lists a number of genetic diseases involving vitamin B$_6$ dependent enzymes.

Deficiency

Vitamin B$_6$ deficiency has been demonstrated in infants inadvertently fed vitamin B$_6$ -deficient formula. The signs included irritability and convulsions. Vitamin B$_6$ deficiency has been induced experimentally in adults fed B$_6$ -deficient diets in the presence of the vitamin B$_6$ antagonist deoxypyridoxine. Vitamin B$_6$ deficiency is characterized by seborrheic dermatitis, cheilosis, glossitis, stomatitis, altered tryptophan metabolism, depression and confusion.

The incidence of vitamin B$_6$ deficiency in alcoholics may be as high as 20-30 %. Up to one-fifth of oral contraceptive users show biochemical evidence of vitamin B$_6$ deficiency. Daily administra-

Table 4-11	
Inborn Errors of Vitamin B$_6$ Metabolism	
Condition	**Metabolic Defect**
Convulsive seizures in infants	Reduced synthesis of gamma-amino butyric acid (GABA) due to abnormal glutamic acid decarboxylase
Vitamin B$_6$-responsive chronic anemia	Impaired synthesis of heme due to reduced formation of its delta-amino-levulinic acid precursor
Xanthurenic aciduria	Decreased conversion of hydroxykynurenine to hydroxyanthranilic acid due to low activity of Kynurenase (in tryptophan metabolism)
Cystathioninuria	Decreased cleavage of cystathionine to cysteine and homoserine due to deficiency of cystathionase (in methionine metabolism)
Homocystinuria	Impaired conversion of homocysteine and serine to cystathionine due to absence of or defect in the enzyme cystathione synthetase (in methionine metabolism)

tion of vitamin B_6 supplements has been reported to restore normal biochemical values. The underlying mechanism by which oral contraceptives affect vitamin B_6 metabolism has not been elucidated.

Toxicity

Oral doses of 1-150 mg/day of vitamin B_6 have been used therapeutically without ill effect in treating pyridoxine responsive anemias, inborn errors of vitamin B_6 metabolism, B_6 deficiency and as a supplement when B_6 antagonists are being administered in clinical situations. A transient dependency has been induced in adults given a supplement of 200 mg pyridoxine hydrochloride per day for 33 days. Megadoses of 2-6 g/day for two months or more have been shown to cause ataxia and severe sensory-nervous system dysfunction in adults.

Health Benefits

Vitamin B_6 has many health benefits. It reduces the symptoms of carpal tunnel syndrome and, although more controversial, some of the symptoms of premenstrual syndrome in some women. As well, supplementation of vitamin B_6 to asthmatic subjects may decrease the frequency and severity of asthma attacks. Furthermore, it is suggested that vitamin B_6 may play a role in cardiovascular health in individuals with inherited homocystinuria and high circulating homocysteine levels, as well as in diabetic neuropathy and in normal immune function.

FOLIC ACID

Folate is a generic name describing folic acid and related compounds which have the biological activity of folic acid. Folic acid (pteroylmonoglutamic acid) is composed of a pteridine nucleus, para-aminobenzoic acid and glutamic acid (Figure 4-13). Folic acid may be conjugated with up to seven molecules of glutamic acid forming pteroyl-polyglutamic acid. The biologically active form of the vitamin tetrahydrofolic acid (THFA) functions as a carrier of one carbon units in biological reactions.

Folic acid in general, is unstable to heat and oxidation although some derivatives such as N^5-methyl-THFA are heat stable.

Sources

Important sources of folic acid include liver and organ meats, yeast, dark green leafy vegetables, legumes, nuts, whole grain

cereals, eggs and fruit (Table 4-12). The bioavailability of folic acid from foods is variable. Significant losses during storage, cooking and processing can occur due to the susceptibility of folic acid to destruction by oxidation and heat.

Folic acid is synthesized by the intestinal microflora of the colon but the biological significance of this source to humans is not clear.

Folic acid is stored in the liver and other body tissues. Excretion of THFA and its derivatives occurs via the feces and urine. Fecal losses reflect biliary secretions plus bacterial synthesis of folic acid in the colon. A significant portion of folic acid is thought to be cycled through enterohepatic circulation.

Functions
THFA is necessary for normal cell division and replication as it is needed in nucleic acid synthesis. The synthesis of both the purines and pyrimidines requires THFA.

THFA is needed in several amino acid conversions, namely, the conversion of (a) serine to glycine (vitamin B$_6$ is also required), (b) histidine to glutamate and (c) homocysteine to methionine (vitamin B$_6$ is also required).

Deficiency
Since THFA is required for nucleic acid synthesis, folate deficiency first manifests itself in tissues with rapid cell turnover such as

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Figure 4-13

Structure of Folic Acid (Pteroylmonoglutamic Acid)

Pteridine nucleus   p-Aminobenzoic acid   Glutamic acid

Pteroic Acid

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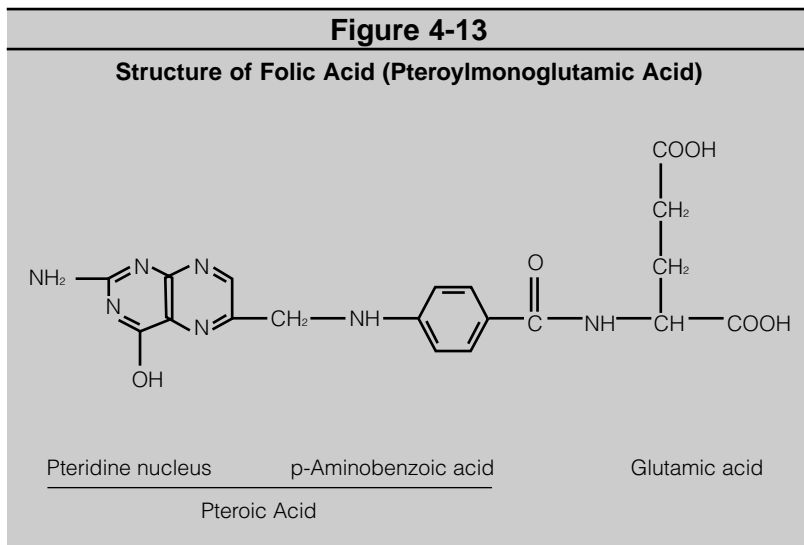
bone marrow (erythropoiesis), and gastrointestinal and oral mucosa. Normal maturation of red blood cells does not take place and hematopoiesis is inhibited at the megaloblast stage. A typical peripheral blood smear is characterized by megaloblastic macrocytic anemia, thrombopenia, leukopenia and old multilobed neutrophils. Glossitis, gastrointestinal disturbances, diarrhea, malabsorption and mood changes such as forgetfulness and paranoia may accompany the anemia.

Megaloblastic anemia during pregnancy is due to folate deficiency and is probably precipitated by an increased physiologic requirement for the vitamin during pregnancy, especially in the last trimester. Increased intake (either dietary or supplemental) is generally advised during pregnancy to prevent folic acid deficiency anemia and to reduce the risk of neural tube defects in the newborn.

Folic acid deficiency is also associated with chronic alcoholism. The underlying mechanism of this association is not clear. It is

Table 4-12

Folate Content of Some Common Foods

Food Items	Folate (µg/100g)
Vegetables	
spinach, raw	194
asparagus, boiled	146
lettuce, romaine, raw	136
Beans	
soybean, roasted	211
chickpeas, dried, cooked or canned	172
lentils, boiled	181
Meats	
liver, chicken, raw	738
liver, beef, raw	248
Miscellaneous	
cornflakes	353
wheat germ	352
all bran	300
almonds, roast	64

See Tables 6-4 and 6-5 for recommended intakes for Canadians and Americans.

Source: U.S. Department of Agriculture, USDA Nutrient Database for Standard References, Release 13, Agricultural Research Service, 1999.

Combs, G.F.Jr. The Vitamins: Fundamental Aspect of Nutrition, 2nd Edition. Academic Press, San Diego, CA, 1998.

likely due to a folic acid deficient diet as well as aberrations in folic acid absorption resulting from chronic alcohol intake.

Toxicity

Doses of folate up to 15 mg/day have been reported with no apparent ill effects. Megadoses of folate reduce the effectiveness of certain anticonvulsant drugs. Animal studies show that high concentrations of folate may result in convulsions. Furthermore, high intakes of folate may mask a deficiency in vitamin B_{12}.

Health Benefits

Folate plays an important role in embryonic development by supporting normal cell division. Adequate folate status is linked to reduced risk of abnormalities in early embryonic development and specifically to risk of malformations of the embryonic brain and/or spinal cord. These include anencephalus, encephaloceles, and spina bifida, all of which result from incomplete neural development and collectively referred to as neural tube defects (NTD). The precise role played by folate in neurulation is not clear, but it has been suggested that defective folate metabolism may cause abnormal methionine synthase activity and homocysteine metabolism. Elevated maternal circulating homocysteine levels have been frequently observed in women with NTD births. Folic acid supplementation is thus important during the periconceptual and early-pregnancy stages in preventing neural tube defects and cleft palate/lip in the newborn. Currently the general recommendation for women is to consume at least 0.4 mg of folic acid per day before and during pregnancy.

There is also some evidence that folic acid is important in reducing the risk of atherosclerosis in individuals with inherited homocystinuria and high circulating homocysteine levels.

VITAMIN B_{12}

Vitamin B_{12} refers to a group of compounds having vitamin B_{12} activity (Figure 4-14). The compounds share a complex structure composed of a cobalt-containing corrin nucleus and a nucleotide attached to a ribose unit. The term cobalamin signifies the presence of the corrin nucleus and cobalt. It is prefixed by the designation of the anionic R group attached to the cobalt. Thus, cyanocobalamin is the permissive name of vitamin B_{12}. The two cobalamins that are coenzymatically active in humans are

5'-deoxyadenosylcobalamin (adenosyl cobalamin) and methyl-cobalamin. Hydroxycobalamin is also present. Vitamin B_{12} is relatively heat stable during normal cooking but is destroyed by heating in acid and alkali.

Sources

Vitamin B_{12} is found almost exclusively in foods of animal origin (Table 4-13). The original source of the vitamin is bacterial fermentation in the intestinal tract of animals. Only bacteria are capable of synthesizing vitamin B_{12}.

Figure 4-14
Structure of Vitamin B_{12}

R = -CN in cyanocobalamin
= -OH in hydroxycobalamin
= -CH₃ in methylcobalamin
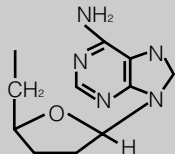
Sources of vitamin B_{12} include organ meats, fish, milk and eggs. Vitamin B_{12} is not found in foods of plant origin unless they have been contaminated by vitamin B_{12}-producing bacteria or vitamin B_{12}-containing substances, or fortified with vitamin B_{12} (e.g. fortified ready-to-eat breakfast cereals). In the human, vitamin B_{12} is synthesized by bacteria in the small intestine and may be absorbed. Synthesis of vitamin B_{12} also occurs in the colon but is unavailable to humans.

Absorption, Storage and Excretion

Vitamin B_{12} is very poorly absorbed from the gastrointestinal tract unless a glycoprotein, the "intrinsic factor" is present concurrently. The "intrinsic factor" is secreted by the gastric parietal cells and binds to vitamin B_{12} forming a complex. Trypsin and bicarbonate also facilitate vitamin B_{12} absorption. The site for vitamin B_{12} absorption is the ileum. The "intrinsic factor" is probably not absorbed. Approximately 1 percent of free vitamin B_{12} may be absorbed by diffusion in the small intestine.

The main form of vitamin B_{12} in the bloodstream is methylcobalamin which circulates in conjunction with a protein, transcobalamin II.

Table 4-13

Vitamin B_{12} Content of Some Common Foods

Food Items	Vitamin B_{12} (µg/100g)
Meat	
liver, chicken	23
liver, beef	69
liver, pork	26
kidney, beef	70
beef, chuck blade, roast	3.4
lamb, leg, roast	2.5
pork, ham, roast	0.7
chicken, roast	0.3
Dairy and Egg Products	
milk, raw	0.36
cheese, cheddar	0.83
cheese, Swiss	1.68
egg, whole	1.00

See Tables 6-4 and 6-5 for recommended intakes for Canadians and Americans.

Source: U.S. Department of Agriculture, USDA Nutrient Database for Standard References, Release 13, Agricultural Research Service, 1999.

Combs, G.F.Jr. The Vitamins: Fundamental Aspect of Nutrition, 2nd Edition. Academic Press, San Diego, CA, 1998.

Vitamin B_{12} is stored in the liver mainly as adenosylcobalamin. It is secreted by the biliary system and enters the enterohepatic circulation. Only very small losses of vitamin B_{12} occur via the urine and feces. As a result, adult vegans who lack a dietary source of vitamin B_{12} may take years to develop vitamin B_{12} deficiency. With damage to the enterohepatic circulation, deficiency may develop more rapidly.

Function

Vitamin B_{12} is required for folic acid metabolism and normal cell division and replication. Vitamin B_{12} is also required in the metabolism of some branched chain amino acids and odd-chain fatty acids.

Deficiency

In vitamin B_{12} deficiency, folate deficiency may also result. The macrocytic megaloblastic anemia seen in B_{12} deficiency may be indistinguishable from folic acid deficiency. Similarly, alterations in fast replicating tissues, such as glossitis and gastrointestinal disturbances, may occur.

The main feature of vitamin B_{12} deficiency which differentiates it from folic acid deficiency is the neurological damage due to inadequate myelin synthesis. Vitamin B_{12} deficiency produces patchy, diffuse and progressive demyelination. Neurological symptoms may include parethesia, unsteadiness, poor muscle coordination, ataxia, forgetfulness, moodiness, confusion, depression, hallucination and psychosis. Vitamin B_{12} deficiency may be due to (a) prolonged inadequate dietary intake, such as a vegan diet (b) interference with vitamin B_{12} absorption, for example, the lack of "intrinsic factor" (pernicious anemia) or (c) interference with the enterohepatic circulation, for example, ileal resection.

Folate fortification of bread and grains has been introduced to prevent NTD. However, there is concern that increased folate intake may mask the development of megaloblastic anemia, thereby delaying diagnosis and treatment and making possible the irreversible neurologic damage caused by vitamin B_{12} deficiency. This is particularly important for the elderly population since vitamin B_{12} malabsorption and prevalence of vitamin B_{12} deficiency increase with age. Preliminary evidence suggests that vitamin B_{12} should be added to foods along with folic acid. Moreover

food fortified with folate and vitamin B_{12} will maximize the reduction of homocysteine which increases benefits of the proposed measures in the prevention of vascular disease and NTD.

Health Benefits

Preliminary research indicates that vitamin B_{12} may be important in the prevention of some cancers and in the treatment of precancerous lesions. Because vitamin B_{12} is required for the remethylation of homocysteine to methionine, supplementation of vitamin B_{12} in individuals with inherited homocysteine and high circulating homocysteine levels may reduce the risk of atherosclerosis. Finally, depression of some immune functions is associated with vitamin B_{12} deficiency.

VITAMIN C

Ascorbic acid is chemically unique among the vitamins as it contains a dienol group which not only contributes reducing action to the compound but also confers an acidic behavior upon the molecule (Figure 4-15). It is readily interconverted between the reduced (ascorbic acid) and oxidized (dehydro-ascorbic acid) form. The anti-scorbutic potency of the two forms is about the same. Vitamin C activity is lost if the dehydroascorbic acid is further oxidized to diketogulonic acid.

Vitamin C is readily soluble in water and is the least stable of the vitamins. It is sensitive to alkali, heat and oxidation, especially in the presence of iron or copper ions which act as catalysts for the oxidation reaction. Vitamin C is fairly stable in acid.

Sources

The foods richest in vitamin C are black currants, citrus fruits and their juices (Table 4-14). Other fruit such as berries, cantaloupe, kiwi fruit, strawberries and raw or minimally cooked vegetables such as peppers, broccoli, cabbage, tomatoes and potatoes are also good sources.

The vitamin C content of fruits and vegetables varies with the conditions under which they are grown and depends on the climate, conditions of the soil, seasonal variations, degree of maturation, freshness of the food and the part of the food which is eaten. For example, the skin of the apple contains more vitamin C than the pulp. Young green leafy vegetables contain more of the

vitamin than the mature leaf. During storage, the vitamin C content in fruits and vegetables will decrease depending on the time and temperature of storage. New potatoes are good sources of vitamin C, but with storage, the level of vitamin C declines. Bruising of soft fruit results in loss of vitamin C, so will slicing and cutting of vegetables. This is due to the release of the enzyme ascorbic acid oxidase and exposure of a greater surface area for oxidation. Prolonged cooking or exposure to heat during food preparation and processing will produce heavy losses. Vitamin C is also lost by leaching into the water.

Absorption, Storage and Excretion
Absorption of vitamin C occurs in the jejunum and ileum by a sodium linked energy dependent carrier. It may also be absorbed by diffusion. The efficiency of absorption decreases with increasing vitamin C intake.

Figure 4-15

Structure of L-Ascorbic Acid, L-Dehydroascorbic Acid and Diketogulonic Acid

Structural formulas and interconversion of the two forms of vitamin C and their inactivation by oxidation
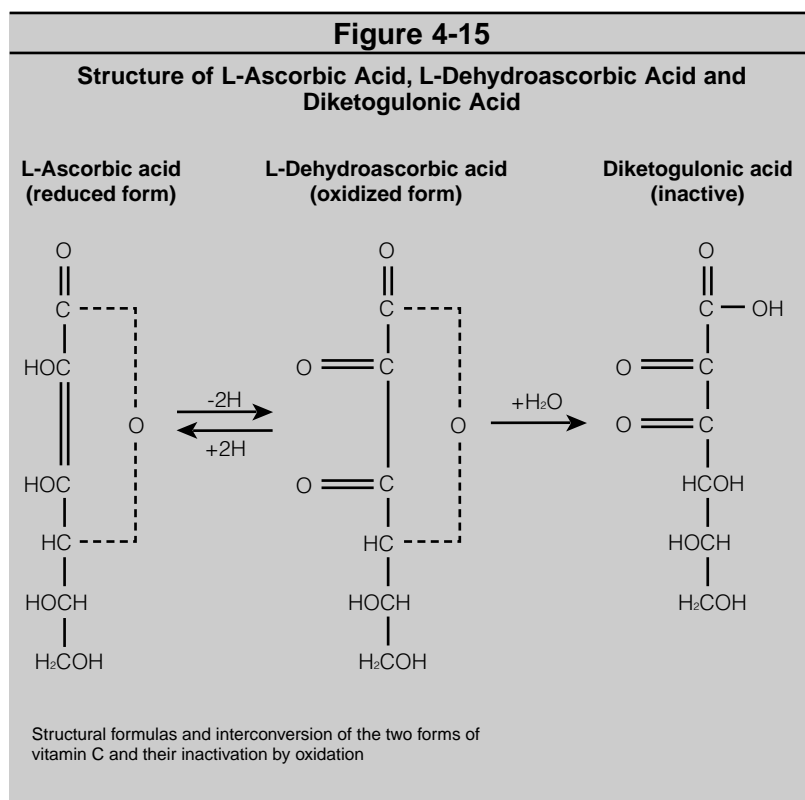
Vitamin C is not stored to any great extent in the body. Relatively high concentrations of vitamin C are found in the retina and other eye tissues, the adrenal and pituitary glands, brain, pancreas, kidney, liver and spleen. It is also present in other tissues such as the testes, ovaries, lungs, platelets, leukocytes, erythrocytes, plasma, etc.

Excess ascorbic acid is excreted mainly in the urine in its original form. Metabolites of the vitamin are similarly excreted.

Table 4-14

Ascorbic Acid Content of Some Common Foods

Food Items	Ascorbic Acid (mg/100g)
Fruits	
strawberries	57
kiwi fruit	98
grapefruit	34
oranges	53
lemons	53
tangerines	31
Fruit Juices	
apple juice, canned	0.9
apple juice with added vitamin C	41.6
grape fruit juice	29.2
orange juice	34.4
tomato juice	18.3
Vegetables	
tomatoes, red	19
cabbage	32
potatoes	20
beans, navy, sprouted	17
spinach, raw	28
broccoli	93
cauliflower	46
collard, boiled	18
parsley	133
peppers, red, sweet	190
turnip greens, boiled	27
asparagus, boiled	11

See Tables 6-4 and 6-5 for recommended intakes for Canadians and Americans.

Source: U.S. Department of Agriculture, USDA Nutrient Database for Standard References, Release 13, Agricultural Research Service, 1999.

Combs, G.F.Jr. The Vitamins: Fundamental Aspect of Nutrition, 2ⁿᵈ Edition. Academic Press, San Diego, CA, 1998.

Function

Vitamin C participates in a number of important body functions. These include: (a) formation of collagen or the intracellular cement substance necessary for body growth, tissue repair and wound healing, (b) conversion of tryptophan to 5-hydroxytryptophan and in the formation of tyrosine from hydroxyphenylpyruvate, (c) synthesis of adrenaline and hydrocortisone, (d) iron and copper metabolism by maintaining them in their reduced forms, thus facilitating absorption, and (e) interaction with folic acid, niacinamide and possibly with vitamins A and D. It also prevents the formation of nitrosamine from nitrates found in some foods. Vitamin C may also play a role in membrane permeability, leukocyte function and prevention of accumulation of histamine in the body.

Deficiency

Scurvy is a vitamin C deficiency disease. It is characterized by weakness, spongy and bleeding gums (scorbutic gum), loose teeth, resorbed dentine, swollen tender joints, hemorrhage (due to capillary fragility), follicular hyperkeratoses in the skin, muscular aches and pains and irritability. These are mainly due to impairment of collagen formation. Other signs are neuritis due to nerve damage, impaired iron absorption, impaired folic acid, serotonin and adrenaline metabolism and psychological changes.

Toxicity

Megadoses of vitamin C have been touted as a prophylactic or curative measure for the common cold and a wide variety of illnesses. However, there is no good scientific evidence to support these claims. Concern has been expressed over routine consumption of megadoses of vitamin C as it has been reported to result in gastrointestinal disturbances, uricosuria, excessive absorption of iron and impaired bactericidal activity of the leukocyte. Prolonged ingestion of high intakes of vitamin C may lead to increased catabolism and excretion of the vitamin. If vitamin C is abruptly reduced a rebound scurvy may develop. Excretion of large amounts of ascorbic acid in the urine, which occurs when individuals consume high doses of vitamin C, may interfere with certain medical tests such as that for glycosuria in diabetic patients.

Health Benefits

A great deal of research indicates a possible beneficial association of vitamin C and cardiovascular health, blood pressure and/or platelets. As well, vitamin C-rich foods may reduce the risk of developing cancer of the gut as well as enhancing immune function. Vitamin C has also been associated with reducing the risk of cataracts and improving nonheme iron absorption and periodontal disease as well as being involved in wound healing and blood vessel formation.

Suggested Readings

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CHAPTER 5

MINERALS

A number of minerals are essential for body functions. They are classified as macrominerals or microminerals (trace elements) depending on their dietary requirement. The macrominerals include calcium, phosphorus, potassium, sodium, chloride, sulfur and magnesium. The trace elements include copper, cobalt, chromium, fluorine, iodine, iron, manganese, molybdenum, nickel, selenium, silicon, tin, vanadium and zinc. Other minerals such as lead, arsenic and cadmium are toxic. The essentiality of several other elements in human nutrition is still under investigation.

Minerals may function as cofactors of enzymes, as components of organic compounds and as structural components of bones and teeth and are catalysts for many biological processes. Minerals may also participate in contraction and conduction of nerve impulses.

Recommended levels of intakes of some of the minerals are shown in Chapter 6 (Tables 6-1 to 6-4).

Increased emphasis is being placed on the importance of minerals. At the International Conference on Nutrition that was held in Rome, 1992, two of the eight Goals of the World Summit for Children focused on the reduction of iron deficiency anemia and virtual elimination of iodine deficiency.

MACROMINERALS

CALCIUM

Calcium is the most abundant cation in the human body. The body of an adult usually contains about 1200 g of calcium. More than ninety-nine percent of the calcium is present in bones and teeth as a component of hydroxyapatite crystals-$Ca_{10}(PO_4)_6(OH)_2$ and amorphous calcium phosphate. The remaining 1% of total body calcium is necessary for a variety of metabolic processes including enzyme activation, hormone function, nerve transmission, muscle contraction, blood clotting and membrane transport.

Sources

Milk and milk products are the richest dietary sources of calcium (Table 5-1). Lesser amounts of calcium are found in shellfish, egg yolk, canned sardines and salmon (with bones), soybeans and certain green leafy vegetables such as turnip and mustard greens, broccoli and kale. Calcium is also found in such foods as spinach and rhubarb; however, the presence of oxalic acid in these foods may bind with calcium rendering it unavailable for absorption.

Table 5-1

Calcium Content of Some Common Foods

Food Items	Calcium (mg/100g)
Dairy Products	
cheese, parmesan, grated	1376
cheese, mozzarella, partly skimmed	645
cheese, cheddar	721
cheese, swiss, process	961
cheese, blue	528
cheese, ricotta, partly skimmed	272
cheese, cottage, creamed	60
ice cream, 50% fat	157
milk, skim	124
milk, lowfat, 2% fat	122
milk, whole, 3.3% fat	119
yogurt, plain, 1.5% fat	182
buttermilk	116
Fruits	
rhubarb, frozen, cooked, added sugar	145*
Vegetables	
beet greens, boiled	114
spinach, boiled	136*
broccoli, boiled	46
Meat/Fish/Poultry and Alternates	
sardines, canned in oil	382
salmon, canned, solids and liquid	213
nuts, Brazil	176
seeds, sunflower, dry	116
almonds, whole	266
beans, red, kidney, cooked	28

See Tables 6-4 and 6-5 for recommended intakes for Canadians and Americans.

* Ca bound by oxalic acid greatly reduces the amount absorbed

Source: U.S. Department of Agriculture, USDA Nutrient Database for Standard References, Release 13, Agricultural Research Service, 1999.

As a result of fortification, infant cereals are excellent sources of calcium. More recently, the fortification of foods such as orange juice and flour, as well as the use of calcium in antacids like Tums™ and Rolaids™, have become additional sources of calcium in the diet. In hard water areas, drinking water may also be a significant source.

Absorption and Metabolism

Absorption of calcium from the gut is influenced by a number of factors. One of the most important factors is the individual's need for calcium. The body can adapt to a wide range of intakes by altering the efficiency of absorption and excretion. The efficiency of calcium absorption in the gut decreases with age.

Calcium absorption is enhanced by an acid pH which keeps calcium in solution. Vitamin D, lactose and certain amino acids such as lysine and arginine, also enhance calcium absorption. Calcium absorption is decreased by foods containing large amounts of phytates or oxalates both of which will form an insoluble calcium salt which is unavailable for absorption. However, this reaction is considered to be of little biological significance if calcium intake is liberal. Excess fat in the diet or impaired digestion of fat may reduce calcium absorption through the formation of insoluble calcium soaps.

Animal studies have linked both a low calcium-to-phosphorus ratio and a high phosphorus intake to bone demineralization and soft tissue calcification. However, results from studies on humans regarding this relationship are not conclusive. A dietary calcium-to-phosphorus ratio of 1:1 is generally recommended.

There is also an interrelationship among calcium, phosphorus and protein. Increasing dietary protein will increase urinary calcium excretion if phosphorus intake is held constant.

Serum calcium levels are maintained between 9-11 mg/dl. Control of serum calcium is dependent upon the hormones, 1,25-dihydroxy D_3 calcitonin and parathyroid hormone (PTH). PTH, in conjunction with 1,25 dihydroxy D_3, raises serum calcium levels while calcitonin lowers them to keep them within the desirable physiologically range.

Calcium is present in the blood in several forms. Ionized calcium accounts for 50% of the total plasma calcium and is the physio-

logically active form of calcium. The remaining calcium is bound to serum proteins (40-45 percent of total plasma levels) or complexed with citrate, bicarbonate or phosphate.

Deficiency
Low dietary calcium intake may produce rickets in children. Severe calcium deficiency in children may also result in stunted growth, muscle weakness, parathyroid hyperplasia, hyperirritability, tetany and death. In adults a calcium deficiency will exacerbate osteomalacia caused by vitamin D deficiency.

Insufficient dietary calcium is thought to be a factor contributing to osteoporosis. Osteoporosis is characterized by a significant reduction in bone density causing bone fragility and susceptibility to fracture. Osteoporosis is a prevalent disease among older women and, to a lesser extent, older men. To reduce the risk of osteoporosis individuals are advised to ascertain the recommended calcium intake from adolescence and to increase intake after menopause. As well, physical activity tends to reduce the risk of osteoporosis.

Skeletal demineralization occurs with prolonged immobilization and bed rest, due to large losses of calcium and phosphorus in the urine.

Among various macrominerals, calcium has attracted significant interest as a potential chemopreventive agent. It has been postulated that low intake of calcium and fiber along with high fat intake predisposes humans to a heightened state of colon cell proliferation leading to the formation of adenomatous polyps with subsequent degeneration to malignant transformation and development of colon cancer. Nutritional data also indicate that increased consumption of calcium-containing foods is associated with a reduced incidence of colon cancer.

It appears that there may be a subpopulation of hypertensives who are sensitive to calcium deficiency. Increasing the dietary calcium intake of these individuals to between 1200 and 1500 mg per day tends to lower their blood pressure.

Toxicity
Hypercalcemia is usually due to hyperparathyroidism or excess vitamin D. It is also associated with patients having neoplasms

with metastases leading to deposition of calcium in the kidneys. Hypercalcemia may result in muscle rigidity, dehydration, lethargy, nausea, vomiting, anorexia and ultimately coma and death.

MAGNESIUM

The human body contains 20 to 28 g of magnesium; fifty-five percent is present in bone while 27 percent is found in muscle. The remainder is found in the cells and extracellular fluids. Magnesium performs a vital role in the enzymatic reactions involving ATP. It is needed for the activation of thiokinases used in fatty acid oxidation as well as amino acid acyl synthetases. Magnesium is required for protein synthesis through its action on ribosomal aggregation. It is required for the formation of cyclic AMP and other second messengers. It plays a role in maintenance of cell membranes and neuromuscular transmission.

Sources
Magnesium occurs widely in foods, particularly those of plant origin (Table 5-2). Sources include green vegetables (magnesium is a component of chlorophyll), cereals, grains, meat, milk, seafood, cocoa and nuts.

Absorption and Metabolism
Absorption of magnesium appears to occur by active transport at low concentrations and passive diffusion at high concentrations. Absorption of magnesium occurs throughout the small intestine, particularly the jejunum and the ileum.

Plasma magnesium concentrations range from 2 to 3 mg/dl. About 55 percent is in the free form, the rest is complexed with protein and other body chemicals. Regulation of body magnesium occurs mainly in the kidney but the mechanisms are not clearly delineated. The gastrointestinal tract also plays a role in magnesium regulation by virtue of its sensitivity to high intakes. Excretion of magnesium is largely via the urine. In hot climates, however, up to 25 percent of total magnesium losses are via the sweat. Fecal magnesium losses represent unabsorbed magnesium. Only 2 percent of absorbed magnesium are lost by the fecal route.

Deficiency
Due to the ubiquitous nature of magnesium in the food supply, primary magnesium deficiency is rare. Magnesium deficiency may

be found in association with a number of diseases involving magnesium absorption or excretion such as malabsorption syndrome, ileal resection, diuretic therapy and kidney disease. Magnesium depletion may be noted in protein-calorie malnutrition, chronic alcoholism, diabetes mellitus, hyperparathyroidism and in individuals maintained on magnesium-free total parenteral nutrition.

Magnesium deficiency results in hypocalcemia and hypokalemia. Magnesium deficiency affects the gastrointestinal, neuromuscular, cardiovascular and hematologic systems. Dysphagia, anemia, cardiac arrhythmias, tremors, weakness, failure to thrive and psychiatric disturbances may be present. Magnesium deficiency ultimately results in tachycardia, fibrillations, convulsions and death.

Table 5-2

Magnesium Content of Some Common Foods

Food Items	Magnesium (mg/100g)
Vegetables	
okra, raw	57
spinach, raw	79
turnip, raw	11
Nuts	
brazil, shelled	225
almonds, dried, shelled	296
cashew, dry, roast	260
peanuts, roasted & salted	176
Cereal and Grain Products	
bran, wheat	611
flour, soya, defatted	290
flour, wheat, all purpose	22
flour, rye	75
Other	
anchovies, canned in oil, drained	56
chickpeas, canned	29
milk, 3.25% fat	13
chicken, drumsticks, roast	24
beef short loin, porterhouse steak, broiled	27

See Tables 6-4 and 6-5 for recommended intakes for Canadians and Americans.

Source: U.S. Department of Agriculture, USDA Nutrient Database for Standard References, Release 13, Agricultural Research Service, 1999.

Toxicity

Magnesium toxicity has been reported in patients with renal failure receiving high doses of magnesium, due to the decreased ability to excrete magnesium. Symptoms of magnesium toxicity are essentially the reverse of a deficiency and include lethargy and decreased nerve impulse transmission. Nausea, vomiting and flushing may be present. Ultimately magnesium toxicity results in paralysis, cardiac arrest and death.

