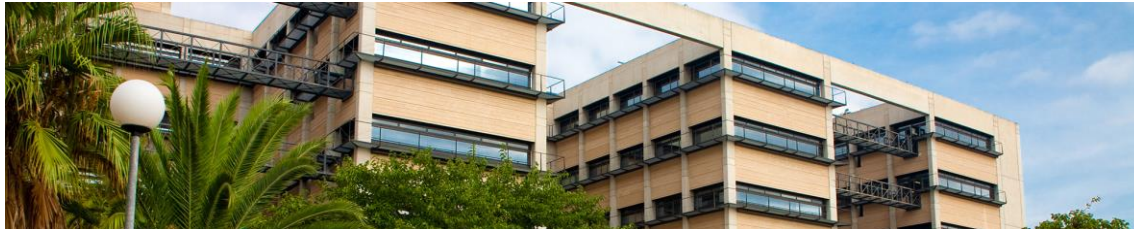




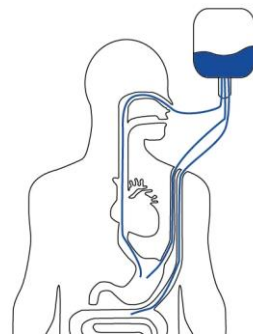
VNIVERSITAT
DE VALÈNCIA

- **Subject** → Clinical Nutrition (Pharmacy Degree -AR group-, 2nd term, 3rd Academic year)
- **Professor** → Dr. Pilar Vila-Donat (Adjunct Professor) on Thursday and Dr. Teresa Climent (Adjunct Professor) on Tuesday.
- **Coordination** → Dr. Ana Frigola, Full Professor
- **Department** → Preventive Medicine, Public Health, Food Sciences, Toxicology and Forensic Medicine, Faculty of Pharmacy
- **mail** → pilar.vila@uv.es
- **Office:** 1st floor (1-64), Faculty of Pharmacy
- **Text book** → Krause's Food and The Nutrition Care Process



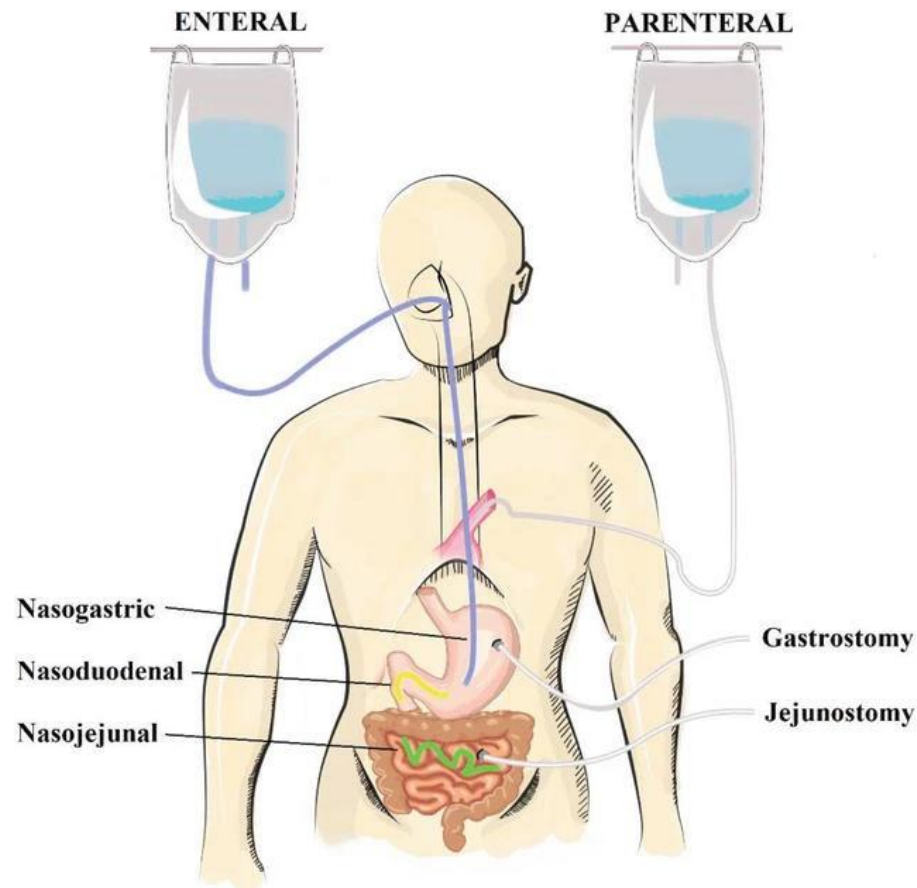
1. INTRODUCTION

1.2 ARTIFICIAL NUTRITION SUPPORT ENTERAL AND PARENTERAL NUTRITION. OBJECTIVES, INDICATIONS AND CHARACTERISTICS.

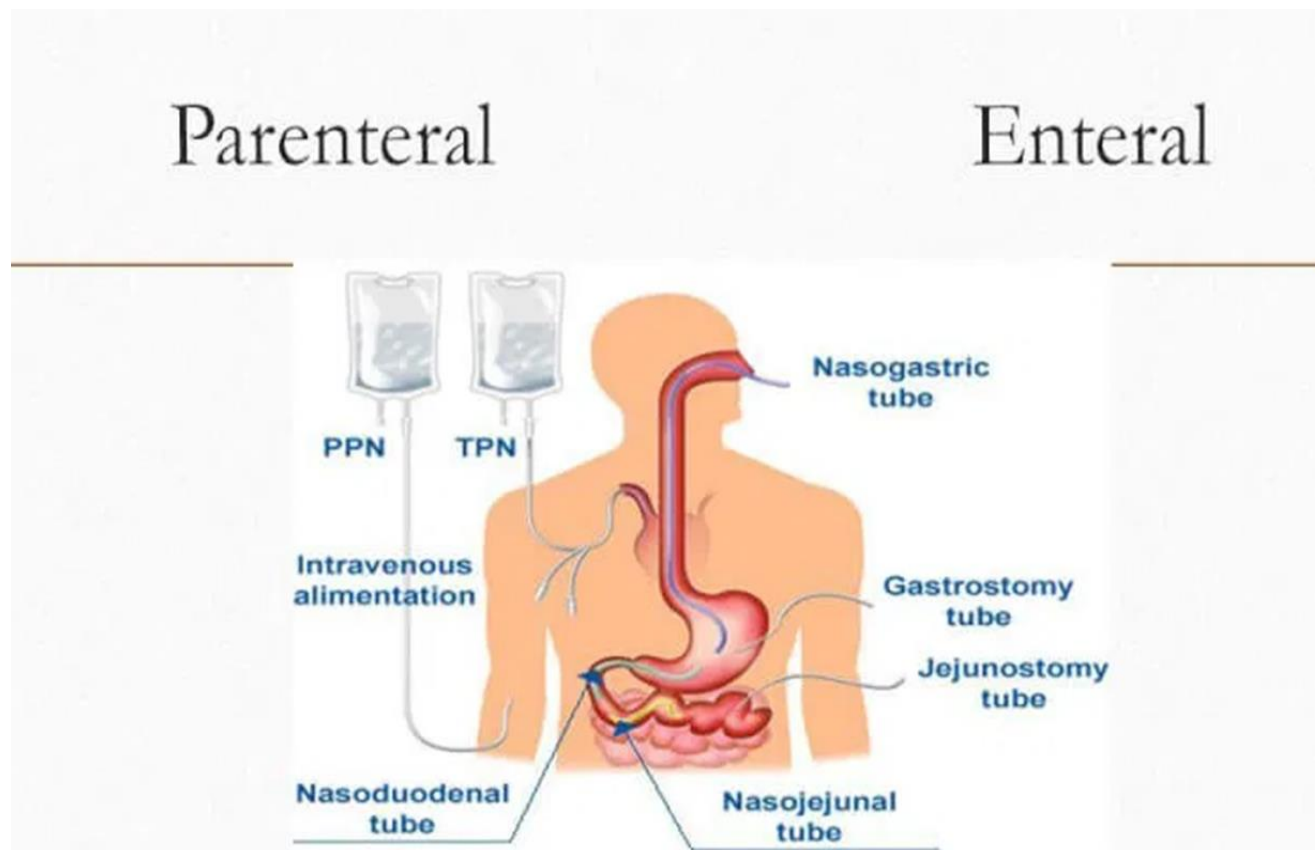


Unit 1.2

Nutrition support is the delivery of formulated enteral or parenteral nutrients to maintain or restore nutritional status.



This support can be given **through total parenteral nutrition (TPN) or via a nasogastric (NG tube) or gastrostomy tube (G-tube or PEG tube).**



➤ When patients are unable to eat enough to support their nutritional needs for more than a few days, nutrition support should be considered.

❖ **Enteral nutrition (EN)** → is the provision of nutrients to the gastrointestinal tract (GIT) through a tube or catheter. In certain instances EN involves the use of formulas as oral supplements or meal replacements.

❖ **Parenteral nutrition (PN)** → is the provision of nutrients intravenously.

→ **EN SHOULD BE THE FIRST CONSIDERATION!**
Use of the gut for nutrition rather than PN alone is preferable for preserving mucosal barrier function and integrity and for preserving immunologic function.



ENTERAL NUTRITION IS THE RECOMMENDED ROUTE OF FEEDING when patients display:

❖ AN INABILITY TO EAT

Neurologic disorders (dysphagia)
Facial trauma
Oral or esophageal trauma
Respiratory failure
Congenital anomalies

❖ AN INABILITY TO EAT ENOUGH

Cancer
Congenital heart disease
Anorexia nervosa
HIV/AIDS
Heart failure
Cystic fibrosis

❖ IMPAIRED DIGESTION, ABSORPTION OR METABOLISM

Pancreatitis
Chron's disease
Inborn errors of metabolism
Severe gastroparesis



PARENTERAL NUTRITION IS THE RECOMMENDED ROUTE OF FEEDING when patients display:

❖ GASTROINTESTINAL INCOMPETENCE

Short bowel syndrome
Severe inflammatory bowel disease
Small bowel ischemia
Intestinal atresia
Severe liver failure
Severe GI bleeding

❖ CLINICAL ILLNESS WITH POOR ENTERAL TOLERANCE OR ACCESSIBILITY

Multiple organ system failure
Major trauma or burns
Bone marrow transplantation
Acute respiratory failure with ventilator dependency and gastrointestinal malfunction
Small bowel transplantation
Immediately after surgery



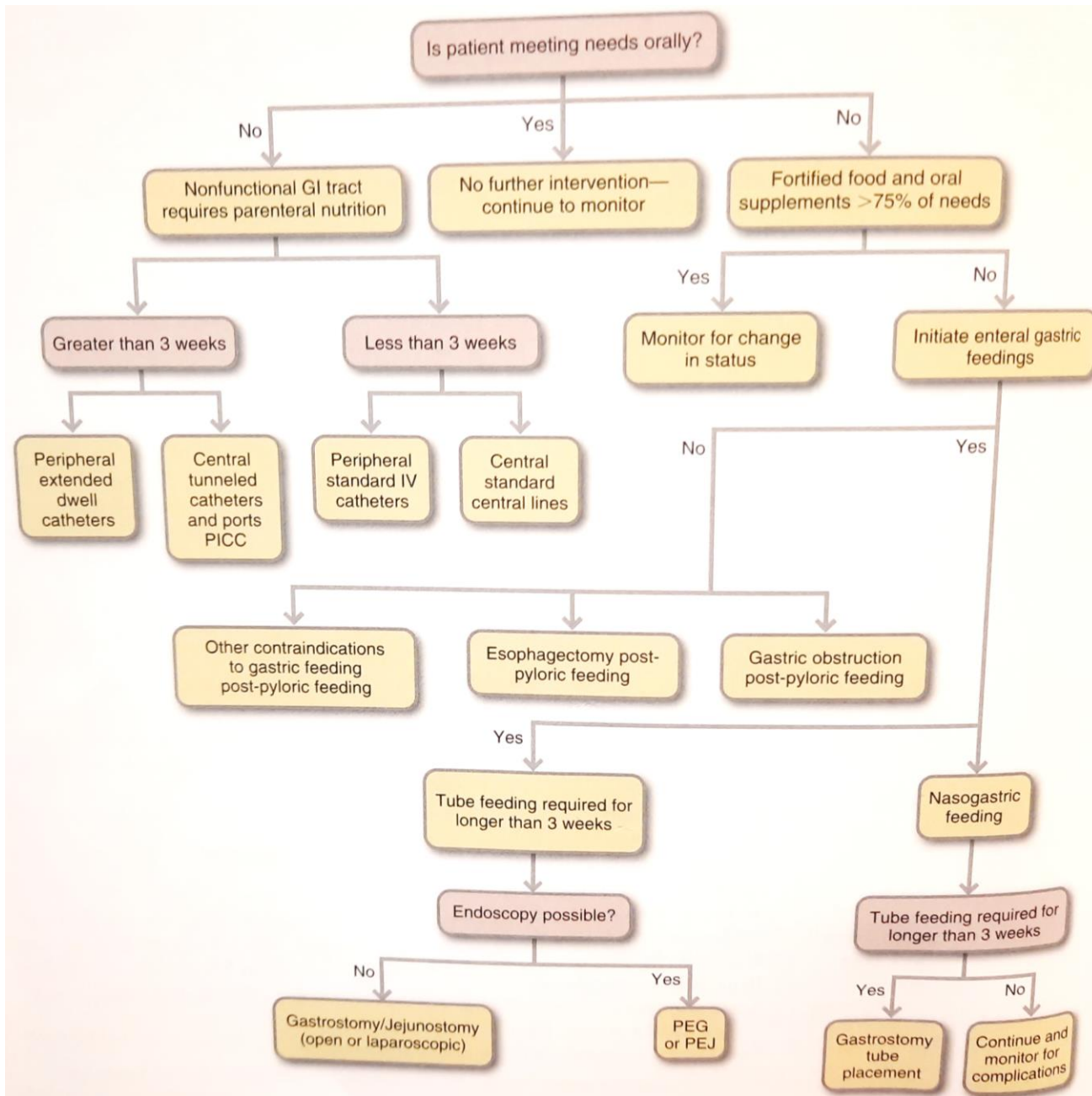
- ❖ **Several criteria should be applied when selecting suitable candidates for nutrition support → EN or PN?**



- PN SHOULD BE USED IN PATIENTS WHO ARE OR WHO WILL BECOME MALNOURISHED AND WHO DO NOT HAVE SUFFICIENT GI FUNCTION TO BE ABLE TO RESTORE OR MAINTAIN OPTIMAL NUTRITIONAL STATUS.**
- PN IS OFTEN USED TEMPORARILY UNTIL GI FUNCTION CAN ADEQUATELY SUPPORT EN OR ORAL INTAKE.**



ALGORITHM FOR SELECTING EN OR PN ROUTES



ENTERAL NUTRITION

By definition, enteral nutrition implies **using the GIT**, primarily by “**tube feeding**”. When a patient is selected as a candidate for EN, the location for nutrient administration and the **type of enteral access** device is also selected. This depends on:

- How long enteral feeding will be needed
- The risk of aspiration
- The patient’s clinical status
- The presence or absence of normal digestion and absorption
- The patient’s anatomy
- Whether a surgical intervention is envisaged



ENTERAL NUTRITION



❑ **SHORT-TERM ENTERAL ACCESS (3-4 weeks)**

→ **NASOGASTRIC ROUTE**

→ Use of NASOGASTRIC TUBES (NGTs) is the most common way to access the GIT.

→ **SMALL-BOWEL FEEDING TUBE**

→ **NASODUODENAL OR NASOJEJUNAL ROUTE:** this route is for patients who are unable to tolerate gastric feedings.

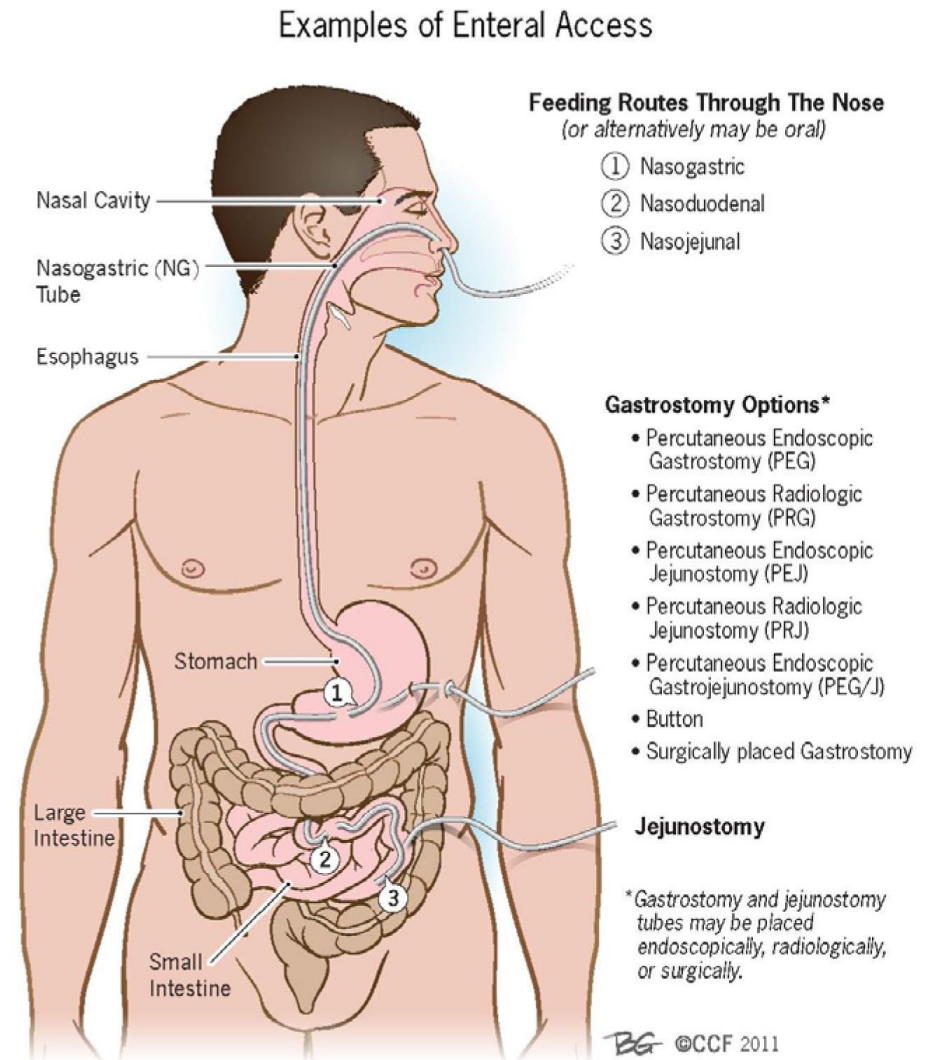
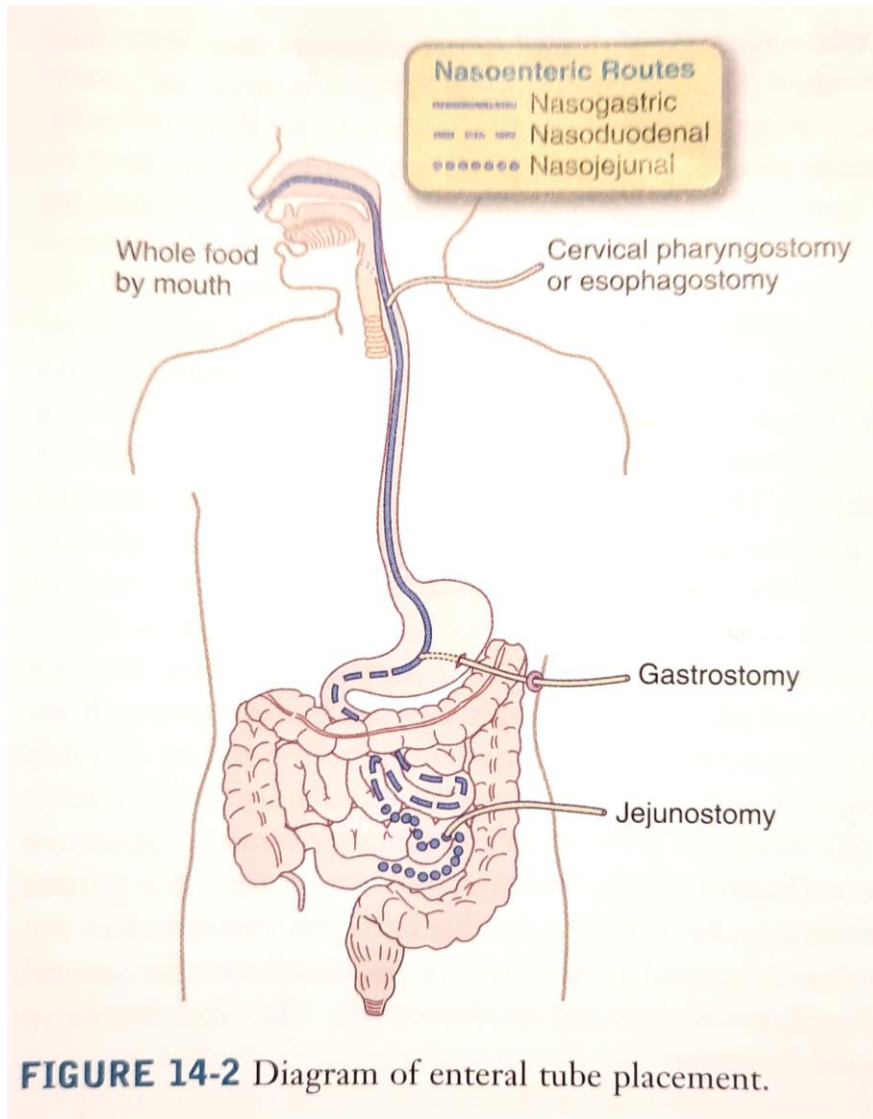
❑ **LONG-TERM ENTERAL ACCESS (more than 3-4 weeks)**

