

Unit 1 – Understanding Micronutrient Nutrition

Welcome to the first unit of this module on *Micronutrient Malnutrition*. We hope that you will find it stimulating and relevant to your field of practice. The overall aim of Unit 1 is to introduce you to micronutrients and their role in health and disease.

In 1998, Carol Bellamy, Executive Director of UNICEF wrote:

There is no one kind of malnutrition. It can take a variety of forms that often appear in combination, and contribute to each other ... Many involve deficiencies of ‘micronutrients’ – substances like vitamin A and iodine that the human body cannot make itself, but that are needed, often only in tiny amounts, to orchestrate a whole range of essential physiological functions.

Each type of malnutrition is the result of the complex interplay of factors involving such diverse elements as household access to food, child and maternal care, safe water and sanitation and access to basic health services.

And each wreaks its own particular kind of havoc on the human body (UNICEF, 1998: 10).

Before reading any further, consider this question: Why do you think micronutrient malnutrition is sometimes called “the hidden hunger”?

This is because micronutrients, which include the whole range of less visible vitamins and minerals, are essential to human health. As Carol Bellamy notes above, the human body cannot synthesise them itself and therefore humans need to *take in* micronutrients through their diet. Without micronutrients, people develop deficiency diseases over time. These may have a negative impact on growth, on mental development and on peoples’ quality of life. So without knowing it, your body may be “hungering for” one of these less visible sources of nutrition. For example, people may have an adequate intake of carbohydrates, proteins and fats, but an inadequate intake of a particular micronutrient.

During the last two decades, the importance of micronutrients in Public Health has been recognised. Research has increasingly focused on understanding the physiological role of micronutrients and the health consequences of micronutrient deficient diets. This knowledge is essential at local, national and global levels: firstly it has been important for establishing dietary requirements and planning targeted intervention programmes to address micronutrient deficiencies; secondly, this knowledge is a starting point for decreasing the burden of micronutrient malnutrition globally; and leading on from this, such

research has helped to develop indicators describing the severity of micronutrient deficiencies in the Public Health context.

The aim of Unit 1 is to provide you with a basic understanding of micronutrients - what they are and how they function. We address good food sources of the different micronutrients and how much humans need for health, or for preventing deficiency and disease. Those of you coming from previous training in nutrition may be able to move through this unit quite quickly.

In Public Health Nutrition, there are certain micronutrients that are considered more critical than others. This is because of the high global prevalence of deficiency of these micronutrients and the resulting impact. These micronutrients will be discussed in more detail.

There are three Study Sessions in this Unit:

Study Session 1: Micronutrients: The Technical Background.

Study Session 2: Key Micronutrients in Public Health Nutrition.

Study Session 3: Micronutrients: Dietary Requirements.

In Session 1, you will be introduced to all the micronutrients, their names, definitions, classifications and food sources. In Session 2, the most critical micronutrients in Public Health will be covered, i.e. vitamin A, iron, iodine and zinc. These micronutrients will be discussed in detail, focusing on their physiology, biochemistry, food sources, clinical signs and manifestations of deficiency and toxicity. In Session 3, issues concerning the recommended intake of micronutrients will be addressed. Two sets of recommendations, the Dietary Reference Intakes (DRIs) and the recommendations of the World Health Organisation (WHO) will be discussed.

Learning Outcomes of this Module

By the end of this unit, you should be able to:

- Define micronutrients.
- Name and classify all the vitamins and minerals.
- Describe the different functions of all the vitamins and minerals.
- Identify food sources of all the vitamins and minerals.
- Identify the important micronutrients in Public Health Nutrition and motivate their importance.
- Understand the biochemistry and physiological roles of vitamin A, iron, iodine and zinc.
- Describe the food sources of vitamin A, iron, iodine and zinc.
- Discuss the factors that influence the bioavailability of vitamin A, iron and zinc.
- Describe the clinical signs of deficiency and the deficiency diseases of vitamin A, iron, iodine and zinc.
- Discuss the clinical manifestations of toxicity of vitamin A, iron, iodine and zinc.
- Understand the concept and scientific basis of the different recommended intakes (RIs), namely the Dietary Reference Intakes (DRIs) and the WHO recommendations.
- Apply the two different recommended intakes, i.e. Dietary Reference Intakes and the WHO recommendations on intakes of micronutrients.

Before starting Session 1, look back at the two assignment topics in the Module Introduction. As you work through the sessions, bear these assignments in mind and prepare while you study. To do this, you could make notes on issues that are relevant to the assignment and record the page numbers of helpful sections of the readings. This will enable you to study with more focus, and ensure that you can work effectively when you start the assignments.

We trust that you will find the unit stimulating and challenging and that it will enable you to gain the knowledge and competence you need to address micronutrient malnutrition in the course of your work in Public Health.

References

UNICEF. (1998). Ch 1 - Malnutrition: Causes, Consequences and Solutions. In Bellamy, C. *The State of the World's Children 1998*. Oxford University Press for UNICEF, New York: 7 - 35.

Unit 1 - Session 1

Micronutrients: A Technical Background

Introduction

Food provides humans with energy and nutrients that are essential for growth, and the development and maintenance of health and life – hence the popular statement: “We are what we eat”. All the food that humans consume consists of macronutrients, micronutrients and water. The macronutrients consist of carbohydrates, proteins and fats, which yield energy and are needed by the human body in recommended amounts, measured in grams. Micronutrients yield no energy, yet they are essential in body function regulation and are needed in amounts measured in milligrams or micrograms, hence they are termed *micronutrients*. In this session we explore micronutrients, in more depth, considering their sources and functions.

Session Contents

- 1 Learning outcomes of this session
- 2 Readings
- 3 Vitamins and minerals: nomenclature and classification
- 4 Functions and significance in human health
- 5 Dietary sources of micronutrients
- 6 Session summary
- 7 References and further reading

Timing of this session

This session contains two readings and three tasks. It should take you about three hours to complete. A logical break is at the end of Section 3.

1 LEARNING OUTCOMES OF THIS SESSION

By the end of this unit, you should be able to:

- Define micronutrients.
- Name and classify all the vitamins and minerals.
- Describe the different functions of all the vitamins and minerals.
- Identify food sources of all the vitamins and minerals.

2 READINGS

You will be referred to the following readings in the course of this session. Use the author's initial to find the page numbers in the alphabetical index of the Reader.

Author/s	Reference details
Gallagher, M. L.	(2004). Ch 4 - Vitamins. In L. K. Mahan & S. Escott-Stump. (eds). <i>Krause's Food, Nutrition & Diet Therapy</i> . 11th Edition. Pennsylvania, USA: Elsevier: 75 - 119.
Anderson, J. J. B.	(2004). Ch 5 - Minerals. In L. K. Mahan & S. Escott-Stump. (eds). <i>Krause's Food, Nutrition & Diet Therapy</i> . 11 th Edition. Pennsylvania, USA: Elsevier: 120 - 163.

3 VITAMINS AND MINERALS: NOMENCLATURE AND CLASSIFICATION

There are two types of micronutrients: vitamins and minerals. Vitamins and minerals are essential nutrients, needed for normal physiological functioning of the human body. *Essential* means that the human body cannot synthesise vitamins and minerals in amounts adequate to meet physiological needs, and therefore micronutrients must be taken in through the diet. Both vitamins and minerals cause specific deficiency syndromes over time, if not consumed in adequate amounts.

Vitamins

Vitamins are organic compounds, classified into two groups, based on their solubility. These are - fat soluble or water soluble vitamins. Vitamins have very specific functions in the human body, which can be summarised as follows:

- They act as constituents of co-enzymes and hormones, ensuring regulation of physiological processes, e.g. the digestive process.
- They facilitate respiration as electron donors and acceptors.

- They act as membrane stabilisers, ensuring that membranes function optimally.

Minerals

Minerals are divided into macro-minerals and micro-minerals. They are found in the human body either in *ionic form* or as components of organic compounds. Minerals are structural in blood, cell membranes and bones. In addition, their functions include maintenance of acid-base balance and osmotic pressure, enzyme regulation and maintenance of nerve and muscular irritability (Anderson, 2004).

The tasks in this session might seem very basic and simple, but they serve as revision or to equip you with a basic knowledge of micronutrients. Use these chapters from Mahan & Escott-Stump's book *Krause's Food, Nutrition & Diet Therapy* (2004) to complete the tasks. Actively engaging in these tasks is worth doing, as it will help you to internalise the information you need for this module. Merely reading the Feedback is probably a waste of your time.

READINGS

Gallagher, M. L. (2004). Ch 4 - Vitamins. In L. K. Mahan & S. Escott-Stump. (eds). *Krause's Food, Nutrition & Diet Therapy*. 11th Edition. Pennsylvania, USA: Elsevier: 75 - 119.

Anderson, J. J. B. (2004). Ch 5 - Minerals. In L. K. Mahan & S. Escott-Stump. (eds). *Krause's Food, Nutrition & Diet Therapy*. 11th Edition. Pennsylvania, USA: Elsevier: 120 - 163.

TASK 1 - MAKE A TABLE OF ALL THE VITAMINS AND MINERALS

Using the above chapters for reference:

a) Make two tables each with four columns; one table should be headed *Vitamins*, the other *Minerals*. You are only going to use two columns for this task.

(Cont'd)

Table 1: Vitamins

Name	Fat or water soluble?		
Vitamin A			
Carotenoids			
Vitamin D			
Vitamin E			
Vitamin K			
Thiamin			
Riboflavin			
Niacin			
Pantothenic acid			
Vitamin B6			
Folate			
Vitamin B 12			
Biotin			
Vitamin C			

Table 2: Minerals

Name	Macro- or micro-mineral?		
Calcium			
Phosphorus			
Magnesium			
Sulphur			
Iron			
Zinc			
Copper			
Iodine			
Manganese			
Fluoride			
Molybdenum			
Cobalt			
Selenium			
Chromium			

TASK 1 - ADD DETAIL TO YOUR TABLE OF VITAMINS AND MINERALS (Cont'd)

- b) Write the name of all the vitamins in column 1 of the Vitamins table using Chapter 4 to guide you; do the same for the Minerals table using Chapter 5.
- c) In the Vitamins table, use column 2 to specify if it is fat or water soluble; in the Minerals table, specify if it is a macro- or micro-mineral.
- d) Separately, define the concepts *fat-soluble*, *water soluble*, *macro-minerals*, *micro-minerals* as each applies to micronutrients.

FEEDBACK

Your Vitamins table should be as follows:

Table1: Vitamins

Name	Classification		
Vitamin A	Fat soluble		
Carotenoids	Fat soluble		
Vitamin D	Fat soluble		
Vitamin E	Fat soluble		
Vitamin K	Fat soluble		
Thiamin	Water soluble		
Riboflavin	Water soluble		
Niacin	Water soluble		
Pantothenic acid	Water soluble		
Vitamin B ₆	Water soluble		
Folate	Water soluble		
Vitamin B ₁₂	Water soluble		
Biotin	Water soluble		
Vitamin C	Water soluble		

Definitions relevant to vitamins:

Fat-soluble vitamins: These are vitamins that are absorbed passively with fatty acids from the gastro-intestinal tract and *must* be transported with lipids (fats),

e.g. lipoproteins in blood. These are found and stored in the lipid-containing portions of cells in the body, e.g. cell membranes and lipid droplets.