PHOSPHORUS

The phosphorus content of an adult man is approximately 700 g. Eighty percent of the body's phosphorus is found in bone as calcium phosphate and hydroxyapatite. In the bone the phosphorus to calcium ratio is 1:2. There is no free elemental phosphorus in the body. It is present as a constituent of various lipids, proteins, carbohydrates, enzymes, nucleic acid and ATP. In its ionic form, phosphorus serves to modify acid-base balance in the blood. Phosphorus acts as a cofactor in a number of enzyme systems involved in carbohydrate, protein and fat metabolism. Phosphorus is important as a component of phospholipids in cell membranes, lipoproteins, etc., and is involved in the renal excretion of hydrogen ions.

Sources

Protein foods of animal origin such as meat, fish, poultry and eggs are excellent sources of phosphorus. Milk and cheese are good sources of phosphorus as are nuts and legumes. The availability of the phosphorus in cereal grains, especially the bran portion, is somewhat doubtful since much of this phosphorus is present as phytic acid which is not well utilized.

Absorption and Metabolism

Absorption of phosphate is dependent upon dietary phosphorus intake and food sources. The efficiency of absorption is increased when dietary phosphorus intake is low and during growth. Lack of 1,25-dihydroxy D_3 will reduce both calcium and phosphorus absorption. Certain minerals such as calcium, aluminum and strontium will bind with phosphorus forming insoluble phosphates, rendering both elements unavailable for absorption.

Serum phosphorus levels range from 2.4-4.4 mg/dl. Approximately half of this amount is present as phosphate ions, with one third complexed to sodium, calcium and magnesium and the remainder bound to protein.

Deficiency
Phosphorus deficiency has been reported in individuals consuming a prolonged and excessive intake of nonabsorbable antacids which bind dietary phosphorus preventing its absorption. Symptoms of phosphorus deficiency include weakness, anorexia, malaise and pain in the bones. Hemolytic anemia, granulocyte dysfunction, erythrocyte glycolysis, neurologic and psychiatric disorders, hypercalciuria and renal calculi may also result from phosphorus deficiency.

Cow's milk is relatively high in phosphorus (the calcium to phosphorus ratio is 1.2:1) compared to breastmilk (calcium to phosphorus ratio is 2:1). The relatively high phosphorus content of cow's milk is thought to interfere with calcium absorption resulting in the development of hypocalcemic tetany in infants fed cow's milk in the first week of life.

Toxicity
Hyperphosphophatemia is associated with certain disease states such as hypoparathyroidism or chronic renal failure. Hypocalcemia is often associated with hyperphosphatemia, since excessive phosphate interferes with calcium utilization. Signs of hyperphosphatemia may include neuroexcitability, tetany and convulsions.

SODIUM AND POTASSIUM
Sodium and potassium are essential for normal growth and body functions. Sodium is the predominant extracellular electrolyte and potassium is the major intracellular cation. They are both involved in regulating water and acid-base balance, in membrane permeability, nerve conduction and muscle action. Changes in extracellular sodium concentration can affect arterial pressure, whereas changes in blood potassium concentration can affect cardiac performance.

Sources

Sodium and potassium are widely distributed in foods (Tables 5-3 and 5-4). Dietary sources of sodium include table salt (sodium chloride) and sodium-based food additives used in commercially processed and cured foods. Foods that have relatively high levels of naturally occurring sodium include milk and milk products, meat, fish (particularly ocean fish), seafood, poultry and eggs. Good sources of potassium are meat, poultry, fish, organ meats, milk and milk products and certain fruits and vegetables. High potassium fruits include avocado, banana, apricot, dried fruit, melons and oranges. Some high potassium vegetables are broccoli, brussels sprouts, parsnips, squash, potatoes, dry beans and peas.

Table 5-3

Sodium Content of Some Common Foods

Food Items	Sodium (mg/100g)
Meat/Fish/Poultry	
bacon, back, sliced, grilled	1546
crab, blue, canned	333
pork, liver	87
haddock, cooked	87
pork, loin	30
chicken, roasted	86
beef, blade, roast	67
Dairy Products	
cheese, cheddar	620
cheese, parmesan, grated	1862
cheese, cottage, creamed	405
milk, cow's, fluid, whole	49
Condiments	
salt	38758
shake & bake, dry	3500
ketchup	1041
dressing, Thousand Island, low calorie	1000
vinegar, cider	1
Beverages	
club soda	21
ginger ale	7
cola	4
tea	3

Source: U.S. Department of Agriculture, USDA Nutrient Database for Standard References, Release 13, Agricultural Research Service, 1999.

Table 5-4

Potassium Content of Some Common Foods

Food Items	Potassium (mg/100g)
Vegetables	
broccoli, raw	325
brussel sprouts, raw	389
parsnips, cooked, boiled, drained	367
pumpkin, canned	206
potato, cooked in skin	379
rutabaga, boiled, drained	326
tomato ripe, raw	222
winter squash, raw	350
Fruits	
apricots, raw	296
avocado, raw	599
banana, raw, common	396
dates, natural, dry	652
figs, dried, uncooked	712
grapefruit, raw pulp, all varieties	139
nectarines, raw	212
oranges, raw	181
peaches, raw	197
pears, raw	125
pineapple, raw	113
prunes, dries, uncooked	745
raisins, seedless	751
Meat	
veal, loin, roast	325
pork, loin, centre cut chop, broiled	358
beef, steak, t-bone	321
Miscellaneous	
wheat bran	1182
molasses	1464
peanuts, roasted with skins	658
walnuts	502
bread, white	119
milk, cow's, whole, 3.3% fat	152

Source: U.S. Department of Agriculture, USDA Nutrient Database for Standard References,
Release 13, Agricultural Research Service, 1999.

Absorption and Metabolism

The small intestine has a great capacity to absorb sodium, the greater the sodium intake, the greater the absorption. In the body, the level of sodium in extracellular fluid is controlled by a complicated system that involves the glomerular filtration rate, the cells of the juxtaglomerular apparatus of the kidneys, the renin-aldosterone system, the sympathetic nervous system, concentrations of catecholamines, sodium and potassium in circulating blood and blood pressure.

Potassium is absorbed in the intestine, probably by diffusion. It is well absorbed and normally only a small amount of dietary potassium is found in the feces. Potassium is readily excreted in the urine. The level of potassium in the blood is carefully maintained. If it rises more than three or four times above normal, the beating of the heart will cease. If as little as 6% of the intracellular potassium escapes quickly in the extracellular space, the organism will die. Regulation of body potassium is interrelated with sodium homeostasis; for example, potassium loss is decreased when sodium intake is low.

Deficiency

Deficiency of both sodium and potassium under ordinary conditions is virtually unknown. However, in excessive sweat loss, severe vomiting and diarrhea, and in renal pathology, depletion of sodium can result in muscular cramps, mental confusion, apathy, anorexia and coma. Potassium depletion which occurs when using diuretics in addition to the conditions mentioned previously, will cause muscular weakness, cardiac arrhythmia and mental confusion.

Toxicity

Under normal conditions, a high intake of sodium has not been proven harmful since the body excretes it readily in the urine. However, signs of hypernatremia can manifest if chronic excessive sodium intake is accompanied by inadequate fluid intake and increased loss of body water. Hypernatremic dehydration in infancy and early childhood is a life-threatening clinical entity. The infant is particularly at risk because of the kidney's limited ability to concentrate urine and to excrete excess sodium. The causes of this problem include:

(a) loss of water in excess of sodium such as in hyperventilation, fever, diarrhea, vomiting and high protein feeding.

(b) excessive intake of sodium or salt, and

(c) central nervous system disturbances such as altered thirst mechanism.

Clinical features of hypernatremic dehydration include dry mucous membranes and dysfunction of the central nervous system. The latter is characterized by irritability, lethargy and even coma and convulsions. Fever is commonly present.

Hyperkalemia is almost always the result of greatly diminished renal excretion of potassium caused by renal dysfunction or by severe dehydration. It is virtually impossible to induce hyperkalemia in individuals with normal circulation and renal function.

Hypertension
Epidemiological studies suggest a relationship between salt intake and hypertension but fail to support a cause and effect relationship. Although lowering salt intake reduces blood pressure in salt-sensitive hypertensive individuals, chronic salt overload does not necessarily result in the development of hypertension. Nevertheless, current recommendation is to limit sodium intake to 3000 mg/day.

Some studies have promoted the theory that potassium may have a protective effect on salt-induced hypertension. Others suggest that inadequate intake of calcium and potassium and not excessive salt intake is important in the development of essential hypertension. Further research is required to appreciate the full implication of levels of sodium, potassium, calcium and other nutrients in the diet on the etiology and treatment of essential hypertension.

Other factors that may adversely influence blood pressure include obesity and excessive alcohol intake which may be stronger determinants of blood pressure.

TRACE ELEMENTS

CADMIUM

Cadmium is probably not an essential nutrient for humans. Essentiality of cadmium for animals has not been established either. Cadmium can activate a number of enzymes; however, the enzymes may also be activated by metals other than cadmium.

Cadmium can enter the body via food, water, air and cigarette smoke. Oysters are very rich in cadmium. Normally, absorption of dietary cadmium is very poor. In the body, cadmium is concentrated in the kidney and liver where it is believed to be bound in metallothionein. The excretion of cadmium is slow, and as a result total body cadmium tends to increase with age.

The main concern about cadmium in human nutrition is toxicity. Cadmium toxicity has been reported in Japan where a number of women developed Itai-Itai disease as a result of consuming rice and water obtained from a river polluted by industrial cadmium waste. The women were noted to have calcium and vitamin D-poor diets. Symptoms of the disease producing painful bone fractures, proteinuria, glycosuria and hypercalciuria.

Cadmium poisoning due to consumption of foods stored in cadmium plated vessels has also been reported. Levels of 55 parts per million (PPM) cadmium in foods will result in nausea, vomiting, diarrhea and prostration.

Long-term exposure to cadmium by industrial workers has resulted in pulmonary emphysema, anemia and kidney damage.

CHROMIUM

Chromium is required for normal glucose metabolism. Chromium is a component of the glucose tolerance factor (GTF), a molecular complex which is believed to function by promoting the interaction of insulin with cellular receptor sites.

Dietary sources of chromium include brewer's yeast, meats, cheese, whole grains and certain condiments, notably pepper. The amount of chromium in plants is variable and is dependent upon the chromium content of the soil. Refined flour and sugar have a very low chromium content.

Chromium is present in the diet in an inorganic $Cr^{3}+$ form and also in a biologically active molecule. The absorption of inorganic $Cr^{3}+$ has been shown to be very low in humans and animals (0.5-1%). In animals, the absorption of biologically active chromium is much higher (10-25 percent) but the efficiency of absorption in humans is unknown. Ingested inorganic $Cr^{3}+$ is converted by the body to its biologically active form. Chromium is transported in the blood bound to transferrin suggesting that it may be transported and stored with ferric iron.

Experimentally induced chromium deficiency in animals results in impaired glucose tolerance, fasting hyperglycemia, impaired growth, decreased longevity, elevated serum cholesterol and triglycerides and increased incidence of aortic plaques. Chromium deficiency in humans has been demonstrated clinically in patients given chromium-free parenteral nutrition. Signs of chromium deficiency in humans include peripheral neuropathy, weight loss, glucose intolerance and a metabolic encephalopathy-like confusion state.

Concern exists as to the availability of chromium in the diet regarding marginal deficiencies in certain target groups. Chromium supplementation of children with protein energy malnutrition, individuals with diabetes and the elderly, may result in improved glucose tolerance tests.

Chromium is one of the least toxic trace metals. Experiments with animals show that large doses of chromium fed over a long time show no toxic effects.

COPPER

Copper is an essential element. The adult human body contains about 75 mg of copper. Copper is a cofactor for a wide variety of enzymes such as cytochrome C oxidase, amine oxidases and cerulloplasmin (ferroxidase I). Copper also serves to activate a number of enzymes.

The copper metalloenzymes are required in a variety of processes. Cytochrome-C oxidase is an enzyme of the electron transport system. Tyrosinase is required for synthesis of the melanin pigment of hair and skin. Lysyl oxidase is responsible for the formation of cross links in connective tissues; in copper deficiency synthesis of

collagen, elastin and keratin is defective. Copper is important in the absorption and mobilization of iron. The copper metalloenzyme converts ferrous to ferric iron prior to its binding to the transport protein transferrin. Copper is also a component of the enzyme superoxidase dismutase. This enzyme converts superoxide radicals to oxygen and hydrogen peroxide protecting the cell from oxidative damage by the superoxide radical. Dopamine-beta-hydroxylase, a copper containing metalloenzyme, is required for synthesis of norepinephrine from dopamine. Copper is also important in phospholipid synthesis and myelination as evidenced by symptoms of copper deficiency.

Sources

Good dietary sources of copper include oysters, nuts, liver, kidney, dried legumes and whole grain cereals. Smaller amounts are present in a wide variety of foods (Table 5-5).

Table 5-5

Copper Content of Some Common Foods

Food Items	Copper (mg/100g)
Meat and Shellfish	
oysters, raw	4.4
liver, beef, fried	4.5
liver, lamb, fried	9.8
Nuts and Seeds	
brazil nuts	1.8
sunflower seeds	1.8
almonds	0.9
Cereal and Grain Products	
flour, soybean, defatted	4.1
wheat germ	0.8
wheat bran	1.0
Vegetables	
beans, lima, canned	0.2
peas, raw	0.2
spinach, raw	0.1
asparagus, cooked, boiled, drained	0.1
Fruits	
avocado	0.3
banana	0.1

Source: U.S. Department of Agriculture, USDA Nutrient Database for Standard References, Release 13, Agricultural Research Service, 1999.

Absorption and Metabolism
The level of copper in the body is controlled by the rate of absorption. A luminal copper binding protein serves to deliver copper to the intestinal brush border. The mechanism of regulation is unknown but is believed to involve a sulfur-rich protein in the mucosal cells. The protein is thought to be similar to metallothreonine. The absorption of copper is increased by acid and decreased by the presence of calcium, cadmium, phytates, fiber, calcium carbonate, vitamin C and zinc. The latter competes with copper for the same binding site.

Once absorbed, copper is bound to albumin and free amino acids and is transported to the liver which is the main storage organ in the body. Cerulloplasmin is the protein that transports copper from the liver to other tissues.

Deficiency
Copper deficiency results in hypochromic anemia and connective tissue damage. The hypochromic anemia is indistinguishable from iron-deficiency anemia. This suggests that copper is essential for iron metabolism. Damaged connective tissue is due to impairment of cross-linking of the connective tissue proteins collagen and elastin. This can result in lung damage and hemorrhage due to damaged blood vessels. The most dramatic sign of copper deficiency is sudden death due to spontaneous rupture of a large blood vessel or the heart itself. Infants are particularly susceptible to copper deficiency anemia, low white blood cell counts and bone demineralization have been reported in malnourished infants in Peru fed modified cow's milk formula low in copper. Copper deficiency has been reported in premature infants (born with low copper reserves) fed modified cow's milk. It has also been observed in individuals receiving total parenteral nutrition deficient in copper.

Menke's syndrome is an inherited disease of defective copper metabolism. Symptoms include abnormal keratinization (resulting in kinky hair), low serum and liver copper levels, bone and skeletal abnormalities and mental deterioration resulting in death. The mechanism of this syndrome is not clear but is partly due to a defect in the release from the gut into the circulation. Intravenous copper administration, however, does not appear to alleviate the syndrome.

Hypocupremia may occur in nephrotic syndrome due to loss of cerulloplasmin in the urine. Low levels of serum copper may also occur in kwashiorkor, sprue and celiac disease due to lack of adequate cerulloplasmin synthesis.

Toxicity
Copper poisoning may occur with the consumption of acidic beverages stored in copper containers. Copper toxicity may result in tachycardia, hypertension, jaundice, hemolytic anemia, uremia and death.

Wilson's disease is a genetic disorder in which excessive amounts of copper are deposited in the liver, kidney, cornea and brain producing symptoms such as tremor, ascites and psychoses. Treatment involves administration of penicillamine which chelates copper.

To avoid toxicity of copper, it has been recommended that copper intake over long periods of time not exceed 2-3 mg/day.

FLUORIDE
Traces of fluoride are normally present in human tissues, notably in the bones, teeth, thyroid gland and skin. The primary significance of this element in human nutrition is in the prevention of dental caries.

Fluoride reduces the risk of dental decay in several ways. First, when sufficient fluoride is present, the enamel formed contains some calcium fluorapatite. This is six to ten times more resistant to acid dissolution than calcium hydroxyapatite. Fluorapatite has a tight crystal structure with a smooth surface, and is free from pits and fissures that allow decay. Once fluoride is incorporated into the enamel, it will remain there throughout the life of the tooth.

The deciduous teeth begin to calcify just prior to birth. The crowns of the permanent first molars and the central and lateral incisors begin to calcify within a few months after birth. Therefore, fluoride should be introduced in infancy and continued until the permanent teeth are calcified at approximately 10-12 years of age.

Another way fluoride functions is to promote the remineralization of the enamel by rehardening enamel which has been subjected to repetitive cycles of acid attack after eating. Additionally, higher concentrations of fluoride, which occur when fluoride is applied directly to the tooth surface, or when a child sucks on a fluoride tablet, reduce the concentration of *Streptococcus mutans* (the most virulent species of caries-producing bacteria) in the dental plaque.

There is emerging evidence that fluoride also strengthens the bone. This may help to reduce the risk of osteoporosis in elderly individuals.

Sources

Most human adults ingest between 2 and 3 mg of fluorine daily. The chief source is usually drinking water, which, if it contains 1 PPM of fluorine, will supply 1 to 2 mg/day and will confer optimal protection against tooth decay. Compared with this source, the fluoride in foodstuffs is of little importance (Table 5-6). Very few foods contain more than 1 PPM; the exception is seafish which may contain relatively large amounts in the order of 5 to 10 PPM. Another significant source is tea, particularly Chinese tea, which in the dry state may contain as much as

Table 5-6		
Fluoride Content of Some Common Foods		

Food Items	Fluoride (mg/100g)	
	mean	range
Fish, Shellfish		
cod, raw	700	
crab, canned	200	
mackarel, raw	2700	
mackarel, canned	1200	
salmon, canned		450-900
sardines, canned		800-4000
shrimp, canned	44	
Beverages		
tea, brewed		120-6300
coffee		20-160

Source: **Bowe's and Church's Food Values of Portions Commonly Used.** 14th ed., revised.
    J.B. Lippincott Co., Philadelphia, 1985.

100 PPM. In Britain and Australia, where tea is a popular beverage, the adult intake from this source may be as much as 1 mg daily.

Anti-fluoridationists argue that fluoride can be poisonous. However, the lethal dose for a one-year-old child is 500 mg or an equivalent of 500 quarts of 1 PPM fluoridated water. No toxic effects from fluoridated water supplies or fluoride supplements have been shown. The one problem of excess fluoride ingestion during tooth formation is mottled enamel (or enamel fluorosis). When doses of over 2 mg/day are ingested during the period of tooth formation, some children will develop white stains on their permanent teeth. Therefore, health professionals recommend controlled dosage with supplementation adjusted according to the fluoride content of the drinking water. During infancy, consideration must also be given to the fluoride content of milk, baby foods and drinking water. Breastmilk and fresh cow's milk are low in fluoride, regardless of fluoride in the water supply. Most ready-to-serve and concentrated infant formulas contain varying amounts of fluoride and therefore supplementation is not recommended. The amount of fluoride in formula made from powder depends on the fluoride content of the local water used to prepare the formula.

Commercial baby foods generally contain less than 0.1 PPM fluoride.

A significant source of fluoride for preschool children is fluoridated toothpaste. As the practice of tooth brushing becomes more widespread among preschool children, as many as 75% of children may be using toothpaste by 18 months of age. However, such young children do not rinse effectively and may be swallowing appreciable quantities of toothpaste, possibly 0.3 to 0.4 g at each brushing . This translates into a daily intake of 0.5 mg fluoride from this one source.

Some infants and preschool children are given tea, which is another source of fluoride. Some children can receive 0.4 mg fluoride or more daily from this source.

The Committee on Nutrition of the American Academy of Pediatrics recommends that infants in non-fluoridated areas (water less than 0.3 PPM) be given a fluoride supplement from about 2 weeks of age. The dosage depends on the concentration of fluoride in the local drinking water. According to the

Nutrition Committee of the Canadian Pediatric Society, the need for fluoride supplements in the breastfed infant has not been substantiated. The Committee does not recommend fluoride supplements for breastfed infants. After weaning and for formula fed infants, a supplement of 0.25 mg fluoride daily if the local water supply has less than 0.3 PPM fluoride is advised from six months.

IODINE

Iodine is essential for all animals, including humans. Iodine is required as a component of the thyroid hormones thyroxine (T_4) and triiodothyronine (T_3). Thyroxine and triiodothyronine are required for normal energy metabolism, thermoregulation, intermediary metabolism, protein synthesis, reproduction, growth, physical and mental development, hematopoiesis and neuromuscular function.

The adult human body contains 10-20 mg iodine. About seventy to eighty percent of the iodine is concentrated in the thyroid gland. The remainder is distributed throughout the body with higher concentrations occurring in the salivary and gastric glands as well as in the dense connective tissue.

Sources

The iodine content of foods is dependent upon the iodine content in the environment in which they grew. The best sources of iodine are seafood and seaweed which are of limited significance in Western diets. Meat, milk and eggs may also provide iodine; their iodine content is influenced by the iodine provided in the animal feed. Table 5-7 contains values for some common foods.

Soils of coastal areas are rich in iodine. Many inland areas have iodine-deficient soils. As a result, iodine deficiency is prevalent in inland plains and mountainous areas but not in coastal areas.

Food fortification with iodine has been most effective in the eradication of iodine deficiency. In Canada, table salt must contain 0.01 percent potassium iodide. In the United States, where non-iodized salt is also available, iodized salt contains 76 µg iodine per gram. Since the introduction of iodized salt, goiter resulting from iodine deficiency has been virtually eliminated.

Certain foods such as cabbage, rutabaga, and other members of the Brassica family contain goitrogens which interfere with thy-

roid hormone synthesis. Under normal circumstances insufficient quantities of the goitrigen are ingested to be of clinical significance.

Absorption and Metabolism

Absorbed iodide is converted to iodine in the gut before it enters the circulatory system. Circulating iodine is taken up by the thyroid gland by an active transport process. The iodine is then oxidized and bound to the thyroid protein, thyroglobulin, and then may undergo oxidative coupling to form thyroxine (T_4) and triiodothyronine (T_3). T_4 and T_3 circulate in the bloodstream bound to thyroxine binding globulin, prealbumin and albumin. Very small amounts of T_4 and T_3 circulate in the free form and it is only the free form which is available to the tissues. T_3 is biologically more potent than T_4. The release of T_4/ T_3 by the thyroid is usually at a ratio of 20/1. However, this ratio is decreased when there is lack of iodine. In the tissue T_4 may be converted to T_3 or its inactive isomer, reverse T_3.

The metabolism of iodine occurs under hormonal feedback control. The uptake, synthesis and release of iodine by the thyroid gland is stimulated by thyroid stimulating hormone (TSH) released by the anterior pituitary gland. The release of TSH is dependent upon release of thyrotropin-releasing factor (TRF) from the hypothalamus. TRF release, in turn, is dependent upon

Table 5-7	
Iodine Content of Some Common Foods	
Food Items	**Iodine (µg/100g)**
Condiments	
salt, iodized	44
Fish, Shellfish	
cod, dried, salted, boiled	100
cod liver oil	700
haddock, raw	250
Dairy Products	
milk, whole cow's*	15
milk, human	7
yogurt, whole milk, fruit*	48

See Tables 6-4 and 6-5 for recommended intakes for Canadians and Americans.

* amount is dependent on the iodine content of the animals diet which in turn reflects the iodine contents of the soil on which its feed is grown

Source: Nutrient Value of Some Common Foods, Health Services & Promotion Branch Ministry of Supply & Services, Canada, 1988.

levels of circulating T_3 and T_4. When circulating T_3 and T_4 levels are low, there is increased release of TRF.

Excretion of iodine is largely via the urine. There is significant recycling of iodine derived from T_4 and T_3 metabolites from the tissues back to the circulation. T_4 and T_3 may be conjugated in the liver to glucuronate and sulfate with some losses via the bile although most of the T_4 is reabsorbed.

Deficiency
Iodine deficiency is the most common cause of endemic goiter and cretinism in the world. Endemic goiter is one of the most prevalent nutritional deficiency problems that afflicts millions of people in many parts of the world. Lack of iodine results in an enlargement of the thyroid gland (goiter). The enlargement is due to over stimulation of the thyroid by TRF and TSH which are present in increased levels in an attempt to produce more circulating T_4 and T_3. Severe iodine deficiency can produce myxedema which is characterized by a dry, waxy type swelling, with abnormal deposits of mucoproteins under the skin. Cretinism may occur when there is an insufficient supply of iodine to infants and young children resulting in lowered BMR, dwarfism and retarded mental development. The use of supplemental iodine such as iodized salt in areas where dietary iodine deficiency is endemic has dramatically reduced the incidence of cretinism in the world. Virtual elimination of iodine deficiency worldwide by the year 2010 is targeted.

Toxicity
Excessive dietary intake of iodine results in inhibition of thyroid hormone synthesis clinically known as the Wolff-Chaikoff effect. Generally, the body will adapt to the higher intake but in a few individuals the effect continues and the individual develops a goiter.

Hyperthyroidism resulting from excessive iodine intake is characterized by increased basal metabolism, goiter and disturbances in the autonomic nervous systems causing hyperirritability and increased creatinine metabolism.

IRON

Iron is an essential element in all cells of the body. It plays a key role in oxygen transport and cellular respiration as a constituent of hemoglobin, myoglobin and the enzymes: cytochrome oxidase, peroxidase and catalase.

Hemoglobin, a component of red blood cells, carries oxygen from the lungs to all tissues. In turn, hemoglobin transports carbon dioxide from the tissue to the lungs for expiration. In the muscles, myoglobin accepts oxygen and acts as a temporary reservoir for oxygen. Iron in the cellular cytochrome system is required for energy metabolism.

In the adult male, total body iron amounts to 3.5 to 5.0 g. In the premenopausal woman approximately 2.5 g of iron is normally present.

Sources

The following are some good food sources of iron: liver, heart, kidney, red meats, shell fish, egg yolk, dried beans, other legumes, dried fruits, nuts, green leafy vegetables, dark molasses and whole grain cereals (Table 5-8). Iron-fortified cereal and cereal products, such as flour, bread, infant and breakfast cereals are also good sources when consumed as a dietary staple.

Cow's milk is a poor source of iron as the iron in milk is poorly absorbed. Although breastmilk supplies only about 0.5 to 0.8 mg of iron per day, the bioavailability of its iron is high. Normally, an infant born with an adequate store of iron will receive sufficient iron from breastmilk in the first 4 to 6 months of life. Thereafter, additional sources such as iron-fortified infant cereals and other iron-rich baby foods are required.

Absorption and Metabolism

Iron absorption is influenced by the combination of a number of factors:

(a) Total iron content in the food ingested. The efficiency of iron is inversely proportional to the amount of iron in the food, i.e. the greater the amount, the lesser is the percent absorbed. However, total iron absorbed will still be higher with increased intake.

(b) Amount of heme iron. Heme iron refers to the iron in hemoglobin and myoglobin and is much more readily absorbed than non-heme iron. Approximately 40% of the iron from animal sources is heme iron and the remaining 60% is non-heme iron. All plant food iron is considered to be non-heme iron. Despite the relatively high meat consumption, heme iron contributes only 1 to 2 mg per day in the Western diet which usually contains 10 to 20 mg iron. Non-heme iron is thus the primary source of iron in the human diet.

Table 5-8

Iron Content of Some Common Foods

Food Items	Iron (µg/100g)
Meat/Poultry/Fish	
beef, liver, fried	6.3
beef, heart, simmered	7.5
beef, chuck blade	2.0
ham, roasted	1.1
lamb, roasted	2.0
chicken, breast meat, without skin, roasted	1.2
clams, breaded, fried	2.6
oysters, raw	6.7
shrimp, canned, solids	2.7
Nuts and Seeds	
sunflower seeds, dried, hulled	6.8
walnuts	2.4
almonds, shelled	3.7
cashew nuts, roasted in oil	4.1
Vegetables	
spinach, cooked, drained	3.6
brussel sprouts. cooked	1.2
broccoli, spears	0.8
Legumes	
beans, lima	3.1
lentils, whole, cooked	3.3
Cereal and Grain Products	
wheat germ	6.3
bread, white, enriched	3.0

See Tables 6-4 and 6-5 for recommended intakes for Canadians and Americans.

Source: U.S. Department of Agriculture, USDA Nutrient Database for Standard References, Release 13, Agricultural Research Service, 1999.

(c) Presence of enhancers and inhibitors. Non-heme iron absorption is greatly influenced by a number of dietary factors. Factors that increase non-heme iron absorption include meat, and ascorbic acid. Some of the known inhibitors are phytates, oxalates, phosphates, dietary fiber, soy protein and tannin. Heme iron absorption is enhanced by the presence of meat in the diet, but is not influenced by the other factors that affect non-heme iron absorption.

(d) Iron nutriture of the individual. More iron is absorbed by iron deficient individuals than by individuals with normal iron status. This is particularly true for non-heme iron.

(e) Source of iron, type of iron compound and the food with which it is eaten. Breast fed infants absorb over 50% of iron from human milk compared to formula fed infants who absorb less than 12% of iron from cow milk-derived formula. The percentage of iron absorbed from soy formula is lower than that from cow milk formula. Iron found in meat sources is better absorbed than from nonmeat sources or in the pyrophosphate form.

For iron to be absorbed the presence of an acidic pH, i.e. gastric acid or concurrent intake of vitamin C, is required. Ferric iron has to be reduced to its ferrous form before it can be adsorbed. Luminal iron binding proteins facilitate the absorption of ferrous iron across the intestinal mucosa. A summary of iron metabolism in humans is shown in Figure 5-1.

Approximately 70 to 75% of body iron is in hemoglobin and myoglobin. About 25 to 30% is stored as ferritin and hemosiderin in the liver, bone marrow and spleen. Only about 1% of the total body iron is bound to the iron-transport protein, transferrin and in enzymes such as cytochrome oxidase and catalase. The functional iron is maintained at fairly constant levels. Functional iron levels begin to fall only when storage is depleted.

Tissue iron is re-utilized continuously. Iron released from hemoglobin from catabolized red blood cells is taken up by transferrin and is transported to the bone marrow for the formation of hemoglobin in the synthesis of new red blood cells. Normally, only small amounts of iron are lost in the feces, skin and urine. However, a significant iron loss can occur in menstruation and in hemorrhages.

Deficiency

Iron deficiency is one of the most prevalent nutritional problems in the world. Sectors of the population most susceptible to this problem are infants and young children, menstruating females, pregnant women and individuals on energy restricted diets and older people. WHO estimates over 1 billion individuals suffer from iron deficiency. Its goal was a drastic reduction of this problem by the year 2010 primarily through food fortification.

The most frequent cause of iron deficiency is nutritional, due either to consumption of a diet lacking in iron or a diet in which the iron is of low bioavailability. Excessive losses of iron from menstruation or blood losses are also common causes of iron deficiency. In developing countries, frequent pregnancies with inadequate iron supplementation may precipitate iron deficiency in women. In children, blood sucking parasitic infestations, such as hookworm, often result in iron deficiency.

Figure 5-1

Iron Metabolism in Man

Dietary Intake (18mg) → Small Intestine → Feces (11mg)

(7mg)

Bone Marrow ← Plasma Iron (Turnover 35mg) → Tissue Store 1 g Iron as Ferritin in Reticulo-Endothelial Cells

(20mg)

All Cells

Hemoglobin

Dead Cells

Menstrual Loss (28mg/Period)

Desquamation, Hair, Nails, Skin, Alimentary and Urinary Tract (1mg)
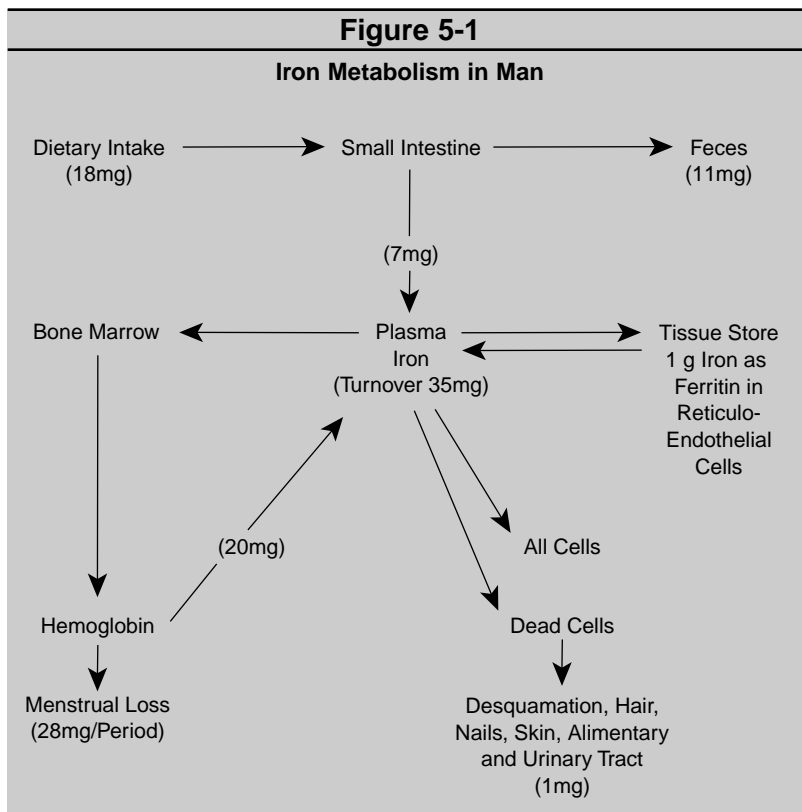
Iron deficiency is characterized by weakness, fatigue, poor work performance and changes in behavior. Iron deficiency anemia is present if the hemoglobin level is less than 11 g/100 ml of blood and erythrocytes are hypochromic and microcytic. Signs of iron deficiency anemia include fatigue, pallor, coldness and parasthesia of the extremities, and greater susceptibility to infections. Iron deficiency may also be associated with oral and gastrointestinal manifestations, such as dysphagia and postcricoid oesophageal stricture. The fingernails may become spoon shaped (koilonychia).