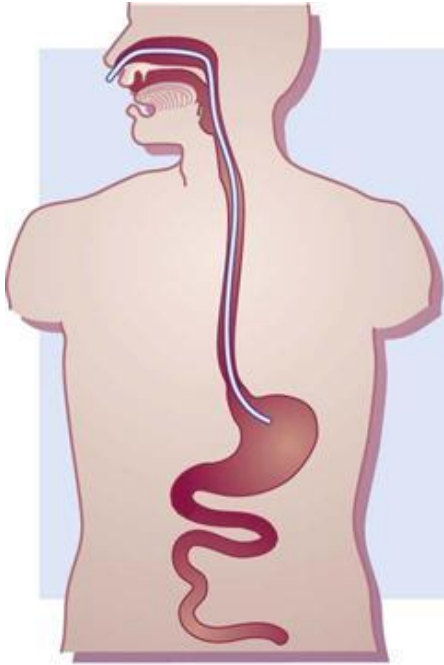
→ **GASTROSTOMY OR JEJUNOSTOMY**

→ **OTHER INVASIVE TECHNIQUES**

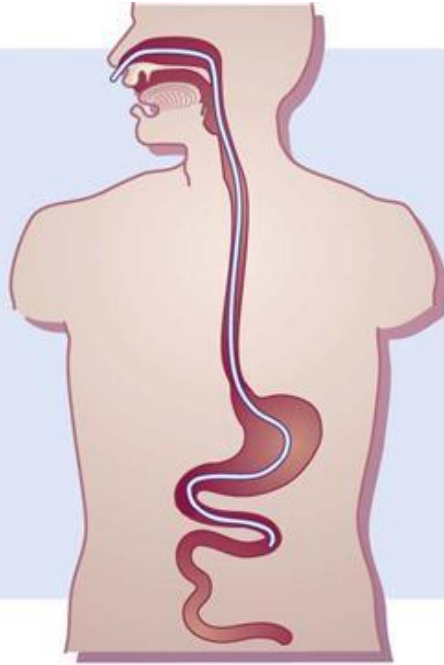
→ **MULTIPLE LUMEN TUBES**



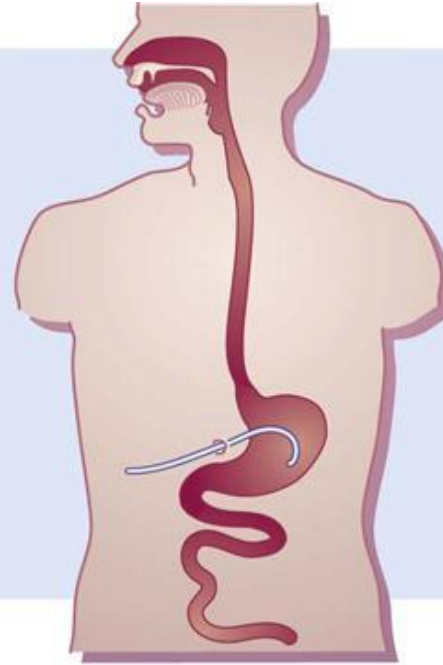




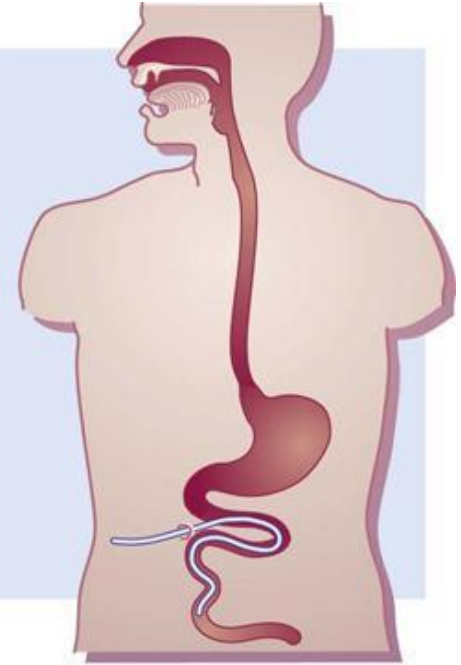
Nasogastric



Nasoduodenal/nasojejunal

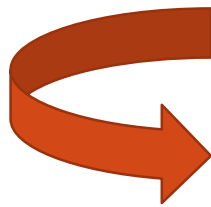
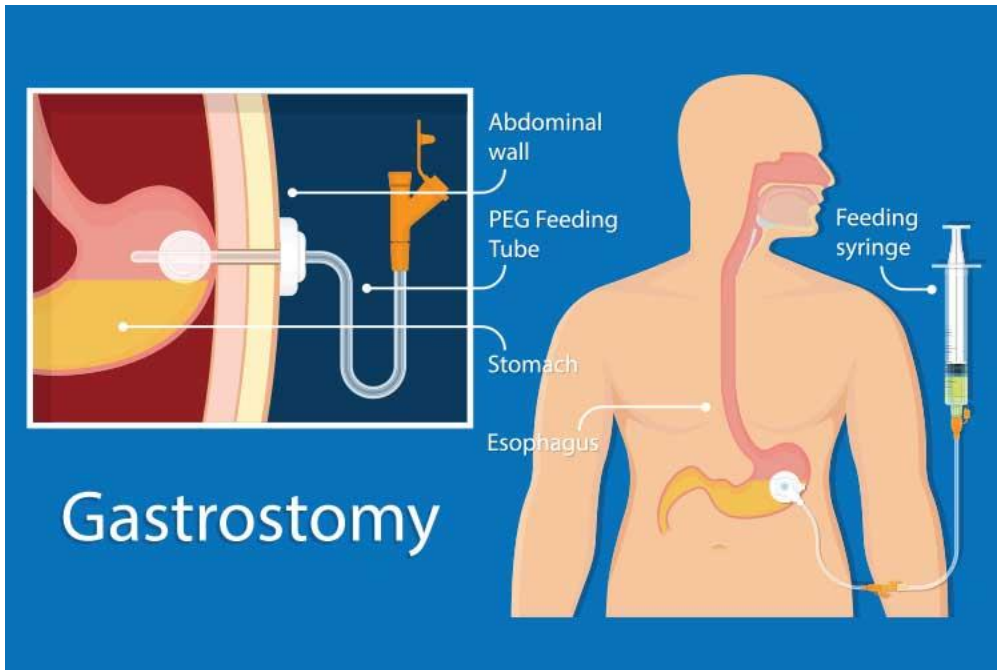


Gastrostomy

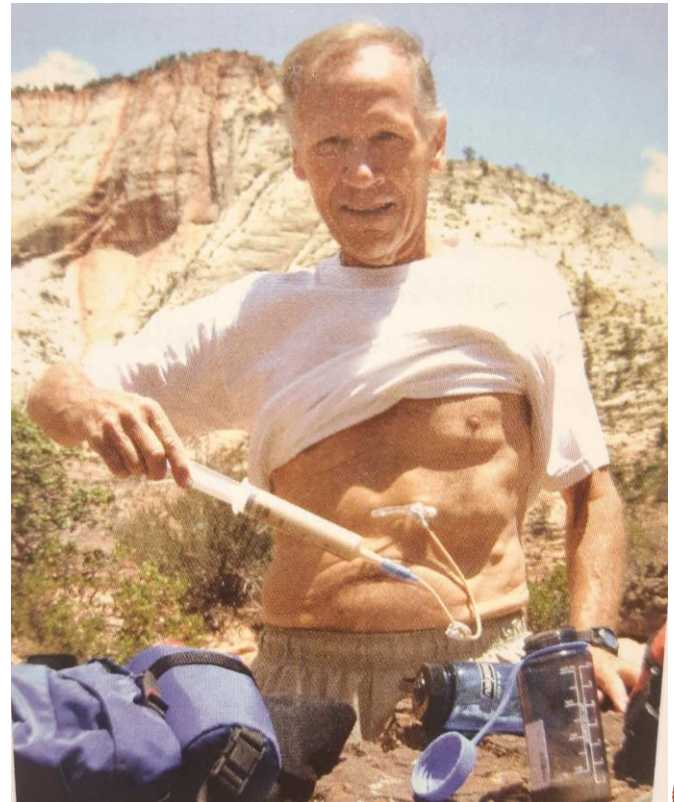


Jejunostomy





A man with a gastrostomy tube out hiking.



POTENTIAL COMPLICATIONS OF NASOENTERIC TUBES

Esophageal strictures

Gastroesophageal reflux resulting in aspiration pneumonia

Incorrect position of the tube leading to pulmonary injury

Mucosal damage at the insertion site

Nasal irritation and erosion

Pharyngeal or vocal cord paralysis

Rhinorrhea, sinusitis

Ruptured gastroesophageal varices in hepatic disease

Ulcerations or perforations of the upper gastrointestinal tract and airway

Adapted from McClave SA et al: Guidelines for the provision and assessment of nutrition support therapy in the adult critically ill patient, JPEN J Parenter Enteral Nutr 33: 277, 2009.



- ❖ **ENTERAL FORMULAS** can be classified as:
- 1) standard polymeric formulas,
 - 2) elemental or predigested formulas, or
 - 3) specialized formulas.



ENTERAL FORMULAS

PROTEIN → 6% TO 25% OF TOTAL KCAL. DERIVED FROM CASEIN, WHEY OR SOY PROTEIN ISOLATE. 1 → INTACT PROTEIN. 2, 3 → DI- AND TRIPEPTIDES AND AA. 3 → CRYSTALLINE AA.

CARBOHYDRATE → 30% TO 85% OF TOTAL KCAL. CORN SYRUP SOLIDS ARE USUALLY THE CH FOUND. FOS. LACTOSE-FREE.

LIPID → 1.5% TO 55% OF THE TOTAL KCAL, USUALLY FROM CORN, SOY, SUNFLOWER OILS. 2 → MINIMUM AMOUNTS OF LONG-CHAIN FAT, AND 2-4% OF DAILY ENERGY INTAKE FROM ω 3 AND ω 6.

VITAMINS, MINERALS AND ELECTROLYTES → MEET THE DRIs. Formulas intended for treating renal and hepatic failure are low in specific vitamins, minerals and electrolytes.

FLUID → 1ML water/KCAL. All sources of fluids given to a patient receiving EF (medications, etc.) should be considered when calculating the patient's intake.



FORMULA CONTENT AND SELECTION

Factors to Consider When Choosing an Enteral Formula

Ability of the formula to meet the patient's nutritional requirements

Caloric and protein density of the formula (i.e., kcal/mL, g protein/mL, kcal:nitrogen ratio)

Gastrointestinal function of the patient

Presence of lactose, which may not be tolerated

Sodium, potassium, magnesium, and phosphorus content of the formula, especially in cardiopulmonary, renal, or hepatic failure

Type of protein, fat, carbohydrate, and fiber in the formula tolerable for the patient's digestive and absorptive capacity

Viscosity of the formula related to tube size and method of feeding



TUBES: FEEDING ADMINISTRATION

→ **BOLUS FEEDING** → the patients are clinically stable.

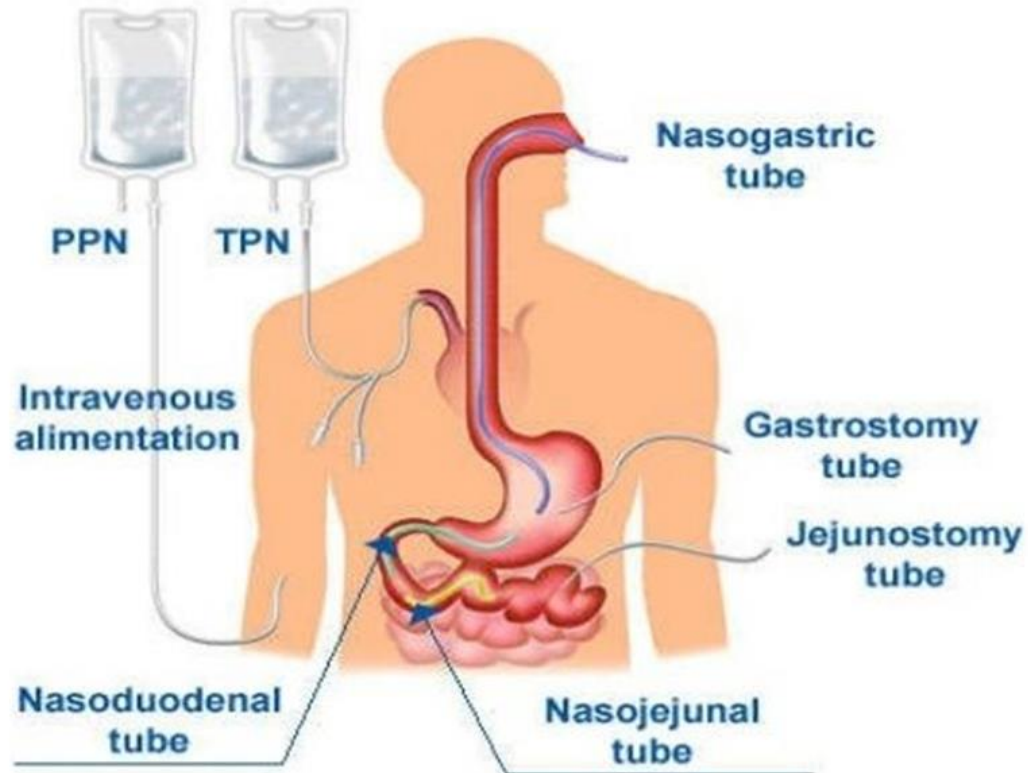
→ **INTERMITTENT DRIP** → administered by pump or gravity drip.

→ **CONTINUOUS DRIP INFUSION** → administration requires a pump

- **The method is selected according to the patient's clinical status, living situation, and quality of life.**
- **Methods can be changed as the patient's status changes.**



ROUTES OF FEEDING



COMPLICATIONS OF ENTERAL NUTRITION

Access Problems

Leakage from ostomy/stoma site
Pressure necrosis/ulceration/stenosis
Tissue erosion
Tube displacement/migration
Tube obstruction

Administration Problems

Microbial contamination
Misplacement of tube, causing infection or aspiration pneumonia or peritonitis
Regurgitation

Gastrointestinal Complications

Constipation
Delayed gastric emptying
Diarrhea
 Osmotic diarrhea, especially if sorbitol is given in liquid drug preparations
 Secretory
Distention/bloating/cramping
Formula choice/rate of administration
High gastric residuals
Intolerance of nutrient components
Maldigestion/malabsorption
Medications
Nausea/vomiting
Treatment/therapies

Metabolic Complications

Drug-nutrient interactions
Glucose intolerance/hyperglycemia
Hydration status—dehydration/overhydration
Hypoalbuminemia
Hyponatremia
Hypoglycemia
Hyperkalemia/hypokalemia
Hyperphosphatemia/hypophosphatemia
Micronutrient deficiencies
Refeeding syndrome



Enteral nutrition

Enteral nutrition guide from the Spanish National Health System

- EN is an alternative for many patients who are **unable to eat food** because of their clinical situation.
- Progress in the formulas, methods and routes of administration have made EN a simple and useful technique for treating many situations, including alterations in intake, digestion or nutrient absorption.
- The current trend towards shortening hospital stays implies that **enteral nutrition should be done at home** for patients who require nutritional support but do not require hospitalization.
- EN is one of the health benefits of the Spanish National Health System.

If EN formulas are to be funded, they must be registered in the General Health Register of Food as “**dietary foods for special medical purposes**”.



PARENTERAL NUTRITION

- ✓ PN administers nutrients directly into the bloodstream intravenously.
- ✓ PN is indicated when the patient requires nutritional support but is unable or unwilling to take adequate nutrients orally or enterally.
- ✓ PN may be used as an adjunct to oral nutrition or EN for meeting a patient's nutritional needs. It may also be the sole source of nutrition during recovery from illness or injury, or as a life-sustaining therapy for patients who have lost the function of their intestine for nutrient absorption.

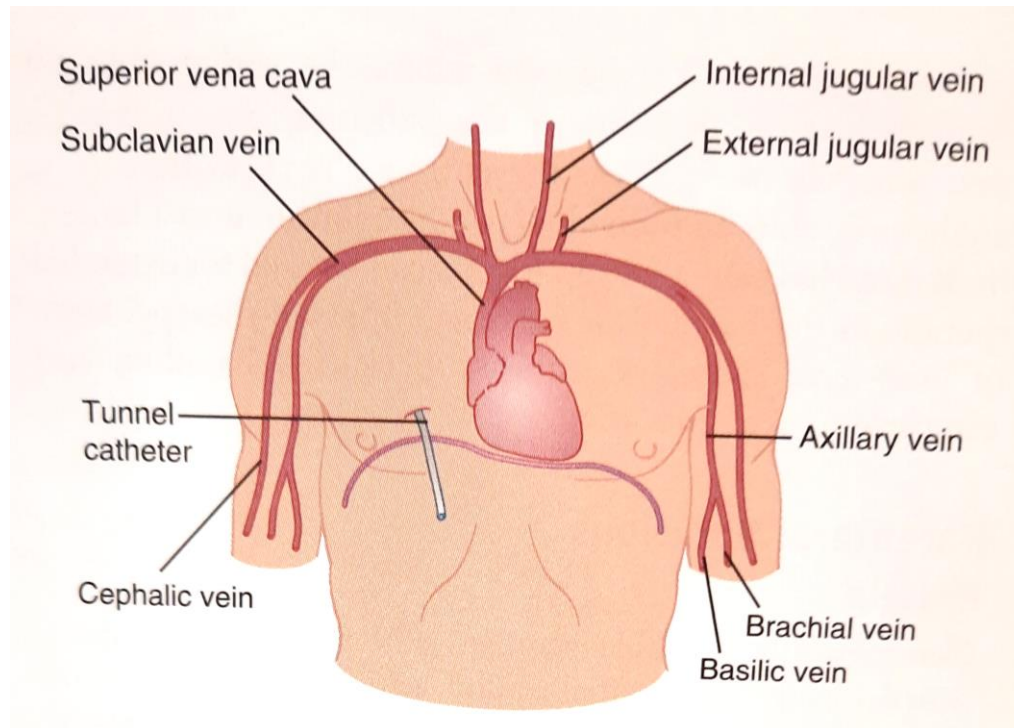


The practitioner must choose between central and peripheral access:

- **Central parenteral nutrition (CPN)** → the catheter tip is placed in a large, high-blood-flow vein such as the superior vena cava.
- **Peripheral parenteral nutrition (PPN)** → the catheter tip is placed in a small vein, usually in the hand or forearm.