Water soluble vitamins: These are vitamins that are either absorbed through active or passive mechanisms from the gastro-intestinal tract; they are transported by carriers in the water phase of blood (the blood plasma) and are not stored in the human body. Excess is excreted in urine as well as small amounts in sweat.

Table 2: Minerals

Name	Classification		
Calcium	Macro-mineral		
Phosphorus	Macro-mineral		
Magnesium	Macro-mineral		
Sulphur	Macro-mineral		
Iron	Micro-mineral		
Zinc	Micro-mineral		
Copper	Micro-mineral		
Iodine	Micro-mineral		
Manganese	Micro-mineral		
Fluoride	Micro-mineral		
Molybdenum	Micro-mineral		
Cobalt	Micro-mineral		
Selenium	Micro-mineral		
Chromium	Micro-mineral		

Definitions relevant to minerals:

Macrominerals are also called *bulk elements*, and are required in amounts of 100mg/day or more. Microminerals are also called trace elements and are required in amounts less than 15mg/day or in microgram quantities.

4 FUNCTIONS AND SIGNIFICANCE IN HUMAN HEALTH

Each of the different vitamins and minerals has a specific function in the human body. These functions usually have to do with the maintenance of the immune system, producing the essential components of enzymes and hormones and maintaining the functioning of specific organs, e.g. liver and eyes.

If any of the micronutrients is too low or absent in the diet over an extended period, the micronutrient levels in the blood will drop below normal levels and a deficiency will develop in the body. The deficiency will be manifest as specific symptoms and if the deficiency in the diet continues, specific clinical signs of deficiency will develop which may become a deficiency disease. The deficiency symptoms and the disease are usually linked to the functions of the micronutrient in the human body (Mahan & Escott-Stump, 2004).

TASK 2 - ADD FUNCTIONS TO YOUR TABLES OF VITAMINS AND MINERALS

Use Chapters 4 and 5 of *Krause's Food, Nutrition & Diet Therapy* (2004) again to find this information.

a) In column 3 of the tables you developed in Task 1, summarise the main functions of each specific vitamin and mineral.

FEEDBACK

Your tables should now look like the table that follows. Remember that you have these tables for reference in the future. Take note of how many of the vitamins and minerals are involved in the maintenance of immune function; also take note of which vitamins and minerals are involved in circulation, and which ones in respiration. You could colour code them. Note the role of the different B vitamins in energy metabolism.

You are not expected to memorise this information, although it will be important to become very familiar with the characteristics of key micronutrients that we will deal with in Session 2.

Table 1: Vitamins

Name	Classification	Functions	Good and excellent food sources
Vitamin A	Fat soluble	<ul style="list-style-type: none"> - Essential for normal growth - Development and maintenance of epithelial tissue - Essential for night vision - Required for normal bone development - Functions as an antioxidant - Builds immune system 	
Carotenoids	Fat soluble	<ul style="list-style-type: none"> - Functions as a provitamin A - Functions as a phyto-chemical 	
Vitamin D	Fat soluble	<ul style="list-style-type: none"> - Functions as a pro-hormone - Supports normal bone development - Phosphorus and calcium metabolism 	
Vitamin E	Fat soluble	<ul style="list-style-type: none"> - Functions as an antioxidant - Protects red blood cells from hemolysis - Maintains epithelial tissue 	
Vitamin K	Fat soluble	<ul style="list-style-type: none"> - Normal blood clotting - Bone metabolism 	
Thiamin	Water soluble	<ul style="list-style-type: none"> - Energy metabolism - Normal growth - Maintains nervous system 	
Riboflavin	Water soluble	<ul style="list-style-type: none"> - Normal growth - Energy metabolism - Functions as a co-enzyme 	
Niacin	Water soluble	<ul style="list-style-type: none"> - Part of the enzyme system - Fat synthesis - Promotes glycolysis 	

Pantothenic acid	Water soluble	- Co-enzyme in metabolism of macronutrients	
Vitamin B ₆	Water soluble	- Functions as a co-enzyme - Tryptophan to niacin conversion - Normal growth	
Folate	Water soluble	- Biosynthesis of nucleic acids - Normal maturation of red blood cells - Co-enzyme	
Vitamin B ₁₂	Water soluble	- Biosynthesis of nucleic acids - Metabolism of nervous cells - Folate metabolism	
Biotin	Water soluble	- Enzyme component - Macronutrient metabolism	
Vitamin C	Water soluble	- Promotes immune response - Wound healing - Capillary integrity - Increases iron absorption	

Table 2: Minerals

Name	Classification	Functions	Good and excellent food Sources
Calcium	Macro-mineral	- Builds and maintains bones and teeth - Acts as a membrane stabiliser - Influences transmission of ions across membranes - Releases neurotransmitters - Enhances function of protein hormones - Release or activation of enzymes - Regulation of heart beat - Maintains muscle tone and controls nerve irritability - Initiates blood clotting	
Phosphorus	Macro-mineral	- Structural role for bone and teeth: hydroxyapatite - Promotes absorption of glucose and glycerol in intestine - Transport of fatty acids, e.g. phospholipids - Energy metabolism: ATP - Helps control acid-base balance - Integrity of cell membranes: phospholipids - Phosphorylation reactions - Phosphate buffer system - DNA and RNA are based on phosphate	
Magnesium	Macro-mineral	- Catalyst in metabolic reactions: co-factor for enzymes - Involved in protein and fatty acid metabolism - Activator for enzymes involved in oxidative phosphorylation - Mg, with Ca, Na, K in balance in extracellular fluids for transmission	

		<p>of nerve impulses and muscle contraction</p> <ul style="list-style-type: none"> - Acts as a physiological Ca channel blocker 	
Sulphur	Macro-mineral	<ul style="list-style-type: none"> - Part of Mucopolysaccharides in cartilage and bone - Anticoagulant, as part of Heparin - Used to produce insulin - Important in many biochemical reactions 	
Iron	Micro-mineral	<ul style="list-style-type: none"> - Role in respiratory transport of O₂ and CO₂. Haemoglobin is present in red blood cells: carries O₂ from lungs to tissues - CO₂ from tissues to lungs - Used as part of Myoglobin (also a heme protein) which is the O₂ reservoir in muscles - Oxidative production of ATP in mitochondria involves many iron-containing enzymes - Cytochromes - functions in respiratory chain through alternate oxidation and reduction of iron - Immune system: neutrophils less effective in Fe-deficiency - action requires iron-dependent steps - Transferrin and lactoferrin (Fe-binding proteins); protect against infection; bind Fe and makes it unavailable to bacteria - Cognitive performance: anaemic children have less attention span, lower learning ability, poor memory 	
Zinc	Micro-mineral	<ul style="list-style-type: none"> - Involved in reactions that synthesise/degrade major metabolites CHO, protein, lipid and nucleic acids - Stabilises protein and nucleic acid structure and integrity of subcellular organelles - Metallothionein - Zn-containing protein; role: detoxification of metals - Influences metabolism of sulphur-containing amino acids - Zn abundant in nucleus: stabilises RNA and DNA - Involved in transcription / replication of genetic material - Direct link between Zn status and growth and development in children and also immunity 	
Copper	Micro-mineral	<ul style="list-style-type: none"> - Essential element in several enzymes involved in oxidative reactions - Conversion of tyrosine to melanin - Essential for the formation of haemoglobin 	
Iodine	Micro-mineral	<ul style="list-style-type: none"> - Integral part of the thyroid hormones, Triiodothyronine (T3), Thyroxine (T4) 	

		<ul style="list-style-type: none"> - Regulates the rate of oxidation in cells - Influences physical and mental growth - Influences functioning of the nervous and muscle tissue - Influences metabolism of all nutrients - Maintains body heat and energy 	
Manganese	Micro-mineral	<ul style="list-style-type: none"> - Component of enzymes, especially in mitochondria - Associated with the formation of connective and skeletal tissue - Role in growth and development 	
Fluoride	Micro-mineral	<ul style="list-style-type: none"> - As a Ca-salt in bone and teeth - One part fluoride per million (1 mg/L): prevents tooth decay (tooth enamel made more resistant to the action of acids produced by bacteria) - Involved in maintenance of bone structure 	
Molybdenum	Micro-mineral	<ul style="list-style-type: none"> - Component of enzymes 	
Cobalt	Micro-mineral	<ul style="list-style-type: none"> - Component of Vitamin B₁₂ 	
Selenium	Micro-mineral	<ul style="list-style-type: none"> - Antioxidant - Plays a role in lipid metabolism 	
Chromium	Micro-mineral	<ul style="list-style-type: none"> - Potentiates insulin action and influences CHO metabolism 	

5 DIETARY SOURCES OF MICRONUTRIENTS

The only way that humans can get micronutrients into their bodies is by consuming food that contains them, by taking a vitamin or mineral supplement or by getting an injection that contains the micronutrient.

Food contains the nutrients that the body needs for normal functioning and to sustain life. However, we generally do not know what nutrients are contained in the food. Usually we eat because we are hungry or find enjoyment in eating, or because of the social interactions that go with eating. In fact, we usually choose food, not because of the nutrients it contains, but because it smells and tastes good.

Humans get hungry, because they need food to provide energy. Appetite is a physiological desire to eat food, not nutrients. We can eat enough food to satisfy our appetite and hunger, but our diet can still be deficient in a specific nutrient or nutrients. This happens quite often in children who are on a cereal based diet (Whitney, Cataldo & Rolfes, 2002).

Before we go on, we need to define several core concepts used to describe food sources in nutrition studies – *affordability*, *accessibility*, *availability* and *appropriateness*.

- Affordability:** This means food or a specific food is affordable to a person or community: they can buy it or trade for it.
- Accessibility:** This means food can be accessed, either through travelling to where it can be bought or by growing it oneself.
- Availability:** This means a food or foods are available to a person or community, either to buy or to grow themselves. A food may be affordable, but not available or accessible and vice versa.
- Appropriateness:** This means a food is appropriate to recommend as a micronutrient source: factors such as religious practices or certain cultural beliefs might make a food inappropriate. Thus a food may be affordable, accessible and available, but may not be appropriate, owing to cultural beliefs.

TASK 3 - INVESTIGATING GOOD FOOD SOURCES OF VITAMINS AND MINERALS

Once again, use the chapters from Mahan & Escott-Stump (2004) for reference.

a) Go back to your tables, and in column 4, next to each vitamin and mineral, list all the *good* and *excellent* food sources of that micronutrient.

b) For each of the critical micronutrients for Public Health Nutrition, i.e. vitamin A, iron, iodine and zinc, reflect on the availability, accessibility, affordability and appropriateness of food sources in a community where you work. Write a paragraph on each, motivating your answers.

FEEDBACK

Your tables should look like this.