Among infants and young children iron deficiency anemia may have an effect on mental development and may result in cognitive and behavioral problems. The greatest prevalence of iron deficiency anemia occurs between 6 and 24 months of age. During this period, rapid brain growth occurs and cognitive and motor skills are developed. Iron is an essential component and is required not only for cellular growth, but also for fundamental aspects of cellular energy metabolism and function. Iron is also essential for proper production and catabolism of several neurotransmitters. Iron deficiency therefore may also alter brain functions during periods of life other than growth and development.

The thalassemias (genetic hemolytic diseases characterized by defective synthesis of hemoglobin) also result in anemia, which may be severe. Approximately 28% of African Americans lack one of the four normal a-globulin genes and more than 25% of the Indochinese refugees now living in North America also have forms of α-thalassemia. β-Thalassemia occurs in about 5% of people of Mediterranean, Southern Chinese, or Southeast Asian origin. The high prevalence of thalassemia in North America should not be mistaken for iron deficiency.

Toxicity
Excessive iron ingestion may result in hemosiderosis or deposition of iron in the tissues without harmful effect. Prolonged excessive intake may result in hemochromatosis in which further iron storage results in tissue damage, particularly the liver and the pancreas. Hyperpigmentation of the skin is another feature of this disorder. Iron overload has been documented in the Bantu due to their use of iron drums used in the manufacture of alcohol. Chronic alcoholics may exhibit iron overload possibly due to the iron content of certain alcoholic beverages and/or altered iron absorption.

Idiopathic hemochromatosis is a genetic disorder causing excessive absorption of iron and tissue damage due to deposition of iron. However, this is a very rare disease.

LEAD

Lead is ubiquitous. It is present in the air, dust, soil and food. Lead is not required by man but is toxic when excessive amounts are inhaled or ingested.

The amount of lead ingested depends on the lead content of individual foods and the quantities consumed. Lead contamination of food and drink may occur at the point of origin, during commercial processing, or during preparation and serving at home. In recent years, public health regulations have reduced or eliminated many of the sources of such contamination in the field; for example, arsenate insecticides on fruit trees, fertilizer made from sewage sludge, and lead exhaust fumes which can spread to leafy vegetable crops such as lettuce. The food industry has taken measures to reduce the risk of lead consumption by reducing or eliminating the use of lead-soldered cans. Infant foods in resealable plastic or cardboard containers further eliminates the risk of exposure to lead in foods in the early years.

Infants and children absorb more of the ingested lead than do adults. Recent balance studies indicate 30 to 50% absorption for children as compared to 10% for adults. The mean daily dietary intake of lead of infants and children increases from 20-46 mg/day for infants under six months, to 60-73 mg/day for children up to five years of age. In addition, normal hand-to-mouth activity, so typical in young children, can add 20 to 500 mg/day of lead from dust and soil. Therefore, although non-dietary sources account for more than half the lead ingested by children, dietary intake may still exceed the FAO/WHO recommended tolerable intake of 25 µg/kg/week.

The human adult contains 120 mg of lead. This may increase to 400 mg with age. Lead is deposited in the bone and to a lesser extent in soft tissues such as liver, kidney, heart, muscle and brain.

Lead is slowly metabolized, therefore once it enters the tissues, it takes months to eliminate. For both adults and children, early signs of lead poisoning are vague and non-specific. They include

increased irritability and fatigue, and some constipation or occasional diarrhea. As a result, parents may overlook the problem in children, attributing it to other factors. As the disease progresses, children may experience vomiting.

It is only after several months that the symptoms of encephalopathy (the most severe form of lead poisoning which causes ataxia, convulsions and semicoma) may develop. Children who are not treated until signs of encephalopathy are manifest have a 30 to 40% chance of developing gross brain damage if they survive treatment.

Nutritionists and nurses who have the opportunity to observe family eating habits should be aware of practices which can result in contamination of foods in the home. Some fatal and non-fatal cases of encephalopathy in young children had been traced to beverages served in improperly lead-glazed vessels. For example, when acidic foods such as fruits, fruit juices, cider, wine and cola drinks are cooked or served in ancient pewter or imperfectly glazed ceramic utensils are used, small quantities of lead can be leached out.

Proponents of health-store style medications have been promoting bone meal and dolomite mineral supplements as sources of calcium, especially for people who do not drink milk. Many of these products contain variable amounts of heavy metals, including lead and arsenic. Ingestion of such mineral supplements with heavy metal contamination can be considered a potential health hazard if consumed for extended periods of time.

Lead can be transferred from the mother to the child. Lead can readily cross the placental tissue to the fetus. It can also appear in breastmilk. Thus, pregnant and lactating mothers can minimize lead exposure to their children by reducing lead exposure to themselves.

Acute lead poisoning is not as common as it was in past years. Nevertheless, continued vigilance by health professionals is needed to reduce all forms of lead contamination of our food supply.

MANGANESE

Manganese is required by animals and is believed to be essential for humans. Manganese functions as a cofactor for a number of enzymes involved in urea, cholesterol, fatty acid biosynthesis and in protein, carbohydrate and energy metabolism. However, its action as a cofactor is often non-specific; other minerals will perform the same function.

Pyruvate carboxylase and manganese superoxide dismutase are manganese containing metalloenzymes. Pyruvate carboxylase is a regulatory enzyme in gluconeogenesis. Manganese superoxide dismutase is important in protecting the cell from damage by superoxide free radicals. Manganese is bound to RNA but the significance of the binding is not known.

Table 5-9

Manganese Content of Some Common Foods

Food Items	Manganese (mg/100g)
Cereal and Grain Products	
wheat germ	13.3
wheat bran, crude	11.5
rice, brown	3.7
oats	4.9
flour, soybean, defatted	3.0
Legumes	
soybeans, dry	2.5
beans, lima, dry	1.2
Nuts and Seeds	
peanuts, roasted & salted	2.1
sunflower seeds	2.0
Vegetables	
sweet potato, cooked, baked in skin	0.6
peas, green, canned, drained	0.3
kale, cooked, boiled, drained	0.4
corn, sweet, cooked, boiled, drained	0.2
lettuce, iceberg, raw	0.2
Fruits	
banana	0.2
prunes, dried	0.2

Source: U.S. Department of Agriculture, USDA Nutrient Database for Standard References, Release 13, Agricultural Research Service, 1999.

Sources

Sources of manganese include nuts, whole grains and tea. The manganese content of fruits and vegetables is somewhat lower and dependent upon the soil manganese content (Table 5-9).

Absorption and Metabolism

Manganese competes with iron and cobalt for absorption. It is transported in the blood complexed to a protein, transmanganin. Levels of manganese in the body are controlled by biliary excretion. Manganese may also be excreted via the pancreas and gut. In the latter instances, manganese is secreted into the intestinal lumen by the enterocytes.

Deficiency

Manganese deficiency induced experimentally in animals results in poor reproduction, depressed growth, congenital malformations in the offspring, abnormal bone and cartilage, and impaired glucose tolerance. Central nervous system abnormalities such as ataxia may be present. Increased susceptibility to convulsions has also been noted in manganese deficient animals. In humans, manganese deficiency was reported in an individual fed an experimental diet inadvertently devoid of this mineral. Symptoms of the deficiency included weight loss, dermatitis, nausea, vomiting, hypocholesterolemia and prolonged clotting time.

Toxicity

Manganese is one of the least toxic elements. Toxicity due to dietary causes has not been reported. Toxicity due to inhalation of manganese may occur in industrial workers. Inhalation toxicity may produce widespread damage including psychic and neurologic disorders, pulmonary changes, cirrhosis and nephritis.

NICKEL

Nickel has been shown to be essential for six animal species (chicken, cow, goat, minipig, rat and sheep), under conditions of strict dietary and environmental control. It is believed to be required by man but its essentiality has not yet been demonstrated. Nickel is believed to function as a cofactor or component of various metalloenzymes.

Dietary sources of nickel include cereal grains, oysters, tea, baking powder, cabbage, mushrooms and kidney beans. Absorption of

nickel is low (less than 10%) and may occur by active transport. The levels of nickel in the body are believed to be controlled by the kidney. Nickel circulates in the free form, bound to albumin or as nickelplasmin.

Symptoms of nickel deficiency in animals may include depressed growth and reproduction as well as impaired function of the liver, bone and connective tissue.

Nickel toxicity in humans has been observed among workers in nickel refineries. Inhalation and contact with nickel dust may produce respiratory tract neoplasia and dermatitis. Toxicity due to excessive dietary nickel ingestion by humans has not been reported.

In animals, signs of acute nickel toxicity include severe gastroenteritis, tremor, paralysis and death. Chronic toxicity produces damage to the heart, brain, lung, kidney and can ultimately result in cancer.

SELENIUM

Selenium is an essential element for humans. It is a component of the enzyme glutathione peroxidase in the red blood cell. Glutathione peroxidase functions as an antioxidant by reducing hydrogen peroxide and lipid peroxides. As a result, the cell and cell membranes are protected from the damaging effects of peroxidation.

Other functions of selenium are less clear. Selenium is a component of various proteins of biological significance including hemoglobin, myosin, cytochrome C and ribonucleoproteins. However, the significance of selenium as a constituent of these proteins is unknown.

Selenium has a role in heavy metal metabolism as demonstrated by the fact the selenium deficient animals are more susceptible to mercury poisoning.

Sources

Dietary sources of selenium include seafood, organ meats, meats, poultry, fish and cereals. The selenium content in grains is dependent upon soil selenium levels. Fruits and vegetables are low in selenium. The various forms in which selenium is present in foods has not been fully investigated. Selenium may be present

as a component of the sulfur containing amino acids; namely selenomethionine, selenocysteine and selenocystine.

The selenium content in soils in different parts of the world is variable. For example, the average selenium intake in New Zealand is 28-30 mg/day, whereas, the average selenium intake in Venezuela is about 218 mg/day. This observed wide discrepancy in selenium intake reflects differences in the soil content of selenium in the two countries.

Deficiency
Dietary selenium is well absorbed. Regulation of selenium levels is by excretion via the urine, feces and sweat.

Lack of selenium in the soil has resulted in animals displaying symptoms of vitamin E deficiency which are selenium-responsive. For this reason, supplementation of animal feed with selenium has been approved by the United States Food and Drug Administration.

Keshan disease, a disease resulting in cardiomyopathy, is found in children in China living in areas with selenium-poor soil. Supplemental selenium has been effective in lowering the incidence of this disease. However, other factors are known to have a role in the etiology of Keshan disease.

Low selenium levels have been noted in children with kwashiorkor due to insufficient protein intake and in children with phenylketonuria and maple syrup urine disease fed formulated diets low in selenium. Symptoms of muscle pain and weakness which were alleviated by selenium supplementation, have been reported in a patient fed selenium-free total parenteral nutrition.

Epidemiological and animal studies suggest that selenium may have anticancer properties possibly from its antioxidant function as well as from its ability to inhibit many enzymes involved in cell division and growth. Furthermore, a higher incidence of cancer and mortality is found in geographical areas with low selenium. However, the role of selenium in this regard has not been elucidated.

Toxicity
Selenium toxicity has been found among animals grazing in areas with very high soil selenium levels. Acute toxicity in animals

produces "blind staggers", a syndrome characterized by loss of appetite, emaciation, blindness, abdominal pain, muscle paralysis and death. Chronic toxicity produces "alkali disease" with symptoms including stiffness of the joints, cirrhosis of the liver, anemia and cardiac atrophy.

Selenium toxicity in humans has occurred as a result of excessive industrial exposure. Acute toxicity may result in fever, vomiting, hypotension, convulsions and death. Symptoms of chronic toxicity include hemolytic anemia and damage to the kidney, spleen and liver.

TIN

The adult human body contains approximately 17 mg of tin. To date, tin has not been shown to be an essential element although there is evidence to suggest that it may be essential to some species of animals.

Tin is poorly absorbed. Over 90% of experimentally administered tin is excreted in the feces. Minute amounts are absorbed and remain in body tissues.

Chronic industrial exposure to tin dust or fumes can cause benign pneumoconiosis. Tin poisoning from ingested food is rare. Tin-canned acidic fruit and vegetable juices have been reported to cause acute gastrointestinal disturbances such as nausea and diarrhea, possibly from local irritation of the mucosa. In all reported cases the tin content in the beverage contained 250 PPM or more of tin. Canned foods exhibiting more than 250 PPM of tin reflect poor manufacturing practices. Lacquer-lined cans used today greatly minimize the possibility of contamination.

ZINC

The biological role of zinc has been known for more than 100 years, but only recently has the importance of this element in human nutrition been recognized. There are over 70 enzymes in which zinc acts as a co-factor. Zinc, therefore, plays diverse roles in carbohydrate, lipid, protein and nucleic acid metabolism and in cell growth. Zinc is a cofactor for aldolase (glycolysis), malate dehydrogenase (in citric acid cycle), cytochrome C (in the electron transport system) and glutamate dehydrogenase (in amino acid synthesis and catabolism). Other enzymes for which zinc is a

cofactor include carboxypeptidases (digestive enzymes), alkaline phosphatase, retinene reductase (which converts retinol to retinal) and superoxidase dismutase (a cellular antioxidant). Zinc is a constituent of carbonic anhydrase which serves to rid the body of carbon dioxide by converting it to carbonic acid. Zinc is required for protein, carbohydrate, mucopolysaccharide, lipid and nucleic acid metabolism. It is necessary for cell division, growth and repair; for example zinc is required for the activity of both DNA and RNA polymerase. Zinc also serves as a ligand, binding to and stabilizing various compounds.

Zinc is widely distributed throughout the body. The average adult body contains 2-3 mg of zinc. Zinc is required for normal growth, sexual maturation, wound healing, appetite, sense of taste and smell, and skin integrity as evidenced by symptoms of zinc deficiency.

Sources and Availability
The richest dietary sources of zinc include red meats, liver, fish, eggs, milk, nuts and legumes (Table 5-10). In general, the concentration of zinc in animal foods is higher and is more available than in vegetable products. The availability of zinc in plant products is decreased by the presence of fiber and phytates which bind to zinc rendering it unavailable. Other inhibitors of zinc absorption include phosphate, calcium, cadmium, copper and ferrous iron. Geophagia (clay eating) is another factor which decreases absorption and has contributed to zinc deficiencies in certain areas of the world. Absorption of zinc is increased by the presence of chelating agents which bind phytate, certain amino acids and a high level of dietary protein.

For young infants, breastmilk is the best dietary source of zinc. Colostrum contains as much as 20 mg of zinc per liter. The level of zinc in breastmilk progressively declines as lactation continues to a level of 1 mg per liter at 3 months and 0.8 mg per liter, averaging about 3.5 mg. Infant formulas are supplemented with zinc to contain no less than 0.5 mg of zinc per 100 kcal or 3.25 mg per liter.

The recommended intake for young infants (0-3 months) is 2 mg of zinc per day and for infants from 3 to 12 months is 3 mg. Infants who consume a sufficient amount of breastmilk, infant

formula or cow's milk and eat a balanced diet containing infant cereals and meat would consume sufficient zinc to meet their requirement. Zinc intake of strict vegetarians is consistently marginal due to the lack of animal foods providing bioavailable zinc.

Table 5-10

Zinc Content of Some Common Foods

Food Items	Zinc (mg/100g)
Meat/Fish/Poultry	
liver, beef	3.9
beef, rib, roasted	5.4
lamb, leg, roasted	4.4
turkey, dark meat with skin roasted	2.1
Cereal and Grain Products	
wheat germ	12.3
wheat, bran, crude	7.3
oats	4.0
bread, whole wheat	1.9
bread, rye	1.1
rice, white, enriched, long grain, cooked	0.5
Dairy and Egg Products	
milk, nonfat, regular, dry	4.1
cheese, cheddar	3.1
milk, whole, fluid	0.4
milk, skim, fluid	0.4
egg, chicken, yolk, fresh, raw	3.1
egg, chicken, whole, fresh, raw	1.1
Vegetables	
peas, green, cooked, boiled, drained	1.1
spinach, cooked, boiled, drained	0.8
corn, sweet, cooked, boiled, drained	0.5
potato, raw	0.4
lettuce, iceberg, raw	0.2
carrot, raw	0.2
cabbage, cooked, boiled, drained	0.1
Fruits	
cherries	0.1
oranges	0.1
pears	0.1

See Tables 6-4 and 6-5 for recommended intakes for Canadians and Americans.

Source: U.S. Department of Agriculture, USDA Nutrient Database for Standard References, Release 13, Agricultural Research Service, 1999.

Absorption and Metabolism

Zinc absorption in the gut is facilitated by the presence of a low molecular weight ligand believed to be secreted by the pancreas. The ligand is present in human breastmilk thereby enhancing the bioavailability of zinc.

Once absorbed by the enterocyte, zinc may be used for cellular processes, complexed to metallothionein or released into the circulation bound to a serum protein. The release of zinc into the portal circulation is believed to be under homeostatic control. With increased zinc intake, zinc is retained by the enterocyte and secreted into the gastrointestinal tract to be excreted in the feces.

Circulating zinc may be bound to albumin (57% of the total plasma zinc), α-2-macroglobulin (40%), various amino acids and possibly other molecules (3%). The main route of zinc excretion is via the gastrointestinal tract. Zinc is also excreted in the urine and sweat. Losses of zinc via the latter route may be significant in hot climates.

Deficiency

Zinc deficiency was first documented in malnourished adolescents in the Middle East. The deficiency was characterized by growth retardation, hypogonadism and delayed sexual maturation. One of the first symptoms of zinc deficiency is poor appetite and changes in the perception of taste and smell. Lethargy, slowing of activity, apathy and depression are also associated with zinc deficiency. Impaired cell-mediated immunity, slow wound healing, dermatitis and failure to thrive are other manifestations of this nutritional problem.

Zinc is prevalent in the brain. There is evidence to indicate that zinc deficiency in infants may lead to deficits in children's neuropsychologic functions, activity or motor development subsequently interfering with their cognitive development and performance.

The etiology of zinc deficiency may be dietary or physiologic. Dietary deficiency can be due to a lack of zinc in foods consumed. It may also be caused by the presence of substances, described earlier, that interfere with zinc absorption.

Various disorders of the gastrointestinal tract may result in zinc depletion; for example, severe intestinal malabsorption syndrome, enterocolitis, short bowel syndrome, celiac disease and cystic fibrosis. Plasma zinc levels may be depressed in infectious hepatitis, alcoholism, protein-energy malnutrition, and sickle cell anemia. Acrodermatitis enteropathica is a rare genetic disease characterized by alopecia, dermatitis, diarrhea, photophobia, psychological changes, failure to thrive, infections and death. The disease is believed to be due to a defect in zinc absorption, most likely due to insufficient synthesis of the low molecular weight zinc ligand. Zinc deficiency in breast-fed only children is a rare disorder affecting mostly premature infants. However, some cases of the disease in term infants have also been reported. Acrodermatitis enteropathica first manifests itself upon weaning from breastmilk. The disease may be treated by continuing breastfeeding or through supplemental zinc.

Toxicity

Zinc has relatively low toxicity with a wide margin of safety between requirement and toxic intake. However, therapeutic use of zinc salts at levels of 50 mg of elemental zinc per day can result in transient nausea.

Suggested Readings

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CHAPTER 6

*DIETARY STANDARDS, FOOD GUIDES
AND DIETARY GUIDELINES*

Dietary standards, dietary goals (guidelines) and food guides are nutritional statements intended to assist in the selection of foods to provide a balance of nutrients in the diet to maintain health. The primary purpose of these statements is nutrition education. Dietary standards provide means to evaluate dietary adequacies and dietary guidelines provide education in relation to nutritionally adequate diets.

Dietary Standards

Dietary standards define the levels of dietary reference intakes (DRI) sufficient to meet the needs of the majority of healthy individuals in the targeted population.

Dietary standards were developed to assist in the planning and procurement of food supplies for individuals and groups. They are used in public health to assess the need for and levels of fortification of a particular nutrient in the diet. They are also used as standards in the food and pharmaceutical industry for formulation of infant formulas and enteral feeding supplements.

However, the philosophy concerning dietary standards has changed. Standards, particularly those in industrialized countries, are also used to reduce the risk of chronic diseases and are more flexible in addressing these multiple uses.

The various dietary standards set by the FAO/WHO, Australia, the United Kingdom, Canada and the United States are shown in Tables 6-1 to 6-5 respectively. Although these and other dietary standards may be based on essentially the same scientific data the actual recommended levels may vary considerably. These apparent discrepancies are due to differing definitions of health, interpretation of data and choice of factors used to adjust the data to cover the needs of society and inefficiencies of nutrient utilization.

Table 6-1

Recommended Intakes of Nutrients (WHO)

AGE	Vitamin A (µg retinol /day)[a,b]		Folate (µg/day)[a]		Vitamin B12 (µg/day)[a]		Vitamin C (mg/day)[c]		Vitamin D (µg/day)[c]		Iron Absorbed (µg/kg/day)[a,d]		Zinc (mg/day)[e]	
	M	F	F	M	F	M	F	M	F	M	F	M	M	F
Infants (months)														
0-3	350		16		0.1		20		10			120	4.0	3.1
4-6	350		24		0.1		20		10			120	4.0	3.1
7-9	350		32		0.1		20		10			120	4.0	2.8
10-12	350		32		0.1		20		10			120	4.0	2.8
Children and adults														
1-2	400		50		1.0		20		10			56	4.0	3.9
3-4	400		50		1.0		20		10			44	4.0	3.9
5-6	400		102		1.0		20		10			40	4.0	3.9
7-10	400		102		1.0		20		2.5			40	4.0	3.9
11-12	500		102		1.0		20		2.5			40	7.0	6.6
13-14	600		170		1.0		30		2.5			34	7.0	6.6
15-16	600	500	170	200	1.0		30		2.5		40	34	7.0	5.5
17-18	600	500	170	200	1.0		30		2.5		40	34	7.0	5.5
19+	600	500	170	200	1.0		30		2.5		43	18	5.5	5.5

Table 6-1 *Continued*

Recommended Intakes of Nutrients (WHO)

AGE	Vitamin A (µg retinol /day)[a,b]		Folate[a] (µg/day)		Vitamin B12[a] (µg/day)		Vitamin C[c] (mg/day)		Vitamin D[c] (µg/day)		Iron[a,d] Absorbed (µg/kg/day)		Zinc[e] (mg/day)	
	F	M	F	M	F	M	F	M	F	M	F	M	F	M
Pregnant women														
	600		370 to 470		1.4		50		10		f		6.4 to 7.5	
Lactating women														
	850		270		1.3		50		10		24		13.7	
Post-menopausal women														
	500		170		1.0		30		2.5		18		5.5	

[a] FAO Food and Nutrition Series No. 23. Rome, Food and Agriculture Organization, 1988.

[b] Minimum level considered safe.

[c] WHO Technical Report Series No. 452. Geneva, World Health Organization, 1970. 2.5µg of cholecalciferol are equivalent to 100 IU of vitamin D.

[d] The amount of absorbed iron is a variable proportion of the intake, depending on the type of diet.

[e] WHO Technical Report Series No. 532. Geneva, World Health Organization, 1973.

[f] Requirements during pregnancy depend on the woman's iron status before pregnancy.

Table 6-2

Recommended Nutrient Intakes for Use in Australia

For Children under 7 years (mean daily intakes)

	0-6 mths Breastfed	0-6 mths Bottlefed	7-12 mths	1-3 yrs	4-7 yrs
Vitamin A[a] (µg retinol equivalents)	425	425	300	300	350
Thiamin[b] (mg)	0.15	0.25	0.35	0.5	0.7
Riboflavin (mg)	0.4	0.4	0.6	0.8	1.1
Niacin[c] (mg niacin equivalents)	4	4	7	9–10	11–13
Vitamin B6[d] (mg)	0.25	0.25	0.45	0.6–0.9	0.8–1.3
Total folate[e](µg)	50	50	75	100	100
Vitamin B12 (µg)	0.3	0.3	0.7	1.0	1.5
Vitamin C (mg)	25	25	30	30	30
Vitamin E (mg alpha-(tocopherol equivalents)	2.5	4.0	4.0	5.0	6.0
Zinc[f] (mg)	3	3-6	4.5	4.5	6
Iron[g] (mg)	0.5	3.0	9.0	6–8	6–8
Iodine (µg)	50	50	60	70	90
Magnesium (mg)	40	40	60	80	110
Calcium (mg)	300	500	550	700	800
Phosphorus (mg)	150	150	300	500	700
Selenium[h] (µg)	10	10	15	25	30
Sodium[i] (mg)	140–280	140–280	320–580	320–1150	460–1730
Potassium (mg)	390–580	390–580	470–370	980–2730	1560–3900
Protein[j] (g)		2.0g/kg body wt	1.6g/kg body wt	14-18	18-24

a Vitamin A can also be supplied as β-carotene.

b Thiamin recommendation based on 0.1 mg thiamin/1000kJ.

c Niacin can also be supplied as tryptophan in dietary protein.

d For vitamin B6 RDI based on 0.02 mg/g dietary protein.

e RDI is for total folate, i.e., for free plus conjugated folate.

f For Zinc RDI reduced 1989 (lower end of range that was recommended in 1982).

g For iron RDI is expressed as a range to allow for differences in bioavailability of iron from different Australian foods. Recommendations for pregnancy are for 2nd and 3rd trimesters.

h Selenium intake should not exceed 600µg/day.

i For sodium the upper end of the recommended range for adults corresponds to 6.0g/day of common salt NaCl.

j Protein RDI essentially follows FAO/WHO/UNU (1985). Figures based on 0.75g protein/kg body weight for adults; 1.0g protein/kg body weight for children 4 to 18 years. No recommendation has been made for protein for breastfed infants under the age of 6 months. Many observations show that infants breastfed by healthy well-nourished mothers will grow at a satisfactory rate for the first 4-6 months. It can therefore be assumed that protein requirements are met if the volume of milk maintains growth at an acceptable rate.

Table 6-2 *Continued*

Recommended Nutrient Intakes for Use in Australia

For Children over 7 years (mean daily intakes)

	Boys			Girls		
	8–11 yrs	12–15 yrs	16–18 yrs	8–11 yrs	12–15 yrs	16–18 yrs
Vitamin A[a] (µg retinol equivalents)	500	725	760	500	725	750
Thiamin[b] (mg)	0.9	1.2	1.2	0.8	1.0	0.9
Riboflavin (mg)	1.4	1.8	1.9	1.3	1.6	1.4
Niacin[c] (mg niacin equivalents)	14–16	19–21	20–22	14–16	17–19	15–17
Vitamin B6[d] (mg)	1.1–1.5	1.4–2.1	1.5–2.2	1.0–1.5	1.2–1.8	1.1–1.6
Total folate[e](µg)	150	200	200	150	200	200
Vitamin B12 (µg)	1.5	2.0	2.0	1.5	2.0	2.0
Vitamin C (mg)	30	30	40	30	30	30
Vitamin E (mg alpha-(tocopherol equivalents)	8.0	10.5	11.0	8.0	9.0	8.0
Zinc[f] (mg)	9	12	12	9	12	12
Iron[g] (mg)	6–8	10–13	10–13	6–8	10–13	10–13
Iodine (µg)	120	150	150	120	120	120
Magnesium (mg)	180	260	320	160	240	270
Calcium (mg)	800	1200	1000	900	1000	800
Phosphorus (mg)	800	1200	1100	800	1200	1100
Selenium[h] (µg)	50	85	85	50	70	70
Sodium[i] (mg)	600–2300	920–2300	920–2300	600–2300	920–2300	920–2300
Potassium (mg)	1950–5460	1950–5460	1950–5460	1950–5460	1950–5460	1950–5460
Protein[j] (g)	27–38	42–60	64–70	27–39	44–55	57

[a] Vitamin A can also be supplied as β-carotene.

[b] Thiamin recommendation based on 0.1 mg thiamin/1000kJ.

[c] Niacin can also be supplied as tryptophan in dietary protein.

[d] For vitamin B6 RDI based on 0.02 mg/g dietary protein.

[e] RDI is for total folate, i.e., for free plus conjugated folate.

[f] For Zinc RDI reduced 1989 (lower end of range that was recommended in 1982).

[g] For iron RDI is expressed as a range to allow for differences in bioavailability of iron from different Australian foods. Recommendations for pregnancy are for 2nd and 3rd trimesters.

[h] Selenium intake should not exceed 600µg/day.

[i] For sodium the upper end of the recommended range for adults corresponds to 6.0g/day of common salt NaCl.

[j] Protein RDI essentially follows FAO/WHO/UNU (1985). Figures based on 0.75g protein/kg body weight for adults; 1.0g protein/kg body weight for children 4 to 18 years. No recommendation has been made for protein for breastfed infants under the age of 6 months. Many observations show that infants breastfed by healthy well-nourished mothers will grow at a satisfactory rate for the first 4-6 months. It can therefore be assumed that protein requirements are met if the volume of milk maintains growth at an acceptable rate.

| Table 6-2 *Continued* |

Recommended Nutrient Intakes for Use in Australia

For adults (mean daily intakes)

	Men		Women			
	19–64 yrs	64+ yrs	19–54 yrs	54+ yrs	Pregnant	Lactating
Vitamin A[a] (µg retinol equivalents)	750	750	750	750	+0	+450
Thiamin[b] (mg)	1.1	0.9	0.8	0.7	+0.2	+0.4
Riboflavin (mg)	1.7	1.3	1.2	1.0	0.3	+0.5
Niacin[c] (mg niacin equivalents)	18–20	14–17	12–14	10–12	+2	+5
Vitamin B$_6$[d] (mg)	1.3–1.95	1.0–1.5	0.9–1.4	0.8–1.1	+0.1	0.7–0.8
Total folate[e](µg)	200	200	200	200	+200	+150
Vitamin B$_{12}$ (µg)	2.0	2.0	2.0	2.0	+1.0	+0.5
Vitamin C (mg)	40	40	30	30	+30	+30
Vitamin E (mg alpha-(tocopherol equivalents)	10.0	10.0	7.0	7.0	+0	+2.5
Zinc[f] (mg)	12	12	12	12	+4	+6
Iron[g] (mg)	7	7	12–16	5–7	+10–20	+0
Iodine (µg)	150	150	120	120	+30	+50
Magnesium (mg)	320	320	270	270	+30	+70
Calcium (mg)	800	800	800	1000	+300	+400
Phosphorus (mg)	1000	1000	1000	1000	+200	+200
Selenium[h] (µg)	85	85	70	70	+10	+15
Sodium[i] (mg)	920–2300	920–2300	920–2300	920–2300	+0	+0
Potassium (mg)	1950–5460	1950–5460	1950–5460	1950–5460	+0	+0
Protein[j] (g)	55	55	45	45	+6	+16

a Vitamin A can also be supplied as β-carotene.

b Thiamin recommendation based on 0.1 mg thiamin/1000kJ.

c Niacin can also be supplied as tryptophan in dietary protein.

d For vitamin B$_6$ RDI based on 0.02 mg/g dietary protein.

e RDI is for total folate, i.e., for free plus conjugated folate.

f For Zinc RDI reduced 1989 (lower end of range that was recommended in 1982).

g For iron RDI is expressed as a range to allow for differences in bioavailability of iron from different Australian foods. Recommendations for pregnancy are for 2nd and 3rd trimesters.

h Selenium intake should not exceed 600µg/day.

i For sodium the upper end of the recommended range for adults corresponds to 6.0g/day of common salt NaCl.

j Protein RDI essentially follows FAO/WHO/UNU (1985). Figures based on 0.75g protein/kg body weight for adults; 1.0g protein/kg body weight for children 4 to 18 years. No recommendation has been made for protein for breastfed infants under the age of 6 months. Many observations show that infants breastfed by healthy well-nourished mothers will grow at a satisfactory rate for the first 4-6 months. It can therefore be assumed that protein requirements are met if the volume of milk maintains growth at an acceptable rate.