Venous sites from where the superior vena cava can be accessed



ADMINISTRATION

- CONTINUOUS INFUSION
- CYCLIC INFUSION



COMPOSITION OF PARENTERAL SOLUTIONS

- ✓ PROTEIN → 3-20% by volume. 15 to 20% of total energy intake should come from protein. Including all essential amino acids (AA) and only some of the non-essential crystalline AA.
- ✓ CARBOHYDRATES → dextrose monohydrate. 5 to 70% by volume.
- ✓ LIPID → source of essential fatty acids. Aqueous suspensions of soybean or safflower oils. 20 to 30% of total Kcal.
- ✓ ELECTROLYTES, VITAMINS, TRACE ELEMENTS
- ✓ FLUID



Adult Parenteral Multivitamins: Comparison of Guidelines and Products

Vitamin	NAG-AMA Guidelines	FDA Requirements	MVI-12	MVI-13 (Infuvite) Baxter
A (retinol)	3300 units (1 mg)	3300 units (1 mg)	3300 units (1 mg)	3300 units (1 mg)
D (ergocalciferol cholecalciferol))	200 units (5 mcg)	200 units (5 mcg)	200 units (5 mcg)	200 units (5 mcg)
E (mcg-tocopherol)	10 units (10 mg)	10 units (10 mg)	10 units (10 mg)	10 units (10 mg)
B ₁ (thiamin)	3 mg	6 mg	3 mg	6 mg
B ₂ (riboflavin)	3.6 mg	3.6 mg	3.6 mg	3.6 mg
B ₃ (niacinamide)	40 mg	40 mg	40 mg	40 mg
B ₅ (dexpantenol)	15 mg	15 mg	15 mg	15 mg
B ₆ (pyridoxine)	4 mg	6 mg	4 mg	6 mg
B ₁₂ (cyanocobalamin)	5 mcg	5 mcg	5 mcg	5 mcg
C (ascorbic acid)	100 mg	200 mg	100 mg	200 mg
Biotin	60 mcg	60 mcg	60 mcg	60 mcg
Folic acid	400 mcg	600 mcg	400 mcg	600 mcg
K		150 mcg	0	150 mcg

From Fed Reg 66(77), 2000

Daily Electrolyte Requirements During Total Parenteral Nutrition—Adults

Electrolyte	Standard Intake/Day
Calcium	10-15 mEq
Magnesium	8-20 mEq
Phosphate	20-40 mmol
Sodium	1-2 mEq/kg + replacement
Potassium	1-2 mEq/kg
Acetate	As needed to maintain acid-base balance
Chloride	As needed to maintain acid-base balance

From McClure SA



COMPLICATIONS OF PARENTERAL NUTRITION

Mechanical Complications

Air embolism
Arteriovenous fistula
Brachial plexus injury
Catheter fragment embolism
Catheter misplacement
Cardiac perforation
Central vein thrombophlebitis
Endocarditis
Hemothorax
Hydromediastinum
Hydrothorax
Pneumothorax or tension pneumothorax
Subcutaneous emphysema
Subclavian artery injury
Subclavian hematoma
Thoracic duct injury



Other complications

Metabolic Complications

Dehydration from osmotic diuresis
Electrolyte imbalance
Essential fatty acid deficiency
Hyperosmolar, nonketotic, hyperglycemic coma
Hyperammonemia
Hypercalcemia
Hyperchloremic metabolic acidosis
Hyperlipidemia
Hyperphosphatemia
Hypocalcemia
Hypomagnesemia
Hypophosphatemia
Rebound hypoglycemia on sudden cessation of PN in patient with unstable glucose levels
Uremia
Trace mineral deficiencies

Gastrointestinal Complications

Cholestasis
Gastrointestinal villous atrophy
Hepatic abnormalities

Infection and Sepsis

Catheter entrance site
Catheter seeding from bloodborne or distant infection
Contamination during insertion
Long-term catheter placement
Solution contamination



PROCESS OF NUTRITIONAL CARE FOR EN AND PN

Assessment

1. Clinical status, including medications
2. Fluid requirement
3. Route of administration
4. Energy (kcal) requirement
5. Protein requirement
6. Carbohydrate/lipid considerations
7. Micronutrient considerations
8. Formula selection or PN solution considerations
 - A. Concentration (osmolarity)
 - B. Protein content
 - C. Carbohydrate/lipid content
 - D. Micronutrient content
 - E. Special formula considerations
9. Calculations
 - A. Energy: use kcal/mL formula
 - B. Protein: use g/1000 mL
 - C. Fat and micronutrient considerations:
units/1000 mL
 - D. Fluid considerations: extra water, IV fluids
(including medications)



The **hospital pharmacist** has always been responsible for preparing **parenteral nutrition** (PN).

PN must be sterile and stable (physicochemical).

The hospital pharmacist reports which drugs can be administered within or with the PN.

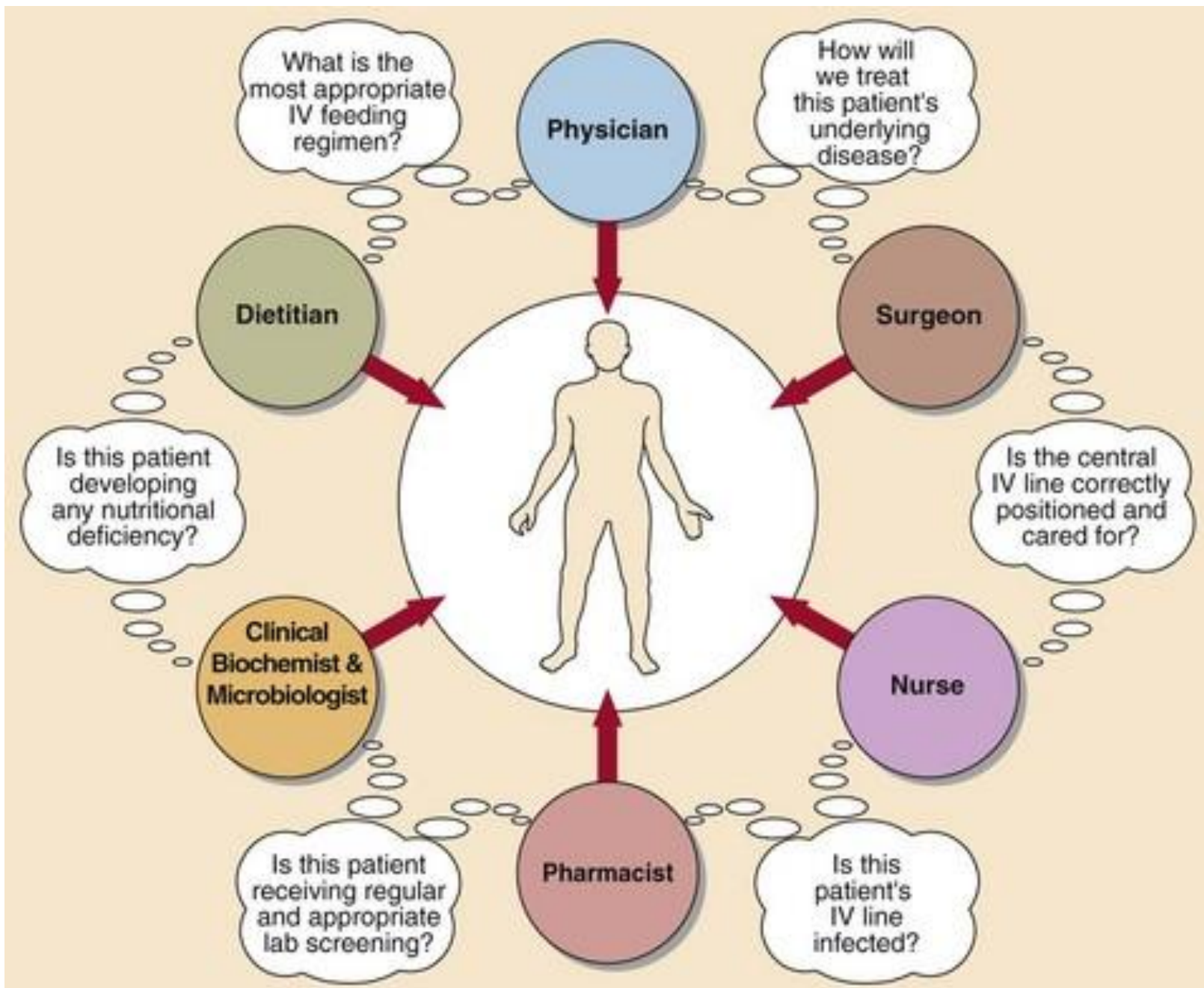
In the case of **enteral nutrition**, the industry sells the final product, which is prepared with all the components.

There are two basic premises for determining who should manage EN and when:

- EN is essential for those who cannot eat food orally.
- EN must be adapted to the patient's illness.
- EN or PN will be selected only in accordance with the **functionality** of the digestive system.
- If the patient has a sufficiently large intestinal surface that functions properly and is accessible, nutritional intake should be enteral.

PN is more aggressive and involves more complications.





All professionals must work together and be coordinated.



ENTERAL FEEDING VERSUS PARENTERAL FEEDING

ENTERAL FEEDING

Any method of feeding, which uses the gastrointestinal tract to deliver part or all of a person's caloric requirements

The method of getting nutrition into the body through veins

Routes: Oral, sublingual, and rectal

Organs: Esophagus, stomach, and small and large intestines

Uses a feeding tube

More physiological, simpler, cheaper and less complicated

Depends on the gastric/intestinal function

Has fewer complications

PARENTERAL FEEDING

The method of getting nutrition into the body through veins

Normal urine is either colorless or pale yellow in color

Routes: Intravenous

Organs: Central or peripheral veins

Uses a catheter

A less physiological, complex, comparatively expensive, and more complicated process

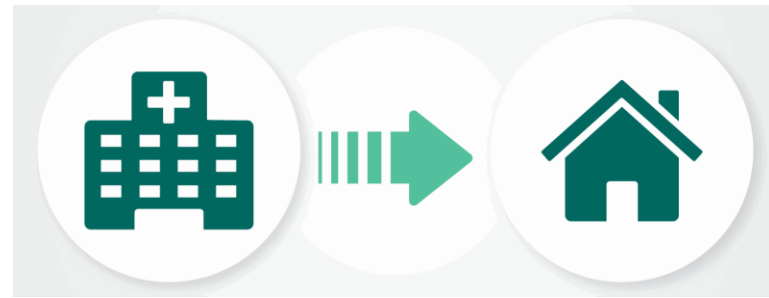
Does not depend on the gastric/intestinal function

Has more complications

Visit www.PEDIAA.com



TRANSITIONAL FEEDING:



 PARENTERAL TO ENTERAL FEEDING

 PARENTERAL TO ORAL FEEDING

 ENTERAL TO ORAL FEEDING

 ORAL SUPPLEMENTS



**INITIAL ORAL DIETS MUST
BE LACTOSE-FREE AND
LOW IN SIMPLE CH AND FAT.**



Thank You
For Your Attention...



2. MODIFIED DIETS

2.1 TEXTURE-MODIFIED DIETS

TYPES. NUTRITIONAL OBJECTIVES. INDICATIONS, CONTRAINDICATIONS AND ADVERSE EFFECTS. CHARACTERISTICS. PRACTICAL DETAILS OF IMPLEMENTATION. PROGRESSIVE DIETS.



Unit 2.1

COMPETENCES:

Block 2 discusses modifications to the composition and texture of diets to determine how they can be used to treat diseases and/or improve patients' quality of life.

This unit will discuss texture-modified diets. Types. Indications. Characteristics. Practical details on their implementation. Progressive diets.



What does a modified consistency mean?

Foods that have been altered physically



Mashed



Chopped



Grounded



Purees



FOODS

Soft foods and process-softened foods

Minced and moist foods

Purees and patés

Jellies and mousses

Creamy foods

Thickened liquids

Soups

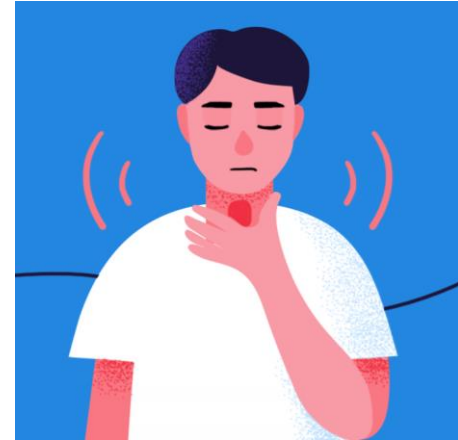
Thin drinks

DRINKS



Texture-modified meals are developed for patients with swallowing difficulties

Difficulty in swallowing, also called dysphagia, is present in older patients as well as in stroke and cancer patients. These patients must be given smaller-sized food with specific textures, such as softer solid foods. This helps to minimize choking and aspiration (inhaling food into the lungs).



Dysphagia

- ✓ **Symptoms of dysphagia include** drooling, choking or coughing during or after meals, the inability to suck from a straw, a gurgled voice, holding pockets of food in the buccal recesses, absent gag reflex, and chronic upper respiratory infections.
- ✓ Dysphagia often leads to **malnutrition** due to inadequate intake (weight loss and anorexia).
- **Swallowing evaluation** is important when assessing and treating swallowing disorders. → This should be done by a speech-language pathologist or registered dietitian.
- Swallowing occurs in three phases:
 - The voluntary or oral phase (the tongue presses food against the hard palate).
 - The involuntary reflex or pharyngeal phase (early, middle or late).
 - The involuntary or oesophageal phase (peristalsis).

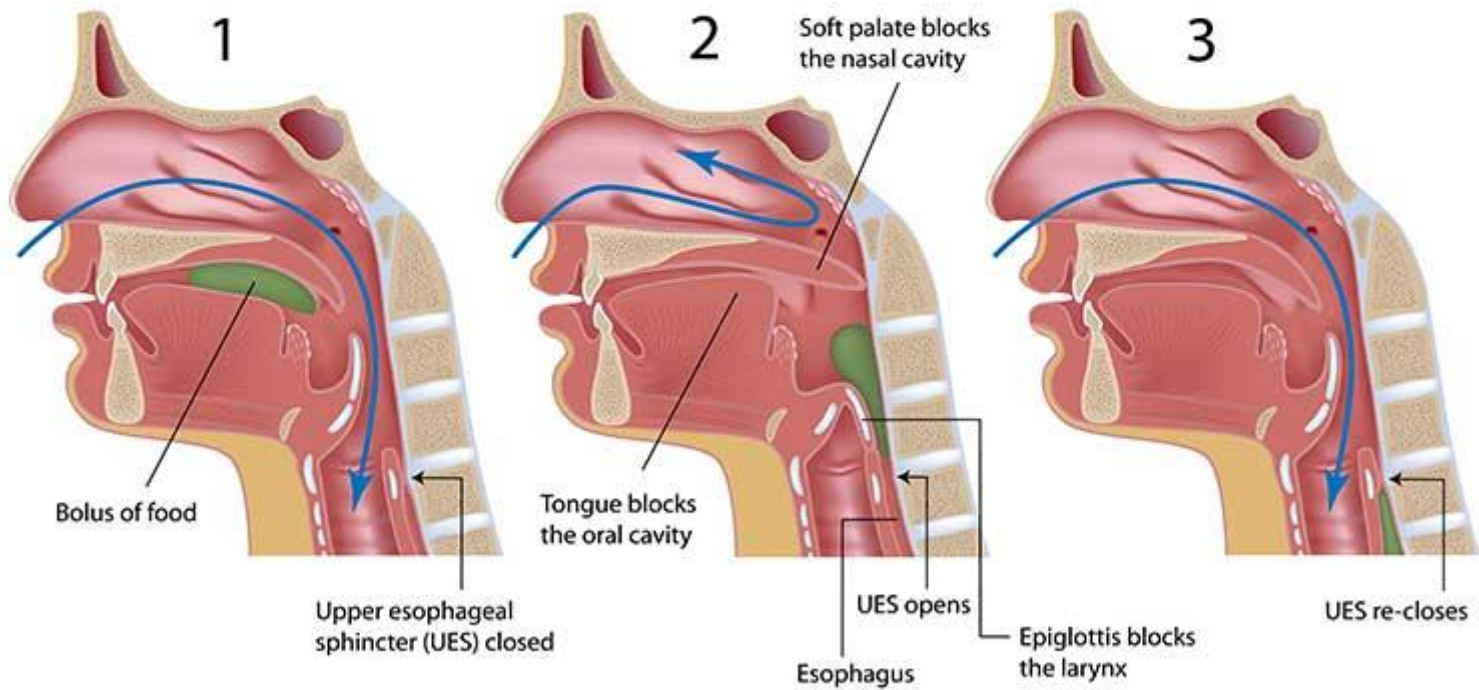


Phase of swallowing	Frequent findings
Oral	Repetitive pump movements of the tongue Oral residue Premature spillage Piecemeal deglutition
Pharyngeal	Residue in valleculae and pyriform sinuses Aspiration in 50% of dysphagic patients Somatosensory deficits Reduced spontaneous swallow (48 vs 71 per hour)
Esophageal	Hypomotility Spasms Multiple contractions

Note: Data from Warnecke.¹⁷⁰



Swallow Function



Symptoms of Dysphagia

- Choking on food or drink
- Coughing during or after swallowing
- Coughing or vomiting up food
- Having a weak, soft voice
- Aspirating (getting food or liquid into your lungs)



- Excessive saliva or drooling
- Difficulty chewing
- Trouble moving food to the back of your mouth
- Food sticking in your throat



TEXTURE-MODIFIED DIETS

These diets are indicated for patients who need a change in the texture of their diet or minimal gastrointestinal stimulation.

Liquid diet

Semi-solid
diet

Easy-chew
diet

Progressive
diet



LIQUID DIETS

INCOMPLETE LIQUID DIETS

- ❖ **Aims:** to keep electrolyte balance and minimal gastrointestinal stimulation.
- ❖ **Indications:**
 - a) Patients recovering from paralytic ileus.
 - b) Patients with acute diarrhoea.
 - c) As an intermediate stage for patients between parenteral nutrition and oral nutrition.
- ❖ **Characteristics:** as these diets cannot satisfy the patient's nutritional needs (energy), they should not be maintained for too long.
- ❖ **Composition:** oral rehydration fluids or food at normal temperature.
- ❖ **Tolerance** depends on osmolality, volume, speed administration and the interval between meals: $>$ osmolality $<$ volume and $<$ speed.



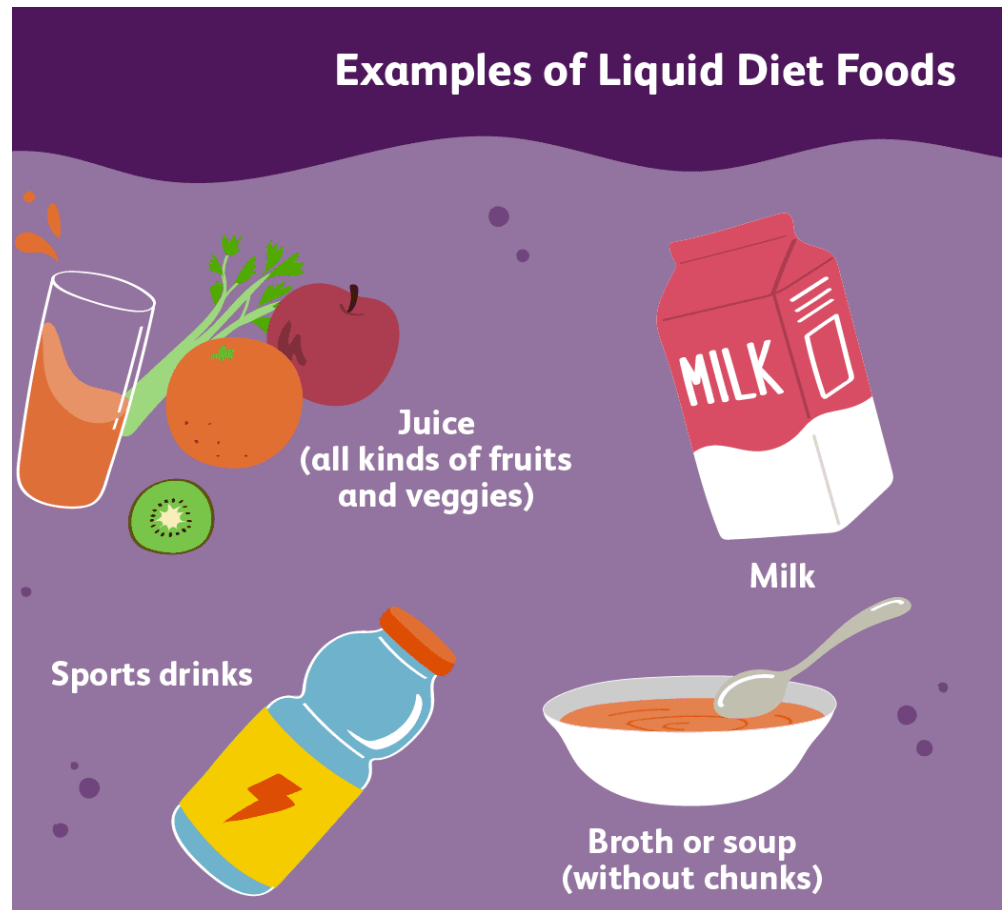
COMPLETE LIQUID DIETS

- ❖ **Aim:** to satisfy the energy and nutritional requirements of patients who cannot chew, swallow or digest solid foods.
- ❖ **Indications:**
 - Patients with oropharynx and oesophagus diseases at risk of aspiration.
 - Overall committing intake (chewing, swallowing, digestion).
 - Patients put on a liquid diet after an incomplete diet.
- ❖ **Characteristics:** These diets can be sustained for a long time; the patient's nutritional needs are assessed; the diets can be adapted to the patient's tastes; flavour and presentation are important; the patient's evolution is monitored.
- ❖ **Composition:** liquid food and liquefied solids, supplements, enteral nutrition formulas, and baby foods.
- ❖ **Problems:** hyper-osmolality, lactose intolerance, hypertriglyceridemia and/or hypercholesterolemia, constipation, danger of infection.