Table 1: Vitamins

Name	Classification	Functions	Good and excellent food sources
Vitamin A	Fat soluble	<ul style="list-style-type: none"> - Essential for normal growth - Development and maintenance of epithelial tissue - Essential for night vision - Required for normal bone development - Functions as an antioxidant - Builds immune system 	Liver; kidneys; milk fat; egg yolk; fortified foods; breastmilk
Carotenoids	Fat soluble	<ul style="list-style-type: none"> - Functions as a provitamin A - Functions as a phyto-chemical 	Green leafy vegetables; yellow and dark orange vegetables and fruits; tomatoes
Vitamin D	Fat soluble	<ul style="list-style-type: none"> - Functions as a pro-hormone - Supports normal bone development - Phosphorus and calcium metabolism 	Liver; fatty fish; fortified foods

Vitamin E	Fat soluble	- Functions as an antioxidant - Protects red blood cells from hemolysis - Maintains epithelial tissue	Wheat germ, vegetable oils; milk fat; egg yolk
Vitamin K	Fat soluble	- Normal blood clotting - Bone metabolism	Liver; soybean oil; green leafy vegetables
Thiamin	Water soluble	- Energy metabolism - Normal growth - Maintains nervous system	Organ meats; legumes; whole grain cereals; fortified foods
Riboflavin	Water soluble	- Normal growth - Energy metabolism - Functions as a co-enzyme	Dairy products; organ meats; green leafy vegetables; fortified foods
Niacin	Water soluble	- Part of the enzyme system - Fat synthesis - Promotes glycolysis	Fish; liver; red meats; poultry; eggs; legumes; fortified foods
Pantothenic acid	Water soluble	- Co-enzyme in metabolism of macronutrients	Eggs; organ meats; salmon; yeast.
Vitamin B ₆	Water soluble	- Functions as a co-enzyme - Tryptophan to niacin conversion - Normal growth	Pork; glandular meats; whole grain cereals; legumes; eggs
Folate	Water soluble	- Biosynthesis of nucleic acids - Normal maturation of red blood cells - Co-enzyme	Green leafy vegetables; organ meats; beef; eggs; legumes
Vitamin B ₁₂	Water soluble	- Biosynthesis of nucleic acids - Metabolism of nervous cells - Folate metabolism	Organ meats; dairy products; eggs; meat; <u>only found in animal products</u>
Biotin	Water soluble	- Enzyme component - Macronutrient metabolism	Liver, mushrooms; peanuts; meat, egg yolk, bananas
Vitamin C	Water soluble	- Promotes immune response - Wound healing - Capillary integrity - Increases iron absorption	Citrus fruits; strawberries, guavas; tomatoes; cabbage; potatoes; kiwi fruit.

Table 2: Minerals

Name	Classification	Functions	Food Sources
Calcium	Macro-mineral	- Builds and maintains bones and teeth - Acts as a membrane stabiliser - Influences transmission of ions across membranes - Releases neurotransmitters - Enhances function of protein hormones	Dairy products; sardines (fish bones); tofu

		<ul style="list-style-type: none"> - Release or activation of enzymes - Regulation of heart beat - Maintains muscle tone and controls nerve irritability - Initiates blood clotting 	
Phosphorus	Macro-mineral	<ul style="list-style-type: none"> - Structural role for bone and teeth: hydroxyapatite - Promotes absorption of glucose and glycerol in intestine - Transport of fatty acids, e.g. phospholipids - Energy metabolism: ATP - Helps control acid-base balance - Integrity of cell membranes: phospholipids - Phosphorylation reactions - Phosphate buffer system - DNA and RNA are based on phosphate 	Dairy products; meat; fish; poultry; whole grain cereals
Magnesium	Macro-mineral	<ul style="list-style-type: none"> - Catalyst in metabolic reactions: co-factor for enzymes - Involved in protein and fatty acid metabolism - Activator for enzymes involved in oxidative phosphorylation - Mg, with Ca, Na, K in balance in extracellular fluids for transmission of nerve impulses and muscle contraction - Acts as a physiological Ca channel blocker 	Whole grain cereals; tofu; nuts; meat; green vegetables; legumes; chocolate
Sulphur	Macro-mineral	<ul style="list-style-type: none"> - Part of Mucopolysaccharides in cartilage and bone - Anticoagulant, as part of Heparin - Used to produce insulin - Important in many biochemical reactions 	Meat; fish; poultry; eggs; milk; legumes
Iron	Micro-mineral	<ul style="list-style-type: none"> - Role in respiratory transport of O₂ and CO₂. Haemoglobin is present in red blood cells: carries O₂ from lungs to tissues - CO₂ from tissues to lungs - Used as part of Myoglobin (also a heme protein) which is the O₂ reservoir in muscles - Oxidative production of ATP in mitochondria involves many iron-containing enzymes - Cytochromes - functions in respiratory chain through alternate oxidation and reduction of iron - Immune system: neutrophils less effective in Fe-deficiency - action requires iron-dependent steps - Transferrin and lactoferrin (Fe-binding proteins); protect against infection; bind Fe and makes it unavailable to bacteria - Cognitive performance: anaemic children have less attention span, lower learning ability, poor memory 	Liver; meat; fish; poultry; egg yolk; breastmilk; legumes; dark green leafy vegetables; molasses; fortified foods

Zinc	Micro-mineral	<ul style="list-style-type: none"> - Involved in reactions that synthesise/degrade major metabolites CHO, protein, lipid and nucleic acids - Stabilises protein and nucleic acid structure and integrity of subcellular organelles - Metallothionein - Zn-containing protein; role: detoxification of metals - Influences metabolism of sulphur-containing amino acids - Zn abundant in nucleus: stabilises RNA and DNA - Involved in transcription / replication of genetic material - Direct link between Zn status and growth and development in children and also immunity 	Shellfish; oysters; liver; legumes; whole wheat products; milk; breastmilk
Copper	Micro-mineral	<ul style="list-style-type: none"> - Essential element in several enzymes involved in oxidative reactions - Conversion of tyrosine to melanin - Essential for the formation of haemoglobin 	Liver; shellfish; whole grain cereals; legumes; kidneys; poultry; chocolate
Iodine	Micro-mineral	<ul style="list-style-type: none"> - Integral part of the thyroid hormones, Triiodothyronine (T3), Thyroxine (T4) - Regulates the rate of oxidation in cells - Influences physical and mental growth - Influences functioning of the nervous and muscle tissue - Influences metabolism of all nutrients - Maintenance of body heat and energy 	Iodised salts and products containing iodised salt; seafood
Manganese	Micro-mineral	<ul style="list-style-type: none"> - Component of enzymes, especially in mitochondria - Associated with the formation of connective and skeletal tissue - Role in growth and development 	Whole grain cereals; nuts; legumes
Fluoride	Micro-mineral	<ul style="list-style-type: none"> - As a Ca-salt in bone and teeth - One part fluoride per million (1 mg/L): prevents tooth decay (tooth enamel made more resistant to the action of acids produced by bacteria) - Involved in maintenance of bone structure 	Drinking water at least 1ppm (part per million); soybeans; spinach; onions
Molybdenum	Micro-mineral	<ul style="list-style-type: none"> - Component of enzymes 	Legumes; cereals; dark green leafy vegetables
Cobalt	Micro-mineral	<ul style="list-style-type: none"> - Component of Vitamin B₁₂ 	Liver; kidney; milk
Selenium	Micro-mineral	<ul style="list-style-type: none"> - Antioxidant - Plays a role in lipid metabolism 	Grains; onions; meat; milk; depends on soil content
Chromium	Micro-mineral	<ul style="list-style-type: none"> - Potentiates insulin action and influences CHO metabolism 	Whole grain cereals; yeast; meat

You were asked to reflect on the availability, accessibility, affordability and appropriateness of food sources containing vitamin A, iron, iodine and zinc, in a community with which you are familiar or work.

Here is an example for you. Study it, and then evaluate your answers. For vitamin A, a good food source is egg yolk.

In a community in a deep rural area, eggs may not be available if there are no chickens and if shops are distant. Eggs are therefore also not accessible because of the distance to shops. In such situations, the eggs that are available might be very expensive and not affordable to poorer communities. In some cultures, eggs are also not allowed as food for young children, teenagers and pregnant women; they are therefore also not appropriate. Thus, even though egg yolk contains Vitamin A, it is not a good food source for this rural community.

In this community, to make eggs affordable, accessible and available, a good intervention would be to consider chicken farming, provided that chicken farming will be accepted by the community. Then eggs will be produced locally, and chicken meat which contains vitamin A, iron and zinc will also become available. The money that the community generates through selling eggs and chickens could also be used to buy other foodstuffs that may provide additional micronutrients. To overcome the cultural beliefs concerning eggs, an education programme could be launched with the chicken farming programme.

6 SESSION SUMMARY

Well done, you have completed the first session of this module. You should, in the process, have been introduced to all the vitamins and minerals and their classifications. It is unlikely that you have committed all this to memory, but at least, you should be able to find this information when you need it.

You have also studied the functions of these micronutrients and investigated their different food sources. For some of the key micronutrients, vitamin A, iron, iodine and zinc, you have considered good food sources and how available, accessible, appropriate and affordable they are. Go back to the learning outcomes of this session and see if you have achieved them!

7 REFERENCES AND FURTHER READING

- Mahan, L. K. & Escott-Stump, S. (eds). (2004). *Krause's Food, Nutrition & Diet Therapy*. 11th Edition. Pennsylvania, USA: Elsevier.
- Semba, R. D. & M. W. Bloem (eds). (2001). *Nutrition and Health in Developing Countries*. Totowa, New Jersey, USA: Humana Press.
- Whitney, E. N., Cataldo, C. B. & Rolfes, S. R. (2002). *Understanding Normal and Clinical Nutrition*. 6th Edition USA: Wadsworth.

Unit 1 – Session 2

Key Micronutrients in Public Health Nutrition

Introduction

At the beginning of the 1990s, there were three international meetings which addressed micronutrient malnutrition. These were the 1990 World Summit for Children at the United Nations in New York, the Ending Hidden Hunger Conference in Montreal in 1991 and the 1992 International Conference on Nutrition at the Food and Agricultural Organisation (FAO) in Rome.

Three micronutrients were singled out because of the high prevalence of their deficiency and their devastating effects. Such effects were identified predominantly in the populations of developing countries, affecting infant and child morbidity and mortality, low productivity, poor quality of life in children, adults and pregnant and lactating women. Representatives of almost all countries at these meetings agreed to take action towards the elimination of:

- Vitamin A deficiency
- Iron-deficiency anaemia in women of childbearing age
- Iodine deficiency.

Since these meetings, zinc started to emerge as another important micronutrient in Public Health, because of its role in child growth and development.

As an outcome of the above strategy, different groups with worldwide membership were formed, and meet every two years to discuss new findings and progress in the fight to eliminate these micronutrient deficiencies. These consultative groups are:

- Vitamin A: International Vitamin A Consultative Group (IVACG).
- Iron: International Iron Consultative Group (INACG).
- Iodine: International Council for Control of Iodine Deficiency Disorders (ICCIDD).
- Zinc: International Zinc Consultative Group (IZiNACG).