Table 6-3

Recommended Nutrient Intakes, United Kingdom

AGE	Protein (g/d)[a]	Thiamin (mg/d)	Riboflavin (mg/d)	Niacin (mg/d)	Vitamin B₆ (mg/d)[d]	Vitamin B₁₂ (µg/d)	Folate (µg/d)	Vitamin C (mg/d)	Vitamin A (µg/d)	Vitamin D (µg/d)
0-3 months	12.5[b]	0.2	0.4	3	0.2	0.3	50	25	350	8.5
4-6 months	12.7	0.2	0.4	3	0.2	0.3	50	25	350	8.5
7-9 months	13.7	0.2	0.4	4	0.3	0.4	50	25	350	7
10-12 months	14.9	0.3	0.4	5	0.4	0.4	50	25	350	7
1-3 years	14.5	0.5	0.6	8	0.7	0.5	70	30	400	7
4-6 years	19.7	0.7	0.8	11	0.9	0.8	100	30	500	-
7-10 years	28.3	0.7	1.0	12	1.0	1.0	150	30	500	-
Males										
11-14 years	42.1	0.9	1.2	15	1.2	1.2	200	35	600	-
15-18 years	55.2	1.1	1.3	18	1.5	1.5	200	40	700	-
19-50 years	55.5	1.0	1.3	17	1.4	1.5	200	40	700	-
50+ years	53.3	0.9	1.3	16	1.4	1.5	200	40	700	**
Females										
11-14 years	41.2	0.7	1.1	12	1.0	1.2	200	35	600	-
15-18 years	45.0	0.8	1.1	14	1.2	1.5	200	40	600	-
19-50 years	45.0	0.8	1.1	13	1.2	1.5	200	40	600	-
50+ years	46.5	0.8	1.1	12	1.2	1.5	200	40	600	**

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Table 6-3 *Continued*

Recommended Nutrient Intakes, United Kingdom

AGE	Protein (g/d)[a]	Thiamin (mg/d)	Riboflavin (mg/d)	Niacin (mg/d)	Vitamin B_6 (mg/d)[d]	Vitamin B_{12} (µg/d)	Folate (µg/d)	Vitamin C (mg/d)	Vitamin A (µg/d)	Vitamin D (µg/d)
Pregnancy[c]	+ 6	+0.1[e]	+0.3	*	*	*	+100	+10	+100	10
Lactation[c]										
0-4 months	+11	+0.2	+0.5	+2	*	+0.5	+60	+30	+350	10
4+ months	+ 8	+0.2	+0.5	+2	*	+0.5	+60	+30	+350	10

[a] These figures, based on egg and milk protein, assume complete digestibility
[b] No values for infants 0 to 3 months are given by WHO. The reference nutrient intake is calculated from the recommendations of Committee on Medical Aspects of Food policy (COMA)
[c] To be added to adult requirement through all stages of pregnancy and lactation
[d] Based on protein providing 14.7% of EAR for energy
[e] For last trimester only
* No increment
**After age 65 the RNI is 10 µg/d for men and women

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Table 6-3 *Continued*

Reference Nutrient Intakes, United Kingdom

AGE	Calcium (mg/d)	Phosphorus[1] (mg/d)	Magnesium (mg/d)	Sodium[2] (mg/d)	Potassium[3] (mg/d)	Chloride[4] (mg/d)	Iron (mg/d)	Zinc (mg/d)	Copper (mg/d)	Selenium (µg/d)	Iodine (µg/d)
0-3 months	525	400	55	210	800	320	1.7	4.0	0.2	10	50
4-6 months	525	400	60	280	850	400	4.3	4.0	0.3	13	60
7-9 months	525	400	75	320	700	500	7.8	5.0	0.3	10	60
10-12 months	525	400	80	350	700	500	7.8	5.0	0.3	10	60
1-3 years	350	270	85	500	800	800	6.9	5.0	0.4	15	70
4-6 years	450	350	120	700	1100	1100	6.1	6.5	0.6	20	100
7-10 years	550	450	200	1200	2000	1800	8.7	7.0	0.7	30	110
Males											
11-14 years	1000	775	280	1600	3100	2500	11.3	9.0	0.8	45	130
15-18 years	1000	775	300	1600	3500	2500	11.3	9.5	1.0	70	140
19-50 years	700	550	300	1600	3500	2500	8.7	9.5	1.2	75	140
50+ years	700	550	300	1600	3500	2500	8.7	9.5	1.2	35	140
Females											
11-14 years	800	625	280	1600	3100	2500	14.8	9.0	0.8	45	130
15-18 years	800	625	300	1600	3500	2500	14.8	7.0	1.0	60	140
19-50 years	700	550	270	1600	3500	2500	14.8	7.0	1.2	60	140
50+ years	700	550	270	1600	3500	2500	8.7	7.0	1.2	60	140

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Table 6-3 *Continued*

Reference Nutrient Intakes, United Kingdom

AGE	Calcium (mg/d)	Phosphorus[1] (mg/d)	Magnesium (mg/d)	Sodium[2] (mg/d)	Potassium[3] (mg/d)	Chloride[4] (mg/d)	Iron (mg/d)	Zinc (mg/d)	Copper (mg/d)	Selenium (µg/d)	Iodine (µg/d)
Pregnancy	*	*	*	*	*	*	*	*	*	*	*
Lactation											
0-4 months	+550	+440	+50	*	*	*	*	+6.0	+0.2	+15	*
4+ months	+550	+440	+50	*	*	*	*	+2.5	+0.3	+15	*

1 Phosphorus RNI is set equal to calcium in molar terms

2 1 mmol sodium=23 mg

3 1 mmol potassium=39 mg

4 Corresponds to sodium 1 mmol=35.5 mg

5 Insufficient for women with high menstrual losses where the most practical way of meeting iron requirements is to take iron supplements

* No increment

The estimation of dietary standards is based on several techniques which have been summarized by the U.S. Committee on Dietary Allowances, Food and Nutrition Board, National Research Council. The techniques include: (1) nutrient intakes of fully breastfed infants and of apparently healthy people from their food supply, (2) epidemiological observations of nutrient status in populations in relation to intake, (3) biochemical measurements that assess degree of tissue saturation or adequacy of molecular function in relation to nutrient intake, (4) nutrient balance studies that measure nutritional status in relation to nutrient intake, (5) studies of subjects maintained on diets containing marginally low or deficient levels of a nutrient followed by correction of the deficit with measured amounts of that nutrient (such studies are undertaken in humans only when the risk is minimal), and (6) in some cases, extrapolation of data from animal experiments. Once the average requirement and variability have been established within the population under study, the data are adjusted to allow for covering the needs of almost all individuals in a society as well as inefficiencies of utilization such as poor absorption, conversion of precursors to active forms of vitamins, etc.

The recommended daily intake (allowances) were designed to meet the needs of almost all individuals (especially healthy individuals); they exceed the requirements of most. Thus, an individual is not necessarily deficient for a particular nutrient if his or her nutrient intake does not meet the standards. However, the risk of deficiency will increase as intake of a nutrient decreases below the recommended level of intake.

Table 6-4

Recommended Nutrient Intakes, Canada

AGE	Sex	Weight (kg)	Protein (g)	Vitamin A (RE)[a]	Vitamin D (µg)	Vitamin E (mg)	Vitamin C (mg)	Folate (µg)	Vitamin B₁₂ (µg)	Calcium (mg)	Phosphorus (mg)	Magnesium (mg)	Iron (mg)	Iodine (µg)	Zinc (mg)
Months															
0-4	Both	6.0	12[b]	400	10	3	20	25	0.3	250	150	20	0.3[d]	30	2[d]
5-12	Both	9.0	12	400	10	3	20	40	0.4	400	200	32	7	40	3
Years															
1	Both	11	13	400	10	3	20	40	0.5	500	300	40	6	55	4
2-3	Both	14	16	400	5	4	20	50	0.6	550	350	50	6	65	4
4-6	Both	18	19	500	5	5	25	70	0.8	600	400	65	8	85	5
7-9	M	25	26	700	2.5	7	25	90	1.0	700	500	100	8	110	7
	F	25	26	700	2.5	6	25	90	1.0	700	500	100	8	95	7
10-12	M	34	34	800	2.5	8	25	120	1.0	900	700	130	8	125	9
	F	36	36	800	2.5	7	25	130	1.0	1100	800	135	8	110	9
13-15	M	50	49	900	2.5	9	30[e]	175	1.0	1100	900	185	10	160	12
	F	48	46	800	2.5	7	30[e]	170	1.0	1000	850	180	13	160	9
16-18	M	62	58	1000	2.5	10	40[e]	220	1.0	900	1000	230	10	160	12
	F	53	47	800	2.5	7	30[e]	190	1.0	700	850	200	12	160	9
19-24	M	71	61	1000	2.5	10	40[e]	220	1.0	800	1000	240	9	160	12
	F	58	50	800	2.5	7	30[e]	180	1.0	700	850	200	13	160	9

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Table 6–4 *Continued*

Recommended Nutrient Intakes, Canada

AGE	Sex	Weight (kg)	Protein (g)	Vitamin A (RE)[a]	Vitamin D (µg)	Vitamin E (mg)	Vitamin C (mg)	Folate (µg)	Vitamin B$_{12}$ (µg)	Calcium (mg)	Phosphorus (mg)	Magnesium (mg)	Iron (mg)	Iodine (µg)	Zinc (mg)
Years															
25-49	M	74	64	1000	2.5	9	40[e]	230	1.0	800	1000	250	9	160	12
	F	59	51	800	2.5	6	30[e]	185	1.0	700	850	200	13	160	9
50-74	M	73	63	1000	5	7	40[e]	230	1.0	800	1000	250	9	160	12
	F	63	54	800	5	6	30[e]	195	1.0	800	850	210	8	160	9
75+	M	69	59	1000	5	6	40[e]	215	1.0	800	1000	230	9	160	12
	F	64	55	800	5	5	30[e]	200	1.0	800	850	210	8	160	9
Pregnancy (additional)															
1st trimester			5	0	2.5	2	0	200	0.2	500	200	15	0	25	6
2nd trimester			20	0	2.5	2	10	200	0.2	500	200	45	5	25	6
3rd trimester			24	0	2.5	2	10	200	0.2	500	200	45	10	25	6
Lactation (additional)															
			20	400	2.5	3	25	100	0.2	500	200	65	0	50	6

a Retinol Equivalents
b Protein is assumed to be from breast milk and must be adjusted for infant formula
c Infant formula with high phosphorus should contain 375 mg calcium
d Breast milk is assumed to be the source of the mineral
e Smokers should increase vitamin C by 50%

Source: Health and Welfare Canada: Nutrition Recommendations. The Report of the Scientific Review Committee. Ottawa, Supply and Services Canada, 1990.

Table 6-5

Recommended Dietary Allowances, United States[a]

Category	Age (Years) or Condition	Weight[b] (kg)	(lb)	Height[b] (cm)	(in)	Protein (g)	Vitamin A (µg RE)[c]	Vitamin D (µg)[d]	Vitamin E (mg α-TE)[e]	Vitamin K (µg)
Infants										
	0.0-0.5	6	13	60	24	13	375	7.5	3	5
	0.5-1.0	9	20	71	28	14	375	10	4	10
Children										
	1-3	13	29	90	35	16	400	10	6	15
	4-6	20	44	112	44	24	500	10	7	20
	7-10	28	62	132	52	28	700	10	7	30
Males										
	11-14	45	99	157	62	45	1000	10	10	45
	15-18	66	145	176	69	59	1000	10	10	65
	19-24	72	160	177	70	58	1000	10	10	70
	25-50	79	174	176	70	63	1000	5	10	80
	51+	77	170	173	68	63	1000	5	10	80

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Table 6-5 Continued

Recommended Dietary Allowances, United States[a]

Category / Age (Years) or Condition	Weight[b] (kg)	(lb)	Height[b] (cm)	(in)	Protein (g)	Vitamin A (µg RE)[c]	Vitamin D (µg)[d]	Vitamin E (mg α-TE)[e]	Vitamin K (µg)
Females									
11-14	46	101	157	62	46	800	10	8	45
15-18	55	120	163	64	44	800	10	8	55
19-24	58	128	164	65	46	800	10	8	60
25-50	63	138	163	64	50	800	5	8	65
51+	65	143	160	63	50	800	5	8	65
Pregnant					60	800	10	10	65
Lactating									
1st 6 months					65	1300	10	12	65
2nd 6 months					62	1200	10	11	65

a The allowances, expressed as average daily intakes over time, are intended to provide for individual variations among most normal persons as they live in the United States under usual environmental stresses. Diets should be based on a variety of common foods in order to provide other nutrients for which human requirements have been less well defined

b Weights and heights of Reference Adults are actual medians for the U.S. population of the designated age, as reported by NHANES II. The median weights and heights of those under 19 years of age were taken from Hamill et al. (1979). The use of these figures does not imply that the height-to-weight ratios are ideal

c Retinol equivalents. 1 RE= 1µg retinol or 6 µg β-carotene

d As cholecalciferol. 10 µg cholecalciferol=400 IU of vitamin D

e α-Tocopherol equivalents. 1mg d-α tocopherol= 1 α-TE

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Table 6-5 *Continued*

Recommended Dietary Allowances, United States[a]

Category Age (Years) or Condition	Vitamin C (mg)	Thiamin (mg)	Ribo-flavin (mg)	Niacin (mg NE)[γ]	Vitamin B_6 (mg)	Folate (µg)	Vitamin B_{12} (µg)	Calcium (µg)	Phos-phorus (mg)	Magne-sium (mg)	Iron (mg)	Zinc (mg)	Iodine (µg)	Sele-nium (µg)
Infants														
0.0-0.5	30	0.3	0.4	5	0.3	25	0.3	400	300	40	6	5	40	10
0.5-1.0	35	0.4	0.5	6	0.6	35	0.5	600	500	60	10	5	50	15
Children														
1-3	40	0.7	0.8	9	1.0	50	0.7	800	800	80	10	10	70	20
4-6	45	0.9	1.1	12	1.1	75	1.0	800	800	120	10	10	90	20
7-10	45	1.0	1.2	13	1.4	100	1.4	800	800	170	10	10	120	30
Males														
11-14	50	1.3	1.5	17	1.7	150	2.0	1200	1200	270	12	15	150	40
15-18	60	1.5	1.8	20	2.0	200	2.0	1200	1200	400	12	15	150	50
19-24	60	1.5	1.7	19	2.0	200	2.0	1200	1200	350	10	15	150	70
25-50	60	1.5	1.7	19	2.0	200	2.0	800	800	350	10	15	150	70
51+	60	1.2	1.4	15	2.0	200	2.0	800	800	350	10	15	150	70

Table 6-5 *Continued*

Recommended Dietary Allowances, United States[a]

Category Age (Years) or Condition	Vitamin C (mg)	Thiamin (mg)	Ribo- flavin (mg)	Niacin (mg NE)[f]	Vitamin B6 (mg)	Folate (µg)	Vitamin B12 (µg)	Calcium (µg)	Phos- phorus (mg)	Magne- sium (mg)	Iron (mg)	Zinc (mg)	Iodine (µg)	Sele- nium (µg)
Females														
11-14	50	1.1	1.3	15	1.4	150	2.0	1200	1200	280	15	12	150	45
15-18	60	1.1	1.3	15	1.5	180	2.0	1200	1200	300	15	12	150	50
19-24	60	1.1	1.3	15	1.6	180	2.0	1200	1200	280	15	12	150	55
25-50	60	1.1	1.3	15	1.6	180	2.0	800	800	280	15	12	150	55
51+	60	1.0	1.2	13	1.6	180	2.0	800	800	280	10	12	150	55
Pregnant	70	1.5	1.6	17	2.2	400	2.2	1200	1200	300	30	15	175	65
Lactating														
1st 6 months	95	1.6	1.8	20	2.1	280	2.6	1200	1200	355	15	19	200	75
2nd 6 months	90	1.6	1.7	20	2.1	260	2.6	1200	1200	340	15	16	200	75

f 1 NE (niacin equivalent) is equal to 1 mg of niacin or 60 mg of dietary tryptophan.

Unlike the recommendations for nutrients, energy intake recommendations are based on the average needs of a population group of average height and weight, and activity level (Tables 6-6, 6-7, 6-8 and 6-9). A margin of safety is not included in view of the undesirability of excessive or inadequate energy intakes.

Recommended nutrient intakes for infants are based on the nutrient content of human milk from healthy women. Although the elderly may have altered requirements for some nutrients, such as minerals due to impairment of their intestinal absorption, there is no evidence that an increased intake of nutrients above the recommended intakes is necessary.

There are a number of important limitations to dietary standards. For example, the values are not based on studies of the entire population. Of necessity only a limited number of individuals have been studied to define the average need and variability of requirement for a particular nutrient; yet these values form the basic data used to set standards for 5 billion people (FAO/WHO).

Levels of recommended intakes have not been set for every nutrient known to be essential to man. In 1989, the United States developed guidelines as to the estimated safe and adequate dietary intake of selected vitamins and minerals (Table 6-10); these values were not expressed as recommended daily allowances due to insufficient data. Some of these nutrients have not been included in standards of other groups such as the FAO/WHO, Canada, Australia and the United Kingdom.

Table 6-6

Energy Expenditure Of a 65-Kg Reference Man
Distributed Over 24 Hours and Effect of Occupation

Distribution of activity	Light activity		Moderately active		Very active		Exceptionally active	
	kilo-calories	mega-joules	kilo-calories	mega-joules	kilo-calories	mega-joules	kilo-calories	mega-joules
In bed (8 hours)	500	2.1	500	2.1	500	2.1	500	2.1
At work (8 hours)	1100	4.6	1400	5.8	1900	8.0	2400	10.0
Non-occupational activities (8 hours)	700 1500	3.0 6.3	700 1500	3.0 6.3	700 1500	3.0 6.3	700 1500	3.0 6.3
Range of energy expenditure (24 hours)	2300 3100	9.7 13.0	2600 3400	10.9 14.2	3100 3900	13.0 16.3	3600 4400	15.1 18.4
Mean (24 hours)	2700	11.3	3000	12.5	3500	14.6	4000	16.7
Mean (per kg of body weight)	42	0.17	46	0.19	54	0.23	62	0.26

Source: FAO/WHO Handbook of Human Nutritional Requirement.
FAO Nutritional Studies, No. 28, Rome,1980.

Table 6-7

Energy Expenditure Of a 55-Kg Reference Woman
Distributed Over 24 Hours and Effect of Occupation

Distribution of activity	Light activity		Moderately active		Very active		Exceptionally active	
	kilo-calories	mega-joules	kilo-calories	mega-joules	kilo-calories	mega-joules	kilo-calories	mega-joules
In bed (8 hours)	420	1.81	420	1.8	420	1.8	420	1.8
At work (8 hours)	800	3.3	1000	4.2	1400	5.9	1800	7.5
Non-occupational activities (8 hours)	580 980	2.4 4.1	580 980	2.4 4.1	580 980	2.4 4.1	580 980	2.4 4.1
Range of energy expenditure (24 hours)	1800 2200	7.5 9.2	2000 2400	8.4 10.1	2400 2700	10.1 11.8	2800 3200	11.7 13.4
Mean (24 hours)	2000	8.4	2200	9.2	2600	10.9	3000	12.5
Mean (per kg of body weight)	36	0.15	40	0.17	47	0.20	55	0.23

Source: FAO/WHO Handbook of Human Nutritional Requirement.
FAO Nutritional Studies, No. 28, Rome,1980.

Table 6-8

Median Heights and Weights and Recommended Energy Intake
For the U.S. Population

Category	Age (years) or Condition	Weight (kg)	Weight (lb)	Height (cm)	Height (in)	Average Energy Allowance (kcal)[a] (per kg)	Average Energy Allowance (kcal)[a] (per day)[b]
Infants							
	0.0-0.5	6	13	60	24	108	650
	0.5-1.0	9	20	71	28	98	850
Children							
	1-3	13	29	90	35	102	1300
	4-6	20	44	112	44	90	1800
	7-10	28	62	132	52	70	2000
Males							
	11-14	45	99	157	62	55	2500
	15-18	66	145	176	69	45	3000
	19-24	72	160	177	70	40	2900
	25-50	79	174	176	70	37	2900
	51+	77	170	173	68	30	2300
Females							
	11-14	46	101	157	62	47	2200
	15-18	55	120	163	64	40	2200
	19-24	58	128	164	65	38	2200
	25-50	63	138	163	64	36	2200
	51+	65	143	160	63	30	1900
Pregnant							
	1st trimester						+0
	2nd trimester						+300
	3rd trimester						+300
Lactating							
	1st 6 months						+500
	2nd 6 months						+500

[a] In the range of light to moderate activity, coefficient of variation is ± 20%
[b] Figure is rounded

Source: Recommended Dietary Allowances, 10th Ed. Food and Nutrition Board,
        National Academy of Sciences, National Research Council, Washington, D.C.,
        National Academy Press, 1989, p.33.

Table 6-9

Average Energy Requirements of Canadians

Age	Gender	Weight (kg)	Requirements (kcal/kg)	(Mj/kg)	(kcal/day)
Months					
0-2	Both	4.5	120-100[a]	0.50-0.42	500
3-5	Both	7.0	100-95	0.42-0.40	700
6-8	Both	8.5	95-97	0.40-0.41	800
9-11	Both	9.5	97-99	0.41	950
Years					
1	Both	11	101	0.42	1100
2-3	Both	14	94	0.39	1300
4-6	Both	18	100	0.42	1800
7-9	M	25	88	0.37	2200
	F	25	76	0.32	1900
10-12	M	34	73	0.30	2500
	F	36	61	0.25	2200
13-15	M	50	57	0.24	2800
	F	48	46	0.19	2200
16-18	M	62	51	0.21	3200
	F	53	40	0.17	2100
19-24	M	71	42	0.18	3000
	F	58	36	0.15	2100
25-49	M	74	36	0.15	2700
	F	59	32	0.13	1900
50-74	M	73	31	0.13	2300
	F	63	29	0.12	1800
75+	M	69	29	0.12	2000
	F	64	23	0.10	1500

[a] First and Second figures are averages at the beginning and at the end of the period.

Table 6-10

Estimated Safe and Adequate Daily Dietary Intakes of Selected Vitamins and Minerals[a]

Category	Age (years)	Vitamins Biotin (µg)	Pantothenic Acid (mg)
Infants			
	0-0.5	10	2
	0.5-1	15	3
Children and adolescents			
	1-3	20	3
	4-6	25	3-4
	7-10	30	4-5
	11+	30-100	4-7
Adults			
		30-100	4-7

Category	Age (years)	Trace Elements[b] Copper (mg)	Manganese (mg)	Fluoride (mg)	Chromium (µg)	Molybdenum (µg)
Infants						
	0-0.5	0.4-0.6	0.3-0.6	0.1-0.5	10-40	15-30
	0.5-1	0.6-0.7	0.6-1.0	0.2-1.0	20-60	20-40
Children and adolescents						
	1-3	0.7-1.0	1.0-1.5	0.5-1.5	20-80	25-50
	4-6	1.0-1.5	1.5-2.0	1.0-2.5	30-120	30-75
	7-10	1.0-2.0	2.0-3.0	1.5-2.5	50-200	50-150
	11+	1.5-2.5	2.0-5.0	1.5-2.5	50-200	75-250
Adults						
		1.5-3.0	2.0-5.0	1.5-4.0	50-200	75-250

[a] Because there is less information on which to base allowances, these figures are not given in the main table of RDA and are provided here in the form of ranges of recommended intakes

[b] Since the toxic levels for many trace elements may be only several times usual intakes, the upper levels for the trace elements given in this table should not be habitually exceeded.

Source: Recommended Dietary Allowances, 10th Ed., Food and Nutrition Board, National Research Council. Washington, D.C., National Academy Press, 1989, p. 284.

Dietary Reference Intakes

The dietary reference intakes (DRI) are a set of at least four nutrient-based reference values that are now being developed for use in planning and assessing diets and for many other purposes. The DRI which are developed with the joint efforts of the Standing Committee on the Scientific Evaluation of Dietary Reference Intakes of the Food and Nutrition Board, Institute of Medicine, National Academy of Sciences, USA and Health Canada, are intended to replace the Recommended Dietary Allowances (RDA) and Recommended Nutrient Intakes for Canadians (RNIC). DRIs encompass the Estimated Average Requirement (EAR), the Recommended Dietary Allowance (RDA), the Adequate Intake (AI), and the Tolerable Upper Level (UL). EAR is the nutrient intake value that meets the requirement of a particular nutrient, in 50% of the individuals at a particular life stage and gender group. RDA is the nutrient intake value sufficient to meet the requirement of a particular nutrient, in nearly all (97%) of the individuals at a particular life stage and gender group. EAR forms the basis of setting the RDA. If the variation in the requirement is well defined, RDA is set at 2 standard deviations above the EAR (RDA = EAR + 2SD$_{EAR}$). On the other hand, if there are inconsistencies in the reported SDs or if sufficient data are not available, a 10% CV is assumed (RDA = 1.2 x EAR). If the scientific data are insufficient for a specific life stage group to set an EAR and therefore an RDA, the AI is used instead. Thus AI is the average observed or experimentally derived nutrient intake by a defined group. The UL is the safe maximum intake of a nutrient for almost all individuals in a specified life-stage and gender group. Tables 6-11 and 6-12 show the new DRI values for the USA and Canada for nutrients and antioxidants respectively evaluated up to this point. The equations for calculating energy needs of the various age groups are shown at the end of Table 6-11. The DRI report for energy and the macronutrients also included Acceptable Macronutrient Distribution Ranges (AMDR). These ranges are shown in Table 6-13. As the DRIs become available they will supercede the Canadian and American standards in Table 6-4 and Table 6-5. It is unclear how they will impact nutrient labeling of food products.

Table 6-11

Dietary Reference Intakes: Recommended Levels for Individual Intake

Life-stage Group	Calcium (mg/d)	Phosphorus (mg/d)	Magnesium (mg/d)	Vitamin D (µg/d)[a,b]	Fluoride (mg/d)	Thiamin (mg/d)	Riboflavin (mg/d)	Niacin (mg/d)[c]	Vitamin B6 (mg/d)	Folate (µg/d)[d]	Vitamin B12 (µg/d)	Pantothenic acid (µg/d)	Biotin (µg/d)	Choline (mg/d)[e]
Infants														
0-6mo	210*	100*	30*	5*	0.01*	0.2*	0.3*	2*	0.1*	65*	0.4*	1.7*	5*	125*
7-12mo	270*	275*	75*	5*	0.5*	0.3*	0.4*	4*	0.3*	80*	0.5*	1.8*	6*	150*
Children														
1-3y	500*	460	80	5*	0.7*	0.5	0.5	6	0.5	150	0.9	2*	8*	200*
4-8y	800*	500	130	5*	1*	0.6	0.6	8	0.6	200	1.2	3*	12*	250*
Males														
9-13y	1300*	1250	240	5*	2*	0.9	0.9	12	1.0	300	1.8	4*	20*	375*
14-18y	1300*	1250	410	5*	3*	1.2	1.3	16	1.3	400	2.4	5*	25*	550*
19-30y	1000*	700	400	5*	4*	1.2	1.3	16	1.3	400	2.4	5*	30*	550*
31-50y	1000*	700	420	5*	4*	1.2	1.3	16	1.3	400	2.4	5*	30*	550*
51-70y	1200*	700	420	10*	4*	1.2	1.3	16	1.7	400	2.4[i]	5*	30*	550*
>70	1200*	700	420	15*	4*	1.2	1.3	16	1.7	400	2.4[i]	5*	30*	550*

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Table 6-11 *Continued*

Dietary Reference Intakes: Recommended Levels for Individual Intake

Life-stage Group	Calcium (mg/d)	Phosphorus (mg/d)	Magnesium (mg/d)	Vitamin D (µg/d)[a,b]	Fluoride (mg/d)	Thiamin (mg/d)	Riboflavin (mg/d)	Niacin (mg/d)[c]	Vitamin B6 (mg/d)	Folate (µg/d)[d]	Vitamin B12 (µg/d)	Pantothenic acid (µg/d)	Biotin (µg/d)	Choline (mg/d)[e]
Females														
9-13y	1300*	1250	240	5*	2*	0.9	0.9	12	1.0	300	1.8	4*	20*	375*
14-18y	1300*	1250	360	5*	3*	1.0	1.0	14	1.2	400[g]	2.4	5*	25*	400*
19-30y	1000*	700	310	5*	3*	1.1	1.1	14	1.3	400[g]	2.4	5*	30*	425*
31-50y	1000*	700	320	5*	3*	1.1	1.1	14	1.3	400[g]	2.4	5*	30*	425*
51-70y	1200*	700	320	10*	3*	1.1	1.1	14	1.5	400	2.4[i]	5*	30*	425*
>70	1200*	700	320	15*	3*	1.1	1.1	14	1.5	400	2.4[i]	5*	30*	425*
Pregnancy														
≤18y	1300*	1250	400	5*	3*	1.4	1.4	18	1.9	600[h]	2.6	6*	30*	450*
19-30y	1000*	700	350	5*	3*	1.4	1.4	18	1.9	600[h]	2.6	6*	30*	450*
31-50y	1000*	700	360	5*	3*	1.4	1.4	18	1.9	600[h]	2.6	6*	30*	450*

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Table 6-11 *Continued*

Dietary Reference Intakes: Recommended Levels for Individual Intake

Life-stage Group	Calcium (mg/d)	Phos- phorus (mg/d)	Magne- sium (mg/d)	Vitamin D (µg/d)[a,b]	Fluoride (mg/d)	Thiamin (mg/d)	Ribo- flavin (mg/d)	Niacin (mg/d)[c]	Vitamin B6 (mg/d)	Folate (µg/d)[d]	Vitamin B12 (µg/d)	Pantothenic acid (µg/d)	Biotin (µg/d)	Choline (mg/d)[e]
Lactation														
≤18y	1300*	1250	360	5*	3*	1.4	1.6	17	2.0	500	2.8	7*	35*	550*
19-30y	1000*	700	310	5*	3*	1.4	1.6	17	2.0	500	2.8	7*	35*	550*
31-50y	1000*	700	320	5*	3*	1.4	1.6	17	2.0	500	2.8	7*	35*	550*

NOTE: This table presents RDAs in **bold type** and AIs in ordinary type followed by an asterisk (*). RDAs and AIs may both be used as goals for individual intake. RDAs are set to meet the needs of almost all (97 to 98%) individuals in a group. For healthy breastfed infants, the AI is the mean intake. The AI for other life stage and gender groups is believed to cover needs of all individuals in the group, but lack of data or uncertainty in the data prevent being able to specify with confidence the percentage of individuals covered by this intake.

a   As cholecalciferol. 1µg cholecalciferol=40 IU vitamin D.
b   In the absence of adequate exposure to sunlight.
c   As niacin equivalents (NE). 1mg of niacin=60mg tryptophan; 0-6mo=preformed niacin (not NE).
d   As dietary folate equivalents (DFE). 1DFE=1µg food folate=0.6µg of folic acid from fortified food as a supplement consumed with food=0.5µg of a supplement taken on empty stomach.
e   Although AIs have been set for choline, there are too few data to assess whether a dietary supply of choline is needed at all stages of the life cycle, and it may be that the choline requirement can be met by endogenous synthesis at some of these stages.
f   Because 10-30% of older people may malabsorb food-bound B12. It is advisable for those older than 50y to meet their RDA mainly by consuming food fortified with B12 or a supplement containing B12.
g   In view of the evidence linking folate intake with NTD in the fetus, it is recommended that all women capable of becoming pregnant consume 400µg from supplements or fortified foods in addition to intake of food folate from a varied diet.
h   It is assumed that women will continue consuming 400µg from supplements or fortified foods until their pregnancy is confirmed and they enter prenatal care, which ordinarily occurs after the end of the periconceptional period-the critical time for formation of neural tube.

Table 6-11 *Continued*

Dietary Reference Intakes: Recommended Levels for Individual Intake

Life-stage Group	Vitamin C (mg/d)	Vitamin E (mg/d)	Selenium (µg/d)	Vitamin A (µg/d)	Vitamin K (µg/d)	Chromium (µg/d)	Copper (µg/d)	Iodine (µg/d)	Iron (mg/d)	Manganese (mg/d)	Molybdenum (µg/d)	Zinc (mg/d)
Infants												
0-6mo	40*	4*	15*	400*	2*	0.2*	200*	110*	0.27*	0.003*	2*	2*
7-12mo	50*	5*	20*	500*	2.5*	5.5*	220*	130*	11	0.6*	3*	3
Children												
1-3y	15	6	20	300	30*	11*	340	90	7	1.2*	17	3
4-8y	25	7	30	400	55*	15*	440	90	10	1.5*	22	5
Males												
9-13y	45	11	40	600	60*	25*	700	120	8	1.9*	34	8
14-18y	75	15	55	900	75*	35*	890	150	11	2.2*	43	11
19-30y	90	15	55	900	120*	35*	900	150	8	2.3*	45	11
31-50y	90	15	55	900	120*	35*	900	150	8	2.3*	45	11
51-70y	90	15	55	900	120*	30*	900	150	8	2.3*	45	11
>70	90	15	55	900	120*	30*	900	150	8	2.3*	45	11

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Table 6-11 *Continued*

Dietary Reference Intakes: Recommended Levels for Individual Intake

Life-stage Group	Vitamin C (mg/d)	Vitamin E (mg/d)	Selenium (µg/d)	Vitamin A (µg/d)	Vitamin K (µg/d)	Chromium (µg/d)	Copper (µg/d)	Iodine (µg/d)	Iron (mg/d)	Manga-nese (mg/d)	Molyb-denum (µg/d)	Zinc (mg/d)
Females												
9-13y	45	11	40	600	60*	21*	700	120	8	1.6*	34	8
14-18y	65	15	55	700	75*	24*	890	150	15	1.6*	43	9
19-30y	75	15	55	700	90*	25*	900	150	18	1.8*	45	8
31-50y	75	15	55	700	90*	25*	900	150	18	1.8*	45	8
51-70y	75	15	55	700	90*	20*	900	150	8	1.8*	45	8
>70	75	15	55	700	90*	20*	900	150	8	1.8*	45	8
Pregnancy												
≤18y	80	15	60	750	75*	29*	1000	220	27	2.0*	50	13
19-30y	85	15	60	770	90*	30*	1000	220	27	2.0*	50	11
31-50y	85	15	60	770	90*	30*	1000	220	27	2.0*	50	11

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Table 6-11 *Continued*

Dietary Reference Intakes: Recommended Levels for Individual Intake

Life-stage Group	Vitamin C (mg/d)	Vitamin E (mg/d)	Selenium (µg/d)	Vitamin A (µg/d)	Vitamin K (µg/d)	Chromium (µg/d)	Copper (µg/d)	Iodine (µg/d)	Iron (mg/d)	Manganese (mg/d)	Molybdenum (µg/d)	Zinc (mg/d)
Lactation												
≤18y	**115**	**19**	**70**	**1200**	75*	44*	**1300**	**290**	**10**	2.6*	**50**	**14**
19-30y	**120**	**19**	**70**	**1300**	90*	45*	**1300**	**290**	**9**	2.6*	**50**	**12**
31-50y	**120**	**19**	**70**	**1300**	90*	45*	**1300**	**290**	**9**	2.6*	**50**	**12**

NOTE: This table presents RDAs in **bold type** and AIs in ordinary type followed by an asterisk (*). RDAs and AIs may both be used as goals for individual intake. RDAs are set to meet the needs of almost all (97 to 98%) individuals in a group. For healthy breastfed infants, the AI is the mean intake. The AI for other life stage and gender groups is believed to cover needs of all individuals in the group, but lack of data or uncertainty in the data prevent being able to specify with confidence the percentage of individuals covered by this intake.