LIQUID DIETS

- Water
- Rice water
- Carrot juice
- Diluted fruits
- Strained dairy milk
- Dairy liquid infusions
- Ice cream
- Watery defatted broths



SEMI-SOLID DIETS

- Complete diets with pureed, more or less thick food and with more food than liquid diets (they are more palatable and easier to tolerate, schedule, develop and retain). They can be used for a long time, and sometimes forever.
- It is easier to control their presentation and organoleptic characteristics.
- Nutritional requirements can be adjusted better.
- Nutritional status can be controlled.

Composition

- Mashed solids are added to liquid to obtain a more or less thick mash.
- They should be given in small, frequent meals and should not include foods that:
 - have a double texture
 - form small fluid bolus
 - have a pungent smell or are unsightly.

Indications

- Patients with dysphagia or difficulty in chewing or who are at an intermediate stage in progressive diets.



SEMI-SOLID DIETS



SOFT DIETS

- These are complete diets based on solid foods that are easily digestible, whole foods, and foods subjected to gentle cooking with little fat and little flavour.

Indications

- Patients at an intermediate stage in progressive diets.
- Patients with digestive pathologies such as ulcers and hiatal hernia.
- Patients with infections or fever.

Characteristics

- These diets include all types of food.
- They may contain fats and be low in fibre.
- They are always cooked.
- They contain almost no spices.
- Fried foods are avoided.
- Patients should eat 5-6 meals that are not copious and are suitably spaced out time-wise;
- The following foods are avoided:
 - Stimulants (e.g. caffeine, theobromine, protein, 'cokes', alcohol).
 - Physical irritants (e.g. fibrous meats, whole grains, nuts, raw vegetables, fruits).
 - Chemical irritants (e.g. acids, smoked foods, salted foods, meat concentrates).
 - Raw olive oil.



Soft diets (*dieta blanda*):

Mashed potato

Semolina

Boiled and poached eggs

Crème caramel

Yogurt

Cooked apple

Purees

Vegetables

Meat or fish

Fruit



→ Food to avoid:

Stimulants (e.g. caffeine, theobromine, protein, 'cokes', alcohol).

Physical irritants (e.g. fibrous meats, whole grains, nuts, raw vegetables or fruits).

Chemical irritants (e.g. acids, smoked foods, salted foods, meat concentrates, etc.).



EASY-CHEWING SOFT DIETS

❖ **Aims:**

To provide a complete, balanced and varied diet.

The only limitation is that they should not contain hard elements.

❖ **Characteristics:** The foods should require minimum chewing.

❖ **Indications:**

Patients committed to chewing as a mechanical process.

Patients with missing tooth pieces.

Patients with a painful or inflamed oral cavity.

Patients with adequate digestion.

❖ **Characteristics:** Implementation depends on the patient:

Some foods may be excluded.

The patient's tastes and preferences are taken into account.

Texture levels should be tolerated.



Easy-chewing soft diets (*Dietas de fácil masticación*)



PROGRESSIVE DIETS

❖ **Aims:** These dynamic diets, which are widely used in hospitals, are based on the need to adapt to various situations that affect patients as their illness evolves.

❖ **Indications:**

Patients at the post-operative stage, especially after major abdominal surgery.

Patients requiring oral refeeding after prolonged fasting.

Patients suffering from severe malnutrition or recovering from total parenteral nutrition.

❖ **Characteristics:**

Texture modifications are increasingly applied.

Modified textures range from oral fluids to the basal diet.



Progressive diets: **IMPLEMENTATION (sequence)**

Liquid diets



Semi-liquid/semi-soft /semi-solid diets



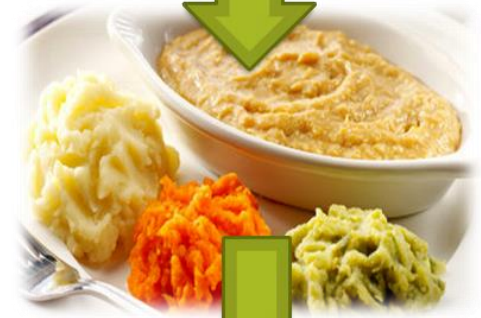
Easy-digestion diets



Soft diets



In-hospital diets (basal diets)



EASY-DIGESTION DIETS (*Dietas de fácil digestión*):

Characteristics:

Gentle cooking: boiled foods, omelettes, etc.

No raw foods

No seasonings

No stimulants

No physical irritants

No chemical irritants

Small volumes taken in 5-6 meals



Texture

≈ Normal

**Easy-digestion foods
culinary processed**

Fibre and lipids not↑



Hospital Basal Diets

- ❖ All culinary options
- ❖ **Avoid food that are:**
 - Flatulent
 - Difficult to digest
 - Spicy
 - Not varied or balanced



❖ **Techniques to improve the acceptance of modified-texture diets:**

- ✓ Take extra care when feeding individuals with dysphagia.
- ✓ Make pureed meals look good, smell good, and taste good.
- ✓ Be creative; serve attractive meals.
- ✓ Create a pleasant atmosphere to increase the patient's appetite and consumption.
- ✓ Use aromatic ingredients such as garlic, pepper, onions and cinnamon.
- ✓ Taste all foods and enhance seasoning as required.
- ✓ Serve foods with strong flavours, such as chili.



❖ **Techniques to improve the acceptance of modified-texture diets:**

- ✓ Give patients small, frequent meals to encourage them to eat more.
- ✓ Bear in mind that cool temperatures facilitate swallowing, so patients may tolerate them better.
- ✓ Remember that sauces lubricate foods and help prevent the fragmentation of food in the oral cavity.
- ✓ Avoid foods that crumble easily since they can increase the risk of choking.



Well-presented modified-texture meals





30+
Soft Food
Recipes



Thank You
For Your Attention...



2.3 HIGH-PROTEIN AND HIGH-ENERGY DIETS.

NUTRITIONAL OBJECTIVES AND INDICATIONS.

CHARACTERISTICS.



Unit 2.3

18/02/21

The current recommended dietary reference intake (DRI)
for healthy individuals is
0.8 grams protein/kilogram of body weight per day.

High-protein and high-energy diets



These are diets with high protein intakes (**>1.5 g/kg/day**) and more energy than is usually recommended for healthy people.



High-protein diets are prescribed for patients with increased energy and nutritional requirements to prevent or correct protein catabolism and weight loss (MN) associated with higher morbidity and mortality rates → metabolic stress produced by some **diseases**.

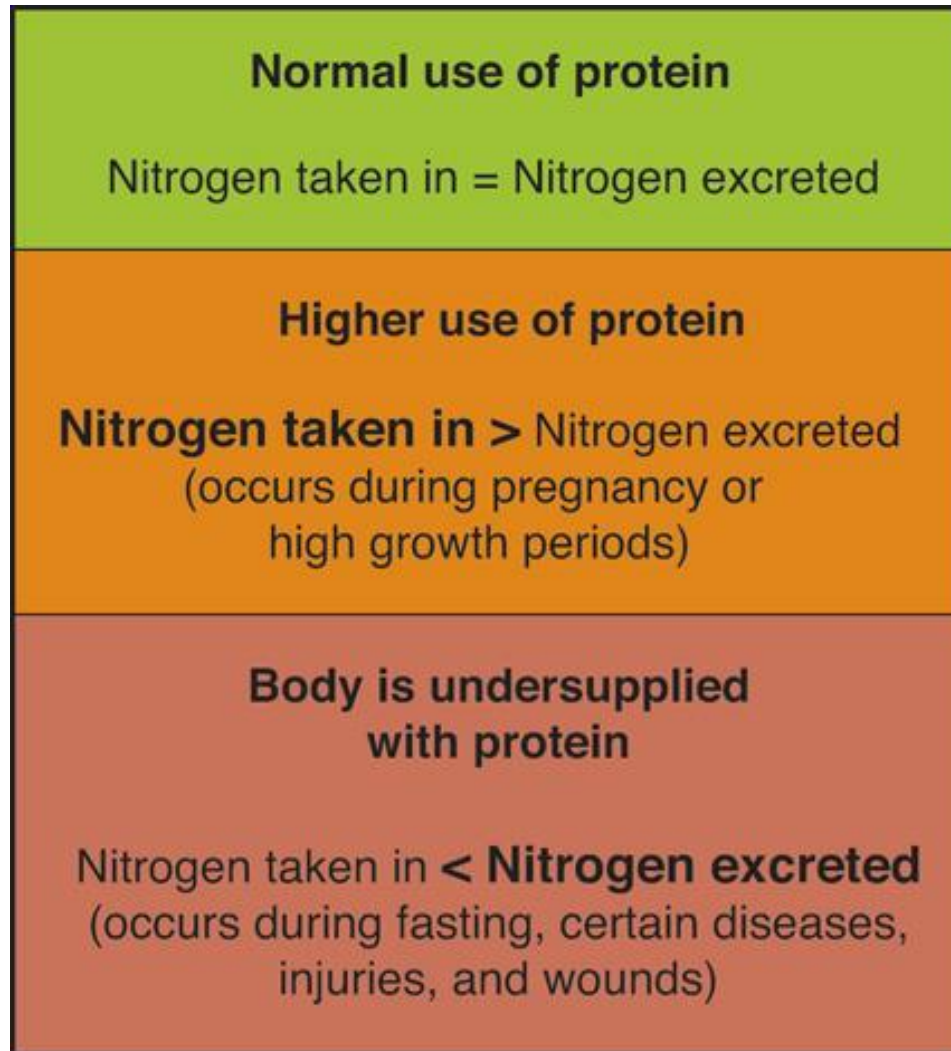


NITROGEN BALANCE

- ✓ Protein supplies nitrogen in the form of amino acids according to the formula (grams N \rightarrow grams protein \rightarrow x 6.25).
- ✓ Over 95% of protein is absorbed normally and enters the synthetic pool. Muscle proteins and visceral (i.e. plasma) proteins are broken down and built up daily. Nitrogen is converted to urea and excreted in the urine.
- ✓ Body protein synthesis and turnover are controlled by homeostatic regulations.

In healthy individuals, the amount of protein ingested is balanced by the amount of protein used for body maintenance and excreted in faeces and urine and from the skin. Nitrogen intake is therefore equal to nitrogen loss, and the individual is in a state of zero protein balance.





**POSITIVE
BALANCE**

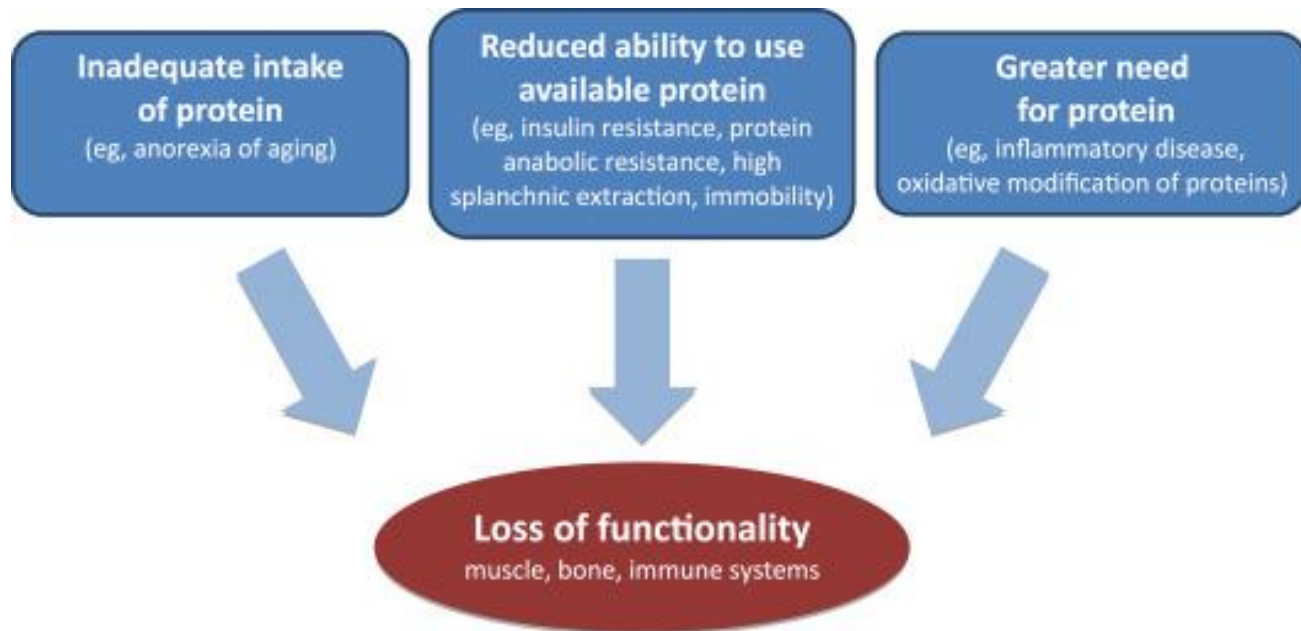
**POSITIVE
BALANCE**

**NEGATIVE
BALANCE**



❖ Factors affecting negative nitrogen balance:

- Metabolic stress
- Inadequate intake of protein
- Deficit of essential amino acids
- Greater needs



A negative nitrogen balance indicates **insufficient PROTEIN** intake (due to illness, malnutrition, or aging).

It means the patient is using protein faster than it is being synthesized.



High-protein and high-energy diets



ARE INDICATED IN
CASES OF:

- ❖ Protein-energy malnutrition
- ❖ Systemic Inflammatory Response Syndrome (SIRS)
- ❖ HIV infection
- ❖ Multiple trauma
- ❖ Burns
- ❖ Cancer



MALNUTRITION

- ✓ Malnutrition **results from a negative balance between dietary intake and body requirements.**
- ✓ Deficiency is generally caused by **insufficient nutrient intake (proteins, vitamins, energy)** but can also be caused by higher energy expenditure.
- ✓ **Protein-energy malnutrition affects every system in the body** and results in greater vulnerability to illness, **increased complications,** and greater morbidity/mortality, especially in the elderly.
- ✓ Malnutrition **affects body composition,** worsening physiological involuntary weight loss due to ageing.



PROTEIN-ENERGY MALNUTRITION

- Muscle proteins tend to be lost earlier than serum proteins.
- **Therapy** → to recover nutritional status, **energy-nutritional contributions should be increased before plasma proteins are decreased → 150-200% DRI**

Functions of plasma proteins	
Function	Example
transport	thyroxine-binding globulin (thyroid hormones) apolipoproteins (cholesterol, triglyceride) transferrin (iron)
humoral immunity	immunoglobulins
maintenance of oncotic pressure	all proteins, particularly albumin
enzymes	renin coagulation factors complement proteins
protease inhibitors	α_1 -antitrypsin (acts on proteases)
buffering	all proteins



PROTEIN-ENERGY MALNUTRITION

Requirements for nutritional recovery of MN:

0.22 g proteins/g tissue lost

5 kcal/g tissue lost

Any
electrolyte and
acid-base
imbalances
must be
corrected first.



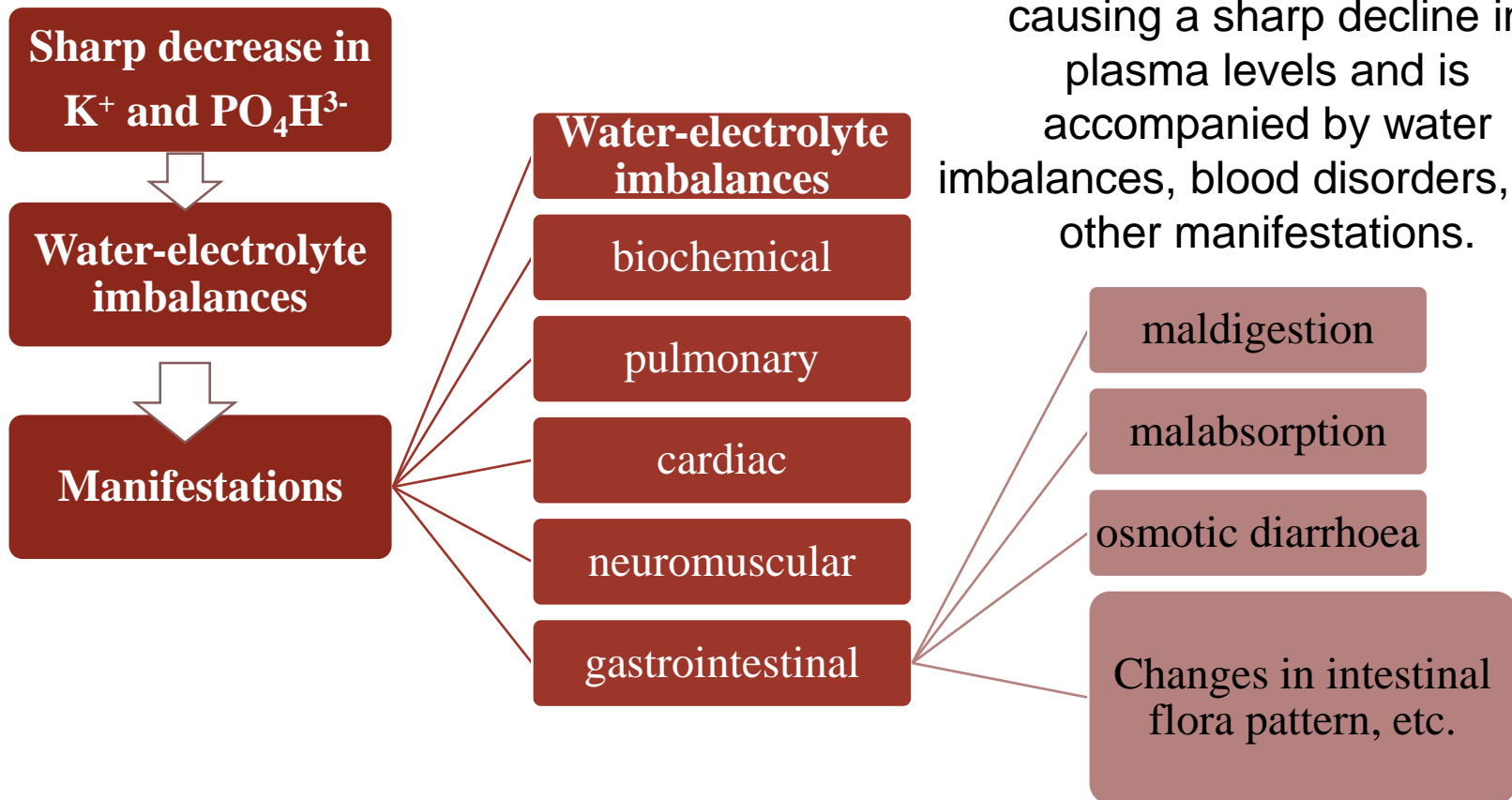
Renutrition should be slow and gradual, especially in severe and/or chronic diseases, to prevent refeeding syndrome (RS), which can be fatal.



→ Correct electrolyte and acid-base imbalances

Renutrition should be slow and progressive to avoid refeeding syndrome (RS):

RS is caused by intense anabolism K^+ and $PO_4H_3^-$ entering massively into cells, causing a sharp decline in plasma levels and is accompanied by water imbalances, blood disorders, and other manifestations.



To avoid refeeding syndrome, intake begins with basal requirements and increases progressively as tolerated.

Basal requirements: 75 kcal/kg/day
 +25 kcal/kg/day

- Roughly 200 kcal/kg/day can be consumed and tolerated.
- Successful treatment requires a gain in weight ($>10\text{g/kg/day}$) that is only obtained with a minimum intake of 150 kcal/kg/day.
- Caloric density of the diet: 1 kcal/mL
- Osmolality: ≤ 350 mOsm/L
- To prevent the use of proteins as a source of energy:
 - Non-protein kcal/protein Kcal = 12-15/1.



Intake pathways:

- Oral intake is always the first choice.
- Enteral nutrition is used if vomiting or digestive intolerance occurs.
- Parenteral nutrition is used if the digestive tract cannot be used or if intake via the above means is insufficient.

Easily digestible liquid formulas supplemented with vitamins (folate and multivitamins) and minerals (Fe, Zn, Mg, Cu, Ca and phosphates) are used. Carnitine supplementation may also be required.