In this study session, we are going to look in detail at the biochemistry, physiology, functions, food sources, bioavailability, deficiency diseases and toxicity of these four micronutrients which are so important in Public Health. Your task is to develop your understanding of: how deficiency in these micronutrients develops in individuals and populations and how to develop indicators of deficiency for planning, implementing, monitoring and evaluating programmes which address deficiencies for each of them. The readings prescribed for this session will provide you with more detailed information on these four micronutrients.

Session Contents

- 1 Learning outcomes of this session
- 2 Readings
- 3 Vitamin A in detail
- 4 More about iron
- 5 Exploring iodine
- 6 More about zinc
- 7 Session summary
- 8 References and further reading

Timing of this session

This session contains nine readings and four tasks. It should take you about six hours to complete.

1 LEARNING OUTCOMES OF THIS SESSION

By the end of this unit, you should be able to:

- Identify the important micronutrients in Public Health Nutrition and motivate their importance.
- Understand the biochemistry and physiological roles of vitamin A, iron, iodine and zinc.
- Describe the food sources of vitamin A, iron, iodine and zinc.
- Discuss the factors that influence bioavailability of vitamin A, iron and zinc.
- Describe the clinical signs of deficiency and the deficiency diseases of vitamin A, iron, iodine and zinc.
- Discuss the clinical manifestations of toxicity of vitamin A, iron, iodine and zinc.

2 READINGS

You will be referred to the following readings in the course of this session. Use the author's initial to find the page numbers in the alphabetical index of the Reader.

Author/s	Reference details
Gallagher, M. L.	(2004). Ch 4 - Vitamins. In L. K. Mahan & S. Escott-Stump. (eds). <i>Krause's Food, Nutrition & Diet Therapy</i> . 11th Edition. Pennsylvania, USA: Elsevier: 75 - 119.
Anderson, J. J. B.	(2004). Ch 5 - Minerals. In L. K. Mahan & S. Escott-Stump. (eds). <i>Krause's Food, Nutrition & Diet Therapy</i> . 11 th Edition. Pennsylvania, USA: Elsevier: 120 - 163.

Solomons, N. W.	(2001). Ch 12 - Vitamin A and Carotenoids. In B. A. Bowman & R. M. Russel, (eds). <i>Present Knowledge in Nutrition</i> . 8 th Ed. Washington, D. C: ILSI Press: 127 - 143.
Yip, R.	(2001). Iron. In B. A. Bowman & R. M. Russel, (eds). <i>Present Knowledge in Nutrition</i> . 8 th Ed. Washington, DC: ILSI Press: 311 - 328.
West, C. E., Jooste, P. L. & Pandav, C. S.	(2004). Ch 12 - Iodine and Iodine-deficiency Disorders. In M. J. Gibney, B. M. Margetts, J. M. Kearney & L. Arab (eds). <i>Public Health Nutrition</i> . Oxford, UK: Blackwell Science: 216 - 226.
Dibley, M. J.	(2001). Ch 31 - Zinc. In B. A. Bowman & R. M. Russel, (eds). <i>Present Knowledge in Nutrition</i> . 8 th Ed. Washington, DC: ILSI Press: 329 - 343.
van het Hof, K. H., West, C. E., Weststrate, J. A. & Hautvast, J. G. A. J.	(2000). Dietary Factors that Affect the Bioavailability of Carotenoids. <i>The Journal of Nutrition</i> , 130(3): 503 - 506.
West, C. E., Eilander, A. & van Lieshout, M.	(2002). Consequences of Revised Estimates of Carotenoid Bioefficacy for Dietary Control of Vitamin A Deficiency in Developing Countries. <i>Journal of Nutrition</i> , 132(9S): 2920S - 2926S.
Academy for Educational Development (AED), USAID.	(Aug 2000). Facts for Feeding. Breastmilk: A Critical Source of Vitamin A for Infants and Young Children. <i>Linkages</i> . Washington, DC: Academy for Educational Development: 1 - 8.

3 VITAMIN A IN DETAIL

In this section, we will explore the biochemistry, physiology, functions, food sources, bioavailability, deficiency diseases and toxicity of vitamin A. Globally over a 100 million children suffer from vitamin A deficiency. Vitamin A deficiency is not only linked to eye health and vision, but plays an important role in human immunity. Sub-clinical vitamin A deficiency is a contributing factor to approximately two million deaths per year from diarrhoea, and also contributes to over a million deaths from measles among children under the age of five years. Severe vitamin A deficiency may result in corneal damage leading to partial or complete blindness. It has been shown that vitamin A supplementation reduces the risk of death from diarrhoea and increases children's resistance to malaria (UNICEF, 1998; WHO, 2000).

The South African Vitamin A Consultative Group (SAVACG) Study found that in South Africa, one in three children between the ages of 1 and 6 years, have marginal vitamin A status; another study, The National Food Consumption Survey (NFCS) of 1999 showed that one out of two children under the age of 9 years had a dietary intake of vitamin A less than two-thirds of the recommended intake (SAVACG, 1995; NFCS, 1999). Vitamin A is involved in regulating many key biological processes in the human body, and neither humans nor animals can survive without it. Vitamin A deficiency is associated with prolonged inadequate intake during periods of high demand - like growth, pregnancy and lactation, and with periods of excessive utilisation and loss, as a result of infection.

To be able to classify vitamin A deficiency as a Public Health problem in a population, region or country, the following WHO classification and minimum prevalence criteria for Xerophthalmia can be applied.

WHO Classification and Minimum Prevalence Criteria for Xerophthalmia and Vitamin A Deficiency as a Public Health Problem

Condition	Abbreviation	Prevalence criteria, %
Xerophthalmia		
Night blindness	XN	> 1.0%
Conjunctival xerosis	XIA	no criteria
Bitot's spots	X1B	> 0.5
Corneal xerosis	X2	
Corneal ulceration/keratomalacia		
< 1/3 corneal surface	X3A	> 0.01
> 1/3 corneal surface	X3B	> 0.01
Corneal scar	XS	> 0.05
Biochemical indicators (c)		
Liver retinol concentration < 35 nmol/g		no criteria
Serum retinol concentration < 0.35 µmol/L		> 5.0
Serum retinol concentration < 0.70 µmol/L		> 20.0
Relative dose response > 15%	RDR	> 20.0
Modified relative dose response > 6%	MRDR	> 20.0
Milk vitamin A concentration:		
based on volume, < 1.05 µmol/L		> 10.0
based on milk fat, < 26 nmol/g		> 10.0

(Sommer, 1995)

In Task 1, you have the opportunity to look at the characteristics of vitamin A in detail. For this task, you will need to study the following readings.

READINGS

Gallagher, M. L. (2004). Ch 4 - Vitamins. In L. K. Mahan & S. Escott-Stump. (eds). *Krause's Food, Nutrition & Diet Therapy*. 11th Edition. Pennsylvania, USA: Elsevier: 75 - 119.

Anderson, J. J. B. (2004). Ch 5 - Minerals. In L. K. Mahan & S. Escott-Stump. (eds). *Krause's Food, Nutrition & Diet Therapy*. 11th Edition. Pennsylvania, USA: Elsevier: 120 - 163.

Solomons, N. W. (2001). Ch 12 - Vitamin A and Carotenoids. In B. A. Bowman & R. M. Russel, (eds). *Present Knowledge in Nutrition*. 8th Ed. Washington, DC: ILSI Press: 127 - 143.

van het Hof, K. H., West, C. E., Weststrate, J. A. & Hautvast, J. G. A. J. (2000). Dietary Factors that Affect the Bioavailability of Carotenoids. *The Journal of Nutrition*, 130(3): 503 - 506.

West, C. E., Eilander, A. & van Lieshout, M. (2002). Consequences of Revised Estimates of Carotenoid Bioefficacy for Dietary Control of Vitamin A Deficiency in Developing Countries. *The Journal of Nutrition*, 132(9S): 2920S - 2926S.

TASK 1 - FAMILIARISE YOURSELF WITH VITAMIN A

Using the readings as your source of information, complete these tasks.

- a) Describe the difference between preformed vitamin A and carotenoids.
- b) Summarise the mechanisms of absorption of preformed vitamin A and carotenoids.
- c) Name the form in which vitamin A is transported and where it is stored in the human body.
- d) List the biochemical indicators used to assess vitamin A status.
- e) Define bioavailability, bioconversion and bioefficacy in the context of vitamin A.
- f) Discuss the factors which affect the bioavailability and the bioconversion of carotenoids, and give the bioconversion factors.
- g) Give the functions of vitamin A. (Refer to your table in Session 1)
- h) List the possible causes of vitamin A deficiency.
- i) Describe the manifestations of vitamin A deficiency.
- j) Critically discuss vitamin A toxicity.
- k) Write a short paragraph on breastmilk as a dietary source of Vitamin A for infants.
- l) List possible approaches in Public Health to prevent vitamin A deficiency.

FEEDBACK

a) The difference between preformed vitamin A and carotenoids:

Preformed vitamin A occurs either as retinol, retinal or retinoic acids. All three compounds have vitamin A activity and are known as preformed vitamin A. Preformed vitamin A is only found in animal products, while plant foods such as vegetables and fruit do not contain preformed vitamin A.

However, carotenoids, a group of compounds found in plants, can be metabolised in the human body to retinoids, and thus vitamin A. There are several hundred types of carotenoids in food, but only some have vitamin A activity, the most active being β -carotene. The amount of vitamin A (retinol) formed from the carotenoids depends on how well the carotenoids are absorbed, and how efficiently it is converted to vitamin A in the body.

b) Absorption and transport of preformed vitamin A and carotenoids:

Passive absorption takes place with lipids into the mucosal cells of the intestine. In the mucosal cells, retinol is bound to retinol-binding protein (RBP). It is then re-esterified and incorporated into chylomicrons, which are transported in the lymph system, and enter blood stream at the thoracic duct. From there it is transported to the liver, where it is metabolised to cellular RBP. The liver is the primary storage place for vitamin A.

In a state of vitamin A deficiency, hepatic RBP is elevated, and peripheral levels are low; thus when a vitamin A rich meal is given, the peripheral levels increase and this forms the basis for a dose-response test.

c) Carotenoid absorption:

Carotenoids can be absorbed intact into the lymph system and circulate with serum lipids. Intact carotenoids are deposited in adipose tissue and the liver. This gives fat a yellow pigmentation. Intact carotenoids are oxidised and degraded over time.

Carotenoids can also be metabolised to retinol in the mucosal cells of the lumen and are transported then as described for vitamin A. This absorption and conversion of carotenoids to vitamin A is influenced by many factors.

d) Vitamin A transportation and storage:

Vitamin A is transported in the blood as Retinol-Retinol binding protein-transthyretin (Retinol-RBP-TTR) complex, and is stored mainly in the liver, with small stores in adipose tissue, lungs and kidneys.

e) The following biochemical indicators are used to assess vitamin A status:

- Serum retinol for assessing deficiency, although it is not a good indicator for hypervitaminoses;
- Relative dose response is more reliable than Serum retinol, and involves assessing the deficiency of liver vitamin A stores;
- Isotope dilution mass spectrometry: This is very expensive and needs technical expertise.

f) Bioavailability is the degree to which a nutrient is available within the small intestine for absorption and physiological use and storage.