Sources: Institute of Medicine. Dietary Reference Intakes for Vitamin A, Vitamin K, Arsenic, Boron, Chromium, Copper, Iodine, Iron, Molybdenum, Nickel, Silicon, Vanadium and Zinc. Food and Nutrition Board. Washington, DC: National Academy Press, 2001.

Institute of Medicine. Dietary Reference Intakes for Calcium, Phosphorus, Magnesium, Vitamin D, and Fluoride. Food and Nutrition Board. Washington, DC: National Academy Press, 1997.

Institute of Medicine. Dietary Reference Intakes for Thiamin, Riboflavin, Niacin, Vitamin B6, Folate, Vitamin B12, Pantothenic Acid, Biotin, and Choline. Food and Nutrition Board. Washington, DC: National Academy Press, 1998.

Institute of Medicine. Dietary Reference Intakes for Vitamin C, Vitamin E, Selenium, and Carotenoids. Food and Nutrition Board. Washington, DC: National Academy Press, 2000.

Table 6-11 *Continued*

Dietary Reference Intakes: Recommended Levels for Individual Intake

Life-stage Group	Protein (g/d)	Total Fat (g/d)	Saturated Fat (g/d)	Trans fat (g/d)	Mono-unsaturated Fat (g/d)	Poly-unsaturated Fat (g/d)	α-Linolenic Acid (g/d)	Linoleic Acid	Cholestrol (mg/d)	Carbo-hydrate (g/d)	Added Sugar[b] (g/d)	Dietary Fiber (g/d)
Infants												
0-6mo	9.1	31	ND	ND	ND	ND	0.5*	4.4*	ND	60*	ND	ND
7-12mo	13.5	30	ND	ND	ND	ND	0.5*	4.6*	ND	95*	ND	ND
Children												
1-3y	13	ND[a]	ND	ND	ND	ND	0.7*	7*	ND	130	ND	19*
4-8y	19	ND	ND	ND	ND	ND	0.9*	10*	ND	130	ND	25*
Males												
9-13y	34	ND	ND	ND	ND	ND	1.2*	12*	ND	130	ND	31*
14-18y	52	ND	ND	ND	ND	ND	1.6*	16*	ND	130	ND	38*
19-30y	56	ND	ND	ND	ND	ND	1.6*	17*	ND	130	ND	38*
31-50y	56	ND	ND	ND	ND	ND	1.6*	17*	ND	130	ND	38*
51-70y	56	ND	ND	ND	ND	ND	1.6*	14*	ND	130	ND	30*
>70	56	ND	ND	ND	ND	ND	1.6*	14*	ND	130	ND	30*

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Table 6-11 *Continued*

Dietary Reference Intakes: Recommended Levels for Individual Intake

Life-stage Group	Protein (g/d)	Total Fat (g/d)	Saturated Fat (g/d)	Trans fat (g/d)	Mono-unsaturated Fat (g/d)	Poly-unsaturated Fat (g/d)	α-Linolenic Acid (g/d)	Linoleic Acid	Cholestrol (mg/d)	Carbo-hydrate (g/d)	Added Sugar[b] (g/d)	Dietary Fiber (g/d)
Females												
9-13y	**34**	ND	ND	ND	ND	ND	1*	10*	ND	**130**	ND	26*
14-18y	**46**	ND	ND	ND	ND	ND	1.1*	11*	ND	**130**	ND	36*
19-30y	**46**	ND	ND	ND	ND	ND	1.1*	12*	ND	**130**	ND	25*
31-50y	**46**	ND	ND	ND	ND	ND	1.1*	12*	ND	**130**	ND	25*
51-70y	**46**	ND	ND	ND	ND	ND	1.1*	11*	ND	**130**	ND	21*
>70	**46**	ND	ND	ND	ND	ND	1.1*	11*	ND	**130**	ND	21*
Pregnancy												
≤18y	**+25**	ND	ND	ND	ND	ND	1.4*	13*	ND	**175**	ND	28*
19-30y	**+25**	ND	ND	ND	ND	ND	1.4*	13*	ND	**175**	ND	28*
31-50y	**+25**	ND	ND	ND	ND	ND	1.4*	13*	ND	**175**	ND	28*

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Table 6-11 *Continued*

Dietary Reference Intakes: Recommended Levels for Individual Intake

Life-stage Group	Protein (g/d)	Total Fat (g/d)	Saturated Fat (g/d)	Trans fat (g/d)	Mono-unsaturated Fat (g/d)	Poly-unsaturated Fat (g/d)	α-Linolenic Acid (g/d)	Linoleic Acid	Cholestrol (mg/d)	Carbo-hydrate (g/d)	Added Sugar[b] (g/d)	Dietary Fiber (g/d)
Lactation												
≤18y	**+25**	ND	ND	ND	ND	ND	1.3*	13*	ND	**210**	ND	29*
19-30y	**+25**	ND	ND	ND	ND	ND	1.3*	13*	ND	**210**	ND	29*
31-50y	**+25**	ND	ND	ND	ND	ND	1.3*	13*	ND	**210**	ND	29*

NOTE: This table presents RDAs in **bold type** and AIs in ordinary type followed by an asterisk (*). RDAs and AIs may both be used as goals for individual intake. RDAs are set to meet the needs of almost all (97 to 98%) individuals in a group. For healthy breastfed infants, the AI is the mean intake. The AI for other life stage and gender groups is believed to cover needs of all individuals in the group, but lack of data or uncertainty in the data prevent being able to specify with confidence the percentage of individuals covered by this intake.

a   ND=Not Determined
b   For adults and children ≥ 1year, added sugars should supply 25% or less of total calories

Source: Dietary Reference Intakes for Energy, Carbohydrates, Fiber, Fat, Protein and Amino Acids (Macronutrients). Food and Nutrition Board, Institute of Medicine, 2002.
http://www.nap.edu/books/0309085373/html/index.html

Table 6-11 *Continued*

Dietary Reference Intakes: Estimated Energy Requirements (EER)

Life-stage Group	Estimated Energy Requirements (EER)
Infants	
0-3 mo	(89 x weight of infant [kg] – 100) + 175 (kcal for Energy Deposition)
4-6 mo	(89 x weight of infant [kg] – 100) + 56 (kcal for Energy Deposition)
7-12mo	(89 x weight of infant [kg] – 100) + 22 (kcal for Energy Deposition)
13-35 mo	(89 x weight of child [kg] – 100) + 20 (kcal for Energy Deposition)
Males	
3-8y	88.5 – 61.9 x Age [y] + PA x (26.7 x Weight [kg] + 903 x Height [m]) + 20 (kcal for Energy Deposition)
	PA^a = 1.00 if sedentary; 1.13 if Low Active; 1.26 if Active; 1.42 if Very Active
9-18y	88.5 – 61.9 x Age [y] + PA x (26.7 x Weight [kg] + 903 x Height [m]) + 25 (kcal for Energy Deposition)
	PA = 1.00 if sedentary; 1.13 if Low Active; 1.26 if Active; 1.42 if Very Active
19+y	662 – 9.53 x Age [y] +PA x (15.9 x Weight [kg] + 539.6 x Height [m]
	PA = 1.00 if sedentary; 1.11 if Low Active; 1.25 if Active; 1.48 if Very Active
Females	
3-8y	135.3 – 30.8 x Age [y] + PA x (10.0 x Weight [kg] + 934 x Height [m] + 20 (kcal for Energy Deposition)
	PA = 1.00 if Sedentary; 1.16 if Low Active; 1.31 if Active; 1.56 if Very Active
9-18y	135.3 – 30.8 x Age [y] + PA x (10.0 x Weight [kg] + 934 x Height [m] + 25 (kcal for Energy Deposition)
	PA = 1.00 if Sedentary; 1.16 if Low Active; 1.31 if Active; 1.56 if Very Active
19+y	354 – 6.91 x Age [y] + PA x (9.36 x Weight [kg] + 726 x Height [m])
	PA = 1.00 if Sedentary; 1.12 if Low Active; 1.27 if Active; 1.45 if Very Active

Table 6-11 *Continued*

Dietary Reference Intakes: Estimated Energy Requirements (EER)

Life-stage Group	Estimated Energy Requirements (EER)
Pregnancy	
14-18y	1st trimester = Adolescent EER + 0 (Pregnancy Energy Deposition)
	2nd trimester = Adolescent EER + 160 kcal (8kcal/wk x 20 wks) + 180 kcal
	3rd trimester = Adolescent EER + 272 (8 kcal/wk x 34 wks) + 180
19-50y	1st trimester = Adult EER + 0 (Pregnancy Energy Deposition)
	2nd trimester = Adult EER + 160 kcal (8 kcal/wk x 20 wks + 180 kcal
	3rd trimester = Adult EER + 272 kcal (8ckal/wk x 34 wks) + 180 kcal
Lactation	
14-18y	1st 6 months = Adolescent EER + 500 – 170 (Milk Energy Output – Weight Loss)
	2nd 6 months = Adolescent EER + 400 – 0 (Milk Energy Output – Weight Loss)
19-50y	1st 6 months = adult EER + 500 – 170 (Milk Energy Output – Weight Loss)
	2nd 6 months = Adult EER + 400 – 0 (Milk Energy Output – Weight Loss)

a PA = Physical Activity Coefficient

Table 6-12

Tolerable Upper Intake Levels (UL[a]) of Antioxidants by Life Stage Group

Life Stage Group	Vitamin C (mg/d)	α-Tocopherol (mg/d)[b]	Selenium (mg/d)
0 through 6 mo	ND[c]	ND	45
7 through 12 mo	ND	ND	60
1 through 3 y	400	200	90
4 through 8 y	650	300	150
9 through 13 y	1,200	600	280
14 through 18 y	1,800	800	400
19 through 70 y	2,000	1,000	400
> 70 y	2,000	1,000	400
Pregnancy			
≦18 y	1,800	800	400
19 through 50 y	2,000	1,000	400
Lactation			
≦ 18 y	1,800	800	400
19 through 50 y	2,000	1,000	400

a The UL is the highest level of daily nutrient intake that is likely to pose no risk of adverse health effects to almost all individuals in the general population. As intake increases above the UL, the risk of adverse effects increases. Unless specified otherwise, the UL represents total nutrient intake from food, water and supplements.

b The UL for α-tocopherol applies to any form of supplemental α-tocopherol.

c ND. Not determined due to lack of data of adverse effects in this age group and concern with regard to lack of ability to handle excess amounts. Source of intake should be from food and formula in order to prevent high levels of intake.

Source: Food and Nutrition Board, Institute of Medicine, Dietary Reference Intakes for Vitamin C, Vitamin E, Selenium and Carotenoids. National Academy Press, Washington, D.C., 2000.

Table 6-13

Dietary Reference Intakes: Acceptable Macronutrient Distribution Ranges (AMDR)

Age	Protein (%)	Carbohydrate (%)	Fat (%)	α-linolenic Acid (%)	Linoleic Acid (%)
1-3 yrs	5-20	45-65	30-40	0.6-1.2	5-10
4-8 yrs	10-30	45-65	25-35	0.6-1.2	5-10
> 9 yrs	10-35	45-65	20-35	0.6-1.2	5-10

Source: Dietary Reference Intakes for Energy, Carbohydrates, Fiber, Fat, Protein and Amino Acids(Macronutrients). Food and Nutrition Board, Institute of Medicine, 2002. http://www.nap.edu/books/0309085373/html/index.html

Dietary Goals (Guidelines)

Dietary goals were introduced in the mid-1970s to provide guidance to the general public for nutritional choices with the goal of reducing the risk of degenerative diseases. They are sets of nutrition recommendations for the effective planning of food supplies and diets in the prevention of prevalent nutrition-related public health diseases such as cardiovascular disease, hypertension, obesity, cancer, dental caries and diabetes mellitus.

Various countries and organizations have established independent dietary goals. The dietary guidelines for Australia, the United Kingdom, China, Canada and the U.S. are shown in Table 6-14. Most countries have included the following recommendations:

(a) Enjoy a variety of foods/maintain a balanced diet

(b) Maintain ideal weight by reducing excessive energy consumption and increasing physical activity

(c) Reduce fat intake to no more than 30% of total energy with no more than 10% of total energy as saturated fat.

(d) Moderate consumption of alcohol

(e) Limit intake of sugar

(f) Avoid too much salt, and

(g) Increase intake of whole grain cereals, fresh fruit and vegetables

A considerable amount of controversy surrounds some of the dietary goals. Some nutritionists suggest that food selection should be left to the individuals. Reduced intake of salt, fat and sugar do not benefit the healthy population but only those individuals who are susceptible to diseases that require dietary modification of these substances. Canada has dropped the guideline restricting sugar while other countries like Australia and the United Kingdom have retained it.

The degree to which these substances should be restricted is another area of contention. Some dietary guidelines specify the amount of energy to be derived from protein, fat and carbohydrate, and the amount in percentages, by which cholesterol, sugar and fat should be lowered. The levels to which polyunsaturated fat and fiber should be raised are also stated in some dietary guidelines.

Table 6-14

Dietary Guidelines for Australia, United Kingdom, Canada, China and the United States

Dietary Guidelines for Australians, 1992.

FOR ALL ADULTS
1. Enjoy a wide variety of nutritious foods.
2. Eat plenty of breads and cereals (preferably whole grain), vegetables (including legumes) and fruits.
3. Eat a diet low in fat and, in particular, low in saturated fat.
4. Maintain a healthy body weight by balancing physical activity and food intake.
5. If you drink alcohol, limit your intake.
6. Eat only a moderate amount of sugars and foods containing added sugars.
7. Choose low salt foods and use salt sparingly.

FOR SOME ADULTS
8. Encourage and support breastfeeding.
9. Eat foods containing calcium. This is particularly important for girls and women.
10. Eat foods containing iron. This applies particularly to girls, women, vegetarians and athletes.

Dietary Guidelines for Canadian, 1992.

1. Enjoy a variety of foods.
2. Emphasize cereals, breads, other grain products, vegetables and fruit.
3. Choose lower-fat dairy products, leaner meats and foods prepared with little or no fat.
4. Achieve and maintain a healthy body weight by enjoying regular physical activity and healthy eating.
5. Limit salt, alcohol and caffeine.

Dietary Guidelines for Chinese, 1997.

1. Eat a variety of foods with cereals as the staple.
2. Consume plenty of vegetables, fruits and tubers.
3. Consume milk, beans or dairy or bean products every day.
4. Consume appropriate amounts of fish, poultry, eggs and lean meat; reduce fatty meat and animal fat in the diet.
5. Balance food intake with physical activity to maintain healthy weight.
6. Choose a light diet that is also low in salt.
7. If you drink alcoholic beverages, do so in limited amounts.
8. Avoid unsanitary and spoiled foods.

Dietary Guidelines for the United Kingdom, 1991.

1. Enjoy your food.
2. Eat a variety of different foods.
3. Eat the right amount to be a healthy weight.
4. Eat plenty of foods rich in starch and fiber.
5. Eat plenty of fruits and vegetables.
6. Don't eat too many foods that contain a lot of fat.
7. Don't have sugary foods and drinks too often.
8. If you drink alcohol, drink sensibly.

Table 6-14 *Continued*

**Dietary Guidelines for Australia, United Kingdom,
Canada, China and the United States**

Dietary Guidelines for Americans, 2000.

AIM FOR FITNESS
1. Aim for a healthy weight.
2. Be physically active each day.

BUILD A HEALTHY BASE
3. Let the Pyramid guide your food choices.
4. Choose a variety of grains daily, especially whole grains.
5. Choose a variety of fruits and vegetables daily.
6. Keep food safe to eat.

CHOOSE SENSIBLY
7. Choose a diet that is low in saturated fat and cholesterol and moderate
    in total fat.
5. Choose beverages and foods to moderate your intake of sugars.
6. Choose and prepare foods with less salt.
7. If you drink alcoholic beverages, do so in moderation.

Nevertheless, there is good agreement that a well-balanced diet comprised of a variety of foods, weight control, moderation in alcohol consumption and regular exercise are healthy practices for the general population.

Food Guides

Food guides translate dietary standards and dietary guidelines (nutrition recommendations) into foods for use by the general population. All foods provide energy and some essential nutrients. No single food provides enough of all the needed nutrients for normal body functions. Thus, simple food guides have been designed to facilitate the selection of a variety of foods daily, that would provide all the necessary nutrients while minimizing the risk of chronic diseases due to poor dietary habits.

Generally, food guides specify a range and the size of servings of foods for different food groups. A pattern for selecting food is recommended to meet the needs of the general population for energy and essential nutrients. Foods selected according to food guides provide between 1800 and 3200 kilocalories. For additional energy, the number and size of servings should be increased.

Nutrient needs vary throughout the life cycle. To meet these needs for various age groups, the number of servings suggested for each food group also varies.

In the U.S., a Food Guide Pyramid was developed to graphically portray the Dietary Guidelines. The food groups are displayed in bands, with grains and cereals at the wide base, vegetables and fruits in the band above, followed by meat and dairy products and finally, fats and sweets in the narrow peak. The pyramid shape conveys the message that the daily diet should include more servings of grains, fruits and vegetables than of meats, dairy products and fats and sweets.

The food guides for Australia, the United Kingdom, Canada, China and the United States are similar. Canada's Food Guide to Healthy Eating is shown in Table 6-15. Basically, the foods in each food group provide similar nutrients. The food groups are (1) Meat and Alternates, (2) Milk Products, (3) Grain Products and (4) Vegetables and Fruit. For the first time, in 1992, Canada joined the U.S. and Australia by including an "Other Food" category to assist consumers in selecting foods such as butter, oil, sweets and snack foods. This group contains foods and condiments high in fat and simple carbohydrates which complement but do not replace foods from other groups.

Meat and Alternates are the main sources of protein in the diet and may also supply iron, niacin, thiamin, riboflavin, vitamin A, vitamin B, fat and fiber (from legumes). Examples of meat alternates include eggs, dried peas, beans, lentils, nuts and seeds. In some food guides cheese is included as a meat alternate based on its protein content. (cheese is also a source of calcium and is found in the Milk Products group). Milk Products are the main dietary sources of calcium and riboflavin. They also supply protein, fat, niacin, folacin, vitamin A and vitamin B_2. Examples of milk products include cheese, yogurt, buttermilk, milk desserts and ice cream. In some countries fluid milk is fortified with vitamin D and serves as the major dietary source of vitamin D. Fortification of skim and 2% milk with vitamin A is also required in some countries so that the vitamin A content of all fluid milk is comparable. It is important to be aware of food fortification practices and policies of countries throughout the world.

Grain Products serve as sources of carbohydrate, protein, thiamin, niacin and fiber. If enriched, they will also supply riboflavin and iron. Examples of the Grain Products food group include rice, barley, oats, corn, potato and wheat. Whole grain products are often promoted in food guides as they supply fiber and additional amounts of minerals.

Vegetables and Fruit are important as sources of vitamin A and Vitamin C. They also supply carbohydrate, thiamin, folate, iron and fiber. Leafy green and yellow vegetables have higher amounts of vitamin A, iron and folate than fruits. Specific recommendations for the daily intake of these vegetables are often included in food guides.

Table 6-15

Major Nutrient Contributions of the Basic Food Groups used in Food Guides

Group	Food Included	Major Nutrient Provided	
GRAIN PRODUCTS	Whole grain and enriched	Protein	Niacin
		Carbohydrate	Folacin
		Fibre	Iron
		Thiamin	Zinc
		Riboflavin	Magnesium
VEGETABLES & FRUIT	Fruits and vegetables	Carbohydrate	Vitamin C
		Fibre	Vitamin A
		Thiamin	Iron
		Folacin	Magnesium
MILK PRODUCTS	Milk, yogurt, other milk products except butter	Protein	Vitamin D
		Fat	Calcium
		Riboflavin	Zinc
		Vitamin B_{12}	Magnesium
		Vitamin A	
MEAT & ALTERNATIVES	Muscle meats (veal, beef, pork, lamb, mutton, venison), fish, poultry, eggs, dried beans and peas	Protein	Folacin
		Fat	Vitamin B_{12}
		Thiamin	Iron
		Riboflavin	Zinc
		Niacin	Magnesium

Countries such as Australia, Canada, New Zealand, United Kingdom, United States, and others have adopted a "5-a-day" concept for eating fruits and vegetables. This concept recommends at least 5 servings of fruits and vegetables per day. Choosing different colored fruits and vegetables daily is in line with general food guide recommendations. A variety of nutrients is ensured with this approach. As well, phytochemicals that have various health benefits are obtained (Refer to Chapter 8).

Some food guides recommend moderate consumption of fat, sugar, salt and alcohol. Moderation means cutting down, not cutting out, and varies from individual to individual. The recommendation to limit consumption of the above list of foods is to reduce the risk of health problems such as cardiovascular disease, dental caries, diabetes mellitus, obesity and hypertension.

Suggested Readings
Australia

National Health and Medical Research Council. **Recommended Dietary Intakes for Use in Australia**. Australian Government Publishing Service, Canberra. 1991.

National Health and Medical Research Council. Dietary Guidelines for Australians. Australian Government Publishing Service, Canberra, 1992.

Canada

Food and Nutrition Board, Institute of Medicine, **Dietary Reference Intakes: A Risk Assessment Model for Establishing Upper Intake Levels for Nutrients**. National Academy press, Washington, D.C., 1998.

Food and Nutrition Board, Institute of Medicine, **Dietary Reference Intakes for Vitamin C, Vitamin E, Selenium and Carotenoids**. National Academy press, Washington, D.C., 2000.

Health and Welfare Canada: **Nutritional Recommendations**. The Report of the Scientific Review Committee., Supply and Services Canada, Ottawa, 1990.

Health and Welfare Canada. **Canada's Food Guide to Healthy Eating**. Supply and Services, Canada, 1992.

China

The Chinese Nutrition Society. **Dietary Guidelines and the Food Pagoda for Chinese Residents**: Balanced Diet, Rational Nutrition, and Health Promotion. Nutrition Today 34:106-115, 1999.

United Kingdom

Health Education Authority. **Eight guidelines for a healthy diet**: A guide for nutrition educators. Abingdon. Health Education Authority (in association with the Ministry of Agriculture, Fisheries and Food and the Department of Health).

Report on Health and Social Subjects: **No. 41, Dietary Reference Values for Food Energy and Nutrients for the United Kingdom**, Report of the Panel on Dietary Reference Values of the Committee on Medical Aspects of Food Policy. Her Majesty's Stationery Office, London, 1991.

United States

Food and Nutrition Board, Institute of Medicine, **Dietary Reference Intakes: A Risk Assessment Model for Establishing Upper Intake Levels for Nutrients**. National Academy press, Washington, D.C., 1998.

Food and Nutrition Board, Institute of Medicine, **Dietary Reference Intakes for Vitamin C, Vitamin E, Selenium and Carotenoids**. National Academy press, Washington, D.C., 2000.

Food and Nutrition Board, National Academy of Sciences, National Research Council, **Recommended Dietary Allowances, 10th Ed.** National Academy press, Washington, D.C., 1989.

USDA: **2000 Dietary Guidelines for Americans**, Human Nutrition Information Service, Hyattsville, MD, 2000. (http://www.usda.gov/cnpp).

CHAPTER 7

GENERAL NUTRITION

In the previous chapters, we discussed the basic aspects of nutritional biochemistry, metabolism and guidelines for food selection in order to achieve a balanced diet and to maintain good health. This section will focus on the special nutritional requirements of infants, the elderly population and athletes.

Infant Feeding
Infancy is a time of rapid growth for the body and individual organ systems, and for physical, physiological and mental development. It is also when the individual begins to develop eating habits that may help determine life-long nutritional status. It is therefore important to provide infants with nutritionally balanced diets to meet demands for their rapid physiological development and to form a firm foundation for good life-long eating habits.

During the first two years of life, the infant's diet changes from an exclusively milk-based diet to one that includes strained solid foods, to a diet consisting of foods of coarser consistency and finally to one that is more or less the same as the rest of the family's. This progression of dietary changes necessarily corresponds to the physical growth and physiological development of the alimentary system and internal organs.

Infant feeding is an art, as well as a science. It must recognize that infants are individuals having individual growth patterns, nutritional needs and food preferences. It must also recognize that the nutritional interaction between the infant and parents can have a significant impact on the infant's psychosocial development.

Growth
The objective of nutrition during the formative years is to achieve optimal health, growth and development. An individual's growth pattern and development are determined by an interplay of hereditary and environmental factors. Nevertheless the normal child will fall within the pattern of growth for all normal children.

An infant's birth weight doubles in the first 4 months and triples by the end of the first year. During the first 4 to 6 months, almost one third of the infant's energy requirement is used for growth. The limitations imposed by the immature gastrointestinal, renal and immune systems of the newborn place unique demands on the food source to supply all the essential nutrients for such rapid growth.

Figure 7-1

CDC Growth Charts: United States

Weight-for-age percentiles: Girls, birth to 36 months

Source: Developed by the National Center for Health Statistics in collaboration with the National Center for Chronic Disease Prevention and Health Promotion (2000).
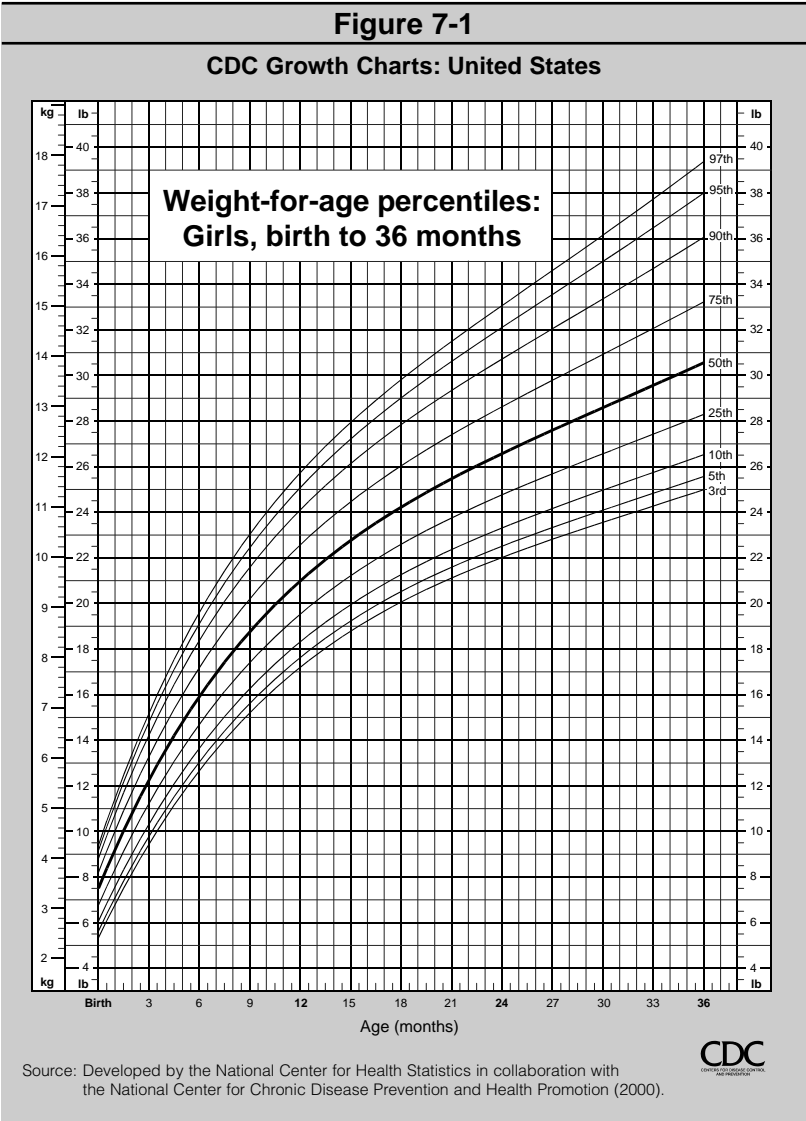
The best means for assessing physical growth and therefore the nutritional adequacy of the diet is to monitor the gain in weight and length of the rapidly growing infant. These physical measurements are usually interpreted by reference to available standards such as the National Center for Health Statistics (NCHS) growth charts presented in Figures 7-1 to 7-4. Significant deviation, upwards or downwards, from normal ranges indicates overnutrition or undernutrition. For example, the degree of retardation of growth due to malnutrition depends on the degree and

Figure 7-2

CDC Growth Charts: United States

Length-for-age percentiles: Girls, birth to 36 months

Source: Developed by the National Center for Health Statistics in collaboration with the National Center for Chronic Disease Prevention and Health Promotion (2000).
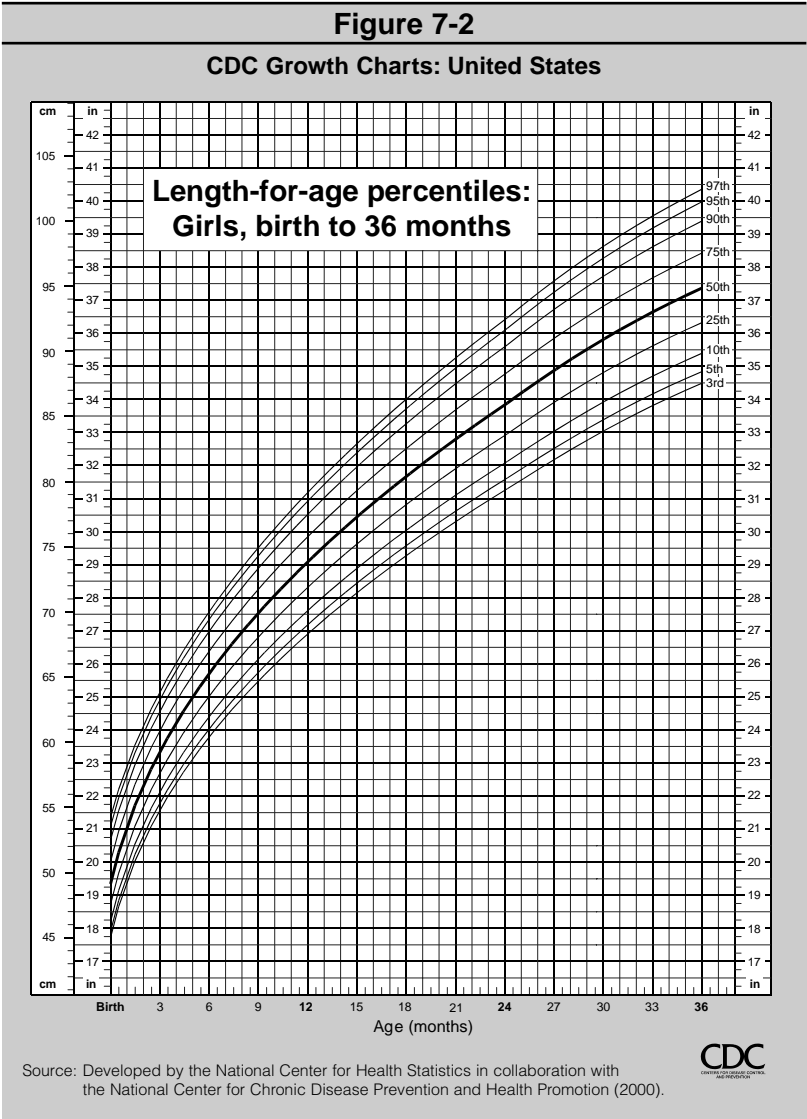
duration of energy or nutrient deprivation. A marked spurt in growth usually occurs when adequate amounts of food are again provided after a period of insufficient food.

MILK FEEDING
Breastmilk
Breastmilk is the best source of nutrition for young infants. Nutritionally, breastmilk (both colostrum and mature breastmilk) contains all the essential nutrients in amounts, with the possible

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Figure 7-3

CDC Growth Charts: United States

Weight-for-age percentiles:
Boys, birth to 36 months

Age (months)

97th
95th
90th
75th
50th
25th
10th
5th
3rd

Source: Developed by the National Center for Health Statistics in collaboration with the National Center for Chronic Disease Prevention and Health Promotion (2000).