Systemic Inflammatory Response Syndrome (SIRS)

The **systemic inflammatory response syndrome** describes a widespread inflammation affecting the whole body. It is the body's response to an infectious or non-infectious insult.

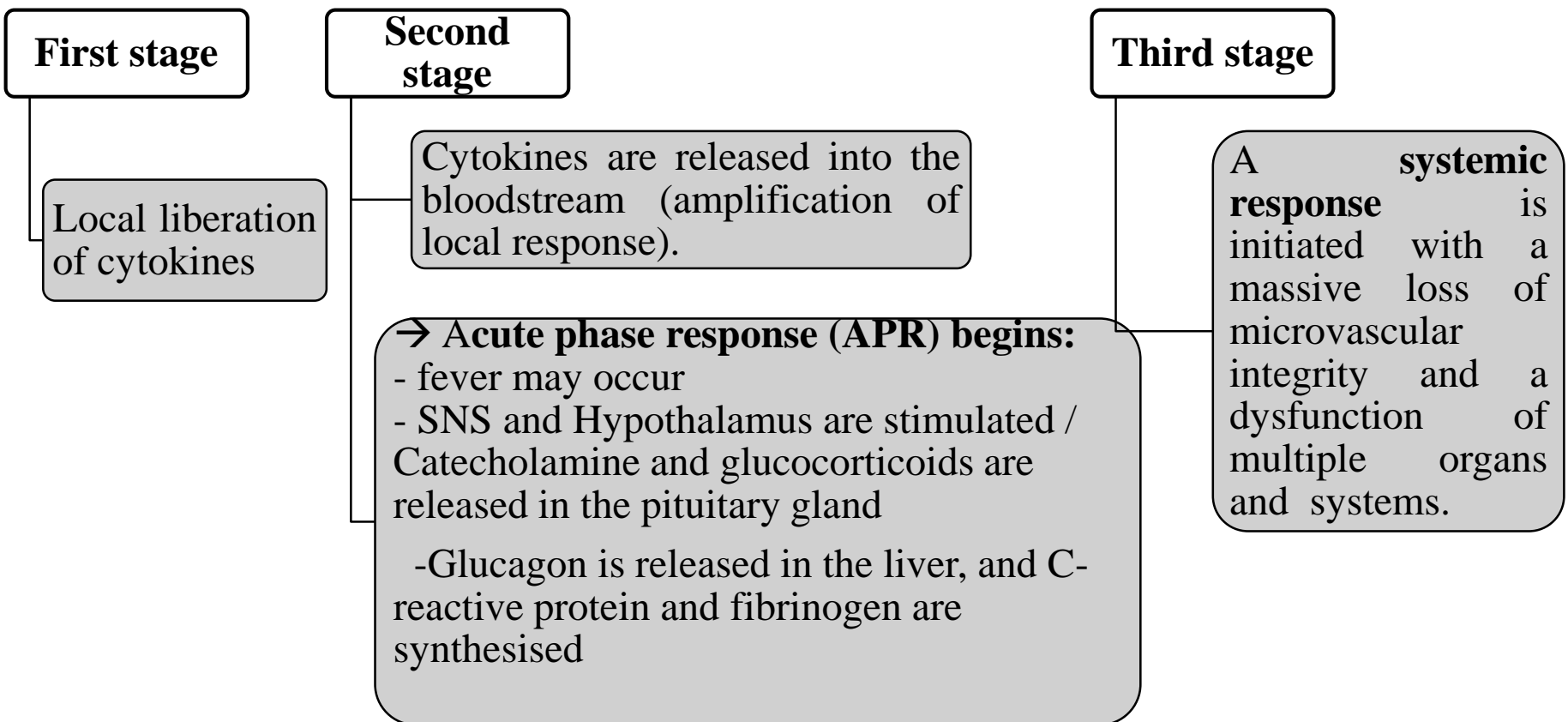
The causes of SIRS are broadly classified as infectious or non-infectious.

Causes of SIRS include:

- ✓ Bacterial infections
- ✓ Severe malaria
 - ✓ Trauma
 - ✓ Burns
- ✓ Pancreatitis
- ✓ Ischemia
- ✓ Haemorrhage



The Systemic Inflammatory Response Syndrome **develops in three main phases** and can be generated by infectious (sepsis) or non-infectious stimuli (trauma, pancreatitis, burns, etc.).



Acute Phase Response (hypermetabolic phase): hormonal and metabolic changes

Oxygen consumption increases by 80%



Metabolism increases (10-15 times)



Protein turnover is increased

- gluconeogenesis
- synthesis of 'acute phase reactants'



Nitrogen balance (-)

- protein intakes 1.5-2 g/kg/day.



Lipolysis increases



Loss of weight occurs



Total energy expenditure increases (25-45%) → each degree above 37 °C increases caloric expenditure by 13%.



Nutritional indications in SIRS

If the patient is able to ingest the recommended amounts, **oral intake (gastrointestinal feeding)** is the best choice because:

- It is cheaper, safer and more physiological.
- It maintains/improves intestinal structure and function.

If anorexia or, less commonly, ileus (intestinal obstruction) occurs, thus impeding nutritional intake, other nutritional support measures are recommended, e.g. the use of:

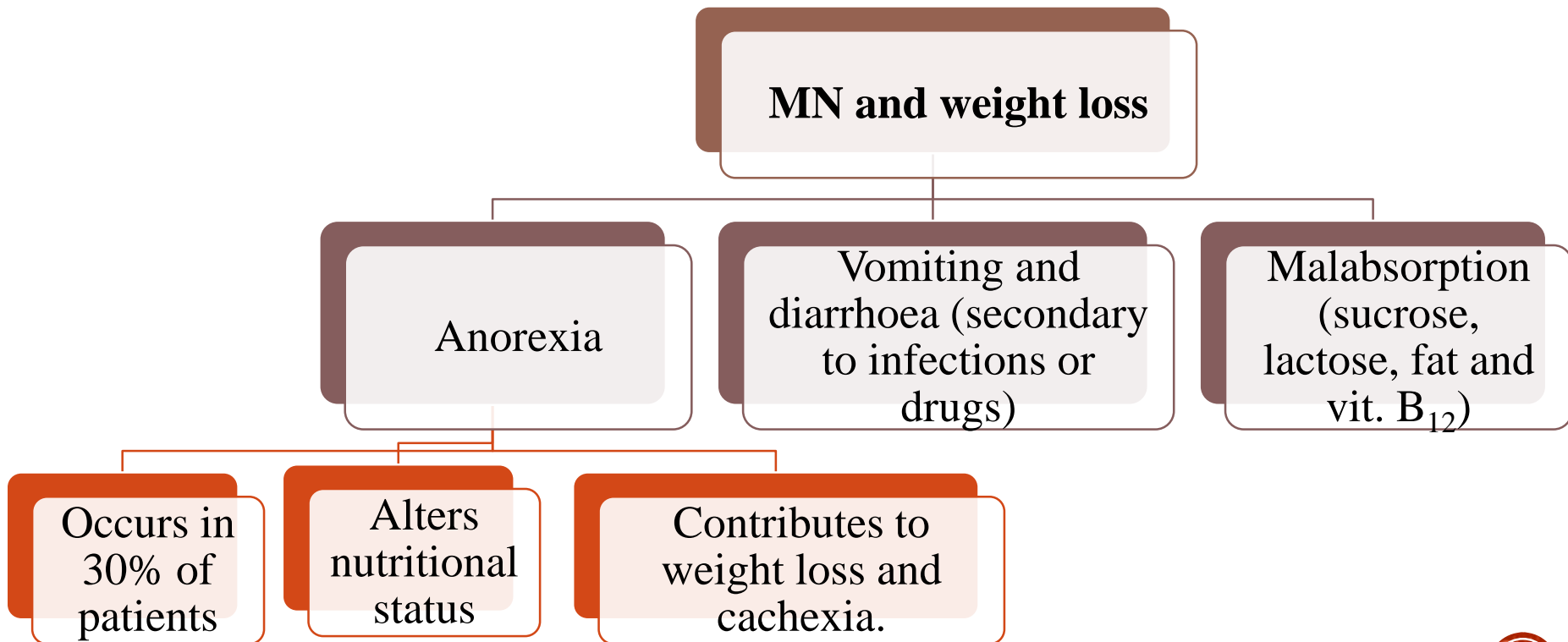
- Nasogastric tubes, ostomy
- Parenteral nutrition



AIDS infection

- Nutritional status is a major determinant of survival.
- MN is a major cause of morbidity.
- Weight loss is a (negative) prognostic indicator.

The main causes of MN, which affect the patient's physical activity and cognitive function, are:



All of these produce:

A reduction in cell mass

Deficiencies of immunostimulant micronutrients, including:

- Se, Zn, Cu
- Vitamin B₁₂
- Vitamin E (intakes <50% DRI)
- Vitamin A:
 - This deficiency is a predictor of mortality, is independent of cachexia, and occurs in 30% of patients.



Aetiology of anorexia in HIV/AIDS

Oro-oesophageal lesions:

- may be fungal (candidiasis)
- may be viral (herpes, cytomegalovirus)
- cause pain, dysphagia and dysgeusia

Fever and associated infections

Symptomatic gastrointestinal disorders

Early satiety

Specific nutritional deficiencies

Fear of pain or diarrhoea

CNS alterations

Psychological disorders

Social and family isolation

Plasmatic changes in appetite
regulatory neuropeptides

Cachexia associated TNF



Protein-energy malnutrition in AIDS

- Affects physical activity and cognitive ability.
- Reduces quality of life.
- Influences the clinical course of the disease.

The diet should try to improve nutritional status and avoid:

- Weight loss and loss of lean mass.
- Micronutrient deficiencies.
- Food-borne infections.

Supplements:

- **Calorie-protein**
- **Formulas with arginine, ω -3 Fatty acids, and vitamin E:**
 - Improve anorexia and immune function.
 - Increase energy intake.
- **Mineral-vitamin:**
 - Can improve immune function.
 - Slows progression to AIDS.
 - Mega doses can have suppressive effects and contribute to rapid disease progression.



Nutritional recommendations for AIDS

- ✓ Patients with asymptomatic HIV infection have the same requirements as healthy people and should be given complete and well-balanced normal diets.
- ✓ Intake and nutritional status must be periodically assessed.
- ✓ If anorexia, weight loss or oral lesions occur, the causes should be investigated and treatments sought.
- ✓ Renutrition should be prudent and progressive and in accordance with the evolving status of dietary and nutritional injuries.



DIETETIC ADAPTATIONS FOR AIDS

- Intake should be divided into frequent servings.
- The consistency of the diet should be adjusted.
- The caloric density of the food should be increased.
- Food the patient dislikes should be changed.
- Excessive liquids should be avoided during meals.
- Foods should be kept at a suitable temperature.
- The following should be avoided:
 - Strong flavours
 - Alcohol
 - Spicy, fatty or irritating foods
- Foods should be well presented.
- The patient should not eat alone.
- Strict hygiene should be maintained in relation to:
 - the patient's mouth
 - the patient's hands, and
 - during food preparation.



TRAUMA

- Trauma is a stress situation that generates an **inflammatory response** and **promotes** haemostasis, exclusion, degradation and lysis of damaged tissues.
- In cases of severe injury, SIRS can occur and metabolic and biochemical processes are significantly accelerated, thus **increasing the patient's nutritional needs**.

Consequences of MN

Weight loss and reduced protein reserves:

- lead to a loss of muscle strength; courage and adaptation are required.
- affect responsiveness, immunity and healing adversely.
- delay recovery.

and increase the risk of potentially serious complications.



Nutritional requirements in TRAUMA

- 35-45 kcal/kg/day.
- 1.5-2 g protein/kg/day.

**Depend on the intensity
of the injury and the
patient's prior nutritional
status**

Nutrients affecting healing capacity

- Arginine, methionine and cysteine: collagen synthesis
 - Arginine deficiency > cytotoxicity of killer lymphocytes
- Fe, Mn, Cu, Mg, Ca: cofactors collagen synthesis
- Zn: promotes epithelialization and resistance of collagen
- Se: deficiency < humoral immunity and peroxidase activity
- I: favours the bactericidal activity of polymorph nuclear
- vitamin C: collagen polymerization
- vitamin A: promotes lymphocyte proliferation
- vitamin E: increase humoral immunity
- nucleotide: synthesis of DNA, RNA and ATP
- ω -3FA: improve the cellular immune response



- ✓ Whenever possible, the oral route is first choice; otherwise, EN or PN is used.
- ✓ To prevent undesirable effects (glucose intolerance, fatty infiltration of the liver, respiratory overwork, or increased CO₂ production), moderate nutritional intake is recommended (calculated using the Harris-Benedict formula multiplied by an aggression factor ranging from 1.2 to 1.6) in both quantity and rate of administration.



BURNS

Major BURNS → severe trauma



SIRS



**Exaggerated protein catabolism, water
and electrolyte losses.**



Increased energy and nutritional needs:

- **Energy:** (2 x TEE)
- **Protein:** 1.5-3 g/kg/day
- **Micronutrients:** Double the fat-soluble vitamins and Zn than the recommended intake (the rest = DRI).



Cancer

Protein-energy malnutrition:

- Is the most common secondary diagnosis in neoplasms.
- Is an important risk factor for mortality.
- Contributes to immunosuppression.
- Promotes the appearance of infectious complications.
- Makes protein synthesis difficult.
- Prevents proper healing and tissue repair after treatment (surgery and/or radiotherapy).
- Favours the occurrence of dehiscence, hernias and fistulas.
- Alters gastrointestinal structure and function (maldigestion and malabsorption).
- Reduces muscle strength (constipation, fatigue and weakness).
- Exacerbates the patient's tendency to depression.
- Worsens the patient's quality of life.



The aims of the cancer patient's **nutritional intervention** are:

- To avoid MN and the complications it causes.
- To improve tolerability and the efficacy of the treatment.
- To improve the patient's quality of life.

Dietary treatments should be individualized and adapted to the circumstances, stage of treatment, and type of cancer.



40-80% of cancer patients suffer malnutrition because cancer causes serious nutritional and metabolic alterations that are expressed clinically as **cancer cachexia**. Relatively independent of tumour type and stage, this is characterized by:

- **Severe anorexia** (85% cases):
 - Anorectic secretion
 - Sickness
 - Feeling full
 - Abdominal pain
 - Alterations in taste and smell
- **Intense asthenia** (90% cases):
 - Loss of muscle mass
 - Cytokines release (biological response tumour)
- **Weight loss**



**Patient-dependent
factors**

Cytokine secretion by:

- macrophages (TNF, interleukins 1 and 6)
 - anorexia
 - decreased muscular and subcutaneous fat mass
 - inhibition of lipoprotein lipase
 - lymphocytes (interferon-alpha)

Varying changes in energy expenditure:

- 35-45 kcal / kg / day (hematopoietic tumours)
- 25-30 kcal / kg / day (solid tumours)
- REE is not reduced when intake is
- energy balance (-)

Increased protein requirements: ≈ 1.5 g/kg/day

micronutrient needs increased

intermediary metabolism disorders:

- increased protein catabolism
- nitrogen balance (-)
- depletion of muscle and visceral protein
- TG lipolysis and increased circulating rate
- decreased lipoprotein synthesis and lipogenesis
- subcutaneous fat depletion with hypertriglyceridemia
- insulin resistance
- increased gluconeogenesis
- Cori cycle activation



Tumoral factors

High anaerobic consumption of glucose

Secretion of lipolytic and anorexigen substances

- (serotonin, bombesin)

Head and neck surgery affects:

- chewing and swallowing

Radiotherapy affects:

- oropharyngeal mucosa
- taste buds (taste disturbance)
- salivary glands (reduced secretion and increased susceptibility to infections)

Oesophageal and/or gastric resection can produce dumping syndrome :

- fullness and abdominal distension
- abdominal cramps and diarrhoea
- sudden decrease in blood volume:
 - cold sweats and tremors
 - paleness and lipothymia

Large bowel resections can cause malabsorption.

Pelvic radiotherapy and chemotherapy cause acute enteritis.

Chemotherapy has emetogenic effects.

Treatment factors



❖ The cancer patient requires:

- Strict INDIVIDUALIZED dietary control
- Highly frequent adaptations
- Information about the importance of adequate caloric intake for maintaining nutritional status
- A climate of trust
- Treatment of anxiety-depressive syndrome

Dietary modifications can lead to:

Anorexia

Disturbance of
taste and smell

Nausea
and
vomiting

Dysphagia

Enteritis

Constipation



Anorexia

- modify meal times
- use foods/supplements with:
 - high energy density that are easy to digest
 - a liquid or pasty consistency that reduces the time and effort needed for eating

Taste and smell alteration

- in hypogeusia: strengthen the usual flavours of dishes
- in dysgeusia: substitute meat with other foods or supplements with high biological value proteins

Nausea and vomiting

- prescribe antiemetics (serotonin antagonists)
- use cold foods (less flavour and aroma)
- avoid fatty and fried foods with excessive flavouring
- eat slowly
- divide intakes (<gastric distension)
- avoid foods and drink liquids during meals
- avoid noisy environments, arguments and pungent or unpleasant food odours

Dysphagia

- edema, ulceration, stomatitis and dental pain can be complicated by local infections
- decreases saliva secretion
- increases salivary viscosity
- causes spasms or muscle fibrosis
- To facilitate swallowing and reduce pain, it is advisable to:
 - maintain good hydration
 - remove irritants or hard food
 - divide intakes
 - use pasty foods or supplements with soft textures
 - eat food at room temperature
 - In the case of neurological dysphagia, commercial thickeners and jellies may be useful

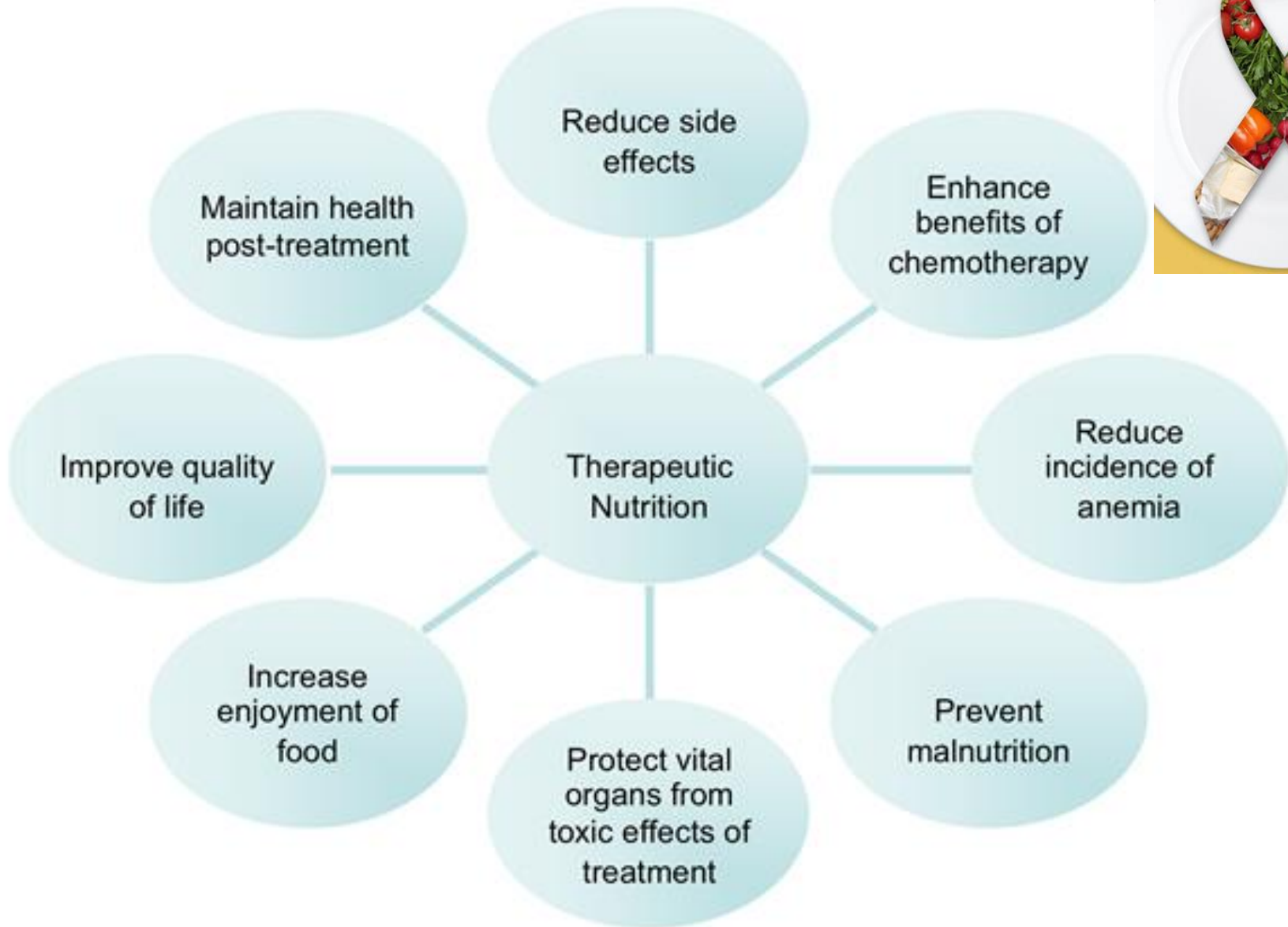


Enteritis

- causes oedema and ulcerations
- causes malabsorption (diarrhoea/abdominal pain)
- Lost fluids and ions should be replaced (with vegetable broths, soups and soothing, sugary degreased teas)
- **Avoid the following:**
 - Stimulating intestinal motility (e.g. with insoluble fibre, spices, tea, coffee, extreme food temperatures, voluminous intakes)
- foods that are difficult to digest (e.g. fats, fried foods);
- milk (lactase deficiency)

Constipation secondary to:

- Low caloric intake, fluids and fibre
- Loss of muscle mass (cachexia)
- Decreased physical activity
- Analgesics, narcotics and some types of chemotherapy
- Increase the volume and hydration of stools and facilitate removal using food or supplements that are rich in fibre and drinking plenty of fluids



Thank You
For Your Attention...