Bioconversion is the amount of carotenoids converted to vitamin A.

Bioefficacy is the efficiency with which carotenoids are absorbed and converted to vitamin A.

g) The factors which affect the bioavailability and the bioconversion of carotenoids:

- Species of carotenoid: carotenoids differ in the extent to which they are absorbed, with lutein and β -cryptoxanthin being absorbed more than β -carotene. Bioconversion to retinol also varies amongst carotenoids. There might also be interaction between the different carotenoids which influences absorption.
- Molecular linkage: Some carotenoids exist as esters such as β -cryptoxanthin in papaya; it is not known to what extent the ester linkage inhibits bioavailability. It is generally agreed that esters are readily hydrolysed, but other linkages such as those of ethers or glycosides would not be split readily.
- The amount of carotene consumed in a meal: The rate of conversion depends on the carotene intake. The effect of the amount of carotene in the meal would possibly be confined to the bioconversion process.
- Matrix in which the carotenoid is incorporated: This is the most important factor reducing the bioavailability of provitamin A carotenoids. In dark-green

leafy vegetables, carotenoids are present in the chloroplasts of leaves, which are not readily digested in the body. In carrots, β -carotene is present in crystal form, and in cells, which are not readily broken down. In contrast, the cell walls of fruits, like papaya, containing carotenoids are more easily digested and available.

- Dietary enhancers of absorption and effectors of bioconversion: absorption of carotenoids is stimulated by dietary fat and reduced by dietary pectins: 3-5g fat per meal is recommended. The type of fat might also play a role. Little is known however about how dietary factors affect bioconversion.
- Nutrient status of the host: Increased retinol status seems to reduce conversion of β -carotene to retinol, while a deficiency of protein and of zinc reduces bioconversion of β -carotene to retinol.
- Genetic factors: Not enough is known about genetic control of absorption in humans, but a deficiency of the cleavage enzyme might play a role.
- Host-related factors: Gastrointestinal infections and parasites can cause maldigestion and malabsorption. Since there is excessive loss of gut epithelium, there is also probably an effect on bioconversion. Little is known about the effect of age on absorption and bioconversion of carotenoids.
- Interactions between the other factors: All of the factors mentioned above can interact with one another to affect carotene bioavailability and bioconversion.

Bioconversion factors:

- 1 Retinol activity equivalent (RAE) = $1\mu\text{g}$ retinol.
- $1\mu\text{g}$ retinol = 3,33 International units (IU).
- β -carotene conversion factor in a mixed diet is 1:12. Thus $12\mu\text{g}$ of β -carotene yields $1\mu\text{g}$ retinol.
- The conversion factor of other carotenoids is 1:24. Thus $24\mu\text{g}$ of the other carotenoids yields $1\mu\text{g}$ of retinol.

h) The functions of vitamin A:

Refer to your completed Table 1: Vitamins from Study Session 1.

i) Possible cause of vitamin A deficiency:

- Primary causes: inadequate intake of preformed vitamin A or carotenoids (taking conversion into account).
- Secondary causes: insufficient dietary fat; biliary insufficiency; liver disease;
- Protein energy malnutrition;
- Parasites;
- Zinc deficiency.

j) Signs of vitamin A deficiency:

Clinical signs of the eye happen consecutively as deficiency progresses: (classification abbreviation is shown in brackets)

- Night blindness, due to impaired dark adaptation (XN)
- Corneal xerosis, caused by reduction in Goblet cells (X1A)
- Bitot's spots, foamy spots on the conjunctiva (X1B)
- Corneal xerosis, due to drying of the cornea (X2)
- Corneal ulceration, due to liquification of process of keratomalacia (X3)
- Corneal scar (XS)

- Xerolpthalmia (XF) - blindness

Signs of the skin:

- Follicular hyperkeratosis or thickening of the hair follicles is a cutaneous manifestation.

Failure in systemic functions:

- Impaired embryonic development and spontaneous abortion
- Anaemia
- Impaired immunocompetence
- Keratinisation of mucous membranes in respiratory tract and gastrointestinal tract, leading to poor growth.

k) Vitamin A toxicity:

Continuous large doses of vitamin A overcome the capacity of the liver to store vitamin A and can lead to liver disease. If large amounts of vitamin A are consumed over time, hypervitaminoses develops. Clinical signs are: cheiloses; dryness of nasal mucosa; scaling and peeling of the skin; hair loss and nails are fragile. Headaches, nausea and vomiting may appear. Hypervitaminose A can be induced by a single dose of vitamin A of 200mg in adults and 100mg in children. High doses of vitamin A > 6000µg /day are teratogenic, thus toxic to the fetus and can cause malformations of the fetus. Thus supplementation during pregnancy is not advised.

l) Breastmilk as a dietary source of vitamin A for infants:

Almost all children are born with low stores of vitamin A. Breastmilk is an excellent source of vitamin A and the needs of an infant that is exclusively breastfed, will be met. Sub-optimal feeding practices will increase the risk of vitamin A deficiency in infants and will threaten survival, growth and development. Colostrum, the so called first milk, is three times richer in vitamin A and ten times richer in β-carotene than mature breastmilk. It therefore supplies the infant with enough vitamin A and antibodies for protection in the first few days of life. Afterwards, the mature breastmilk will supply the vitamin A needs, providing that the mother is well nourished. A postpartum high dose of vitamin A to the mother will ensure that the vitamin A concentration in the breastmilk is adequate.

m) Strategies to prevent vitamin A deficiency:

- Vitamin A supplementation;
- Dietary diversification: increase intake from local available foods;
- Fortification of fortifiable foods.

4 MORE ABOUT IRON

Iron Deficiency Anaemia (IDA) is the most common nutritional deficiency disorder in the world. It affects approximately two billion people. Almost half of the women of childbearing age and young children in developing countries suffer from Iron Deficiency Anaemia and it is also the one nutritional disease that has a high prevalence in almost all developed countries.

The NFCS showed that between 36% and 57% of children had iron intakes of less than two-thirds of the recommended dietary intake (SAVACG, 1996; Labadarios et al, 2001). In South Africa, the SAVACG study has shown that one in 20 children have Iron Deficiency Anaemia and one in 10 children is iron deficient.

Although the causes and burden of iron deficiency (ID) and Iron Deficiency Anaemia are well described and established, the prevention and control of it is still remains a challenge. In children, iron deficiency lowers resistance to disease and influences learning ability and physical stamina. It is also a significant cause of maternal mortality, and increases the risk of haemorrhage and infection during childbirth. The highest risk groups for iron deficiency are pre-term infants, low birthweight infants, infants generally, children during periods of rapid growth, women of child bearing age, pregnant women and children and adults with nematode infections in the gastro-intestinal tract, children with a sensitivity to cow's milk and people who consume diets low in meat and high in phytates, which are found in the outer layer of cereal grains (WHO, 2000).

To be able to classify iron deficiency as a Public Health problem in a population, region or country, the following WHO classification and minimum prevalence criteria, can be applied.

WHO Classification and Minimum Prevalence Criteria for Iron Deficiency Anaemia as a Public Health Problem

Category	% Percentage prevalence
Severe	> or = 40
Moderate	20.0 - 39.9
Mild	5.0 - 19.9
Normal	< or = 4.9

(WHO, 2001)

Iron is essential in humans for the development of normal red blood cells and for the oxygen carrying capacity of these red blood cells. Therefore, a deficiency in iron negatively influences the productivity of people, the development of the fetus during pregnancy, and growth and mental development in children. In Task 2, you are asked to explore the characteristics of iron in detail using the following readings as reference material.

READINGS

Gallagher, M. L. (2004). Ch 4 - Vitamins. In L. K. Mahan & S. Escott-Stump. (eds). *Krause's Food, Nutrition & Diet Therapy*. 11th Edition. Pennsylvania, USA: Elsevier: 75 - 119.

Anderson, J. J. B. (2004). Ch 5 - Minerals. In L. K. Mahan & S. Escott-Stump. (eds). *Krause's Food, Nutrition & Diet Therapy*. 11th Edition. Pennsylvania, USA: Elsevier: 120 - 163.

Yip, R. (2001). Iron. In B. A. Bowman & R. M. Russel. (eds). *Present Knowledge in Nutrition*. 8th Ed. Washington, DC: ILSI Press: 311 - 328.

TASK 2 - CLARIFY THE CHARACTERISTICS OF IRON

- a) List the major iron-containing compounds in the human body and specify their functions.
- b) Describe step by step or use a diagram to show the mechanism of absorption, transport, storage and loss of iron in the human body.
- c) Define anaemia and Iron Deficiency Anaemia. List possible causes and describe the manifestations of iron deficiency.
- d) List the biochemical and haematological indicators used for the assessment of iron status, and indicate the relationship of these indicators to iron status. Also indicate the normal values.
- e) Name the good food sources of iron. See your Minerals Table 2 from Session 1. Specify the heme-iron sources and the non-heme iron sources.
- f) Define the bioavailability of minerals.
- g) Discuss the factors which enhance and inhibit the bioavailability of dietary iron.
- h) Discuss iron overload and iron toxicity.
- i) List possible approaches to address ID and IDA in Public Health.

FEEDBACK

- a) **Major iron containing compounds in the human body:**
 - Heme: nonprotein, the insoluble iron protoporphyrin constituent of haemoglobin.
 - Myoglobin: a ferrous protoporphyrin globin complex present in muscle that stores oxygen.
 - Transferrin: a protein synthesised in the liver that transports iron in the blood to the erythroblasts for use in heme synthesis.
 - Ferritin: an iron-apoferritin complex that is a major storage form of iron.
 - Hemoglobin: conjugated protein containing four heme groups and globin-propcity of reverse oxygenation.
 - Hemosiderin: an insoluble form of storage iron.
- b) **The mechanism of absorption, transport, storage and loss of iron in the human body:** see page 138 of Chapter 5 by Anderson in Mahan & Escott-Stump (2004).
- c) **Anaemia and IDA - causes and manifestations:**

Anaemia is a deficiency in the size or number of red blood cells (erythrocytes) or the amount of haemoglobin they contain, that limits the exchange of oxygen and carbon dioxide between blood and the tissue cells. There are different types of anaemia of which Iron Deficiency Anaemia (IDA) is one. IDA is characterised by the production of small (microcytic) red blood cells, pale in colour (hypochromic) and a low level of circulating haemoglobin. IDA is the end point of a long period of iron deprivation and iron deficiency.