CDC
CENTERS FOR DISEASE CONTROL AND PREVENTION

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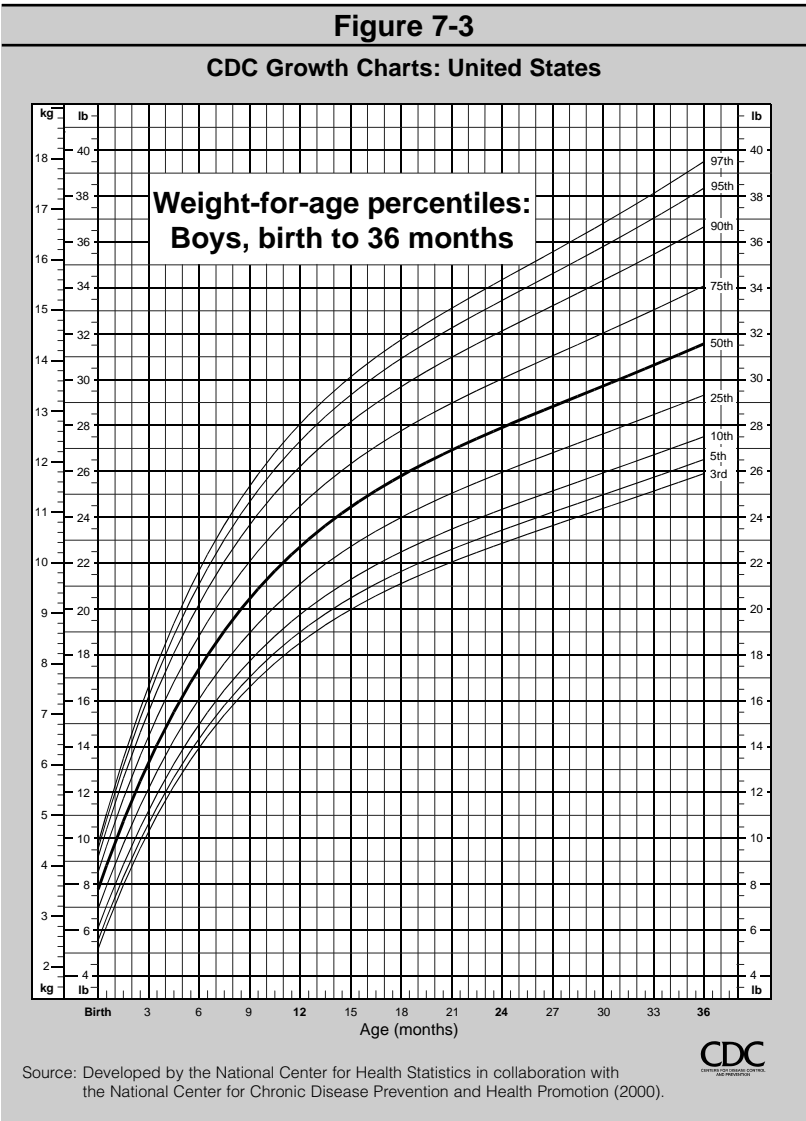
exception of vitamin D, necessary for optimal growth (Table 7-1). Exclusive breast feeding is recommended from birth to about 6 months of age, with prolonged feeding being encouraged for as long as possible. Complementary foods may be introduced from about 6 months of age. Exclusively breast fed infants follow a slower but normal growth pattern than bottle fed infants at around 4 to 6 months of age and may therefore require a separate growth curve. The difference in size of breast and bottle fed infants tends to disappear when solid foods are introduced on a regular basis.

Figure 7-4

CDC Growth Charts: United States

Length-for-age percentiles: Boys, birth to 36 months

Source: Developed by the National Center for Health Statistics in collaboration with the National Center for Chronic Disease Prevention and Health Promotion (2000).
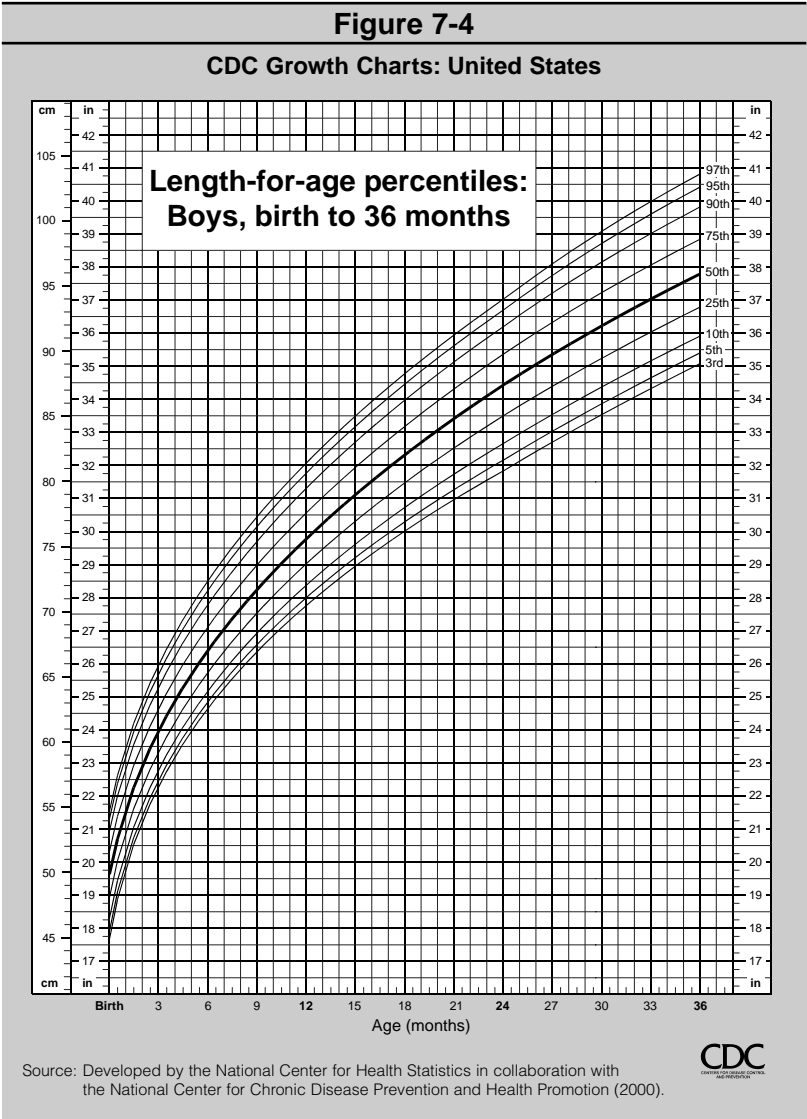
Table 7-1

Composition of Mature Breastmilk

Composition	Amount
Gross composition (% w/w)	
Water	87.5
Calories	69.6
Protein	1.0
Fat	4.4
Carbohydrate (lactose)	6.9
Vitamin (water-soluble) (per 100ml)	
Vitamin C (ascorbic acid) (mg)	5.0
Thiamin (vitamin B_1) (µg)	14.0
Riboflavin (vitamin B_2) (µg)	36.0
Niacin (µg)	177.0
Vitamin B_6 (µg)	11.0
Pantothenic acid (µg)	223.0
Vitamin B_{12} (µg)	0.045
Biotin (µg)	0.8
Folic acid (µg)	5.2
Choline (mg)	9.0
Inositol (mg)	45.0
Vitamin (fat-soluble) (per 100 ml)	
Vitamin A	64
Retinol Equivalents (µg)	241
IU	0.42
Vitamin D (IU)	0.90
Vitamin E (mg)	1.5
Vitamin K (µg)	0.1
Mineral (per 100 ml)	
Calcium (mg)	32.7
Phosphorus (mg)	13.7
Magnesium (mg)	3.4
Sodium (mg)	16.9
Potassium (mg)	51.2
Chloride (mg)	39.0
Iron (mg)	0.030
Trace mineral (select) (µg 100 ml)	
Copper	52
Iodine	4-5
Manganese	26
Fluoride	5-30
Selenium	1.8
Boron	8-9

Continued on next page

Table 7-1 *Continued*

Composition of Mature Breastmilk

Composition	Amount
Protein and other selected nitrogenous compounds	
Protein, total (g/100 ml)	1.06
Nitrogen, total (g/100 ml)	0.14-0.21
Casein nitrogen (% of total nitrogen)	35
Whey protein nitrogen (% of total nitrogen)	40
Whey proteins (g/100 ml)	0.30-0.80
Alpha-Lactalbumin	0.15
Serun albumin	0.030-0.040
Lactoferrin	0.10-0.20
Transferrin	0.005
Lysozyme	0.04
Immunoglobulins, (select)	
IgA	
colostrum	0.41-0.47
3-4 weeks	0.036
IgG	
colostrum	0.006-0.021
3-4 weeks	0.006
IgM	
colostrum	0.010-0.049
3-4 weeks	0.004
Creatinine	0.021
Fat related components	
Cholesterol (mg/100 ml)	14
Carnitine (nmol/100 ml)	590

Source: V.S. Packard. **Human Milk and Infant Formula**.
Academic Press, New York, 1982.
U.S. Department of Agriculture, USDA Nutrient Database
for Standard References, Release 13,
Agricultural Research Service, 1999.

Breastmilk contains digestive enzymes such as amylase and lipase, not present in infant formulas, that facilitate digestion of ingested starch and fat in the infant. Breastmilk contains immunologic substances such as IgA, lactoferrin, lysozymes, macrophages and other antibodies that confer passive immunity to the infant. The Lactobacillus factor in breastmilk promotes colonization of the infant's intestine with the bacteria Lactobacillus. These organisms maintain an environment that discourages growth of pathogenic species which cause gastroenteritis and other infections. Breastfed infants are less susceptible than bottle fed infants to res-

piratory, enteric and ear infections. Breastfed infants are also at lesser risk of food allergy since they are spared from exposure to food antigens at a time when macromolecules are readily absorbed. On a long term basis, breastfed infants may have reduced risk of diabetes mellitus, certain types of cancer and raised serum cholesterol later in life.

Apart from the nutritional and immunological benefits, breastmilk is readily available, relatively safe and may enhance intestinal development. Furthermore, breastfeeding is satisfying for both the mother and child. The closeness of this coupling exposes both individuals to a wide range of physical and emotional stimuli that enrich maternal-infant interaction.

Infant Formula

Commercial infant formulas fortified with iron are the best alternative to breastmilk. They are safe, easily digestible and convenient. Regular infant formulas are cow's milk-based but modified to resemble human milk. Current formulas have a whey protein to casein ratio of 60 to 40% which is similar to human milk but very different from the ratio of 18% to 82% found in cow's milk. Furthermore, the levels and types of fats, minerals and vitamins are adjusted closely to resemble the nutrient composition of breastmilk. Where necessary the levels of the added nutrients are adjusted to their bioavailability. Thus, when fed in adequate amounts, formulas meet the recommended nutrient intakes for infants.

Soy-based formulas contain a soy protein isolate and are lactose-free. They are designed for use by infants who are allergic to cow's milk and infants in vegetarian families in which animal protein formulas are not desired, and for infants who suffer from galactosemia or lactase deficiency.

Less allergenic formulas prepared from protein hydrolysates are now available. Special formulas are also available for low birthweight infants and infants with specific inborn errors of metabolism.

Today, the recommendation is to delay the introduction of cow's milk to between 9 and 12 months. Follow-up formulas, which have higher levels of protein but lower levels of fat than regular formulas, are now available for infants 6 months and older.

Cow's Milk

Cow's milk is not recommended for infants less than 9 months of age. The principle reasons for this recommendation are the high protein content and the accompanying renal solute load in cow's milk; the risk of an allergic reaction; and the risk of a blood-losing enteropathy in some infants that is induced by a heat-labile factor in the protein.

Whole cow's milk may be used for feeding infants who are 9 months and older, and who are eating supplemental foods consisting of a balanced mixture of vegetables, fruits and other foods. Skim and partially-skimmed cow's milk are not suitable for infants less than 2 years of age as they are of low energy density, lack the essential fatty acids and have high renal solute load. In some countries, skimmed milk is not recommended for children under five years.

Complementary Feeding

The time of introduction of complementary foods should be determined by the need of infant and not by chronological age. Large, rapidly growing infants consume comparatively larger quantities of milk and yet, may remain hungry. These infants may need complementary foods earlier than smaller, slower growing infants. Complementary foods are nutritionally unimportant before 4 to 6 months of life if the volume of breastmilk consumed is adequate.

Introduction of complementary foods is not synonymous with termination of breastfeeding. Mothers should be encouraged to breastfeed for as long as possible. Breastfeeding remains advantageous, despite the presence of complementary foods in the infant's diet. Nutritionally, breast-milk remains suitable for the older infant. Besides providing essential nutrients, breast milk contains numerous functional components such as the long chain polyunsaturated fatty acids, bifidus factor, nucleotides, oligosaccharides, macrophages, lymphocytes, antioxidants, flavonoids, and others that may have health benefits for both the mother and her infant (Refer to Chapter 8). Breastfeeding is satisfying to both mother and infant and therefore continues to promote maternal-child interaction. Recent research shows that breastmilk contains alpha-amylase which is present throughout lactation. The breastmilk amylase is stable in the gastric environment, i.e. it is not inactivated

by the stomach's harsh contents. Thus, breastmilk may facilitate starch digestion in infants as starch-containing cereals are introduced. Additionally, breastmilk contains immunoglobulins which may still be immunologically active despite changes in the intestinal microflora due to the presence of complementary foods.

Iron-fortified infant cereals should be introduced as the first complementary food. Infant cereals are easily mixed with fluid. As such, they are fed in a consistency that is easily manageable by the infant. Infant cereals provide energy, protein, and, if fortified, iron, thiamin, riboflavin and niacin as well. They are therefore excellent complements to milk for those infants who are not satisfied with milk alone.

Concern has been raised that young infants may not be able to digest starch adequately because of the observed low concentration of duodenal alpha-amylase. However, research has indeed shown that young infants, including premature infants can digest starch. Starch may be hydrolyzed by salivary amylase which is present in variable amounts in the young infant. It may also be digested by breastmilk amylase which is present throughout lactation. An alternate pathway of starch hydrolysis in young infants may be the glucoamylase activity of the intestinal mucosa.

The Committee of Nutrition of the American Academy of Pediatrics recommends that infants be fed iron-fortified infant cereals up to 2 years of age to ensure an adequate iron intake and reduce the risk of dietary iron deficiency. Iron deficiency anemia is one of the main nutritional problems in the world. Dietary iron deficiency is prevalent among older infants and preschoolers.

Elimination of iron-fortified infant cereals from the diet of infants is the principal cause of dietary iron deficiency among infants. The number of foods that are rich sources of iron is small. Thus, there is a limited choice of foods that can fulfill the iron needs of infants. Organ meats are excellent sources of iron; however, they are not commonly part of the infant diet. Iron-fortified infant cereals are by far the most important sources of iron. Unfortunately, in many instances, they are displaced by adult cereals when table foods are introduced. Adult cereals are also fortified with iron, but at a substantially lower level. Because of this, and the relatively small amounts of foods generally consumed by infants, the amount of

iron consumed in adult cereals is not sufficient to satisfy the dietary iron requirement for most infants. Dietary iron deficiency could be easily eliminated if parents are advised to feed their infants infant cereals up to 2 years of age.

According to the Nutrition Committee of the Canadian Pediatric Society, chewable foods should be started by the age of 6 to 8 months when the infant has acquired opposing teeth and can make meaningful chewing movements. Broad clinical experience suggests that 6 to 8 months is the sensitive learning period for chewing and feeding. Delay in the introduction of foods that require chewing beyond this age may result in later feeding difficulties.

Commercially-prepared foods for older infants and toddlers are designed as transitional foods between strained baby foods and table foods. They have a chunky texture which is designed to encourage infants to chew and exercise their gums. Their pieces are large enough to encourage chewing, yet completely digestible if swallowed. To help harden gums and exercise the jaw, baby biscuits or toast are suitable.

Older infants should be fed a variety of foods selected from the basic food groups: Milk Products, Vegetables and Fruit, Grain Products and Meat and Alternates. This will ensure a diet that provides the more than fifty essential nutrients for health and growth. Variety reduces monotony in the diet, while it provides varied experience in flavor, taste and texture to the infant. Variety also minimizes the risk of toxicity of nutrients which can be potentially harmful if their intake is habitually excessive.

Incorporating a rainbow of colors of fruits and vegetables in the infant diet ensures variety in intake. As a result, infants will consume nutrients and functional components that may have health benefits for the infant. (Refer to Chapter 8). During the weaning process, the amount of breastmilk and therefore functional components declines. Thus, selection of a variety of fruits and vegetables will help to maintain a good supply of functional components in the infant's diet. Various colored fruits and vegetables may be introduced to the infant's diet in stages. Infants who receive all the necessary nutrients as well as many functional components during early life may be imprinted for a lifetime of good health.

Small hard pieces of food can cause choking or asphyxiation in infants and young children. Foods that have been implicated include nuts (alone or in other foods such as cookies), pieces of raw vegetables, kernel corn, raw peas or beans, popcorn, hot dogs and small hard candies.

Vitamin Supplements
Under normal feeding conditions, an infant does not require vitamin supplementation. However, a source of vitamin D is required for infants who have little or no exposure to sunlight and are solely fed breastmilk, because breastmilk lacks sufficient vitamin D. Formula-fed infants do not require supplementation as it is fortified with vitamin D.

Vitamin A and vitamin C are present in sufficient amounts in the diets of infants to satisfy the recommended daily intake for most infants. Generally vitamin supplements provide an additional 25 to 30% of the two vitamins to the total intake of infants. Chronic vitamin A intoxication has been reported in infants occasionally. The problem is usually associated with habitually feeding infants with the same vitamin A-rich foods, such as organ meats, plus a vitamin A-containing supplement. The large amount of vitamin A ingested from such a diet over 3 to 4 months can accumulate to a level such that vitamin A toxicity will manifest. Thus, vitamin A supplementation is largely unnecessary and is potentially harmful. On the other hand, vitamin C is non-toxic since it is readily excreted in the urine. However, since fruits and vegetables alone provide ample vitamin C to satisfy body needs, the additional vitamin C from supplements is unnecessary and wasteful.

General Considerations
Infant feeding should be an enjoyable experience for both parent and child. It is one aspect of parent-child bonding. Every positive feeling the parent and infant associates with feeding enhances their closeness, well-being and security.

In practice, infant feeding varies with the infant and the family situation. The choice of breast or bottle feeding is a personal decision. Granted, breast-feeding is preferable, but, if a mother cannot or does not wish to breastfeed, she should not be impaired with a feeling of guilt or inadequacy.

The ultimate aim in infant feeding is to provide a nutritionally adequate diet for optimal health and growth of the infant. To this end, variety and moderation in food choices are key principles. Food pyramids for infants and young children may be used as a guideline for healthy eating (Figures 7-5, 7-6).

The decision to introduce complementary foods, foods for older infants and toddlers and table foods should be done without the pressure of friends, neighbors or relatives with infants of similar age who have already done so. Each infant is a unique individual and needs vary with the rate of growth, physical development and activity. Large, active or energetic infants may need complementary foods earlier than smaller, quieter or more passive infants. Similarly, infants who develop teeth earlier may be started on chewable foods sooner than other infants.

An infant's appetite will fluctuate from day to day. Changes in meal patterns and variations in food choices will make the meals more interesting for the infant. It also introduces new experiences. For the parent, it varies the routine which may otherwise be monotonous and unfulfilling.

Figure 7-5

Building Block for Infant Feeding and Guidelines for Healthy Eating

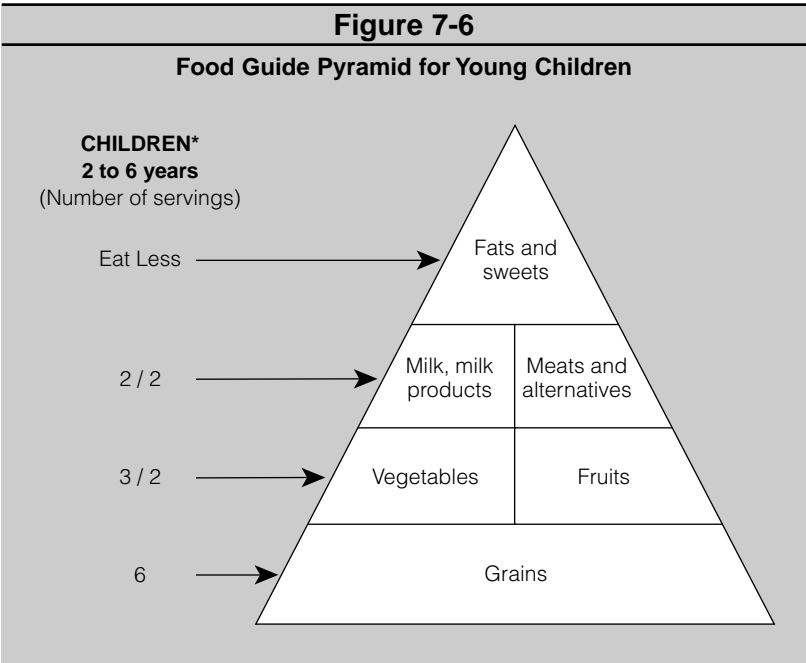| BABIES (Time to Start)	
AVOID	Sweets, salt, flavour enhancers, artificial colours, preservatives
Around 8 Months	Meats, meat alternatives
Around 6-7 Months	Vegetables / Fruits
Around 6 Months	Infant cereals and cereal products
From Birth	Breast milk / Formula
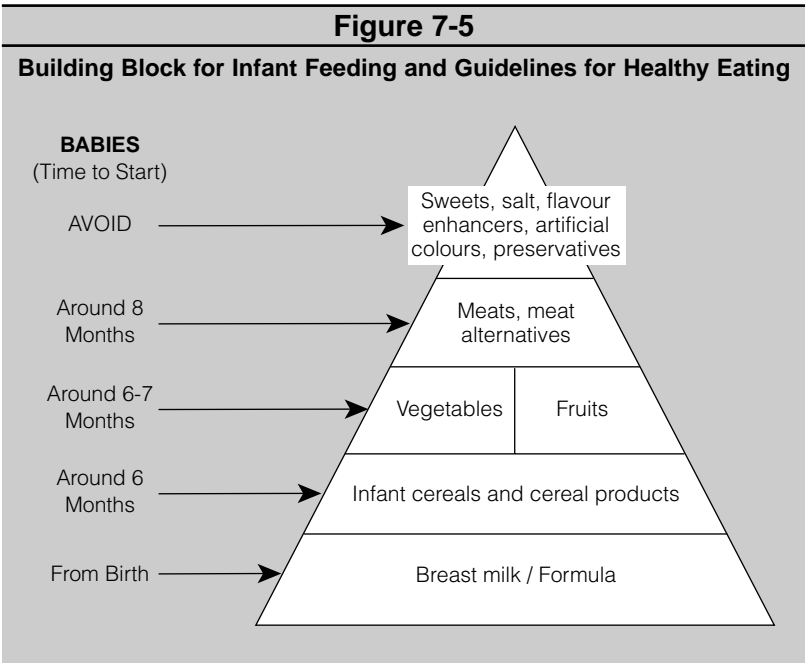
Each feeding period should be a peaceful and unhurried time for both parent and infant. The environment should be warm and relaxed. Anxiety on the part of the lactating mother will reduce her milk output as milk ejection can be influenced by emotion. Infants are sensitive individuals. The emotion of the feeder can easily be transmitted to the infant who will eat accordingly. Positive and negative signals about food, be they verbal or physical expressions about food, can affect likes and dislikes of infants.

As infants grow older and begin to explore their environment, they should be allowed to touch and to feel food. This is an important part of learning. In this situation, patience as well as restraint will need to be exercised.

Food Allergy

The term food allergy refers to an immunologic reaction to a food or part of a food. If a non-allergenic mechanism is involved, the response is a food idiosyncrasy. The term food intolerance encompasses any adverse reaction to food including those of an allergenic and non-allergenic nature.

Figure 7-6

Food Guide Pyramid for Young Children

CHILDREN*
2 to 6 years
(Number of servings)

Eat Less ———→ Fats and sweets

2 / 2 ———→ Milk, milk products | Meats and alternatives

3 / 2 ———→ Vegetables | Fruits

6 ———→ Grains

Source: U.S. Department of Agriculture, Center for Nutrition Policy
　　　and Promotion. Program Aid 1650, March 1999.

The incidence of food allergy has been estimated to be less than 1% for the general population, but 2 to 4% for the pediatric population. The tendency to an allergic reaction is often inherited. It has been estimated that the tendency to develop an allergic reaction is 12.5% if neither parent is atopic, 38% if one parent is atopic and 58% if both parents have allergies.

Food allergy is difficult to diagnose even in clinical situations. A double-blind placebo controlled oral food challenge under carefully supervised conditions provides the most objective and definitive diagnosis. A single-blind placebo controlled oral food challenge may also be performed but is prone to bias. These tests are contraindicated where there is a history of anaphylaxis.

Certain tests such as the scratch test, intradermal test and RAST procedure may be of assistance in the diagnosis of a food allergy. However, these tests are not sufficiently accurate to be used alone. The symptoms of food allergy include the following:

– Systemic: anaphylaxis (allergic shock reaction);

– gastrointestinal: vomiting, abdominal pain, diarrhea, malabsorption, enteropathies;

– respiratory: rhinitis, sinusitis, secretory otis media, cough, wheezing, pulmonary infiltration;

– cutaneous: rash, uticaria, eczema.

The reason for selective reactivity of one system over another is unknown. It should be noted that the above symptoms are not exclusively due to food allergies.

Allergic responses have been grouped according to the time between food ingestion and the appearance of symptoms as: immediate (minutes to an hour); intermediate (within hours); and late (days to weeks).

Manifestation of an allergic reaction can occur at anytime during infancy and childhood. In general, infants are more susceptible than older individuals, probably because of immaturity of the immune system and because of permeability of the intestinal mucosa which allows allergens and immunoglobulins to cross through. However, food allergies that are seen in infancy generally do not persist into childhood or adult life. Most infants do outgrow them.

All foods are potentially allergenic; however, certain foods are more likely to act as sensitizers because of the frequency of use and because they contain large amounts of the allergenic substance. In infancy, the most important allergen is probably cow's milk. Milk allergy is typified by its onset at an early age and spontaneous disappearance of the symptoms and reactions usually within two years. Other foods that are commonly reported to induce allergies in infants are eggs, fish, some fruits, meats, nuts, chocolate and gluten-containing cereals.

Treatment of food allergy lies in avoidance of the food allergen. Patients must be instructed on possible cross-reacting foods and hidden sources of the allergen in the diet. For patients with severe chronic allergies an overall elimination diet may be recommended on a short-term basis. Foods are gradually re-introduced and tolerance monitored. Some foods may contain an allergen as a hidden ingredient and its ingestion may be life-threatening. Patients may be instructed to have antihistamines and/or an epinephrine filled syringe available at all times.

Many allergists recommend exclusive breastfeeding for at least six months particularly for infants with allergic parents. As a means of minimizing the risk of an adverse reaction to foods, infants should be introduced to single foods one at a time. When tolerance of these foods is established mixed varieties may be introduced. If a food intolerance is suspected, the food should be immediately withdrawn from the diet and the physician consulted before the child is rechallenged with the offensive food.

Gluten Sensitivity
Gluten sensitive enteropathy, more commonly known as celiac disease, is a food allergy that affects children and is life-lasting. In adults, gluten-induced enteropathy is referred to as nontropical sprue. This disease causes histologically detectable damage to the small intestinal mucosa. However, the damage to the small intestine only comes into play following exposure to gluten. Consequently, treatment involves a diet which eliminates gluten and gluten-containing products for the patient's lifetime. A few cases of transient gluten intolerance have been described but there is little data available on their long-term outcome.

The generally recognized toxic agent causing gluten-sensitive enteropathy is the protein moiety, gluten, found in wheat, rye, oats and barley. Actually, it is the alcohol soluble fraction of gluten, gliadin, that is offensive to the sensitive individual.

The etiology of gluten-sensitive enteropathy remains a mystery, although several theories have been proposed. While an enzyme deficiency has not been identified, brush border enzymes are decreased in the acute stages of the disease, probably secondary to mucosal damage. Immunological theories abound due to findings of circulating antibodies to gluten, autoantibodies, increased levels of IgA and IgM. Genetic factors also play a role as a predisposing factor in gluten sensitivity.

Children with celiac disease commonly present with diarrhea and abnormal stools that are pale, greasy, bulky and foul smelling. Anorexia and irritability are also common signs. Concomitant with the intestinal lesions caused by the ingestion of gluten is the diminished absorptive capacity of the small intestine which can lead to malabsorption, muscle wasting, stunted growth, weight loss and abdominal distension.

Gluten sensitivity in infants can result in extreme malnutrition unless it is detected and treated early. Infants are particularly sensitive to the insult of some of the allergenic foods such as wheat due to immaturity of their gastrointestinal tract. One of the nutritional consequences of gluten-sensitive enteropathy is a reduction in fat absorption. Undigested fat is passed in the feces resulting in steatorrhea. Mucosal damage also results in poor carbohydrate and protein absorption. Excess undigested carbohydrate becomes available for fermentation by bacteria in the bowel; the resultant gas makes the stools frothy and offensive smelling.

The malabsorption of fat influences the fat-soluble vitamin status of the infant as well. Malabsorption of the fat-soluble vitamins results in deficiency syndromes. Calcium is also poorly absorbed resulting in a negative calcium balance and a tendency to develop rickets and osteopenia. Vitamin D deficiency only serves to aggravate calcium malabsorption. Anemia is usually present because of iron and folate deficiency. B vitamin malabsorption, particularly folate, is also a common finding in gluten-sensitivity.

This is due to the fact that folate is absorbed in the jejunum, the portion of the small intestine which is affected by the gluten-induced enteropathy.

Early detection of gluten-sensitivity is necessary in order to limit the extent of malnutrition to which the infant and young child is exposed. Once gluten-sensitivity has been diagnosed a diet free of gluten is prescribed. Dietary elimination of wheat, oats, barley and rye has historically been found to result in dramatic relief from the effects of gluten-sensitivity and is usually very effective in children. This includes marked histological improvement of the intestinal mucosa which occurs within weeks of starting the diet. The mucosa will eventually return to a normal state if the diet is maintained.

Successful treatment depends on an ability to select foods free of gluten-containing products, and the ability of the caregiver to help the patient abide by the diet. The list of unacceptable foods for gluten-sensitive individuals includes wheat, wheat flour, wheat germ, wheat bran, rye, barley, kasha, oats and malt. The acceptability of millet and sorghum is uncertain. Cereals are the main food group which naturally contain gluten. Very few cereals can be introduced in the low-gluten diet. Among the cereals, only corn and rice are acceptable.

Caution is advised to all parents faced with having to provide a gluten-free diet to their infant. There is the hazard of providing a nutritionally inadequate diet if foods are excluded "just in case". Professional advice on diet planning is important to ensure a balanced and nutritionally sound gluten-free diet. As a precaution, gluten-containing foods are generally not advised for feeding infants less than six months of age, especially if the family has a history of allergy.

Weight Control and Physical Activity

Obesity is an important health problem and is associated with the development of several diseases including diabetes mellitus, hypertension, hyperlipidemia, coronary artery disease, obstructive sleep apnea, and cancer. It is also associated with several physiological disorders, such as depression, anorexia nervosa, and bulimia. Moreover, it is an independent risk factor for increased mortality. The obese person may also be subject to psychological

stresses. Obesity in children is of special significance because obese children remain obese as adults.

The prevalence of obesity is increasing world wide. There are over 300 million obese and many more overweight adults around the world. In developing countries, it is estimated that over 115 million people are obese. Prevalence rates range from a low of less than five percent in rural China, Japan, and some African countries to a high of 75% of the adult population in urban Samoa. Even in countries with low prevalence rates, both over-weight and underweight people can now be seen. In many countries, the increase in obesity has occurred within the last few years. The main causes are a nutrition transition to fat-rich diets and reduced physical activity in urban populations. The prevalence of obesity varies with socioeconomic status. In developed countries, poverty is associated with increased prevalence, whereas in developing countries it is the relative affluence that carries the greater risk.

Obesity is usually defined as 20% above desired weight for height as determined by weight tables. A limitation of the standard weight tables often cited is the bias introduced as a result of the highly selected population on which the information is based. Additionally, weight tables do not take into account percentage of body fat. For example, an athlete may be above the ideal weight due to muscle mass and not due to excess body fat. Overfatness has been defined as a percent body fat composition greater than 20 for men and greater than 30 for women.

The 1999 Metropolitan Life company height and weight table is shown in Table 7-2. The method for the determination of body frame size appears on Table 7-3. The weights shown have been increased from the previous 1959 tables. Some groups such as the American Heart Association continue to endorse the 1959 tables on the basis that the 1999 tables do not consider the non-fatal risks of increased weight.