**2.6 A. Electrolyte minerals (K, Ca and P)
in controlled diets.
Characteristics and implementation.
Dietary recommendations.**

1

Unit 2.6 A

INDEX 2.6

- **Electrolyte balance**
- **Reference values in blood and dietary sources**
- **Dietary recommendations**
- **Potassium: hyperkalaemia and hypokalaemia**
- **Calcium: hypercalcemia and hypocalcaemia**
- **Phosphorus: hyperphosphatemia and hypophosphatemia**
- **Iron metabolism. anaemia**
- **Copper metabolism. Wilson's disease.**

Competences acquired in unit 2.6

- ✓ Understand the basis of electrolyte balance and homeostasis.
- ✓ Know the main reference values of electrolytes and dietary sources.
- ✓ Be able to initially assess deficit or excess in:

Potassium

Calcium

Phosphorus

Iron

Copper

- ✓ Be able to correct deficit or excess and make dietary recommendations.
- ✓ Identify treat complications due to electrolyte imbalance.

MINERALS

- ✓ Minerals represent approximately 4% to 5% of body weight, or 2.8 to 3.5 kg in adult women and adult men, respectively.
- ✓ 50% of this weight is **calcium**, and another 25% is **phosphorus**.
- ✓ The **five other essential macro minerals** (>100 mg/day required) (magnesium, sodium, potassium, chloride and sulphur) and the 11 established **micro-minerals** (<100 mg/day required) (iron, zinc, iodide, selenium, manganese, fluoride, molybdenum, copper, chromium, cobalt and boron) account for the remaining 25%.
- ✓ **Sodium, potassium and calcium** form positive ions (cations), whereas other minerals form negative ions (anions).

... REMEMBER ...

Table: 10 Classification of essential minerals

Macro minerals (> 100 mg/day)*	Micro minerals (<100 mg/day)*
Calcium	Iron
Phosphorus	Zinc
Magnesium	Copper
Sulfur	Iodine
Sodium*	Fluoride
Potassium*	Manganese
Chloride*	Selenium
	Chromium
	Molybdenum



ELECTROLYTE BALANCE

- ✓ **Electrolytes** play a vital role in maintaining **homeostasis** in the body.
- ✓ They help to regulate heart and neurological function, **fluid balance**, **oxygen delivery**, **acid-base balance**, and much more.
- ✓ Electrolyte imbalances can develop by **consuming** too little or too much electrolyte or by **excreting** too little or too much electrolyte.

- ✓ **Electrolyte disturbances are involved in many disease processes.** The causes, severity, treatment, and outcomes of these disturbances can differ greatly depending on the electrolyte.
- ✓ The most serious electrolyte disturbances involve abnormalities in the levels of sodium, potassium and calcium.
- ✓ The kidney is the most important organ for maintaining appropriate fluid and electrolyte balance but other factors, such as hormonal changes and physiological stress, also play a role.

THE ROLE OF ELECTROLYTES IN THE BODY

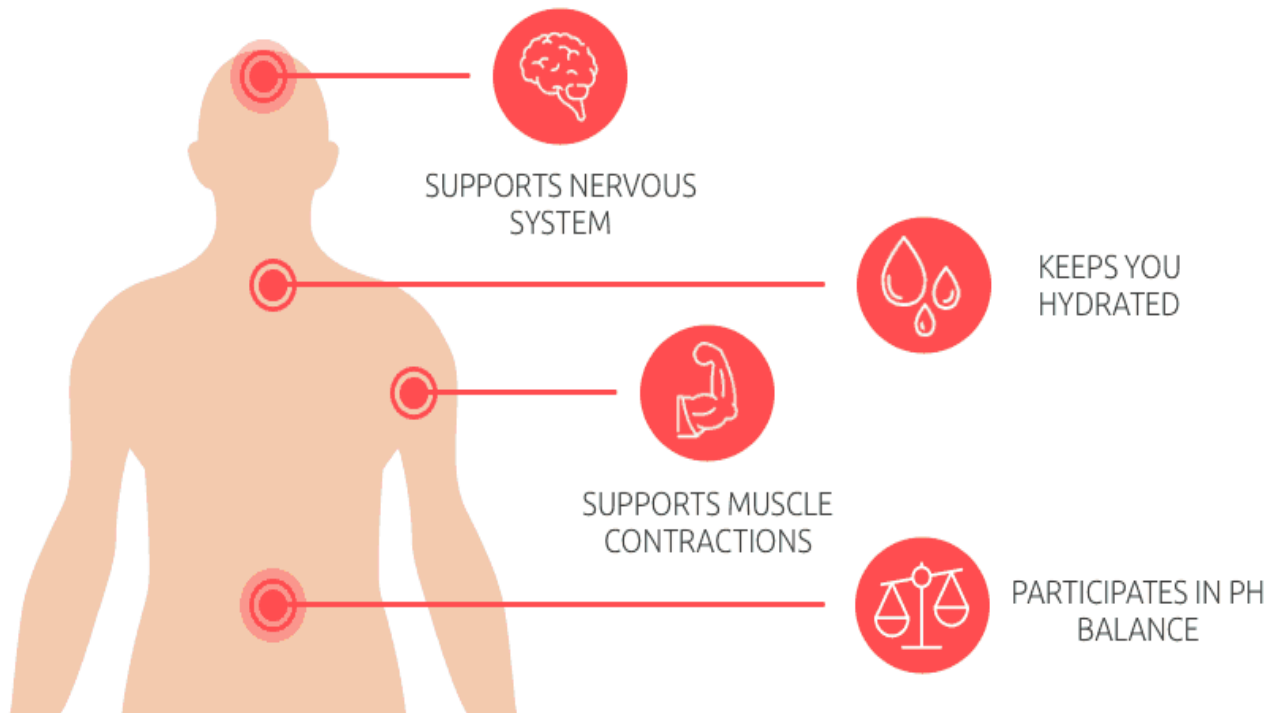


TABLE 6-1

Concentrations of Extracellular and Intracellular Electrolytes in Adults

Electrolyte	Extracellular Concentration*	Intracellular Concentration*
Sodium	135–145 mEq/L	10–14 mEq/L
Potassium	3.5–5.0 mEq/L	140–150 mEq/L
Chloride	98–106 mEq/L	3–4 mEq/L
Bicarbonate	24–31 mEq/L	7–10 mEq/L
Calcium	8.5–10.5 mg/dL	<1 mEq/L
Phosphate/ phosphorus	2.5–4.5 mg/dL	4 mEq/kg [†]
Magnesium	1.8–3.0 mg/dL	40 mEq/kg [†]

*Values may vary among laboratories, depending on the method of analysis used.

†Values vary among various tissues and with nutritional status.

Electrolyte minerals (dietary sources)

Sodium: pickled foods, cheese and table salt.

Chloride: table salt.

Potassium: fruits and vegetables such as bananas, avocado and sweet potato.

Magnesium: seeds and nuts.

Calcium: dairy products, fortified dairy alternatives, and green leafy vegetables.

Phosphorus: milk, milk products, meat, beans, lentils, nuts, vegetables and fruits.

Iron: meat products, pâté, black pudding

Copper: oysters, lobster, nuts, seeds, liver, leafy greens and dark chocolate.

POTASSIUM



- ✓ **Potassium** is an **important** mineral that functions as an **electrolyte**. It helps regulate fluid balance, nerve signals and muscle contractions.
- ✓ Most of the body's potassium is located inside the cells. Potassium is necessary for the normal functioning of cells, nerves, and muscles.
- ✓ The body must keep the potassium level in the blood within a narrow range. A blood potassium level that is too high (**hyperkalaemia**) or too low (**hypokalaemia**) can have serious consequences, such as abnormal heart rhythm or even cardiac arrest (when the heart stops).
- ✓ The body can use the large reservoir of potassium stored in cells to help maintain a constant level of potassium in the blood.

Overview:

Intracellular cation (140 mEq/l) vs extracellular cation (3.5-5.5 mEq/L)

Intracellular content is proportional to lean mass.

Essential nutrient: plentiful vegetables and protein foods (e.g. meat, fish and eggs).

Absorption: Absorbed by diffusion (especially in the colon). The same amount is eliminated, mostly in urine.

Daily requirements

Healthy individual

3.5-4.7 g/day (3500- 4700 mg/day)

- Hyperkalaemia:** $[K^+]_{PL} > 5.5$ mEq/ml
- Hypokalaemia:** $[K^+]_{PL} < 3.5$ mEq/ml

Potassium (hyperkalaemia)



Hyperkalaemia is an elevated level of potassium (K^+) in the blood. Normal K levels are between 3.5 and 5.0 mmol/L (3.5 and 5.0 mEq/L), with levels above 5.5 mmol/L defined as hyperkalaemia (>5.5 mEq/L).

❖ Aetiology:

Use of K-sparing diuretics.

Drugs that contain K (KCl as a substitute for salt).

Acidosis (H^+ exchange by intracellular K^+).

Excessive intake (fruits and vegetables).

Renal failure requiring dialysis.

❖ Symptoms:

Muscle weakness, or abdominal cramps

Paresthesia

Irritability

Diarrhoea

Mental confusion

Hypotension

Arrhythmia

Potassium (hypokalaemia)



Low level of K in the blood serum (<3.5 mEq/L).

→ **Hypokalaemia is one of the most common water-electrolyte imbalances.**

Aetiology:

Vomiting or profuse diarrhoea.

Use of diuretics.

Corticosteroids or laxatives.

Cushing's disease: a disorder caused by the body's exposure to an excess of the hormone cortisol.

Symptoms: leg cramps, tiredness, hypotonicity, weakness, arrhythmias, constipation, cardiac arrest.

Recommended diets:

Use foods rich in K.

Use salt substitutes

Use cooking water.

POTASSIUM Food Sources

→Potassium is widely available in many foods, especially **fruits and vegetables**. Leafy greens, beans, nuts, dairy foods, and starchy vegetables such as winter squash are rich sources.

- Dried fruits (raisins, apricots)
- Beans, lentils
- Potatoes
- Spinach, broccoli
- Beet greens
- Avocado
- Bananas
- Oranges, orange juice
- Tomatoes
- Yogurt
- Cashews, almonds
- Chicken
- Salmon



How can we prevent or treat chronic hyperkalaemia?

→ By managing the diet of patients **with chronic kidney disease**. **Hyperkalaemia** is the medical term that describes potassium levels in a patient's blood that are higher than normal.

- ✓ These patients should be kept off foods that are rich in potassium.
- ✓ The normal amount of potassium in a typical healthy diet is **3,500 to 4,800 mg per day**. In a **potassium-restricted diet** it is usually **2,000-2,500 mg per day**.
- ✓ Among foods that are rich in potassium and therefore likely targets for restriction are many fruits and vegetables, including such mainstays as bananas, avocados, and oranges.

LOW-POTASSIUM DIET

CHARACTERISTICS OF THE DIET:

→ Know the patient's dietary habits well.

A) Closed diet

B) List the ingredients with their densities in K.

List foods that can be consumed freely.

Describe forms of food preparation.

Recommend portions of meat and fish.

List daily quantity of milk and dairy products.

List daily allowed quantity of fruits and fresh legumes.

List allowed condiments.

POTENTIAL PROBLEMS:

1. Inaccuracies in the loss of K^+ due to culinary treatment.
2. Difficulty in increasing caloric intake: \uparrow fat, \downarrow CH \rightarrow \downarrow pleasant.
3. Monotonous diets.
4. Supplement products with low K^+ content.

DIETS RICH IN POTASSIUM

- 3,900–6,825 mg/day: to prevent hypokalaemia and depletion of K^+ from the organism.

CHARACTERISTICS OF THE DIET. USE:

- Food rich in K.
- A varied diet.
- Raw food rather than fresh food: cooked foods have $\uparrow Na^+$ and $\downarrow K^+$.
- The cooking water.

CALCIUM



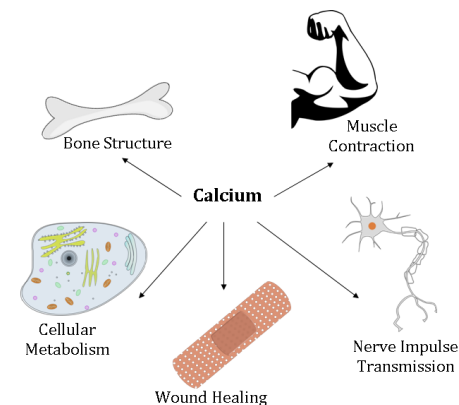
- ✓ **Calcium (Ca)** is the most abundant mineral element in our body.
- ✓ It accounts for roughly 1.5-2% of body weight.
- ✓ 99% of the calcium is in the bones and teeth. The remaining 1% is in the blood and extracellular fluids, and in the cells of all tissues, where it regulates many important metabolic functions.

The functions of calcium are:

- a) skeletal
- b) regulatory

CALCIUM:

- Promotes optimal gains in bone mass and bone density during the adolescent years.
- Helps postmenopausal women maintain bone health and suppress PTH.
- Helps bone structure to develop and grow during pregnancy, lactation, infancy, childhood and adolescence.
- Assists in the transport functions of cell membranes.
- Helps to transmit ions across the membranes of cell organelles.
- Is involved in nerve transmission and the regulation of heart muscle function.
- Is a cofactor for several enzymatic reactions.



CALCIUM AND PHOSPHORUS

Overview:

Calcium and phosphorus are essential elements, representing 99% and 85%, respectively, of bone tissue.

Other functions:

Ca: enzymatic activation, blood coagulation, muscle contraction, secretion and cell division, phagocytosis.

P: energy transport, component nucleic acids, protein structure stabilizer.

Requirements:

Variable needs: **Ca:** 400–1500 mg/day **P:** 300–1200 mg/day.

Hormone regulation:

PTH, calcitonin and vitamin D.

Regulation of serum calcium

- Ca in bones is in equilibrium with Ca in the blood.
- PTH plays the major role in maintaining serum Ca.
- When the concentration of blood Ca falls below this level, PTH stimulates the transfer of exchangeable Ca from the bone to the blood.
- At the same time, PTH promotes renal tubular resorption of Ca and indirectly leads to increased intestinal absorption of Ca by increasing the kidney's production of vitamin D.

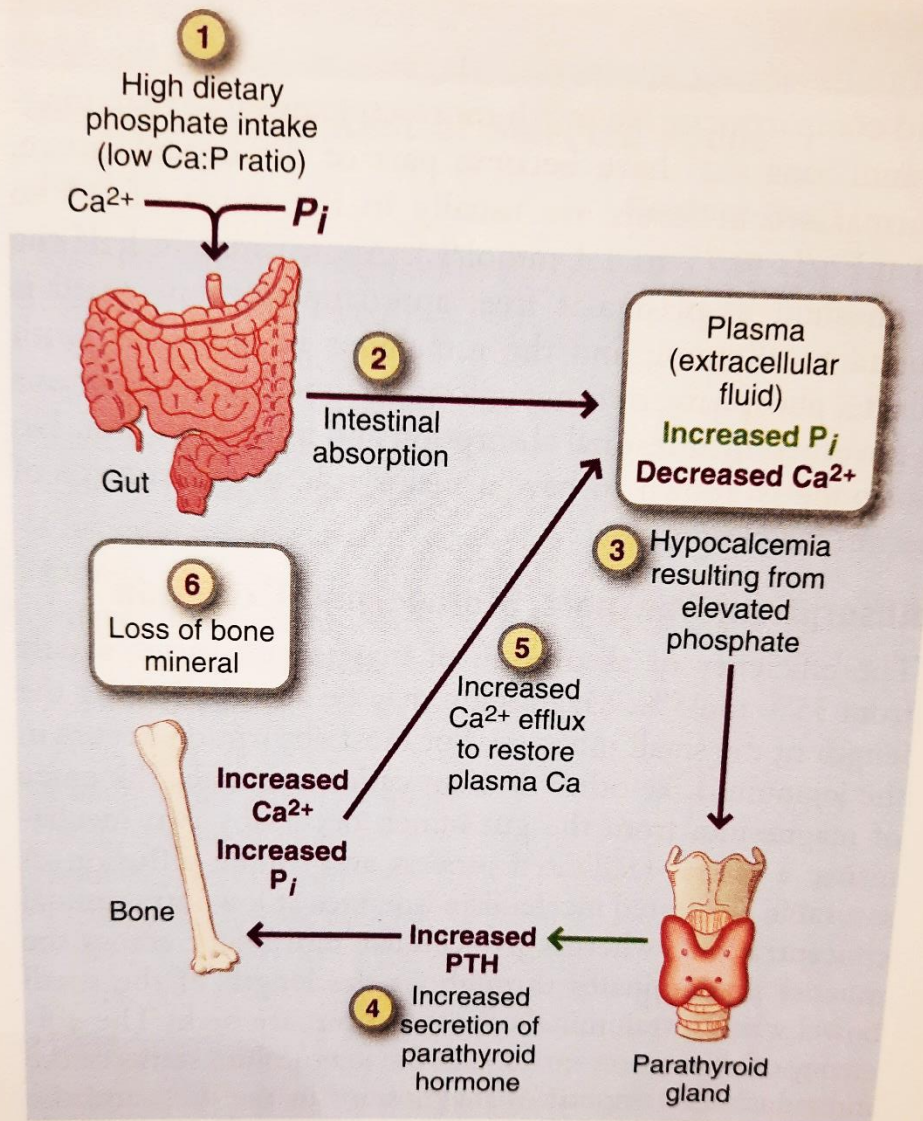
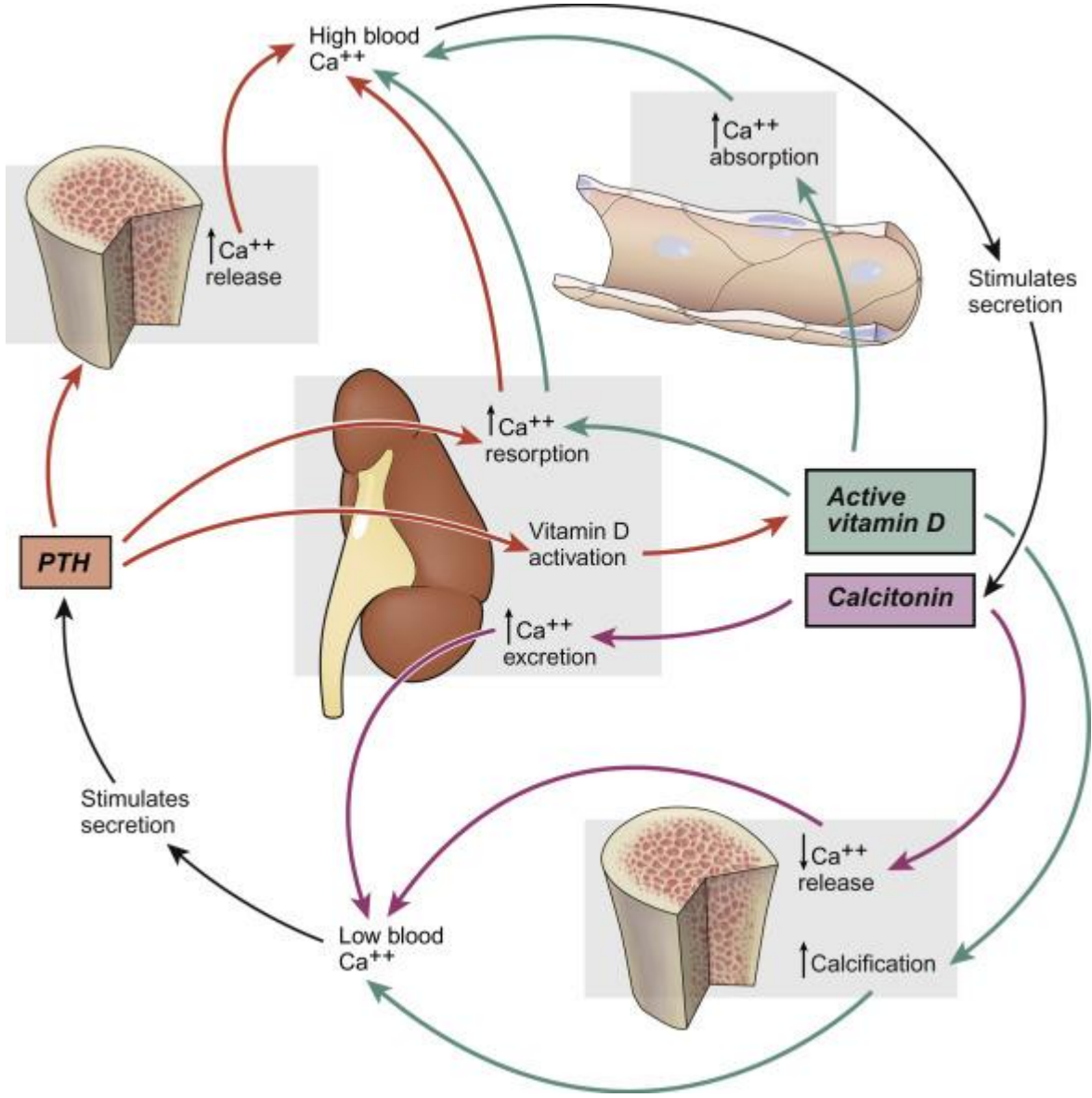


FIGURE 3-29 Mechanism through which a low dietary calcium/phosphorus ratio contributes to the development of a persistently high parathyroid hormone concentration.