Possible causes

- Accidental haemorrhage
- Chronic disease, e.g. TB, ulcers
- Excessive menstrual losses
- Excessive blood donation
- Parasites such as hookworm
- Infant: introduction of whole milk before about six months
- Deficiency of iron in diet during periods of accelerated demand
- In infancy: rapid expanding blood volume
- In adolescence: rapid growth and onset of menses in girls
- Pregnancy because of development of fetus.
- Inadequate absorption of iron
- Diarrhoea
- Lack of acid secretion by the stomach
- Antacid therapy
- Nutritional deficiency such as severe protein depletion
- Protein Energy Malnutrition
- Blood loss

Manifestations:

Iron deficiency (ID) usually remains unrecognised. ID usually has subtle symptoms such as pallor, listlessness and fatigue and is not regarded as life-threatening. Work performance is influenced and work capacity is reduced. In infants and children, behaviour and intellectual performance is negatively influenced. Iron Deficiency Anaemia substantially increases the risk of lead poisoning, particularly in young children because iron-deficient children absorb more lead.

Iron deficiency has a profound effect on the immune response and cell-mediated immunity is often impaired before anaemia is detected and overt clinical signs are seen. There is also a decreased resistance to infection. Body temperature regulation is compromised and there is an impaired capacity to maintain body temperature in a cold environment. This abnormality appears to be related to decreased secretion of thyroid-stimulating hormone and thyroid hormone.

Untreated iron deficiency is associated with infant and child mortality and maternal mortality peri-partum. Anaemia during early pregnancy leads to adverse pregnancy outcomes such as preterm deliveries, low birth weight infants and foetal death.

d) The biochemical and haematological indicators used for the assessment of iron status, and their relationship to iron status

Indicator	Overload	Normal	Depleted stores	Iron deficiency	Iron deficiency anaemia
Hemoglobin	Normal	Normal	Normal	Normal	↓↓
Serum ferritin	↑	Normal	↓	↓	↓↓
Transferrin saturation	↑↑	Normal	Normal	↓	↓

MCV (mean cell volume)	Normal	Normal	Normal	Normal	↓
Erythrocyte protoporphyrin	Normal	Normal	Normal	↑	↑↑
Serum transferrin Receptor	Normal	Normal	Normal	↑	↑

Key	↓ If low ... ↓↓ If very low ...	↑ If high ... ↑↑ If very high ...
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Examples of reading this table:

If the hemoglobin level is very low, this indicates Iron Deficiency Anaemia. If the erythrocyte protoporphyrin is high, this indicates possible iron deficiency. Clinically, these indicators are read in conjunction with one another, but for most community studies, only Hemoglobin and Serum ferritin indicators are used.

The relationship of these indicators to iron status:

The development of iron deficiency is characterised by sequential changes in the amount of iron in the various iron compartments of the body. In the first stage, iron stores become depleted but there is enough iron to meet the needs of red cell production. When iron stores are exhausted, the amount of iron in the circulation system starts to fall and red cell production becomes compromised (iron deficient erythropoiesis); the function of iron-containing enzymes becomes impaired. In the final stage of exhausted iron stores, the amount of iron in the circulation is very low and red cell production is drastically reduced.

e) Sources of iron in food:

There are two broad categories of iron in food. Heme iron, derived mainly from hemoglobin in meat and fish, and non-heme iron which takes the form of iron salts, iron in other proteins and iron derived from processing or storage methods. Heme iron enters the mucosal cells by a different mechanism and is better absorbed than non-heme iron. It is also less influenced by the body iron status, and is not affected by other constituents in the diet (see Bioavailability below). Non-heme iron, a form of iron found in plants, is less well absorbed than heme-iron.

Take note that animal sources of iron, e.g. meat, fish and poultry contain 40% heme iron (which is absorbed as such) and 60% non heme iron; on the other hand, plant food iron, e.g. legumes and dark green leafy vegetables, are 100% non heme iron, and are therefore a poorer source of iron. Plant foods would, however, usually make the larger contribution of iron to the diet.

f) Bioavailability of minerals:

This means the availability of a mineral within the small intestine for absorption, and the amount actually absorbed. Minerals are usually

absorbed in the ionic state. Organic molecules or inorganic compounds are not absorbed, and are excreted in faeces.

g) Factors affecting absorption/bioavailability:

The presence of the following increase iron absorption:

- Ascorbic acid (vitamin C)
- Animal proteins
- Human milk (lactoferrin)
- Increased need for growth, pregnancy or deficiency.

The presence of the following decrease iron absorption:

- Gastric acidity and antacids
- Phytate
- Tannins/caffeine
- Calcium
- Increased intestinal motility

h) Iron overload and toxicity:

Iron overload is the opposite of iron deficiency. Iron overload leads to excess accumulation in the body and results in organ damage. In genetic Hemochromatosis (HFE), iron absorption is defective. This leads to iron accumulation and iron is deposited in the liver, heart, and leads to pancreas damage, liver cirrhosis, cancer and heart failure. It is treated by bleeding repeatedly.

In Hemosiderosis there is an increased ingestion of highly bioavailable iron that is deposited in the organs. When associated with tissue damage, it is called Hemochromatosis. African dietary iron overload is a less common form of iron overload and is caused by the ingestion over a long period of large amounts of highly bioavailable iron in low alcohol beer, brewed traditionally in iron pots. The toxic effects are similar as that of Hemochromatosis.

Thalassaemia is a genetic disorder of iron metabolism and is described as secondary iron overload. It is also called iron-loading anaemia. Excessive amounts of iron are absorbed because of the increased turnover of red cells. Repeated blood transfusions, because of the anaemia add to the iron burden. The iron overload occurs more rapidly and most victims die from iron-induced heart failure.

Acute iron toxicity is seen when excessively large doses of iron is ingested, usually from supplements, usually by children who thought the supplements were sweets. This causes organ damage and death.

i) Ways of addressing iron deficiency and IDA through the Public Health system:

- Control of intestinal helminth (worm) infection.
- Nutrition education and promotion, specifically addressing dietary selection.
- Fortification of basic foods.

5 EXPLORING IODINE

Iodine deficiency is described as the leading cause of preventable brain damage and mental retardation. In 1999, it was estimated that 740 million people worldwide were affected by iodine deficiency and that 500 million people were at risk of developing iodine deficiency. It has been stated that less severe iodine deficiency in children and adults can mean a loss of 10 IQ points. Legislation on salt iodisation by different countries has had a major impact on decreasing the prevalence of Iodine-deficiency Disorders (IDD) world-wide. The epidemiology of IDD is currently described as being in a transitional phase and the latest data indicate a goitre rate of 20% in Africa (UNICEF, 1998; West et al, 2004).

Definition of Iodine Status of a Population Based on Median Urinary Iodine Concentration

Iodine Status	Median Urinary Iodine Concentration ($\mu\text{g/l}$)
Severe iodine deficiency	< 20
Moderate iodine deficiency	20-49
Mild iodine deficiency	50-99
Ideal iodine intake	100-200
More than adequate iodine intake	201-299
Excessive iodine intake	> 300

(West et al, 2004)

Iodine is an essential constituent of the thyroid hormones which are involved in a wide range of biological functions in the human body. The effects of iodine deficiency are most pronounced, as with the other micronutrients, during periods of increased need such as rapid growth of the fetus during pregnancy, during infancy and in young children. Deficiency may have an effect on brain development during these periods.

Task 3 requires you to explore iodine in detail using the following texts as your references.

READINGS

Gallagher, M. L. (2004). Ch 4 - Vitamins. In L. K. Mahan & S. Escott-Stump. (eds). *Krause's Food, Nutrition & Diet Therapy*. 11th Edition. Pennsylvania, USA: Elsevier: 75 - 119.

Anderson, J. J. B. (2004). Ch 5 - Minerals. In L. K. Mahan & S. Escott-Stump. (eds). *Krause's Food, Nutrition & Diet Therapy*. 11th Edition. Pennsylvania, USA: Elsevier: 120 - 163.

West, C. E., Jooste, P. L. & Pandav, C. S. (2004). Ch 12 - Iodine and Iodine-deficiency Disorders. In M. J. Gibney, B. M. Margetts, J. M. Kearney & L. Arab (eds). *Public Health Nutrition*. Oxford: Blackwell Science: 216 - 226.

TASK 3 - UNDERSTANDING KEY ISSUES ABOUT IODINE

- a) Describe the metabolism of iodine and the role it plays in the synthesis of the thyroid hormones.
- b) List the dietary sources of iodine. See your Table 2 of Minerals in Study Session 1.
- c) Define goitrogens and list food sources thereof.
- d) List the spectrum of IDD at the different life stages.
- e) Name the outcome indicators used to assess IDD.
- f) List possible approaches in Public Health to address IDD.

FEEDBACK

- a) **The metabolism of iodine and the role it plays in the synthesis of the thyroid hormones:** See West et al (2004), page 220.

The uptake of iodine by the cells of the thyroid gland, involving the iodine pump, is stimulated by thyroid-stimulating hormone (TSH) released by the pituitary gland. Once taken up by the thyroid cells, the iodine is released into the colloid of the thyroid gland where it is oxidised. Iodide is then incorporated into tyrosine of thyroglobulin to form monoiodotyrosine (MIT) and diiodotyrosine (DIT). The MIT and DIT are coupled to form tetraiodothyronine or thyroxine (T_4) and triiodothyronine (T_3). This coupling reaction is inhibited by the goitrogens. The thyroglobulin is then taken up by the thyroid cells by a process known as pinocytosis. In the thyroid cells, T_4 and T_3 are produced by a process of proteolysis. The secretion of T_4 and T_3 is under the influence of TSH, the secretion of which is controlled by the thyrotropin releasing hormone (TRH) from the hypothalamus and by a feedback mechanism involving T_4 . As the concentration of T_4 in the blood falls, TSH secretion increases and vice versa. In severe iodine deficiency, T_4 remains low and TSH high. A low T_4 and high TSH are indicative of hypothyroidism at all stages of the life cycle. When the iodine supply to the thyroid gland is limited, it produces more T_3 and less T_4 . When T_4 levels are low, target tissues also convert T_4 to T_3 . The brain can take up T_4 but not T_3 , so brain function is affected when T_4 levels are low, even though there may be sufficient T_3 to carry out the function of thyroid hormones in other organs/tissues. Also when the iodine supply to the thyroid gland is limited, the gland releases thyroglobulin into the circulation. An elevated thyroglobulin level is also an indicator of iodine deficiency.

b) Iodine is derived from the soil and may be found:

- In drinking water.
- In plant and animal products such as cereals and other staples, e.g. legumes,
- fruit, vegetables, meat, milk and eggs.
- The intake of iodine depends, to a large extent, on the content of iodine in
- the soil where people live.
- Seafoods such as fish and shellfish are rich sources of iodine.
- Iodised salt contains iodine.

c) Goitrogen types and their food sources:

- There are goitrogen types that produce substances which compete with iodine uptake by the thyroid gland. Food sources are cassava, maize, bamboo shoots, sweet potatoes, lima beans and millet.
- Other food sources which produce goitrogens which block the uptake of iodine by the thyroid gland are from the Brassica genus and from the family Cruciferae which includes cabbage, rapeseed and mustard.

d) The spectrum of IDD at different life stages:

See Table 12.3 on page 222 of West et al, 2004.

e) Outcome indicators for IDD:

- Urinary iodine
- Thyroid size
- Thyroid stimulating hormone levels
- Thyroglobulin levels
- T₄ and T₃ levels
- Cretinism prevalence

f) Public Health approaches to address iodine deficiency:

Public Health approaches to address iodine deficiency are food fortification in the form of iodisation (usually of salt), or oral iodised oil or iodised oil injections.