Today, the use of BMI (Body Mass Index), calculated as BW/Ht^2 (kg/m^2), is gaining acceptance as an index for use in diagnosing obesity. A normal BMI ranges from 18.5 to 24.9. Individuals with a BMI ≥ 25 are considered overweight and those with a BMI ≥ 30 are obese (see Table 7-4). The International Task Force on

Table 7-2

1999 Metropolitan Life Insurance Company Height and Weight Tables

Height	Small Frame	Medium Frame	Large Frame
Men*			
5' 2"	128-134	131-141	138-150
5' 3"	130-136	133-143	140-153
5' 4"	132-138	135-145	142-156
5' 5"	134-140	137-148	144-160
5' 6"	136-142	139-151	146-164
5' 7"	138-145	142-154	149-168
5' 8"	140-148	145-157	152-172
5' 9"	142-151	148-160	155-176
5' 10"	144-154	151-163	158-180
5' 11"	146-157	154-166	161-184
6' 0"	149-160	157-170	164-188
6' 1"	152-164	160-174	168-192
6' 2"	155-168	164-178	172-197
6' 3"	158-172	167-182	176-202
6' 4"	162-176	171-187	181-207
Women**			
4' 10"	102-111	109-121	118-131
4' 11"	103-113	111-123	120-134
5' 0"	104-115	113-126	122-137
5' 1"	106-118	115-129	125-140
5' 2"	108-121	118-132	128-143
5' 3"	111-124	121-135	131-147
5' 4"	114-127	124-138	134-151
5' 5"	117-130	127-141	137-155
5' 6"	120-133	130-144	140-159
5' 7"	123-136	133-147	143-163
5' 8"	126-139	136-150	146-167
5' 9"	129-142	139-153	149-170
5' 10"	132-145	142-156	152-173
5' 11"	135-148	145-159	155-176
6' 0"	138-151	148-162	158-179

* Weights at ages 25 to 59 based on lowest mortality. Weight in pounds according to frame (in indoor clothing weighing 5 lb, shoes with 1" heels).

** Weights at ages 25 to 59 based on lowest mortality. Weight in pounds according to frame (in indoor clothing weighing 3 lb, shoes with 1" heels).

Courtesy of Metropolitan Life Insurance Company, 1999.

Table 7-3

How to Determine Your Body Frame by Elbow Breadth*

To make a simple approximation of your frame size:

Extend your arm and bend the forearm upwards at a 90 degree angle. Keep the fingers straight and turn the inside of your wrist toward the body. Place the thumb and index finger of your hand on the two prominent bones on either side of your elbow. Measure the space between your fingers against a ruler or a tape measure. Compare this measurement with the measurements below.

These tables list the elbow measurements for men and women of medium frame at various heights. Measurements lower than those listed indicate that you have a small frame, while higher measurements indicate a large frame.

Height (in 1" heels)	Elbow breadth in.	Height (in 2.5-cm heels) cm.	Elbow breadth cm.
Men			
5' 2" - 5' 3"	2-1/2 - 2-7/8	158 -161	6.4 - 7.2
5' 4" - 5' 7"	2-5/8 - 2-7/8	162 - 171	6.7 - 7.4
5' 8" - 5" 11"	2-3/4 - 3	172 - 181	6.9 - 7.6
6' 0" - 6' 3"	2-3/4 - 3-1/8	182 - 191	7.1 - 7.8
6' 4"	2-7/8 - 3-1/4	192 - 193	7.4 - 8.1
Women			
4' 10" - 4' 11"	2-1/4 - 2-1/2	148 - 151	5.6 - 6.4
5' 0" - 5' 3"	2-1/4 - 2-1/2	152 - 161	5.8 - 6.5
5' 4" - 5' 7"	2-3/8 - 2-5/8	162 - 171	5.9 - 6.6
5' 8" - 5' 11"	2-3/8 - 2-5/8	172 - 181	6.1 - 6.8
6' 0"	2-1/2 - 2-3/4	182 - 183	6.2 - 6.9

* Source of basic data: Data tape, HANES 1.
Courtesy of Metropolitan Life Insurance Company, 1999.

Obesity suggests that BMI is an accurate and easily available screening index for childhood obesity. BMI has been found to correlate well with the complications of childhood obesity such as hypercholesterolemia, hypertension and long term development of cardiovascular diseases.

Skin-fold thickness measurements are also used to estimate human body composition. It is assumed that a direct relationship exists between total body fat and fat deposited in depots just beneath the skin. The sites most commonly used for measuring skin-fold thickness are the triceps, abdomen, thigh, subscapula (below the tip of the right scapula) and suprailiac (just above the hip bone).

Table 7-4

Body Mass Index and Grades of Chronic Protein-Energy Malnutrition and Obesity

Grade	Body Mass Index (Body weight/ height^2) or (kg/m^2)
Obesity class III	≥ 40.0
Obesity class II	35.0-39.9
Obesity class I	30.0-34.9
Overweight	25.0-29.9
Normal	18.5-24.9
Chronic Energy Deficiency (Thinness)	<18.5

Source: WHO Expert Committee. Physical status: the use and interpretation
of anthropometry: report of a WHO Expert Committee. Geneva.
World Health Organization; 1995. WHO Technical Report Series 854.

Etiology of Obesity

While the immediate cause of obesity is energy imbalance, it is recognized that a number of factors may predispose a person to obesity. Current hypotheses include genetic and psychological factors, hormonal disturbances and others.

Heredity has been shown to play a role in obesity. While a genetic predisposition to obesity exists, it is not clear how and to what extent environmental factors interact to produce the obese state. In addition, psychological factors need to be considered. Psychosocial researchers suggest that individuals overeat for comfort, control and/or to cope with stressful situations.

Despite popular belief, hormonal disturbances are a rare cause of obesity. One such disturbance, Cushing's syndrome (an overactivity of the adrenal gland), is characterized by central obesity. Hypothyroidism may also lead to some increased adipose mass but water is the main cause of weight gain. Another hormonal cause of obesity, although mild, is hypogonadism. Finally, lesions to the hypothalamus by a tumor or infection may lead to obesity.

One popular concept in human obesity was the "Set Point" theory which maintains that each individual has an inherent weight which reflects a balance between energy input and output. The body physiologically strives to maintain this specific weight. Thus any attempt to lower the set point quickly is considered to be futile.

Methods of Weight Reduction

As body weight is a function of energy balance, the input-output equation must be at equilibrium for maintenance of body weight. If input rises, output must also rise. Otherwise excess energy will be stored. If input is less than output, weight loss occurs.

The equation then is a balance between food energy consumed and metabolic and physical activities which expend energy. However, this is not a simple two-step equation. Indeed it consists of multiple steps, beginning with food intake, digestion, absorption, systemic transport, cellular uptake and finally storage and utilization. Alteration in any one step can change the net result.

Over the years, scientists have sought ways to combat obesity through suppressing or stimulating one or more steps in this chain. Diets, fibers, drugs, homeopathic supplements, massage therapy and surgical procedures have also been used. Basically, these reduce energy intake, suppress appetite, decrease absorption, or increase metabolism and/or excretion and increase physical activity. Unfortunately, individuals who are subjected to the non-dietary methods of weight reduction often suffer from side effects or complications. Relapse is frequent once the regimen is relaxed or discontinued.

In extreme cases distorted body image and behavior can result in eating disorders such as bulimia and anorexia nervosa. These psychologically and physiologically debilitating disorders primarily affect adolescent and young adult females. However, older females and males are not spared, although their occurrence is much less frequent.

Under more normal circumstances many dietary regimens for the treatment of obesity have been popularized and commercialized. Basically they can be divided into two main categories: (1) Those that promote rapid weight loss and (2) Those that promote sensible gradual weight loss.

In the first group are methods that prescribe starvation, semi-starvation, high protein or high carbohydrates. Starvation or fasting is reported to be an effective method in treating refractory obesity. This method must be used under the supervision of qualified medical doctors. Complications, such as loss of lean body mass,

liver dysfunction, gallstones, anemia, high levels of uric acid in the blood, postural hypotension, ketosis and electrolyte disorders have been described. The long-term effects of weight reduction and maintenance of lower body weight are generally poor. The main reason is failure to instruct obese patients to adopt good eating habits. Frequently, a pattern of repeated food restriction and refeeding results.

The high protein, low carbohydrate diets range from semi-starvation to unlimited intake of allowable high protein, high fat foods. These diets are ketogenic since the low carbohydrate content in the diet does not allow complete oxidation of fat and some proteins. The result is an accumulation of substances called ketone bodies. Some believe that these ketone bodies suppress appetite while others are unable to demonstrate an effect.

One advantage of the high protein diets is the rapid weight loss, especially during the initial period. The initial rapid weight loss on high protein diets is due to diuresis; water loss rather than fat loss. The ease with which carbohydrate can be eliminated from the diet and the quick initial weight loss have made these diets very popular. However, on a long term basis, there are many side effects that are potentially dangerous, such as: high blood cholesterol, hypokalemia, ketoacidosis, postural hypotension and potential gout development. In addition, long term adherence to a high protein, low carbohydrate diet is very poor because subjects develop a craving for carbohydrates.

Another quick weight loss technique is the high carbohydrate, vegan diet. A vegetarian diet, if well-planned can be nutritious. However, some forms are restrictive and extremely low in protein and fat, and possibly other nutrients as well. Vitamin B_{12} is deficient in pure vegan diets. Furthermore, the high fiber content of these diets may interfere with utilization of minerals and provide inadequate levels of certain vitamins and protein, if the essential amino acids are not properly complemented. Today, there is much emphasis on high fiber diets. Indeed vegetables, fruits and cereals that are relatively high in fiber are pleasant and nutritious contributions to any diet. Fiber itself has no biological property that promotes weight loss. By eating high fiber foods one may take longer to eat a meal and therefore have less time to eat fat or high caloric foods. Such foods may also be satisfying causing

one to eat less. Hence it is the low calorie content of the diet that promotes weight reduction.

It is clear that diets promising rapid weight loss in a short period of time are harmful but do have the advantage of rapid initial loss. However, weight loss tends to decrease with time and the quick weight loss is not maintained. This can cause frustration and return to earlier eating habits thus resulting in weight regain.

Generally, quick weight loss methods are not successful for permanent weight reduction and maintenance, in part because they do not teach eating habits that support weight maintenance after the desired weight is achieved. It is widely believed that losing and regaining weight, commonly referred to yo-yo dieting, may do more harm both physiologically and psychologically and is more stressful on the individual than remaining obese.

If these regimes are to be used, they should only be used for short durations, as a prelude to more sensible weight reduction programs. This should be done under qualified professional supervision.

There is no magic diet or quick and easy way to lose weight and to sustain a lower body weight. The ultimate nutrition principle in weight reduction is sensible weight loss through moderate intake of a variety of foods that provide an adequate balance of all essential nutrients. This should be complemented by physical activity. Slow weight reduction means a loss of 1 to 2 pounds of body weight per week. This approach to gradual weight loss requires dedication, perseverance and an effective behavioral modification program that slowly allows the person to assume a lifestyle of good eating habits and physical activity.

Physical activity has an important role in weight loss. Exercise will enhance the metabolic rate which normally declines during dieting. It decreases the loss of muscle tissue during dieting and it serves to improve blood pressure, and overall cardiovascular health. Exercise increases caloric expenditure but the amount is not normally high enough for weight loss to be achieved without caloric restriction as well. The energy expenditure of various activities is shown in Table 7-5 and is directly related to body size.

Table 7-5

Energy Expenditure of Selected Activities
(Calories expended/minute activity)

Activity	kcal/min/kg	45 kg	55 kg	65 kg
Basketball	0.138	6.2	7.6	9.0
Cycling 5.5mph	0.064	2.9	3.5	4.2
9.4mph	0.100	4.5	5.5	6.5
Dancing (twist)	0.168	7.6	9.2	10.9
Football	0.132	5.9	7.3	8.6
Running 11.5 min./mile	0.135	6.1	7.4	8.8
8 min./mile	0.208	9.4	11.4	13.6
Sitting quietly	0.021	0.9	1.2	1.4
Walking, normal pace	0.080	3.6	4.4	5.2
Writing, sitting	0.029	1.3	1.6	1.9
Vacuuming, females	0.045	2.0	2.5	2.9
males	0.048	2.2	2.6	3.1
Ironing, females	0.033	1.5	1.8	2.1
males	0.064	2.9	3.5	4.2

Source: Shils ME, Olson JA, Shike M and Ross, AC (eds.). Modern Nutrition
in Health and Disease, 9th ed. Williams and Wilkins, Philadelphia, 1999.

A safe dietary weight loss regimen should provide a daily intake of all nutrients from a variety of foods including milk and dairy products, grains, fruit and vegetables and protein sources. A minimum energy level of 1200 kilocalories for the long-term is necessary to achieve this goal. A slow rate of weight loss is recommended as higher losses may result in nutrient deficiency, fatigue and loss of muscle rather than fat tissue.

The development of macronutrient substitutes such as non-nutritive sweeteners, sugar replacers and fat substitutes represent attempts to reduce the caloric contents of the diet. If used wisely, they can be effective in weight control. However dietary compensation on days when these low calorie foods formulated with macronutrient substitutes is utilized is more common than not and obviates any advantages of their use.

Despite some concerns of the possible adverse health effects of artificial sweeteners, they are in widespread use today. Saccharin is 300 to 500 times sweeter than sucrose and is excreted unchanged, contributing zero calories to the food item. Aspartame, a very low calorie sweetener is metabolized to phenylalanine and therefore carries a risk for persons with PKU. Other

non-nutritive sweeteners include acesulfame-K, cyclamate and sucrose polymers.

Fat substitutes can partially replace fat in food and maintain the smell, taste, palatability and visual appeal, while decreasing the caloric content of the food item. A wide range of fat substitutes including gums, emulsifiers, starches and proteins are in current use. Although not universally approved for consumption, zero-calorie fat substitutes which have the same sensory and physical properties as traditional fats, with the advantage that they are suitable for frying, are presently under investigation. One such fat substitute, a sucrose polyester immune to pancreatic lipase hydrolysis and absorption, has received official approval for commercial use in selected products in the United States.

Among the many commercial organizations that are involved in counseling for weight reduction over the years, Weight Watchers International has been shown to be very effective, especially in terms of support and motivation. The Weight Watchers system consists of a nutritious food plan, eating management skills and an exercise plan delivered in a group setting. Once individuals reach their goal weight, a maintenance plan helps them avoid regaining weight. Lifetime membership is offered to individuals who maintain their weight successfully. Shape Up America has an internet web site (http//www.shapeup.org) designed to provide the latest information about weight management, increased activity and physical fitness. The web site also has an interactive, custom designed weight loss program to help people to achieve personal weight loss and shapeup goals through food plans and physical activity plans.

Childhood Obesity
Childhood obesity is the most prevalent nutritional problem among children and adolescents in the USA. The most recent data from the 1999-2000 National Health and Nutrition Examination Survey (NHANES) suggest that 15% of children and adolescents aged 6-19 years are overweight, defined as children with BMI values at or above the 95th percentile of sex-specific BMI growth charts. This represents a 4% increase from the overweight estimates of 11% from NHANES III (1988-1994). The prevalence of obesity is particularly high if both parents are obese. A 5–fold increase in the prevalence of obesity is reported

among children of obese parents compared with children of lean parents.

Both genetic and environmental factors are considered as important determinants for obesity in both adults and children. There is accumulating evidence that individuals with lower basal metabolic rates are more likely to develop obesity. Among environmental factors, lack of physical activity and increased energy and fat intake have a direct relationship with obesity. Socioeconomic and social factors also play a major role in the development of childhood obesity.

Childhood obesity is associated with serious health effects. Childhood obesity is also followed by serious consequences in adulthood. Overweight children and adolescents are at increased risk of early death and morbidity independent of adult BMI. The metabolic consequences of childhood obesity become very apparent in young children. Hypertension, hypercholesterolemia and hyperinsulinemia all occur at a young age and slowly lead to coronary artery disease and diabetes. Gallstones, hepatitis, sleep apnea and increased intracranial pressure may also develop in obese children. In fact only a few organ systems are unaffected by childhood obesity. Teasing, discrimination, and social victimization of obese children are also of serious concern. Treatment of obesity involves improved eating habits, greater physical activity, correct family environment, child motivation and parental support.

Nutrition and Sports

Physical activity is an important aspect of weight control, cardiovascular fitness, and general fitness and health. The nutritional needs of adult athletes have been reviewed by the American Dietetic Association. They include an increased energy requirement to balance the high level of physical activity. In some instances weight gain may be desirable. With increased energy requirements, there is a concomitant increased need for thiamin, riboflavin and niacin which are required for energy metabolism. The American Dietetic Association recommends that these increased requirements be met by increased consumption of breads and cereals, fruits and vegetables with moderate increases in fats and sugar.

Despite much belief to the contrary, sufficient protein is supplied by the recommended dietary protein level standards. An intake of 1 g/kg body weight per day for a mature athlete and 2 g/kg body weight per day for the growing athlete are sufficient to promote synthesis of lean tissue during training. Intake of greater amounts of protein will not enhance physiologic work performance.

Of primary concern is maintenance of an adequate state of hydration. A five percent loss of body weight as fluid can result in heat exhaustion; 10 percent loss can lead to heat stroke and circulatory collapse. Guidelines as to water replacement before, during and after exercise have been proposed. It should be noted the weigh-ins before and after exercise are necessary to assess hydration status and that thirst sensation is not a reliable indicator.

With large sweat losses there is significant loss of the electrolytes sodium, chloride and, on occasion, potassium. The American Dietetic Association recommends their replacement with use of ordinary table foods including use of table salt and high potassium foods. However, where sweat losses exceed 4 liters, it is recommended that electrolyte supplements be administered at the discretion of a physician.

A number of products have been promoted as having a special capacity to increase athletic performance such as wheat germ, vitamin E, ascorbic acid, honey, etc. Such claims are unsubstantiated.

Carbohydrate loading has been suggested to improve performance in endurance events. The traditional regimen has three phases. About one week before a competitive event, the muscles become depleted of glycogen through training to exhaustion. Then, for three days, a high protein, high fat, low carbohydrate (100 g) diet is followed. Three days before the competitive event, carbohydrate loading begins with moderate protein, low fat and high carbohydrate (250-525 g) diet. In this way, the muscles become supersaturated with glycogen, which is stored energy. Recently, the necessity of the high protein, high fat and low carbohydrate phase has been questioned. Additionally, it is not recommended to follow the full sequence more than two to three times per year.

It has also been recommended that a light pre-game meal be consumed 3 to 4 hours before a competitive event. The foods should be low in bulk with a minimal amount of fat. Caffeine containing beverages are not recommended due to their diuretic effect. Simple sugars, such as candy bars which are often promoted as a source of "quick energy" are also not recommended immediately prior to competition as they may result in hyperinsulinemia. The hyperinsulinemia may interfere with the ability of the muscles to use fat. Furthermore, hypoglycemia may follow as a result of hyperinsulinemia.

Nutrition of the Elderly
As a group, individuals over 60 years of age are one of the fastest growing segments of the population. For example, in 1995 there were 14.1% male and 17.7% female Canadians 60 years or older. In the year 2020, it is projected that 24.7% of the male and 28.5% of the female population in Canada will be over 60 years of age. In the United States, 25 million Americans or 11% of the population are 65 years or older. This age group is expected to reach 57 million in the year 2030, an increase of 32 million. Australia also has a considerable aging population segment. By the year 2020, it is estimated that 21.4% of male and 24.7% of female Australians will be over 60 years of age.

Our understanding of the aging process remains poor. Nevertheless studies on aging have shown that it is associated with the progressive loss of physiologic capacity or function in all organs of the body. There is a progressive loss of cells in all tissues resulting in diminished functioning units. However, nutrition research suggests that optimal nutrition and physical fitness can slow the aging process and promote optimal health.

Nutrient Requirements
The current recommended daily nutrient intakes for older adults are similar to those for younger adults. The exception is energy; the need decreases with age. The nutrients that are commonly observed to be inadequate in diets of elderly individuals include: protein, calcium and iron.

Energy
Energy requirements decrease as one ages for three main reasons. With aging, the body tissues tend to change in composition to

relatively more fat and less muscular tissue. The reduction in active tissue alone will lead to a reduction in energy expenditure and energy requirement. There is also a decrease in the basal metabolic rate which relates to the decrease in active cell mass. The basal metabolic rate decreases about 20% between the ages of 25 and 65. As well, decline in physical activity is common. The process of replacement of lean mass with fat can be delayed if the person remains physically active.

As people grow older, they tend to eat less and this compensates, to some extent, for the reduced energy needs. However, this can be accompanied by inadequate consumption of the essential nutrients if nutrient dense foods are not selected. This is especially true for women who have an even lower energy requirement than men.

Protein
The protein needs of older persons are not different from those of younger, healthy adults. The recommended level of protein intake is 0.8 g/kg body weight. The mean protein intake of individuals beyond 65 years of age, particularly women, tends to fall below the recommended level. Foods of high protein density, such as eggs or meat, should be included in the diet.

Calcium
Inadequate calcium intake is a concern as it may contribute to bone diseases such as osteoporosis and osteomalacia often seen in elderly people. Osteoporosis is characterized by a reduction of bone mass, bone fragility and pain. Approximately 25% of post-menopausal women are afflicted with this problem. Although evidence to demonstrate a direct relationship between decreased calcium intake and osteoporosis is not available, a habitually low calcium intake is generally considered a major contributory factor in this disease. The recommended dietary calcium intake for elderly individuals is 50% higher than that of younger adults. However, the level of calcium in the diets of elderly people is commonly low. Furthermore, their reduced ability to absorb dietary calcium due to decreased gastric acidity compounds the problem.

Iron
The recommended iron intake for older men is similar to that for younger adult men. The recommendation for postmenopausal

women is lower than for menstruating women and is similar to the recommended iron intake for men. Iron deficiency is a common problem among the elderly. It results from decreased absorption and/or inadequate iron intake. Iron deficiency in the elderly can cause fatigue, disorientation, confusion and loss of memory.

Folate

Folate supplementation in amounts 1-2 times the RDA is regarded as helpful in lowering elevated plasma homocystine levels and thus may lower the risk for vascular diseases and neural tube defects (NTD). As of January 1996, the FDA mandated fortification of wheat flour and grain products with folate. However, due to malabsorption of vitamin B_{12}, prevalence of vitamin B_{12} deficiency in older adults is twice as common as in younger adults. High folate intake may mask vitamin B_{12} deficiency and may thus delay its diagnosis and treatment. The Food and Nutrition Board has therefore advised that elderly people should also receive vitamin B_{12} through fortified food and/or vitamin B_{12} supplementation.

Use of Supplements

Many older adults are motivated to use dietary supplements to slow the aging process, improve health, increase energy level or prevent chronic diseases. Studies have shown that 35 to 70% of older Americans regularly use one or more nutritional supplements in the form of pills, capsules or drinks. Women tend to use nutritional supplements more than men. These supplements include antioxidants, vitamins, minerals and also homeopathic formulations. Evidence of benefits is scant, however.

Factors Influencing Food Intake

Many of the nutrition-related problems observed among the elderly are related to poor life-long eating habits. However, there are other factors (both social and health) that lead to nutritionally inadequate diets. Some of the social problems include:

(a) limited income and, therefore, purchasing power;

(b) loneliness, unhappiness and bereavement;

(c) social isolation;

(d) decreased mobility and activity;

(e) lack of support from family, friends and community; and

(f) food fads.

Some of the health-related factors include:

(a) chronic health problems;

(b) gastrointestinal disturbances and discomfort, hypochlorhydria;

(c) mental disturbances such as depression, confusion, dementia;

(d) physical incoordination and immobility;

(e) drug use;

(f) restricted diets;

(g) alcohol abuse;

(h) taste and smell aversion and sensory loss; and

(i) poor dental health and ill-fitting dentures.

Principles for Feeding the Elderly

In general, for "healthy" elderly individuals, diets should follow available food guides. Special consideration is given to:

(a) adequate energy intake;

(b) nutrient dense foods, especially for protein, iron and calcium;

(c) adequate fluid intake; and

(d) good sources of dietary fiber.

Attention should be given to the following in planning successful diets for the elderly:

(a) individual preferences. Foods that do not conform to established habits may be physically and emotionally disturbing;

(b) Social, religious, racial and psychological factors, in addition to lifestyle;

(c) Preparation and presentation of meals. They must be attractive, appetizing and provide a consistency and texture acceptable to the consumer;

(d) Variety of foods. A wide choice of food would tend to provide balanced nutrition and a greater chance of acceptance. This would also avoid monotony in eating; and

(e) Flexibility in the number and time of meals. For some individuals small feedings of four or five times a day may be desirable.

Of considerable importance for the well-being of the elderly is regular physical activity. Keeping fit maintains cardiovascular fitness, muscle tone and normal bone density. Active individuals are less likely to suffer from osteoporosis.

Suggested Readings

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CHAPTER 8

NEW HORIZONS IN NUTRITION

In recent years, there has been a major change in the consumption of foods and also in the attitudes towards food. The nutritional emphasis has shifted from survival and hunger contentment to health maintenance and promotion of better health. Food that was traditionally viewed as nourishment needed for growth and strength, energy and nutrients, is now also considered to have beneficial physiological and psychological effects in helping to reduce the risk of chronic diseases such as cardiovascular disease, cancer, obesity and diabetes. These new concepts are important particularly in view of the increasing consumer concern for health, increasing cost of health care, continuous increase in life expectancy and demographic distribution towards the elderly population.

Functional Foods

A food is considered a functional food if it provides nutrition and also has a beneficial physiological effect that improves health and/or reduces risk of chronic disease. The International Life Sciences Institute of North America (ILSI) defines functional foods as 'foods that, by virtue of physiologically active food components, provide health benefits beyond basic nutrition'. Thus, a functional food is a food, which has successfully demonstrated health effects when consumed, in normal amounts, as part of the regular diet. A functional food can be a natural food or a food in which one or more components have been modified. Numerous functional components have been studied and classified. However, most have not been sufficiently researched to bear health claims. Table 8-1 provides a listing of foods that have been approved by the FDA for making health claims.

Dietary Fiber

Dietary fiber is a mixture of many complex organic substances with unique physical and chemical properties. They are resistant to hydrolysis by digestive enzymes. Plant cell wall materials containing cellulose, hemicellulose, pectin and lignin are the major components of dietary fiber. In addition, mucilages, gums, algal

Table 8-1

Health Claims for Functional Foods Currently Approved by the FDA

Functional Food	Key component(s)	Potential health benefits
Low-fat foods as part of a low-fat diet (e.g. cheese, snack foods, meat, fish, dairy)	Low in total fat or saturated fat	Reduced risk of cancer and heart disease.
Food containing sugar alcohols in place of sugar (gum, candies, beverages, snack foods)	Sugar alcohols	Reduced risk of tooth decay
Oat meal/oat bran/ whole oat products	Beta glucan soluble fiber	Reduce cholesterol
Milk-low fat	Calcium	Reduced risk for osteoporosis
Vegetables and fruits	Vitamins, phyto-chemicals, fiber	Reduced risk of cancer and heart disease.
Cereal with added folic acid	Folic acid	Reduced risk for neural tube defect
Juice, pasta, rice, snack bars and other foods with calcium	Calcium	Reduced risk for osteoporosis
Psyllium-containing products (e.g. pasta, bread, snack food)	Psyllium fiber	Reduced risk of heart disease

Source: Position of the American Dietetic Association: functional foods.
Journal of The American Dietetic Association 99:1278-1285, 1999.

polysaccharides, and synthetic polysaccharides are also considered dietary fiber. Except for lignin, dietary fibers are carbohydrate in nature. Epidemiological studies have indicated that a high fiber intake exerts preventive effects against most of the chronic diseases such as cancer, heart disease and diabetes (NIDDM).

Dietary fibers are classified as water soluble or gel forming viscous fibers and water insoluble fibers having very different physiological actions. Insoluble or non-digestible dietary fibers such as cellulose and lignin decrease gut transit time and increase fecal bulk. They reduce fat absorption by binding to bile acids that are needed for emulsification of fat globules. Insoluble fibers are hydrophobic and can absorb hydrophobic carcinogens such as pyrene derivatives or hetrocyclic aromatic amines and thus prevent colon carcinogenesis. Being insoluble they are very poorly fermented by the colonic microflora and are thus almost not metabolized. Whole grains are the major food sources of dietary insoluble fibers.

Soluble fibers include pectin, resistant starches and other polysaccharides. They are not digested in the small intestine, increase the transit time through the gut, delay gastric emptying, and slow glucose absorption. They can be degraded by colonic bacteria and thus lower luminal pH, stimulate proliferation of colonic microflora (prebiotic effect) and produce short chain fatty acids (SCFA) such as acetic, butyric and propionic acid. The SCFA exert a beneficial effect on the health of the colon by providing nourishment to the colon. The effects of soluble fibers are related to viscosity rather than fecal bulking. Fruits, vegetables and oats are the main dietary sources of soluble fiber. Table 8-2 shows the physiological effects of dietary fibers. About 60 to 75% of the dietary fiber in a typical mixed-food-diet are insoluble.

Dietary guidelines for maintaining good health and prevention of chronic diseases recommend inclusion of about 30 g fiber per day or 10-13 g dietary fiber per 1000 kcal. Fruits and vegetables, and bread and cereals are the two main food groups that contribute soluble and insoluble fiber respectively to the diet. These have been divided into high, medium and low fiber food categories. Diets for healthy eating recommend consumption of dietary fiber at about 30 g per day of which 50% fiber intake from grains, 30% from vegetables and beans and 20% from fruits (Table 8-3).

Pro- and Prebiotics

The human intestinal tract is greatly influenced by functional foods. The intestinal microflora represents a rich ecosystem composed of a wide range of microorganisms that play an important role in influencing the health of the host. Some strains have pathogenic effects, whereas others are considered to promote

Table 8-2

Physiological Effects of Common Dietary Fibers

Effects	Soluble Fiber			Insoluble Fiber		
	Pectin	Gum	Oat Bran	Wheat Bran	Cellulose	Lignin
Cholesterol Lowering	↓	↓	↓	↓/↑	↓/↑	↓/↑
Intestinal Absorption	↓	↓	↓	↓/↑	↓/↑	↓/↑
Gut Transit Time	↑	↑	↑	↓	↓	↓
Fecal Bulking	↑	↑	↑	↑	↑↑	↑↑
Bile acid Excretion	↑	↑	↑	↑↑	↑↑	↑↑
Carcinogen Binding	↓/↑	↓/↑	↓/↑	↑	↑↑	↑↑
SCFA Production	↑	↑↑	↑	↓/↑	↓/↑	↓/↑

health. *Clostridia*, *staphylococci*, *vibrionaceae* and *Ps. aeruginosa* are the main pathogenic bacteria involved in the production of toxins and carcinogens and that can cause diarrhea, infections, liver damage, cancer and intestinal putrefaction. *Lactobacilli* and *Bifidobacteria* are main colonic bacterial species which are thought to promote health and are referred to as probiotics. For obvious reasons, there is interest in increasing the number and activity of probiotics in the gut. Foodstuffs provide the principle growth substrates for these colonic bacteria, they influence microfloral composition and thus have a major role in the functional food concept. Prebiotics are dietary components that promote the growth of probiotics. Probiotics and prebiotics are the main dietary approaches to selectively influence the growth of beneficial bacteria.

Probiotic
A probiotic is a live bacteria contained in a food (e.g. functional food such as yogurt) or a food supplement which beneficially affects the host by improving its intestinal microbial balance. Today many different microorganisms are added to yogurts for

Table 8-3	
Guidelines for Fiber Intake on Fiber First Diet©	
Adult	**Children**
Breakfast	
Wheat Bran Cereal (8 g fiber)	2 cup wheat bran cereal (4 g fiber)
1 fruit (4 g fiber)	2 fruits (2 g fiber)
1 slice whole wheat toast (2 g fiber)	
Lunch	
2 slice whole wheat bread (4 g fiber)	2 slice whole wheat bread (4 g fiber)
1 fruit (3 g fiber)	1 fruit (3 g fiber)
Snack	
	1/2 cup fruit (2 g fiber)
Dinner	
1 cup vegetable or legume (4 g fiber)	1/4 cup vegetable or legume (1 g fiber)
1 baked potato w/skin (4 g fiber)	1 baked potato no skin (2 g fiber)
2 tomatoes (1 g fiber)	2 cup fruit (2 g fiber)
1 cup lettuce (1 g fiber)	
Total dietary fiber = 31 g	**Total dietary fiber = 20 g**

The listed foods are fiber-containing foods which should be complemented with other foods and drinks to complete the dietary requirements.

Source: Williams et al. Diet and cancer prevention: The Fiber First Diet. Toxicological Sciences 52:72S-86S, 1999.

their probiotic potential. These include: a) Lactobacilli, b) Gram positive cocci, and c) Bifidobacteria. The probiotic supplemented should thrive in the gastric environment and remain metabolically active. Probiotics stimulate the growth of beneficial bacteria and inhibit the growth of harmful bacteria. They also stimulate the immune system, help in the absorption of certain ions and the synthesis of vitamins such as B vitamin and folic acid.

Prebiotic
A prebiotic is a non-digestible food ingredient that beneficially affects the host by selectively stimulating the growth and/or activity of bacteria in the colon that can improve host health. Inulin and oligofructose are described as prebiotics because they are non-digestible and selectively stimulate the growth of potentially health promoting intestinal bacteria. They are present as plant storage carbohydrates in a number of vegetables and plants including wheat, onion, banana, garlic and chicory. Inulin and oligofructose are respectively long chain, and short and medium chains of β-D fructans in which fructosyl units are bound by β 2-1 osidic linkage and thus remain undigested in the small intestine. Prebiotics are often used in combination with probiotics. These combinations have synergistic effects, referred to as symbiotic, because in addition to the prebiotic action they promote the growth of existing strains of beneficial bacteria in the colon. Inulin and oligofructose also improve the survival, implantation and growth of newly added probiotic strains. These symbiotic concepts are currently being used by many dairy drink and yogurt manufacturers. However, a recent report has indicated adverse immune reactions of inulin and has cautioned its indiscriminate use.