CALCIUM BALANCE



Hormonal regulation:

- PTH, calcitonin and vitamin D
small intestine, skeletal, kidney
- \downarrow [Ca] pl. \rightarrow \uparrow PTH \rightarrow \uparrow bone resorption \rightarrow renal Ca conservation and vitamin D \rightarrow \uparrow intestinal Ca absorption
- \uparrow [Ca] pl. \rightarrow \uparrow calcitonin \rightarrow \downarrow bone resorption

Nutritional objectives:

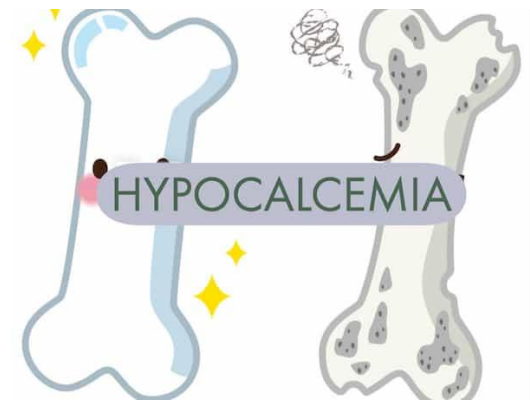
- It prevents osteoporosis and osteopenia.
- Needs are maximum during the foetal period, childhood, the prepubertal period and the menopause.
- Needs vary (Ca 400–1500 mg/day).

Metabolism:

- It is absorbed from 5 to 40% by the proximal intestine.

What are the symptoms of hypocalcemia?

- Confusion or **memory loss**.
- **Muscle spasms**.
- **Numbness** and **tingling** in the hands, feet, and face.
- Depression.
- Hallucinations.
- **Muscle cramps**.
- Weak and brittle nails.
- Easy fracturing of the bones.



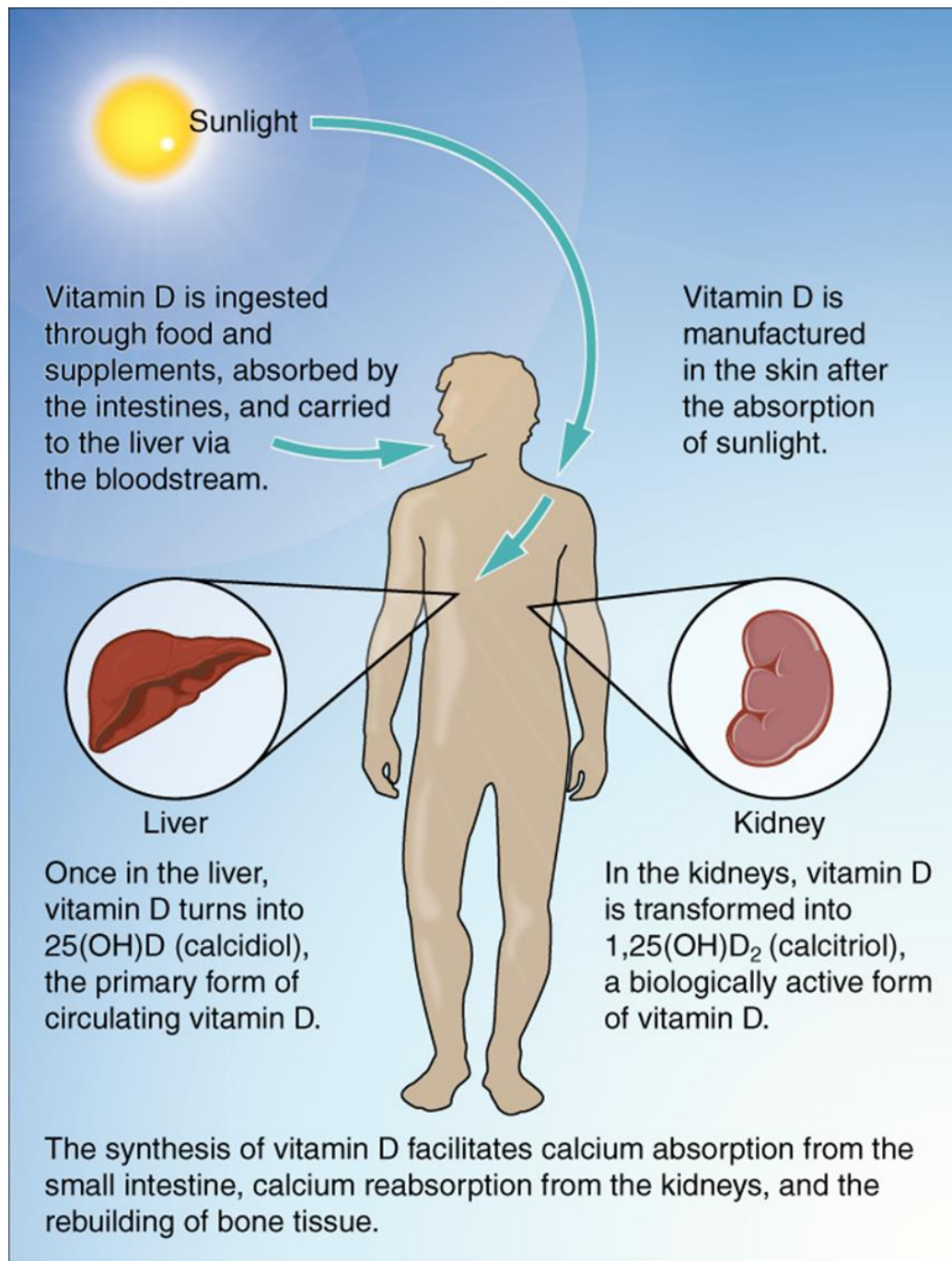
HYPOCALCAEMIA → HIGH CALCIUM DIETS

Aetiology:

- Increased **needs** (growth, pregnancy, lactation, menopause, ageing).
- Intestinal malabsorption (low birth weight or preterm).
- Hyperphosphatemia (reduced ionic Ca concentrations).
- Strict vegetarian diets.
- Corticosteroids, antacids, hypochloridria (gastrectomy, etc.).
- Osteoporosis: Ca²⁺ oestrogen and/or calcitonin.
- Hypertension: Ca²⁺ decreased blood pressure.
- Colon cancer: diets rich in Ca seem to offer protection.
- Osteomalacia (Vit. D, Ca and P).

Recommendations:

- Use a diet rich in dairy products and/or oral supplements of Ca (with meals). 1/1 Ca/P ratio (normally 1/2).
- Provide good exposure to sunlight
- Avoid the joint ingestion of Ca with fat, phytates or oxalates (which reduce Ca absorption).



➤ Sources of calcium

- ✓ Milk and other dairy products.
- ✓ Green leafy vegetables such as curly kale, okra, spinach, broccoli, and collards.
- ✓ Clams, oysters, kale, turnip greens, mustard greens, and tofu.
- ✓ Bread and anything made with fortified flour.
- ✓ Fish where you eat the bones, such as sardines and pilchards.
- ✓ Soybean.
- ✓ Fortified foods.



→ Oxalic limits the availability of Ca in rhubarb, spinach, chard and beet greens.

Food	Serving Size	Calories per Portion	Calcium Content (mg)
Dairy foods			
Milk			
Whole milk	8 oz	149	276
Reduced fat milk (2%)	8 oz	122	293
Low-fat milk (1%)	8 oz	102	305
Skim milk (nonfat)	8 oz	83	299
Reduced-fat chocolate milk (2%)	8 oz	190	275
Low-fat chocolate milk (1%)	8 oz	158	290
Yogurt			
Plain yogurt, low-fat	8 oz	143	415
Fruit yogurt, low-fat	8 oz	232	345
Plain yogurt, nonfat	8 oz	127	452
Cheese			
Romano cheese	1.5 oz	165	452
Swiss cheese	1.5 oz	162	336
Pasteurized processed American cheese	2 oz	187	323
Mozzarella cheese, part skim	1.5 oz	128	311
Cheddar cheese	1.5 oz	171	307
Muenster cheese	1.5 oz	156	305
Nondairy foods			
Salmon	3 oz	76	32
Sardines, canned	3 oz	177	325
White beans, cooked	1 cup	307	191
Broccoli, cooked	1 cup	44	72
Broccoli, raw	1 cup	25	42
Collards, cooked	1 cup	49	226
Spinach, cooked	1 cup	41	249
Spinach, raw	1 cup	7	30
Baked beans, canned	1 cup	680	120
Tomatoes, canned	1 cup	71	84
Calcium-fortified food			
Orange juice	8 oz	117	500
Breakfast cereals	1 cup	100–210	250–1000
Tofu, made with calcium	0.5 cup	94	434
Soy milk, calcium fortified ^a	8 oz	104	299

Data source: Dietary Guidelines for Americans, 2010. Available at: www.ndb.usda.gov.

^a Not all soy beverages are fortified to this level.

DRI OF CALCIUM

1300 mg for children aged 9 to 18.

1000 mg for adults aged 19 to 50.

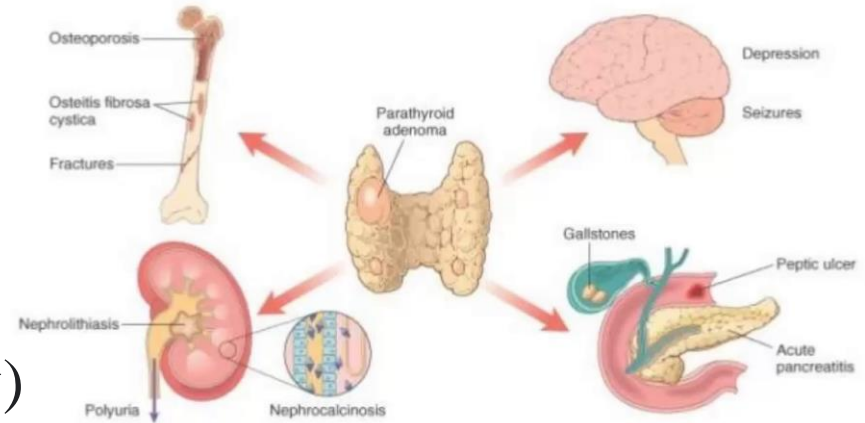
1200 mg for women aged over 51 and for all adults over 70.

→ HIGH dietary calcium intakes are associated with a lower prevalence of overweight and obesity (Heaney and Rafferty, 2009).

What are the symptoms of hypercalcemia?

- Loss of appetite.
- Nausea and vomiting.
- Constipation and abdominal (belly) pain.
- The need to drink more fluids and urinate more.
- Tiredness, weakness, or muscle pain.
- Confusion, disorientation, and difficulty thinking.
- Headaches.
- Depression.

Hypercalcemia



Hypercalcemia (low-calcium diets)

In **renal lithiasis**, dietary treatment is effective if the following **recommendations** are followed:

- ✓ Increase the intake of liquids.
- ✓ Limit the intake of proteins (favour hypercalciuria) to try to acidify the urine ($>$ solubility Ca).
- ✓ Decrease the intake of salt (calcium-dependent Na).
- ✓ Limit the consumption of foods rich in oxalates, e.g. cocoa, coffee, tea, nuts, peanuts, strawberries, cabbage, spinach, thistles, parsley, carrots, etc.).

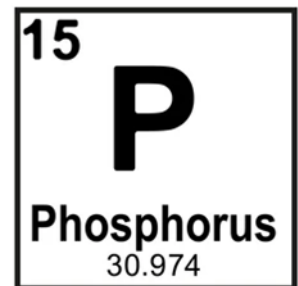
- ✓ In **renal lithiasis** (75% hydroxyapatite and/or calcium oxalates stones) secondary to hypercalciuria (> 300 mg Ca/ day) due to increase (genetic) in Ca absorption (men > 30 years old).
- ✓ In **fat malabsorption** there may be hyperoxaluria without an increase in Ca absorption, so intake of oxalate should be carefully restricted (FA + Ca form soaps and free oxalate is easily absorbed in the colon and eliminated in the urine).
- ✓ Ca restriction is not effective in hypercalcemia of **bone** or **paraneoplastic** origin.

PHOSPHORUS P

- ✓ Phosphorus, an **essential mineral**, is naturally present in many foods and available as a dietary supplement. Phosphorus is a component of bones, teeth, DNA, and RNA.
- ✓ Approximately 700 g of phosphorus exists in adult tissues. In humans, phosphorus makes up about **1 to 1.4% of fat-free mass**. Of this amount, **85% is in the bones and teeth**, and the other 15% is distributed throughout the blood and soft tissues.
- ✓ **Phosphorus and calcium** are interrelated because hormones, such as **vitamin D** and **parathyroid hormone (PTH)**, regulate the metabolism of both minerals.
- ✓ In addition, phosphorus and calcium make up **hydroxyapatite, the main structural component in bones and tooth enamel**.
- ✓ The combination of **high phosphorus intakes with low calcium intakes increases serum PTH levels**, but evidence is mixed on whether the increased hormone levels decrease bone mineral density.

FUNCTIONS OF PHOSPHORUS

- Phosphorus participates in numerous essential functions of the body.
 - DNA and RNA are based on phosphate; Phosphorus is a component of adenine and guanine.
- ATP, the major cellular form of energy, contains high-energy phosphate bonds.
- Phosphorus is a component of phospholipids in membrane cells.
- Phosphorus is active in the enzyme system.
- Phosphate buffer system
- Phosphorus combines with Ca to form hydroxyapatite for healthy teeth and bones.



DRI of Phosphorus

→ The DRI of phosphorus is somewhat lower than that of calcium for all groups.

700 mg/day adults

→ Phosphate **deficiency is rare.**

→ Hypophosphatemia may be common among older adults. It could also develop in individuals who take phosphate binders for renal disease or in older adults due to poor intake in general.

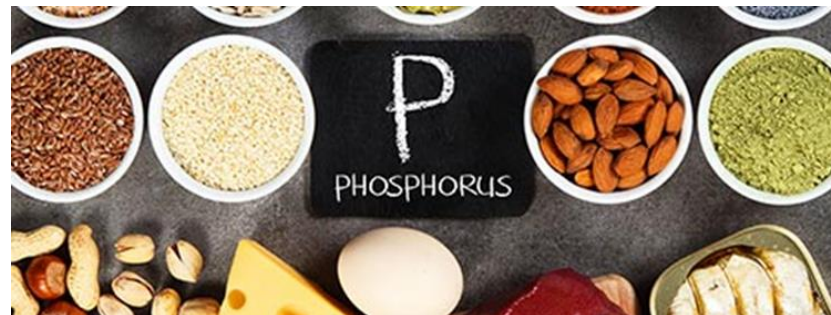
PHOSPHORUS

Metabolism:

- Intestinal absorption: 50-90%.
- Renal elimination: 80%.
- Renal regulation: \downarrow 50% excretion = intake x 3.
- PTH \rightarrow \uparrow renal excretion of P \rightarrow \downarrow [P] pl.

Nutritional objectives:

- Hypophosphatemia: alleviate pluriorganic symptoms.
- Hyperphosphatemia: improve the toxic-metabolic symptoms.



HYPOPHOSPHATEMIA

Aetiology:

Hypophosphatemia (increased synthesis of vitamin D that mobilizes phosphorus from the bone) secondary to:

- ✓ Malabsorption.
- ✓ Chronic intake of aluminium-based antacids.
- ✓ Reduced dietary intake: this is difficult to increase, so it is better to reduce renal excretion (hypercalcemia and hypermagnesemia)
→ massive entrance of P into the cells
- ✓ Excessive urinary losses: alcoholism, hyperparathyroidism: PTH reduces tubular reabsorption of P
- ✓ Treatments with calcitonin: increase renal elimination of P
- ✓ Polyuria in diabetics or large burns, etc.

Recommendations:

•It is recommended to correct the coexistent hypocalcaemia (using different delivery pathways since $\text{Ca} + \text{P}$ precipitates).

Hyperphosphatemia (low-phosphate diets)

Aetiology:

Hyperphosphataemia secondary to:

- Use of P (laxatives, enemas, supplements, etc.).
- Important cellular lysis (rhabdomyolysis, tumour lysis).
- Tubular reabsorption of P (severe dehydration, hypoparathyroidism, hyperthyroidism, acromegaly, etc.).

Symptoms:

Symptoms are due to the accompanying hypocalcaemia and soft tissue calcification.

Recommendations:

If, despite protein reduction, high levels of P remain, P chelators (Ca, Mg or Al salts) are recommended and always correct coexistent hypocalcaemia.

DIETARY SOURCES OF PHOSPHORUS

- In general, good sources of protein are good sources of phosphorus.
- Examples are meat, poultry, fish, eggs, milk, milk products, nuts, legumes, cereals and grains.



What are high phosphorus foods?

- Dairy products such as milk, cheese, custard, cottage cheese, yogurt, ice cream, pudding



- Nuts, seeds, peanut butter



- Dried beans and peas such as baked beans, black beans, chick peas, garbanzo beans, kidney beans, lentils, limas, northern beans, pork and beans, split peas and soybeans



- Bran cereals, whole grain products



- Beverages such as cocoa, ale, beer, chocolate drinks, and dark cola drinks



What are low phosphorus foods?

- Fresh fruits such as apples, apricots, blackberries, grapes, tangerines, pears, peaches, pineapple, plums and strawberries



- Fresh vegetables such as cauliflower, carrots, cucumber, celery, green beans and broccoli



- Popcorn, crackers



- Rice cereal



- Sherbert



- Coffee or tea without milk, light-colored sodas (such as ginger ale), fruit juices



→ For the next lesson we will meet face-to-face in the Faculty of Pharmacy, where we will finish unit 2.6B (iron and copper). See you next week!



Thank You
For Your Attention...

**UNIT 2.6 B. IRON AND COPPER.
METABOLISM. OBJECTIVES AND
INDICATIONS. DEFICIENCY AND
OVERLOAD. FOOD SOURCES. DIETETIC
RECOMMENDATIONS.**



UNIT 2.6 B
Prof. Pilar Vila-Donat

UNIT 2.6

Electrolyte balance

Reference values in blood and dietary sources

Dietary recommendations

Hyperkalaemia and hypokalaemia

Hypercalcemia and hypocalcemia

Hyperphosphatemia and hypophosphatemia


Iron metabolism. Anaemia.

Copper metabolism. Wilson's disease.