6 MORE ABOUT ZINC

Zinc deficiency is now recognised by the United Nations Children's Fund (UNICEF) as a Public Health problem in many countries, especially developing countries. The extent of zinc deficiency is, however, undetermined. Zinc deficiency is increasingly recognised as widespread among women in developing countries and contributes to growth failure and susceptibility to infections in malnourished children. Trials in different developing countries have shown that zinc supplements can reduce the severity and duration of diarrhoea in children (UNICEF, 1998).

Zinc is present in almost every cell of the human body and plays a pivotal role in cellular growth and differentiation. In the last decade there has been a rapid

expansion in Public Health literature on the beneficial effects of zinc supplementation in populations.

In Task 4 of this session you will look at Zinc in detail using the following texts as references.

READINGS

Gallagher, M. L. (2004). Ch 4 - Vitamins. In L. K. Mahan & S. Escott-Stump. (eds). *Krause's Food, Nutrition & Diet Therapy*. 11th Edition. Pennsylvania, USA: Elsevier: 75 - 119.

Anderson, J. J. B. (2004). Ch 5 - Minerals. In L. K. Mahan & S. Escott-Stump. (eds). *Krause's Food, Nutrition & Diet Therapy*. 11th Edition. Pennsylvania, USA: Elsevier: 120 - 163.

Dibley, M. J. (2001). Ch 31 - Zinc. In B. A. Bowman & R. M. Russel, (eds). *Present Knowledge in Nutrition*. 8th Ed. Washington, DC: ILSI Press: 329 - 343.

TASK 4 - BECOMING FAMILIAR WITH THE CHARACTERISTICS OF ZINC

- a) Describe the absorption and transport of zinc.
- b) List the good food sources of zinc. Refer to the Minerals Table you developed in Study Session 1.
- c) List the factors which affect the bioavailability of zinc.
- d) List the functions of zinc. Refer to your Minerals table from Study Session 1.
- e) Discuss the manifestations of zinc deficiency.
- f) Discuss zinc toxicity.

FEEDBACK

a) Absorption and transport of zinc:

Zinc is carrier-mediated and diffused into the blood portal system, where it is found in the erythrocytes and leukocytes. It is taken up by the liver and then transported to the other tissues. The major carrier of zinc in the blood is albumin. Its availability is affected by both the zinc level in the diet and the presence of interfering substances.

b) Food sources of zinc:

Rich sources: oysters, liver, high-protein foods, whole grain cereals, fortified food products.

Good sources: legumes, peanuts, peanut butter, dairy products.

Poor sources: fruits and most vegetables.

Zinc in plant proteins is less available than that in animal proteins.

Vegetarian diets

and low-protein diets are likely to be low in zinc.

c) Factors that influence bioavailability:

Inhibiting factors

- Fibre/phytate

- High doses of copper inhibit absorption because it uses the same carrier protein.
- Iron competes with zinc for absorption

Enhancing factors

- Glucose
- Lactose
- Soy protein
- Red wine
- Breastmilk.

d) Functions:

- Zinc is involved in reactions that synthesise/degrade major metabolites, e.g. carbohydrates, proteins and lipids and nucleic acids.
- It stabilises protein and nucleic acid structure and integrity of sub-cellular organelles' transport processes.
- Metallothionein is the most abundant zinc and contains protein. Its role is detoxification of metals.
- It influences the metabolism of sulphur-containing amino acids.
- Zinc is abundant in the nucleus and stabilises RNA + DNA.
- Zinc is involved in transcription / replication of genetic material.
- Zinc appears in the structure of bone and is needed for adequate osteoblastic activity.
- There is a direct link between zinc status and growth and development in children as well as immunity.

e) Clinical manifestations associated with zinc deficiency:

- growth retardation
- poor appetite
- delayed wound healing
- abnormal dark adaptation
- loss of hair
- hypogonadism in males
- mental lethargy
- susceptibility to infections
- skin lesions
- disturbances in smell and taste acuity, e.g. Hypogeusia, a decrease in taste acuity and Dysgeusia which is unpleasant or perverted tastes.
- Acrodermatitis Enteropathica: Autosomal Recessive Disease of zinc malabsorption.

Those at risk for deficiency: alcoholics, pregnant and elderly people, young children.

f) Toxicity:

It can be considered as a relatively non-toxic micronutrient, however:

- Acute toxicity results in gastric distress, dizziness and nausea.
- Chronic excess affects pancreatic enzymes, inhibits copper and iron absorption and impairs immune functions.

7 SESSION SUMMARY

In this session, you have investigated the biochemistry, physiology, functions, food sources, bioavailability, deficiency diseases and toxicity of the key Public Health micronutrients including vitamin A, iron, iodine and zinc. You should now be able to motivate why these micronutrients are important for human health and what the challenges are to overcoming deficiencies.

8 REFERENCES AND FURTHER READING

- Hambidge, M. (2000). Human Zinc Deficiency. *Journal of Nutrition*, 130(5): 1344 (S) - 1321(S).
- Joint Food and Agricultural Organisation of the United Nations and World Health Organisation (FAO/WHO). (1996). *Progress Report on the Implementation of the ICN World Declaration and Plan of Action for Nutrition*. Rome: FAO/WHO.
- Labadarios, D., Steyn, N., MacIntyre, U., Swart, R., Gericke, G., Huskisson, J., Dannhauser, A., Voster, H. H., Nesamvuni, A. E. (2001). The National Food Consumption Survey (NFCS) – Children aged 1-9 years, South Africa, 1999. *South African Journal of Clinical Nutrition*, 14(2): 62 - 75.
- Sommer, A. (1995). *Vitamin A Deficiency and its Consequences: A Field Guide to Their Detection and Control*. 3rd Ed. Geneva: WHO.
- SAVACG (South African Vitamin A Consultative Group). (1996). Anthropometric, Vitamin A, Iron and Immunisation Coverage Status in Children 6 - 71 months in South Africa. *South African Medical Journal*, 86(4): 354 - 357.
- United Nation Administrative Committee on Coordination/Subcommittee on Nutrition: (ASS/SCN). (1999). *Ending Malnutrition by 2020: An Agenda for Change in the Millenium Final report to ACC/SCN by the Commission on the Nutrition Challenges of the 21st Century*. Geneva: ACC/SCN.
- UNICEF. (1990). *Strategy for Improved Nutrition of Children and Women in Developing Countries: A UNICEF Policy Review*. New York: UNICEF.
- UNICEF. (Bellamy, C.) (1998). *The State of the World's Children 1998*. Oxford University Press for UNICEF, New York.
- West, C. E., Jooste, P. L. & Pandav, C. S. (2004). Ch 12 - Iodine and Iodine-deficiency Disorders. In M. J. Gibney, B. M. Margetts, J. M. Kearney & L. Arab (Eds). *Public Health Nutrition*. Oxford, UK: Blackwell Science: 216 - 226.
- WHO. (1996). *Indicators for Assessing Vitamin A Deficiency and Their Application in Monitoring and Evaluating Intervention Programmes*. (Document WHO/NUT/96.10). Geneva: WHO.

- WHO. (1999). *Progress Towards the Elimination of Iodine Deficiency Disorders*. (Document WHO/NHD/99.4). Geneva: WHO.
- WHO. (2000). *Nutrition for Health and Development: A Global Agenda for Combating Malnutrition*. (Document WHO/NHD/00.6). Geneva: WHO.
- WHO. (2001). *Assessment of Iodine Deficiency Disorders and Monitoring Their Elimination*. (Document WHO/NHD/01.1). Geneva: WHO.
- WHO. (2001). *Iron Deficiency Anaemia: Assessment, Prevention and Control. A Guide for Programme Managers*. (Document WHO/NHD/01.3). Geneva: WHO.

Unit 1 – Session 3

Micronutrients: Dietary Requirements

Introduction

Welcome to Session 3 of Unit 1. In the first two sessions, we have studied the characteristics of the different micronutrients in detail. In this session, we are going to look at the daily dietary requirement of each micronutrient to maintain health, and to prevent deficiencies and toxicity. This is essential knowledge for planning, monitoring and evaluating micronutrient intervention programmes, whether they be food based interventions programmes or supplementation programmes.

Many countries have their own recommended intakes (RIs) for micronutrients, while other countries, including South Africa, use the Dietary Reference Intakes (DRIs) and/or the World Health Organisation's (WHO) set of Recommended Nutrient Intakes (RNIs).

Session Contents

- 1 Learning outcomes of this session
- 2 Readings
- 3 Recommended dietary intakes for micronutrients
- 4 WHO recommendations
- 5 Session summary
- 6 References and further reading

Timing of this session

This session contains three readings and three tasks. It should take you about two hours to complete.

1 LEARNING OUTCOMES OF THIS SESSION

By the end of this unit, you should be able to:

- Understand the concept and scientific basis of two sets of recommended intakes (RIs), namely Dietary Reference Intakes (DRIs) and the WHO recommendations.
- Apply the WHO recommendations on nutrient intakes.
- Describe the application and limitations of the RIs.

2 READINGS

You will be referred to the following readings in the course of this session. Use the author's initial to find the page numbers in the alphabetical index of the Reader.

Author/s	Reference details
Institute of Medicine, Food and Nutrition Board.	Dietary Reference Intakes (DRIs). <i>Recommended Intakes for Individuals of Vitamins and Minerals</i> . Washington DC: National Academy Press. [Online], Available: http://www.nap.edu/books/0309069351/html/21.html [2 pages]
WHO/FAO	(2004). Recommended Nutrient Intakes. <i>Vitamin and Mineral Requirements in Human Nutrition</i> . 2nd edition. Rome: WHO: 338 - 341.
Solomons, N. W.	(2001). Ch 12 - Vitamin A and Carotenoids. In B. A. Bowman & R. M. Russel, (eds). <i>Present Knowledge in Nutrition</i> . 8 th Edition. Washington, DC: ILSI Press: 127 - 143.

3 RECOMMENDED DIETARY INTAKES FOR MICRONUTRIENTS

To address micronutrient requirements, recommended nutrient intakes were determined to serve as standards of good micronutrient nutrition and to guide dietary planning and evaluation. The Food and Agricultural Organisation (FAO) and the World Health Organisation (WHO) of the United Nations established micronutrient recommendations in 1973 and revised them in 1988, 1996 and 2004. The Food and Nutrition Board of the Institute of Medicine (IOM) in the USA has also developed recommended intakes, first published in 1941, and formerly known as the Recommended Dietary Allowances (RDAs). In 1994 they published nutrient recommendations now called the Dietary Reference Intakes (DRIs), which include the RDAs. These recommended intakes (RIs) are based on scientific research findings. Both the WHO recommendations and the DRIs make recommendations according to age, sex and life stage, as well as recommendations for pregnant and lactating women.