Long Chain Polyunsaturated Fatty Acids
Lipids are essential components of our diets. They are required for normal growth and function. Essential fatty acids such as linoleic acid and linolenic acid are critical components of lipids. They belong to the omega-3 and omega-6 series of fatty acids. The examples of physiologically important long chain poly-unsaturated fatty acids (LCPUFA) are eicosapentaenoic acid (EPA), docosahexaenoic acid (DHA), and arachidonic acid (ARA). These are produced by desaturation and elongation of fatty acids or obtained from the diet.

Omega-3 fatty acids are important structural components of cellular membranes in the body. These LCPUFA, particularly DHA, are especially rich in the retina, brain and spermatozoa making up to 36.4% of total fatty acids. They provide membrane fluidity, which is vital for proper functioning of these tissues. They are necessary from conception through pregnancy, infancy and throughout whole life. These omega-3 fatty acids play an important role in the prevention and modulation of several common adult diseases such as cardiovascular disease, cancer, inflammation and rheumatoid arthritis, autoimmune disorders, asthma, Crohn's disease, and hypertension. Their deficiency in the retina can result in decreased vision. Both omega-3 and omega-6 LCPUFA are vital for brain growth and functional development of infants. Both neural integrity and function can be permanently disrupted by an inadequate supply of these fatty acids during fetal and neonatal development.

Estimates of Paleolithic nutrition suggest that humans originally consumed a diet low in saturated fatty acids and rich in LCPUFA. Their diet contained a 1:1 ratio of omega-6 to omega-3 fatty acids. The current Western diet is very high in omega-6 fatty acids with an omega-6 to omega-3 ratio of 20:1. This imbalance may result in a deficiency of omega-3 fatty acids. Although some omega-6 fatty acids are essential, their excess intake stimulates the formation of ARA, the precursor of prostaglandins and other eicosanoids that are involved in inflammation. The eicosanoids that are formed from ARA are biologically active in small quantities, but in large amounts, they contribute to the development of allergic and inflammatory disorders. The World Health Organization is now recommending a ratio of between 3:1 and 4:1 for omega-6 to omega-3 fatty acid intake. The Canadian Nutrition Recommendations suggest infant dietary intakes of total omega-3 fatty acid of 1% of energy of which at least half should be ALA.

The major dietary sources of omega-3 fatty acids are fish (contains EPA and DHA) and vegetable oils (contain ALA). Nuts and seeds, vegetables and some fruits, egg yolk, poultry and meat, and grains contain significant quantities of omega-3 fatty acids

and collectively contribute to the diet. It is also present in breast milk Table 8.4 lists the omega-3 fatty acid content of commonly consumed seafoods.

In the US, recommendations for adult dietary intake of PUFA is 7% (not to exceed 10%) of total energy intake. The recommended LCPUFA intakes are not universal. The most recent DRIs for linoleic acid are 17 g/d for men and 12 g/d for women. For alpha-linolenic acid, the DRIs are 1.6 g/d and 1.1 g/d respectively. Canada recommends a total omega-3 fatty acid intake of 1.2-1.6 g/d but does not distinguish between individual omega-3 fatty acids. The United Kingdom recommends 1% of total energy intake from ALA (omega-6) and 0.5% of total energy from EPA and DHA combined (omega-3) or an intake of 0.2 g/d. Australia recommends an increased intake of omega-3 fatty acids from plant food. A combined intake of 0.8 g/d or 0.27% of energy is recommended for EPA and DHA by the NATO Advanced Workshop on LCPUFA. Table 8-5 provides a listing of approximate quantities of omega-3 sources to meet current Canadian and U.S. recommendations.

Table 8-4	
Omega-3 Fatty Acid Content of Selected Seafoods	
Seafood	**Omega-3 fatty acid ALA, EPA and DHA total (g/100g)**
Mackerel	1.8-5.3
Herring	1.2-3.1
Salmon	1.0-1.4
Tuna	0.5-1.6
Trout	0.5-1.6
Halibut	0.4-0.9
Shrimp	0.2-0.5
Cod	0.2-0.3

Source: Kris-Etherton et al. Polyunsaturated fatty acids in the food chain
in the United States. Am J Clin Nutr 71S:179S-188S, 2000.

Table 8-5

**Approximate Quantity of Fish or Vegetable Oil
(high in omega-3 fatty acids) Needed to Meet
the Current Recommendations**

|	Canadian Recommendations 1.2-1.6 g/d omega-3 fatty acids	U.S. Recommendations ALA 2.2 g/d EPA + DHA 0.65 g/d
Fish		
Halibut	100-131	46-62
Mackerel	45-60	20-28
Herring	57-74	26-34
Salmon	90-117	42-56
Tuna	130-170	60-80
Shrimp	371-485	170-228
Oils		
Canola	14-18	24.2
Menhaden	5-7	2.6-3.4
Soybean	19-25	32.2
Walnut	12-16	21.9

Source: Kris-Etherton et al. Polyunsaturated fatty acids in the food chain
in the United States. Am J Clin Nutr 71S:179S-188S, 2000.

Phytochemicals

There is growing evidence that greater consumption of fruits and vegetables, whole grains and nuts is related to reduced risk of certain chronic diseases. Plant foods are rich sources of phytochemicals, which are non-nutritive substances that possess health- promoting effects. The chemopreventive potential of naturally occurring phytochemicals in foods is a major area of scientific interest. Phytochemicals possess an array of biochemical and pharmacological qualities like antioxidative, anticarcinogenic, antimicrobial, cholesterol lowering and antithrombotic activities.

Organosulfur Compounds

Organosulfur compounds like diallyl sulfides, thiosulfinates, sulfoxides, and S-allyl-cysteine are mainly present in allium vegetables such as garlic, chives and onions. A large number of epidemiological studies, and animal studies have suggested a protective role of garlic in the prevention of cancer and heart disease. The chemopreventive potential of garlic is attributed to the presence of organosulfur compounds. Figure 8-1 shows the chemical structure of diallyl disulfide (DADS).

Figure 8-1

Structure of Diallyl disulfide
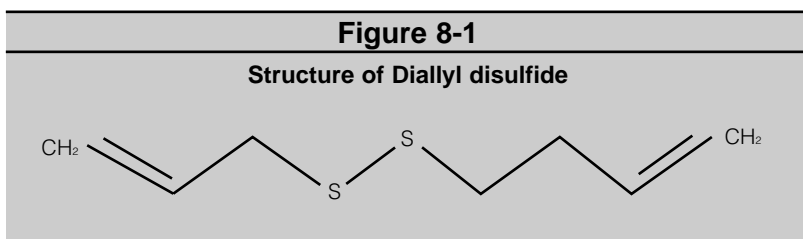
The essential oil of garlic contains 60% DADS. Intake of garlic varies substantially among populations and is generally higher in Asia than in Europe and North America. Chinese people consume as much as 24 kg garlic per year. Intake of up to 100 g of garlic extract is considered to be a maximum tolerable dose. It has, however, been suggested that consumption of excess amounts of allium vegetables could potentially induce pemphigus, a group of chronic, relapsing skin diseases.

The health benefiting properties of DADS include inhibition of platelet aggregation, and cholesterol synthesis. DADS have been shown to inhibit the growth of tumor cells by causing alterations in calcium homeostasis leading to apoptosis. Moreover, aged garlic extract has also been reported to stimulate immune functions such as proliferation of lymphocytes, cytokine release, NK activity and phagocytosis. In experimental animal studies, aged garlic extracts were found to prolong life span and prevent brain atrophy.

Flavonoids
Flavonoids are potentially bioactive polyphenols that are ubiquitously in plants and are therefore an integral part of the human diet. Phenolic compounds were traditionally considered anti-nutrients because of the adverse effects of tannins on protein digestibility. However, recent interest in food phenolics has increased greatly owing to their antioxidant potential and their possible implication in human health, especially in the prevention of cancer and cardiovascular disease.

Flavonoids are low molecular weight compounds and usually occur bound to sugar molecules. They contain several phenolic ring structures, designated as "A, B and C", containing several phenolic hydroxyl functions (Figure 8-2). Structural variations within the rings subdivide them into different subclasses, anthocyanins and anthoxanthins. Anthocyanins are red, blue and purple

pigments. Anthoxanthins are colorless or white to yellow molecules and include flavonols, flavones, flavanols and isoflavones.

Flavonols are the most widely distributed flavonoids. Quercetin, kaempferol and myricetin are the three most common dietary flavonols. Onions, apples and black tea are the major sources of dietary flavonols. Celery is a rich source of two important **flavones**, luteolin and apigenin. The dietary **flavanols** catechin and epicatechin occur in combined form in green tea, or as condensed tannin polymers in fruit, legumes and grains. Their concentration is higher in immature fruits than in ripe or stored fruits. Catechins are more abundant in skin tissues of plants.

Figure 8-2

Structures of Flavonoids

Flavones

Flavonols

Flavanones

Isoflavones

Anthocyanidin

Catechins
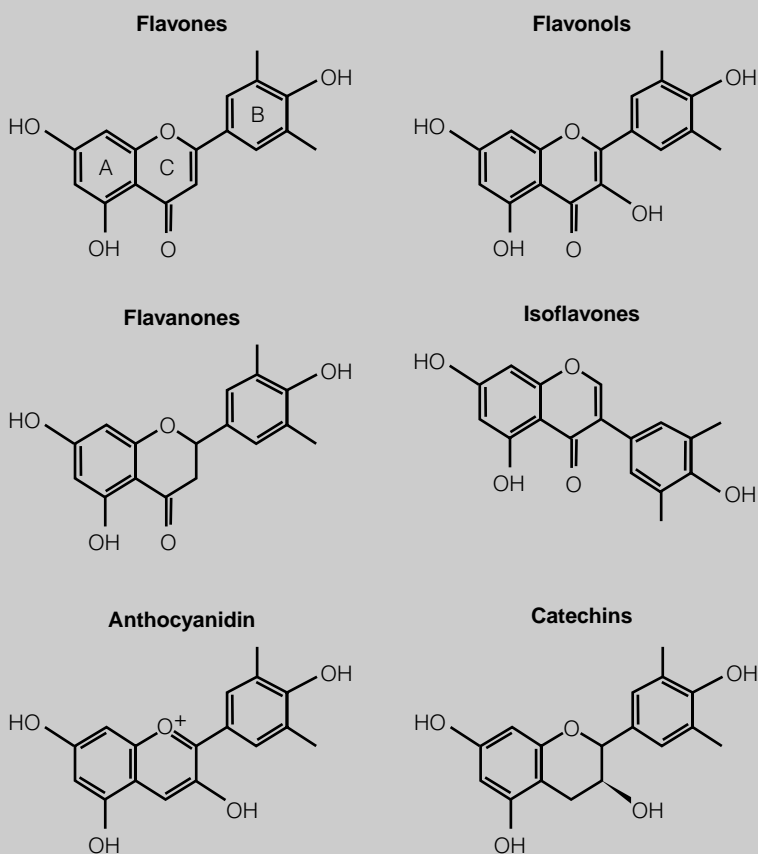
Isoflavones are found exclusively in the legume family and in high amounts in soybeans. Genistein and daidzein are the most common isoflavones present in soybeans and soy-foods **Anthocyanidins** are mainly found in cherries and grapes.

The levels of flavonoids in food are greatly influenced by genetic factors, environmental conditions, and other factors such as germination, degree of ripeness, variety, food processing and storage. These flavonoids are partially responsible for the sensory and nutritional qualities of plant foods. Table 8-6 presents the flavonoid contents of selected foods.

The absorption and the subsequent distribution, metabolism and excretion of flavonoids in humans is not well understood. Dietary flavonoids, except catechins, were long considered to be non-absorbable because they are bound to sugar and only sugar free flavonoids were thought to be absorbable. Moreover, hydrolysis occurs only in the colon by bacteria, which at the same time

Table 8-6			
Flavonoid content of selected foods			
Class	**Phytochemicals**	**Food or beverage**	**Quantity (mg/kg or mg/l)**
Flavonols			
	Quercetin, kaempferol, myricetin	Olives	270-830
		Onions	347
		Kale	321
		Lettuce	308
		Cranberry	249
		Broccoli	102
		Apple	21-72
		Green tea leaves,dry	30-45 g/kg
Flavones			
	Apigenin, luteolin	Celery	130
		Olives	6-29
Flavanol			
	Catechin, epicatechin	Red wine	274
		Green tea leaves,dry	128-226 g/kg
		Pear	70-420
Isoflavones			
	Genistein, daidzein	Soybean, dry	888-2,407
		Soy nuts	1,437-2,363
		Tofu	280-499

Source: King A, Young G. Characteristics and occurrence of phenolic phytochemicals, J Am Diet Assoc 99:213-218, 1999.

degrade flavonoids. However recent investigations suggest about 52% of quercetin glycoside from onion is absorbable. Absorbed quercetin is eliminated only slowly from blood.

Flavonoids have been reported to have anticarcinogenic and antiatherogenic properties. In addition, they have also been shown to have vasodilatory, anti-inflammatory, antibacterial, immune stimulatory and antiviral properties. It is well known that diets rich in fruits and vegetables are protective against CVD and cancers. These protective effects have been attributed, in large part, to their antioxidant potential. Dietary flavonoids are excellent antioxidants. The phenolic hydroxyl groups attached to the ring structures confer the antioxidant activity. Isoflavones are also strikingly similar in chemical structure to mammalian estrogen and are thus considered as potential alternate therapeutic agents for a range of hormone-dependent conditions including cancer, menopausal symptoms, CVD and osteoporosis. Epidemiological studies have shown that these diseases are less prevalent among women in soy-consuming countries than those who are not.

Resveratrols are the non-flavonoid phenolic compounds, which are synthesized by plants (mainly spermatophytes such as grapes) in response to injury or fungal attack. Since it is mostly present in grape skin and not in the flesh, white wine contains very small amounts of resveratrol compared to red wine. In red wine, the concentrations generally range between 0.1 and 15 mg/l. Epidemiological observations showing an inverse correlation between red wine consumption and incidence of CVD (French paradox) sparked the interest into the biological properties of resveratrol. As a phenolic compound, it contributes to the antioxidant potential of red wine and it is thus hypothesized to play a role in the prevention of CVD. It has also been shown to modulate lipid metabolism, inhibit LDL oxidation and platelet aggregation. Resveratrol has also been shown to possess anti-inflammatory, anticancer and phytoestrogenic properties.

Saponins
Saponins are a class of naturally occurring amphiphillic compounds present in many plant foods and especially in legumes. These compounds are known to be biologically active and their role in prevention of chronic diseases is currently under investi-

gation. Saponins are thought to possess anticarcinogenic, hypoc-holesterolemic, hypoglycemic and immune-stimulating properties.

Saponins are steroidal or triterpenoid glycosides, possessing a lipid-soluble steroidal or triterpenoid aglycone (sapogenin) and water-soluble sugar residues differing in type, amount and position of the sugar. Sometimes they may have an additional functional group also attached to the sapogenin (Figure 8-3). They are widely distributed in plant foods, herbs and a particular species of marine starfish. Table 8-7 lists the major food sources and their saponin contents. The saponin contents in legumes are affected by the varietal differences and by cooking and processing operations. Average daily intake of saponins are highly variable according to the diet and range from 10-15 mg/day to about 100-110 mg/day in western or vegetarian diets respectively.

Saponins are poorly absorbed in the intestine. Scientific evidence indicates that they are either excreted unchanged or broken down by the gut microflora, intestinal enzymes and/or gastric juices releasing the free sapogenin unit. Because of their amphiphillic nature they are surface active and have emulsion-stabilizing properties. They have long been thought to influence blood lipids, especially in hyperlipidemic patients. Saponins are thought to form insoluble complexes with cholesterol in the gut and thus limit its absorption. They precipitate bile acids and facilitate their excretion in the feces and thereby exert hypoc-holestrolemic properties. The saponins have also been demonstrated to have immune modulatory, cytotoxicity and tumor cell growth inhibitory, and antioxidant effects in various in-vitro and in-vivo studies.

Carotenoids

Carotenoids are important pigments that are synthesized by plants and microorganisms but not by animals. They are present in fruits and vegetables and are thought to contribute to the inverse relationship between fruit and vegetable consumption and the risk of CVD, cancer and other chronic diseases. Carotenoids are important dietary sources of provitamin A and are antioxidant phytochemicals. In plants, carotenoids are part of the photosynthetic machinery and are responsible for the yellow, orange and red colors of fruits and vegetables.

Table 8-7

Saponin Content of Selected Beans and Vegetables

Beans or Vegetables	Saponin Contents (g/kg)
Beans	
Broad beans	3.5
Broad beans, canned	3.1
Chickpea	0.8 – 60.0
Cowpea	1.2
Faba bean	4.3
Green bean	13.0
Kidney bean	1.7 – 3.5
Lentil	1.1 – 5.1
Lima bean	1.1
Moong bean	0.5 – 5.7
Navy bean	4.5 – 21.0
Pea	1.8
Peanut	1.0 – 16.0
Soybean	2.4 – 43.0
Soy flour	4.7 – 5.3
Soy protein isolate	8.1
Soy milk	2.6 – 3.9
Soy tofu	3.0 – 3.3
Soy miso	1.5
Vegetables	
Cauliflower	24.0
Garlic	0.9 – 2.9
Mustard	4.6
Sesame	3.0
Spinach	23.5 – 47.0

Source: Rao AV, Gurfinkel D. Saponins in human health, In: Oleszek W, Marston A (eds.)
  Saponins in Food, Foodstuffs and Medicinal Plants.
  Kluwer Academic Publishers, Dordrecht, 2000.

There are more than 600 carotenoids found in nature, about 40 of these are present in a typical human diet and about 20 have been identified in blood and tissues. β-carotene, α-carotene, lycopene, α-cryptoxanthin and lutein are the most common carotenoids, accounting for over 90% of the carotenoid content of our diets and our body stores. All carotenoids posses certain common chemical features: a polyisoprenoid structure, a long conjugated chain of double bonds in the central position of the molecules, and a near bilateral symmetry around the central dou-

ble bond. Modifications in the base structure by cyclization of the end groups and by introduction of oxygen functions yield different carotenoids (Figure 8-4). Carotenoids, because they are rich in conjugated double bonds, can undergo *cis-trans* isomerization. The *trans* form, which is more stable, is the most common form in most foods. Very little is known about the specific roles and relative importance of *cis-trans* isomers of carotenoids in humans.

Dietary carotenoids are mostly provided by deeply pigmented vegetables and fruits. The carotenoid pattern of common fruits and vegetables is shown in Table 8-8. In general, yellow-orange vegetables and fruits provide most of the dietary β-carotene and

Figure 8-4

Structure of Carotenoids

ß-Carotene

α-Carotene

Lycopene

ß-Cryptoxanthin
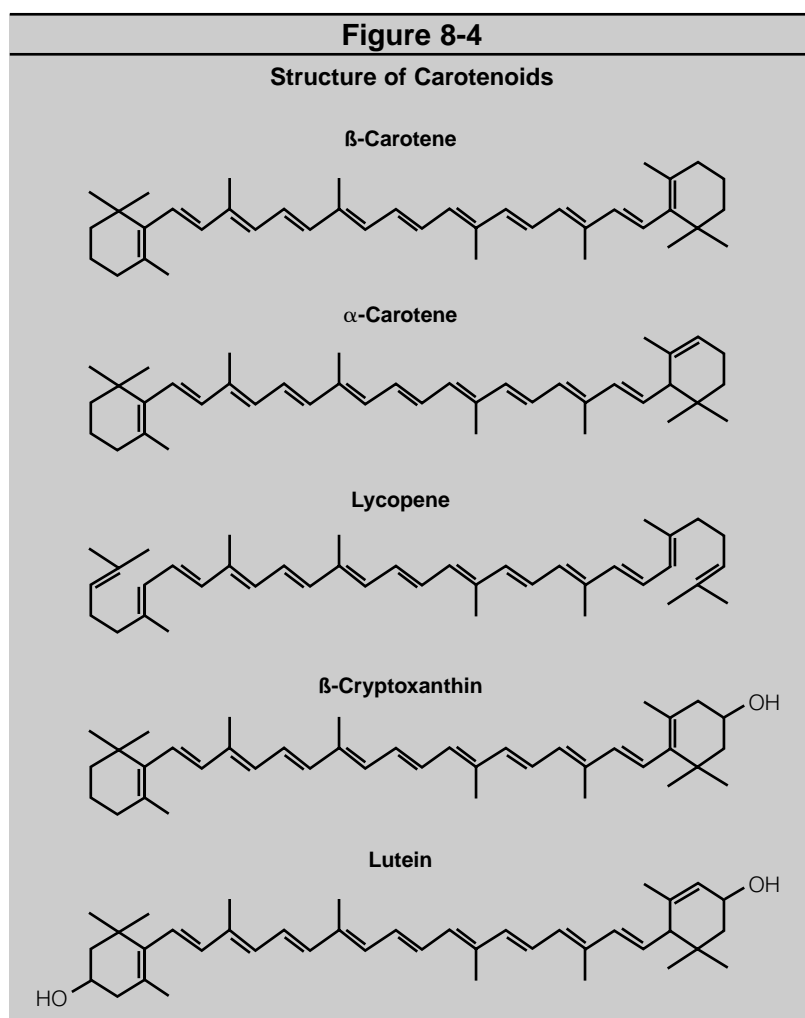
Lutein

α-carotene; orange fruits provide α-cryptoxanthin; dark green vegetables provide lutein; and tomatoes and tomato products are major sources of lycopene in the diet. Cooking and processing of foods affect carotenoid content. In general the most common household cooking methods, such as microwave cooking, steaming and boiling in a small amount of water, do not drastically alter the carotenoid contents of vegetables. However, drying, extreme heat or excessive cooking time results in their oxidative

Table 8-8

Examples of Major Contributors of Carotenoids in the North American Diet

Carotenoid	Food Source	Amount (µg/100g)
β-carotene		
	Apricot, dried	17,600
	Carrots, cooked	9,771
	Spinach, cooked	5,300
	Green Collard	5,400
	Canteloupe	3,000
	Beet Green	2,560
	Broccoli, cooked	1,300
	Tomato, raw	520
α-carotene		
	Carrots, cooked	3,723
Lycopene		
	Tomatoes, raw	3,100
	Tomato Juice	10,000
	Tomato Paste	36,500
	Tomato Ketchup	12,390
	Tomato Sauce	13,060
β-cryptoxanthin		
	Tangerine	1,060
	Papaya	470
Lutein		
	Spinach, cooked	12,475
	Green collard	16,300
	Beet, green	7,700
	Broccoli, cooked	1,839
	Green peas, cooked	1,690

Source: Clinton SK. Lycopene: chemistry, biology and implications for human health and diseases. Nutrition Review 56(2): 35-51, 1998.

U.S. Department of Agriculture, USDA Nutrient Database for Standard References, Release 13, Agricultural Research Service, 1999.

destruction. At present there is no DRI for carotenoids. Quantities of carotenoids in the diet are difficult to estimate partially because the methods used for estimation are neither sensitive nor specific. Also the given values do not always take into account the seasonal and cultural variations.

Very little is known about absorption and transport of major carotenoids except for β-carotene. In humans appreciable amounts of carotenoids are absorbed intact by mucosal cells and subsequently appear unchanged in the circulation and peripheral tissues. Food processing and cooking operations such as mechanical homogenization or heating, which release carotenoids from the food matrix, enhance their bioavailability. In the intestine, they are incorporated into micelles, formed from dietary fat and bile acids, which facilitate absorption via passive diffusion. The intact carotenoids are incorporated into chylomicrons, released into the lymphatic system and then taken up by the liver. They are incorporated into lipoproteins and released into the blood stream for transport to different tissues. Carotenoids are taken up differentially by different tissues, but little is known of the factors that determine this process. Adipose tissue is the primary storage depot for the carotenoids.

Carotenoids play important physiological roles in human health. Not all carotenoids serve as dietary sources of provitamin A. They also exhibit biological activity as antioxidants, in gap junction communication, in cell growth regulation, in modulating gene expression and immune response. Carotenoids have been implicated as protective agents against cancer, cardiovascular disease (CVD), age related macular degeneration (AMD) and cataract formation. Epidemiological evidence has suggested a positive link between higher dietary intakes and tissue concentrations and reduced risk of cancer and CVD. Recent intervention trials using high doses of β-carotene supplements did not show protective effects as hypothesized, against cancer or CVD. On the other hand, pharmacological supplementation of β-carotene increased lung cancer incidence in smokers in the Alpha Tocopherol Beta Carotene (ATBC) trial and increased mortality from CVD in a group of smokers, former smokers and asbestos-exposed individuals in the β-Carotene and Retinol Efficiency Trial (CARET) trial. It appears that carotenoids may promote

health when taken at dietary levels but may have adverse effects when taken in higher amounts under certain physiological conditions. Of all the carotenoids, lycopene has recently received the greatest interest from the scientific community.

Lycopene

Lycopene is a carotenoid naturally present in a number of fruits and vegetables. However, tomatoes and tomato products are the main sources of lycopene in the North American diet. Lycopene gives the tomato its red color. Among the carotenoids, lycopene is a major component found in human serum and tissues. In several recent studies, dietary intakes of tomatoes and tomato products containing lycopene have been shown to be associated with decreased risk of chronic diseases such as cancer and cardiovascular disease. Lycopene was identified as the main phytochemical in tomatoes responsible for the protective effects observed. Unlike β-carotene, it does not have provitamin A activity. It is one of the most potent antioxidants among dietary carotenoids. It is readily absorbed from different food sources, present in different tissues and maintains its antioxidant properties in the body. It is suggested to have anti-cell proliferative, anticarcinogenic and antiatherogenic activities. Epidemiological, animal and human studies have provided evidence in support of its protective role in heart disease, cancer and other chronic diseases. Several epidemiological studies have indicated that higher intakes of tomatoes or tomato products as well as high serum levels of lycopene are related to reduced risk for several human cancers and CVD. Although the antioxidant properties of lycopene are thought to be primarily responsible for its biological effects, evidence is accumulating to suggest other mechanisms such as modulation of intercellular gap junction communication, hormonal and immune system and metabolic pathways may also be involved.

Table 8-9 lists various phytochemicals and the health benefits associated with fruits and vegetables by color.

Table 8-9

Functional Components of Fruits and Vegetables by Color

Functional Component	Foods	Health Benefits
RED		
Lycopene	Tomatoes and tomato products Pink grapefruit Watermelon	• Reduces risk of heart disease, cancer, and asthma • Improves eye health and male fertility
Anthocyanins	Strawberries Cherries Pomegranates Apples Red onion	• Reduces risk of cancer and heart disease
Ellagic Acid	Strawberries Pomegranates	• Reduces risk of cancer
Quercetin	Tomatoes Cherries Apples Red onion	• Reduces risk of cancer and heart disease
Chlorogenic acid	Tomatoes Cherries Pomegranates Red peppers	• Inhibits nitrosamine formation (prevention of cancer)
Betacyanins	Beets	• Cancer 'fighting' properties
ORANGE		
Beta-carotene	Carrots Sweet potato Mangoes Cantaloupe Pumpkins Apricot Squash	• Improves vision (night) • Reduces risk of cancer
Alpha-carotene	Pumpkins Cantaloupe Carrots	• Reduces risk of cancer
Lutein	Carrots Oranges Pumpkins	• Improves heart health • Reduces risk of cancer
Beta-cryptoxanthin	Carrots Oranges Mango	• Improves heart health • Reduces risk of cancer
Limonoids	Oranges	• Reduces risk of cancer

Continued on next page

Table 8-9 *Continued*

Functional Components of Fruits and Vegetables by Color

Functional Component	Foods	Health Benefits
YELLOW		
Lutein/Zeaxanthin	Yellow potatoes Corn	• Improves eye health (reduces risk of aging macular degeneration) • Reduces risk of heart disease and cancer
Quercetin	Bananas Yellow grapefruit	• Reduces risk of cancer and heart disease
Limonoids	Lemon, yellow grapefruit	• Reduces risk of cancer
Alpha-lipoic acid	Yellow potatoes	• Reduces oxidative stress (reduces risk of cancer)
Ferulic acid	Yellow potatoes, yellow grapefruit,corn, pineapple	• Reduces the risk of cancer
GREEN		
Lutein/Zeaxanthin	Spinach Avocados Asparagus Broccoli Kale Watercress Brussel sprouts Kiwi	• Improves eye health (prevents aging macular degeneration) • Reduces risk of heart disease and cancer
Sulforaphane	Broccoli Watercress Kale	• Reduces risk of cancer
Glutathione	Asparagus Avocados Spinach	• Reduces oxidative stress (reduces risk of cancer)
BLUE-PURPLE		
Anthocyanins	Blueberries Blackberries Grapes Plums Prunes Eggplant	• Reduces risk of cancers and heart disease
Chlorogenic acid	Blueberries Eggplant Blackberries Grapes Plums Prunes	• Inhibits nitrosamine formation (cancer prevention)
Ellagic acid	Blackberries	• Reduces risk of cancers
Quercetin	Blueberries Raisins Blackberries Grapes	• Reduces risk of cancer • and heart disease

Herbal and Botanical Supplements

The increasing concerns for general health, chronic disease prevention and aging has generated consumer interest in alternative therapies, one of which is herbal remedies. It is estimated that 1 of every 3 Americans uses at least one unconventional therapy per year. Unconventional therapies are mostly used for chronic rather than life-threatening conditions. The WHO estimated that for primary health care, 80% of the world's population relies on traditional medicine involving the use of plant extracts or their active components. Many of today's conventional drugs also had their origins in plants. In herbal medicine the term 'herb' is used loosely to refer to herbaceous plants and also to barks, roots, leaves, seeds, flowers, and fruits of trees, shrubs and woody vines.

Herbs have been used as sources of nutrients and for medicinal purposes for centuries. Self prescribed herbs are used today for several common ailments and conditions such as anxiety, arthritis, colds, coughs, constipation, fever, headaches, infections, insomnia, intestinal disorders, premenstrual syndrome, stress, ulcers, and general malaise. Some of the more popular herbs used today are aloe vera, Echinacea, feverfew, garlic, ginseng, ginkgo, and St. John's wort. Herbs, such as basil, caraway, cilantro, coriander, cumin, dill, oregano, rosemary, sage and thyme, are rich in essential oils and oleoresin and therefore are also used to flavor foods. Some herbs such as saffron, paprika, and turmeric are used to color food. Table 8-10 provides a list of commonly used herbs, their usage and effectiveness.

Herbs for cardiovascular diseases

A plant based diet, rich in fruits, vegetables, legumes and grains, low in saturated fat, and high in dietary fiber is generally recommended for cardiovascular problems. In addition there are a few herbs available that may provide help in cases of hyperlipidemia, impaired blood flow, excess blood clotting and other problems related with the development of CVD. Garlic (*Allium sativum L*) has been suggested to be effective against CVD because it lowers cholesterol, increases fibrinolytic activity and inhibits platelet aggregation. Ginkgo (*Ginkgo biloba*) extract and hawthorn (*Crataegus supp.*), which are rich in flavones, are used to improve blood flow. Some hypercholesterolemic patients have shown improvement from using psyllium (*Plantago psyllium*).

Herbs for immune systems

Several herbs, such as garlic, *Echinacea*, licorice and cat's claw that are rich in flavonoids, vitamin C or carotenoids may enhance immune function. The flavonoid-rich herbs may also have antiinflammatory properties. *Echinacea* (purple coneflower) has been used to treat symptoms of the common cold, flu and sore throat. Cat's claw (*Uncaria*) is from northern regions of South America and has immunostimulant properties and has attracted much attention in treating AIDS and leukemia patients.

Herbs with anticancer activity

The National Cancer Institute has identified cancer-preventing properties of several commonly used herbs. These herbs include members of *Allium sp* (garlic, onions and chives), mint family (basil, mints, oregano, rosemary, sage, and thyme), Zingiberaceae family (ginger and turmeric), *Umbelliferae* family (anise, caraway, celery, chervil, cilantro, coriander, cumin, dill, fennel, and parsley), licorice root, green tea, flax and tarragon. These herbs contain a variety of phytochemicals which have been shown to be chemopreventive. These phytochemicals inhibit tumor formation by their antioxidant properties, stimulating phase I or II enzymes, inhibit hormonal actions and metabolic pathways associated with the development of cancer.

Table 8-10		
Commonly Used Herbs and Their Uses		
Herb	**Use**	**Some side effects**
Echinacea	Immune stimulant	No documented side effects, should not be taken continuously for more than 8 weeks
Feverfew	Migraine headache	Not recommended during pregnancy, induces menstruation, caution with anticoagulants.
Garlic	Atherosclerosis	May increase postoperative bleeding
Ginkgo	Cerebral insufficiency	Transient headache, caution with aspirin
Ginseng	Stimulant, aphrodisiac	hypoglycemic
St. John's wort	Depression	Caution with other antidepressants

Safety Issues

Today, herbalism is gaining attention as 'natural' or alternative therapy for health and healing. A significant number of Americans self-medicate with herbal preparations for preventive or therapeutic purposes. Herbs are often assumed safe because they are natural. However, many medicinal herbs are therapeutic at one dose and toxic at another. Herbs may also interact with pharmaceutical drugs and these interactions may either increase or decrease the pharmacological or toxicological effects of either component. Several safety concerns exist with herbal products. These include lack of safety data, absence of quality-control requirements for potency and purity and lenient labeling standards. Since Canada, the Unites States and most other countries do not have a regulatory system for herbal products, physicians and consumers must therefore educate themselves about the risks of using herbal remedies. Although discriminate and proper use of some herbal products may be safe and may provide some therapeutic benefits, indiscriminate or excessive use of herbs can be unsafe and even dangerous.

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