Because humans have large daily variation in micronutrient intake, recommended intakes refer to an average intake over time. In addition, the RIs are recommendations for *healthy* people, meaning for people where disease, as well as symptoms and signs of micronutrient deficiency or toxicity, are absent. In this section, we focus on the DRIs.

3.1 Dietary Reference Intakes (DRIs)

The term DRI is a collective name and refers to a set of four nutrient based reference values, namely: Estimated Average Requirement (EAR); Adequate Intake (AI); Tolerable Upper Intake Level (UL); Recommended Dietary Allowance (RDA).

Estimated Average Requirement (EAR)

The EAR is the nutrient intake value that is estimated to meet the nutrient requirements in 50% of the individuals in a given life stage and gender group, defined by a specified indicator of adequacy. At this particular level of intake, the remaining 50% of the respective group will not have its nutrient needs met. In deriving the EARs, reducing the risk of chronic disease was among the factors considered.

The EAR is expressed as a daily value for most nutrients, averaged over time of at least one week. It is used as the basis for setting the RDA. If sufficient scientific evidence is *not* available to establish an EAR, no RDA can be set. The EAR may be used as one of the tools for assessing the adequacy of intakes of population groups, and for planning adequate intakes by groups.

Recommended Dietary Allowance (RDA)

The RDA is the daily dietary intake level which is sufficient to meet the nutrient requirements of nearly all (97 – 98%) individuals of a particular gender at a given life stage. The EAR serves as the basis to calculate an RDA.

It is important to recognise that whereas the EAR applies to groups, the RDA is the goal for dietary intake by the individual. If the standard deviation (SD) of the EAR is available, the RDA is set by using the value for the EAR, and then adding a value that is calculated by multiplying the standard deviation of the EAR by 2. The equation is as follows: the RDA = EAR + 2 SD EAR. Thus the risk of inadequacy in using the RDA is negligible at 2 - 3%. If the intake of a nutrient lies between the RDA and the UL, the risk for excess is also negligible.

If you would like to understand how these values are set, you could consult the following reference: Institute of Medicine. Food and Nutrition Board. (1997). *Dietary Reference Intakes for Calcium, Phosphorus, Magnesium, Vitamin D and Fluoride*. Washington D.C: National Academy Press. [Online], Available www.nap.edu.

Adequate Intake (AI)

In a case where the scientific evidence is inadequate to set an EAR, the Adequate Intake (AI) reference is used instead of an RDA. The AI is based on experimentally derived intake levels or approximations of observed mean nutrient intakes by a group of healthy people. For example, for young infants for whom milk is the sole source of most nutrients during the first 4 to 6 months of life, the AI is based on the daily mean nutrient intake supplied by human milk for healthy, full-term infants, who are exclusively breastfed.

Both, the RDA and the AI are used as the goal for the nutrient intake of an individual. However, there is much less certainty about the numerical value of an AI than that of an RDA value. Since the AI is dependent to a greater extent on judgment than is the case in determining an EAR, and subsequently an RDA, the AI value is expected to be numerically larger than an EAR and possibly even larger than the respective RDA. An AI is seen as an indication that substantially more research is required in order to have an EAR established and to have an RDA calculated.

Tolerable Upper Intake Level (UL)

The UL is the highest level of nutrient intake that is considered unlikely to pose any risk of adverse health effects to almost all individuals in the general population. This assumes that if the intake of a given nutrient increases above the UL, then the risk of adverse effects is thought to gradually increase as well. ULs are based on the total intake of a nutrient derived from food, fortified food and food supplements. The need for setting ULs has arisen because of the increasing practice of fortifying foods with nutrients, as well as the increased use of dietary supplements by a larger number of people. The UL applies to long term (chronic) intake of a nutrient over time and the risk of toxicity. The estimates of adverse health effects in humans are based on the extrapolation of data obtained from experimental studies (Vitamin Information Centre, Roche, 1999).

Using these tables as your reference, explore the RDAs for the nutrients which are key to Public Health by doing Task 1. Remember, that a nutrient requirement, as expressed by the DRIs or RNIs, is the lowest intake level for a nutrient that will "... maintain a defined level of nutriture in an individual" (Institute of Medicine, Food and Nutrition Board). The underlying approach in setting these reference intakes is to maximize health and to improve quality of life.

READING

Institute of Medicine. Food and Nutrition Board. Dietary Reference Intakes (DRIs). *Recommended Intakes for Individuals of Vitamins and Minerals*. Washington D.C: National Academy Press. [Online], Available: <http://www.nap.edu/books/0309069351/html/21.html> [2 pages]

TASK 1 - EXPLORE THE RDAs

List the RDAs for vitamin A, iron, iodine and zinc for children of 7-12 months.

FEEDBACK

Micronutrient	DRI
Vitamin A	500µg/day = AI
Iron	11mg = RDA
Iodine	130µg = AI
Zinc	3.0mg = RDA

Take note that there are also ULs for these micronutrients, and that one must be careful of how much the micronutrients are supplemented.

In Task 2 you will apply the DRIs using Solomons (2001) as a reference.

READING

Solomons, N. W. (2001). Ch 12 - Vitamin A and Carotenoids. In B. A. Bowman & R. M. Russel, (eds). *Present Knowledge in Nutrition*. 8th Ed. Washington, DC: ILSI Press: 127 - 143.

TASK 2 - APPLYING THE DRIs

The Department of Health of South Africa have a vitamin A supplementation programme and have developed the recommend the schedule below:

- Comment on the dosage of vitamin A in relation to the DRI for the different groups.
- Motivate why no supplementation is scheduled during pregnancy, but rather post-partum and then why within a specific time frame. You may need to look back at the basics of Vitamin A in Study Session 2.

Department of Health, South Africa's Vitamin A Supplementation Programme

Target group	Dosage Vitamin A	Schedule
Non-breastfed children 0-5 months	50 000 IU (International Units)	A single dose at the age of 6 weeks
All infants 6-11 months	100 000 IU	A single dose at the age of 6 months (or up to 11 years)
All children 12 – 60 months	200 000 IU	A single dose at the age of 12 months and then every 6 months until 60 months
All postpartum women	200 000 IU	A single dose at delivery (not later than 6-8 weeks after delivery)

Conversion factors: (See Task 1 Session 2)
1 Retinol activity equivalent (RAE) = 1µg retinol
1µg retinol = 3,33 International units (IU)

FEEDBACK

a) Comments on the dosages

Target group	Comments
Non-breastfed children 0-5 months	50 000 IU/ 3,33 = 15 000 µg retinol UL = 600µg/day. Breastfed children will get Vitamin A from the mother through breastmilk.
All infants 6-11 months	All infants 6-11 months: 100 000IU = 30 000 µg retinol, UL = 600µg/day. This dose is given at this age, because breastfeeding will not be exclusive anymore, with the introduction of solids.
All children 12 – 60 months	200 000IU= 60 000 µg retinol. UL = 900-1700µg/day.
All postpartum women	200 000IU = 60 000µg retinol. UL = 3 000µg/day

In relation to the DRIs, the vitamin A content of the supplement is much higher than the UL; however, this is a high dose because it is preventative and is not to be repeated within six months. It will therefore not be toxic because toxicity develops when there is a chronic over consumption of vitamin A. The UL applies to long-term (chronic) nutrient intake, which increases the risk of toxicity.

b) Vitamin A is teratogenic and can cause harm to the fetus, thus no high dose supplementation is encouraged during pregnancy. The post-partum dose will increase the vitamin A content of the breastmilk. It must be given within eight weeks postpartum, as after that, the women may fall pregnant again.

4 WHO RECOMMENDATIONS

The WHO has one set of reference values for all the different micronutrients, called the Recommended Nutrient Intakes (RNIs). This set of RNIs were also developed using EARs and ULs. The WHO use the same definitions for these as are used for the DRIs.

RIs are used as reference values for the evaluation of dietary intakes or to make recommendations concerning *dietary intake of nutrients for individuals or groups, but they are only references and not standards*. The RIs differ for countries and the United Kingdom, Canada, Australia and the Asian countries each have their own set of RIs.

In Task 3 look at the different RIs for the nutrients important in Public Health, using the WHO Recommendations.

READING

WHO/FAO. (2004). Recommended Nutrient Intakes. *Vitamin and Mineral Requirements in Human Nutrition*. 2nd edition. Rome: WHO: 338 - 341.

TASK 3 - USING THE RECOMMENDED INTAKES

List the WHO RNIs for vitamin A, iron, iodine and zinc for children 7-12 months. Note the unit in which the recommendations are made, as well as the ULs for these micronutrients.

FEEDBACK

Micronutrient	WHO	DRI
Vitamin A	400µg RE/day	500µg/day = AI
Iron	6,2 – 18,6mg/day. This recommendation depends	11mg = RDA

	on the bioavailability of iron.	
Iodine	90µg/day	130µg = AI
Zinc	0, 8 – 8,4mg/day. This recommendation depends on bioavailability	3.0mg = RDA

Did you notice the differences in RIs for the different life stages and in relation to gender? Also take note of the slight differences between these two sets of RIs. Once again, remember that there are ULs for these micronutrients, and that they must not be supplemented in excess of these Tolerable Upper Intake Levels.

5 SESSION SUMMARY

In this session, reference intakes for the different micronutrients were introduced. You should now know that there are different sets of Recommended Intakes (RIs) and you should also be able to apply these RIs to different situations.

6 REFERENCES AND FURTHER READING

- Institute of Medicine. Food and Nutrition Board. (1997). *Dietary Reference Intakes for Calcium, Phosphorus, Magnesium, Vitamin D and Fluoride*. Washington DC: National Academy Press. [Online], Available www.nap.edu. [Downloaded 15 June 2004].
- Institute of Medicine. Food and Nutrition Board. (1998). *Dietary Reference Intakes for Thiamine, Riboflavin, Niacin, Vitamin B6, Pantothenic Acid, Biotin and Choline*. Washington DC: National Academy Press. [Online], Available www.nap.edu. [Downloaded 15 June 2004].
- Institute of Medicine. Food and Nutrition Board. (2000). *Dietary Reference Intakes for Vitamin C, Vitamin E, Selenium and Carotenoids*. Washington DC: National Academy Press. [Online], Available www.nap.edu. [Downloaded 15 June 2004].
- Institute of Medicine. Food and Nutrition Board. (2001). *Dietary Reference Intakes for Vitamin A, Vitamin K, Arsenic, Boron, Chromium, Copper, Iodine, Iron, Manganese, Molybdenum, Nickel, Silicon, Vanadium and Zinc*. Washington DC: National Academy Press. [Online], Available www.nap.edu. [Downloaded 15 June 2004].
- Institute of Medicine. Food and Nutrition Board. (2002). *Dietary Reference Intakes for Energy, Carbohydrate, Fibre, Fat, Fatty Acids, Cholesterol, Protein and Amino Acids*. Washington DC: National Academy Press. [Online], Available www.nap.edu. [Downloaded 15 June 2004].
- Vitamin Information Centre, Roche Products. (June 1999). *Medical Update*, 32, South Africa: Isando.