Remember ...
MICROMINERALS
<100mg/day required

Table: 10 Classification of essential minerals



Macro minerals (> 100 mg/day)*	Micro minerals (<100 mg/day)*
Calcium	Iron
Phosphorus	Zinc
Magnesium	Copper
Sulfur	Iodine
Sodium*	Fluoride
Potassium*	Manganese
Chloride*	Selenium
	Chromium
	Molybdenum



IRON

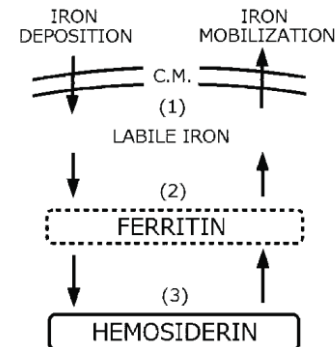


- ✓ Iron is recognized as an **essential** nutrient that promotes blood production.
- ✓ Iron-deficiency is the most common disease caused by nutritional deficiency → **Anaemia**
- ✓ The adult human body contains iron in two major pools:
 - **Functional iron** → haemoglobin, myoglobin, enzymes.
 - **Storage iron** → ferritin, hemosiderin, transferrin.
- Roughly 70% of our **body's iron** is found in the red blood cells → **haemoglobin** and in muscle cells → **myoglobin**.
- Haemoglobin is essential for transferring the oxygen in our blood from the lungs to the tissues.



IRON

- ✓ Healthy adult men → 3.6 g
- ✓ Healthy adult women → 2.4 g → Adult women store much lower amounts of iron than men do.
 - **Heme IRON** (hemoglobin, myoglobin, enzymes)
 - **Ionic form or nonheme IRON** (predominant in plant foods but also in animal foods as *nonheme* enzymes and ferritin).
- ✓ 70% is considered functional iron, while the rest is reversibly stored as *ferritin* (70-80%) or irreversibly stored as *hemosiderin*.

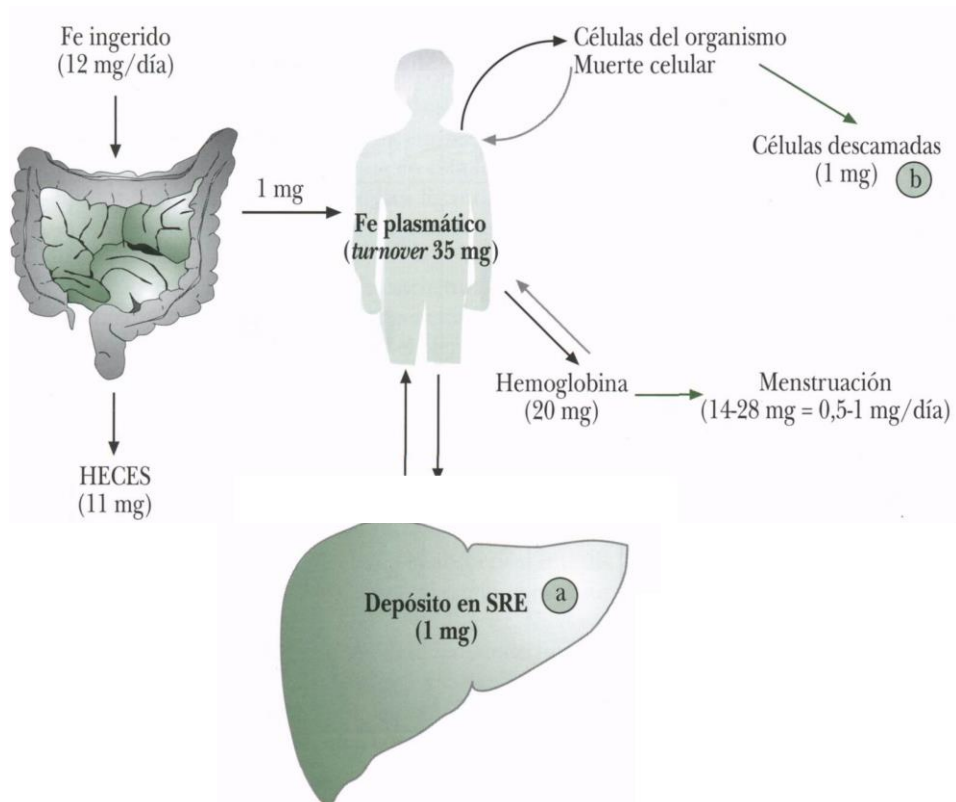


IRON METABOLISM

Absorption: duodenum and proximal jejunum (0-5 mg/day).

Absorption depends on:

- The size of body deposits.
- The rate of blood production.
- The dietary amount and the type of dietary Fe (heme or ionic).



- a** Depósito de hígado y bazo
- b** Corresponde al hierro presente en células de descamación de la piel y del tracto urinario fundamentalmente
- Reutilización del hierro
- Pérdidas obligadas



ABSORPTION, TRANSPORT, STORAGE AND EXCRETION OF IRON

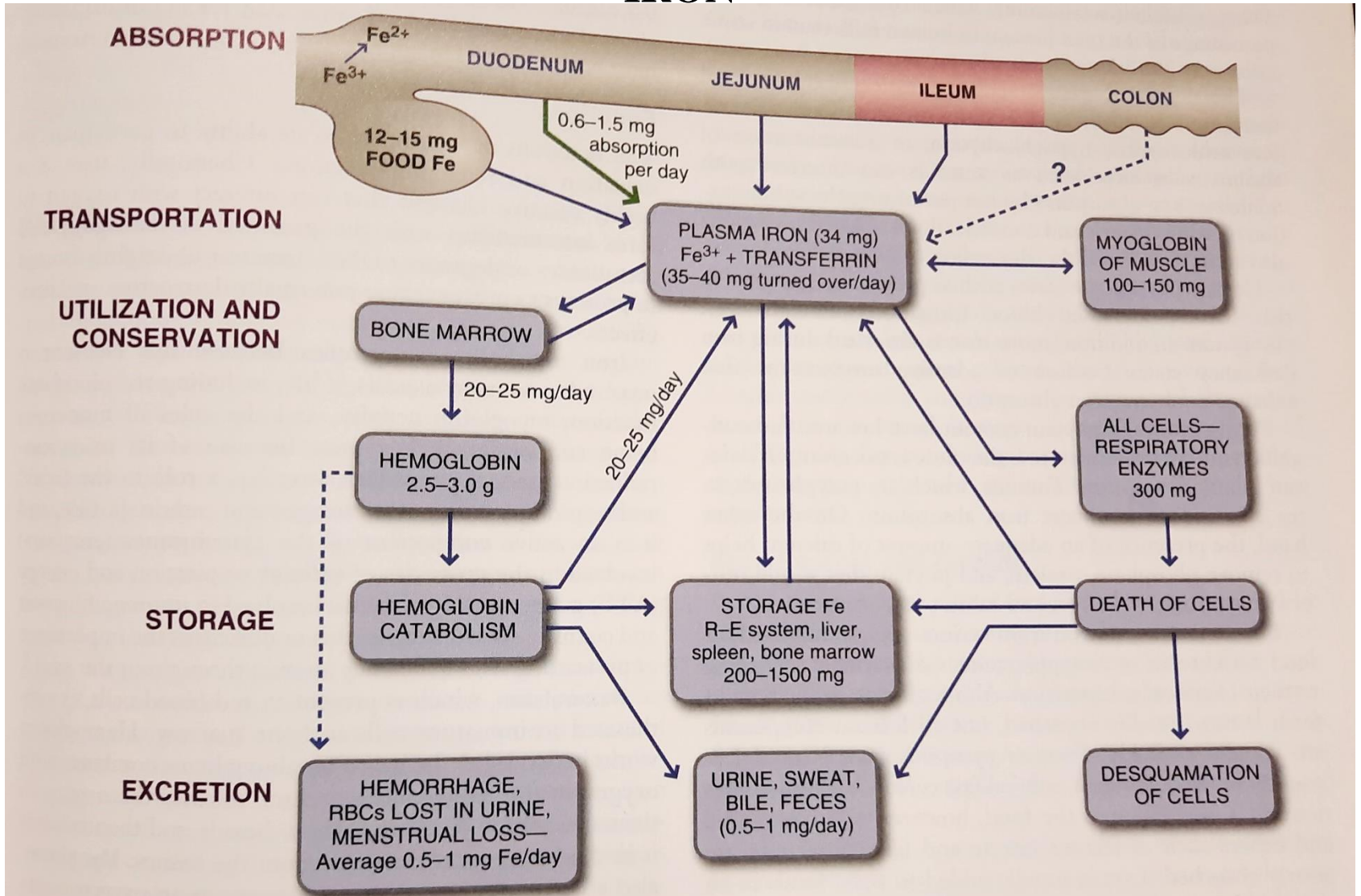


FIGURE 3-33 Iron metabolism in adults. Most iron is absorbed from the duodenum and jejunum, after which it is transported as plasma iron or bound to transferrin. *RBCs*, Red blood cells; *R-E system*, reticuloendothelial system.



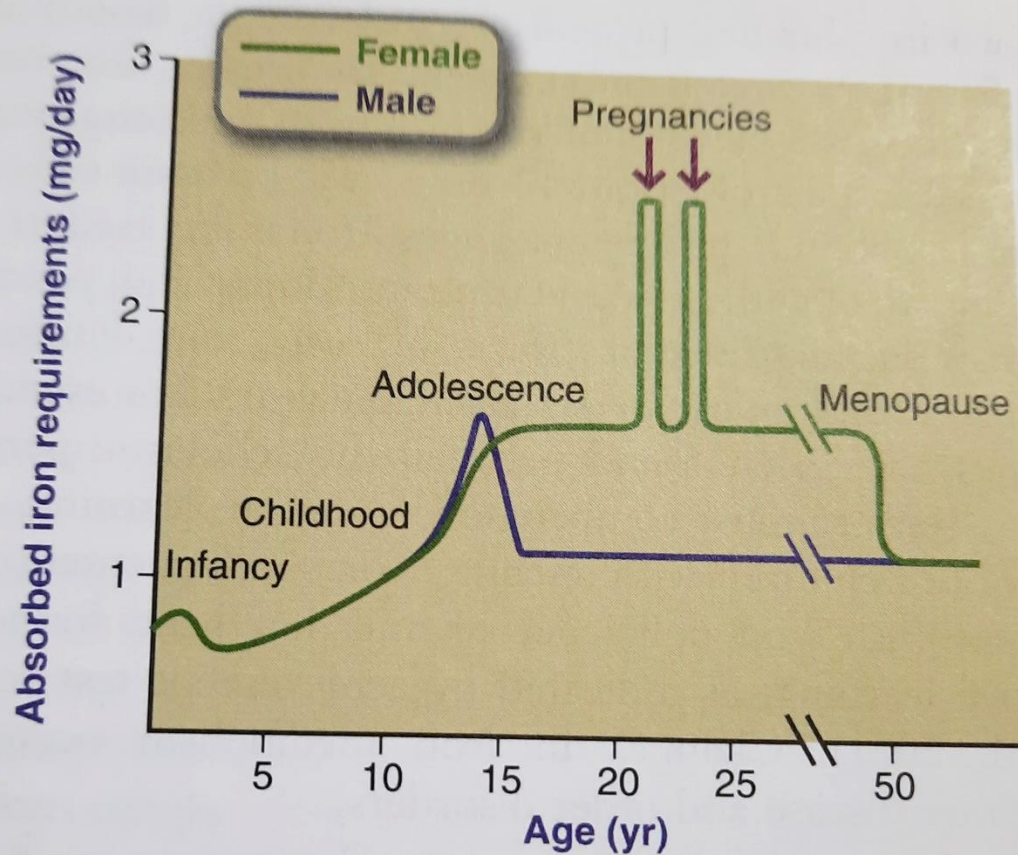


FIGURE 3-34 The absorbed iron requirement for various ages. The greatest requirements for iron occur during infancy. During childhood, requirements are the same for boys and girls. During the adolescent growth spurt, iron needs increase and are greater for boys than girls. However, because of menstruation, the requirements after adolescence remain high for females but decrease for males.



MAIN FUNCTIONS OF IRON

- Iron participates in **oxidation and reduction reactions**.
- It participates in **red blood cell function**.
- **Hemoglobin** → transports oxygen from the lungs to the tissues.
- **Myoglobin** → transports and stores oxygen in the muscle.
- The roles of **numerous *heme non-heme* enzymes** depend on their oxidation-reduction (redox) properties:
 - *Heme*: electron transport, oxidative degradation of drugs
 - *Non heme*: oxidative metabolism
- It is involved in **immune function and cognitive performance**.
- **Transferrin** is involved in the transport of iron and other minerals.



IRON

FUNCTIONS



BLOOD
PRODUCTION



HEALTHY
IMMUNE SYSTEM



NORMAL BRAIN
FUNCTION



MUSCLES
FUNCTION



ENERGY INCREASE



HEALTHY
PREGNANCY



❖ **The two forms of dietary iron are *heme* iron and non-*heme* iron:**

➤ ***Heme* iron** is found only in meat, poultry, seafood, and fish
→ it comes from animal proteins in our diet.

40% *heme* iron and 60% *non-heme* iron

➤ **Non-heme iron** is found in plant-based foods such as grains, beans, vegetables, fruits, nuts, and seeds.

Only *non-heme* iron

***Non-heme* iron** is also found in animal products such as **eggs and milk/dairy products** and comprises over half the iron contained in **animal meat**.



HEME IRON VERSUS NONHEME IRON

HEME IRON

Iron that comes from the animal sources

Occurs in oysters, red meat, poultry, beef liver, and fish like sardines

Consists of a heme protein attached to the iron

Absorption rate is high

Excess heme iron can cause health risks

NONHEME IRON

Iron that comes from plant sources

Occurs in beans, nuts, lentils, greeny-leaves such as spinach, and pumpkin seeds

Does not contain a heme protein attached to the iron

Absorption rate is comparatively low

Does not cause health risks



FOOD SOURCES OF IRON

- Liver
- Seafood
- Kidney, heart, etc.
- Meat and poultry
- Dried beans and vegetables
- Green-leaved vegetables
- Eggs yolks
- Dried fruits
- Dark molasses
- Whole-grain and enriched breads
- Wine
- Cereals and fortified breakfast cereals



Iron Content of Selected Foods

Food	Content (mg)
Cereal, ready-to-eat, fortified, 1 cup	1-22
Clams, canned, 3 oz	23.7
Rice, white, enriched, 1 cup	9.73
Baked beans, 1 cup	8.2
Braunschweiger, 2 slices	6.35
Oysters, cooked, 3 oz	5.9
Bagel, enriched, 1, 4-inch	5.38
Beef liver, fried, 3 oz	5.24
Fast food roast beef sandwich, 1	4.23
Refried beans, 1 cup	4.18
Potato skin, 1	4.08
Burrito, bean, 1	1.13
Ground beef, lean, 3 oz	1.8
Oatmeal, unfortified, 1 cup	1.6
Spinach, cooked, 1 cup	6.43
Corn dog, 1	6.18
Macaroni and cheese, 1 cup	1.86
Egg, 1	0.92
Peanuts, dry roasted, 3 oz	0.8
Blueberries, frozen, ½ cup	4.5
Chicken, breast, roasted, ½	0.64
Broccoli, fresh, cooked, ½ cup	0.64
Wine, red, ½ cup	0.5
Cheese, cheddar, 1 oz	0.2
Milk, 2% fat, 1 cup	0.07





BEANS, PEAS, LENTILS



SPINACH, BOK CHOY, BROCCOLI, KALE



QUINOA



TOFU

IRON

FOR VEGANS AND VEGETARIANS



OLIVES



NUTS, SEEDS



POTATOES



MUSHROOMS





OATS

Good **plant sources of iron** include lentils, chickpeas, beans, tofu, cashew nuts, chia seeds, ground linseed, hemp seeds, pumpkin seeds, kale, dried apricots and figs, raisins, quinoa and fortified breakfast cereals.



Heme iron and non heme iron

Heme iron (25 % absorption)	Non Heme iron (< 25 % absorption)
Fish / Seafood 	Dark green leafy vegetables (Spinach and Kangkung) 
Meat (Beef/ Lamb/ Pork)	Legumes (Bean, dried fruits and Seed)
Poultry	Eggs



❖ Factors affecting iron absorption

Absorption activators:

Meat stimulates the absorption of heme iron.

Organic acids (ascorbic and citric acids, fruits) and animal proteins (meat, fish) stimulate the absorption of non-heme iron.

Absorption inhibitors:

Ca inhibits the absorption of heme iron.

Ca, Mn, some proteins (egg phosphoproteins), phytates, oxalates, tannates and other phenolic compounds inhibit the absorption of non-heme-iron.

PLANT-BASED IRON + VITAMIN C FOR MAXIMUM ABSORPTION



Fortified Cereal +
Strawberries

Fortified bread +
nut butter +
oranges



Legume or iron
fortified pasta +
marinara sauce



Roasted chickpeas
+ lemon juice

Sweet potato +
bean tacos



Broccoli +
tahini dip

- Try to combine cereals, bread and vegetables with milk or egg.

- Finish meals with a fruit rich in vitamin C that increases iron absorption.

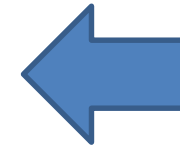
- Take dried fruit and nuts between meals to provide iron all day.

FOOD COMBINATIONS THAT INCREASE IRON ABSORPTION

Alimentos ricos en hierro no hemo	Combinar/cocinar con	Evitar consumir a la vez
Legumbres: <ul style="list-style-type: none"> ▪ Lentejas ▪ Garbanzos ▪ Judías 	Cocinar con pimiento, tomate. Acompañar con morcilla, chorizo Fruta rica en vitamina C: naranja, mandarina, kiwi, fresas.	Té, café, vino tinto.
Verduras: <ul style="list-style-type: none"> ▪ Acelgas ▪ Espinacas 	Cocinar con patatas, aceite de oliva, jamón serrano. Fruta rica en vitamina C.	Té, café, vino tinto.
Cereales de desayuno	Consumir con leche entera o semidesnatada. Zumo cítrico.	Té, café.

DAILY RECOMMENDED INTAKES OF IRON

Edad	Sexo	Condición	Hierro
			Comité Científico AESAN (2019)
			INR
			mg/día
Valor de referencia:			
Unidades:			
0-6 meses	-	-	4,3
7-12 meses	-	-	8
1-3 años	-	-	8
4-5 años	-	-	8
6-9 años	-	-	10
10-13 años	Hombre	-	11
	Mujer	-	15
14-19 años	Hombre	-	11
	Mujer	-	15
20-29 años	Hombre	-	9,1
	Mujer	-	18
30-39 años	Hombre	-	9,1
	Mujer	-	18
40-49 años	Hombre	-	9,1
	Mujer	-	18
50-59 años	Hombre	-	9,1
	Mujer	-	15
60-69 años	Hombre	-	9,1
	Mujer	-	9
>70 años	Hombre	-	9,1
	Mujer	-	9
-	Mujer	Embarazo	27
-	Mujer	Lactancia	15

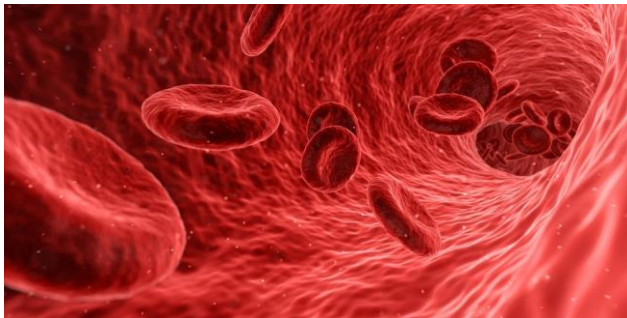


IRON DEFICIENCY

- Iron deficiency is the **most common nutritional deficiency**.
- Those most at risk of iron deficiency are **infants under two years of age, adolescents girls, pregnant women, and older adults**.
- Iron deficiency can be caused by injury, haemorrhage or illness. It is aggravated by an unbalanced diet with insufficient iron, protein, folate and vitamin C.
- Anaemia typically develops because of an inadequate amount of dietary iron or faulty iron absorption.
- The final stage of iron deficiency includes **hypochromic microcytic anaemia**.
- Individuals should be given advice about a diet rich in iron.

IRON DEFICIENCY → FERROPENIA

- This is the most prevalent nutritional deficiency worldwide, affecting:
 - 15% of the total population, and
 - 15-50% of the population in developing countries.
- In Spain it effects:
 - 15% of children,
 - 5% of women of childbearing age, and
 - almost 2% of men.



Anemia ferropénica

The infographic shows a child's body with various symptoms labeled: Mareos (dizziness), Palidez llamativa (pale skin), Tensión arterial baja (low blood pressure), Cansancio (fatigue), Falta de apetito (loss of appetite), Caída de cabello (hair loss), and Menor rendimiento escolar (poor school performance).

Causas

- ▲ Bajo peso al nacer
- 💧 Hemorragias en recién nacidos
- 🍲 Escaso aporte de hierro en la alimentación
- 🍼 Intolerancia a las proteínas de la leche
- 👩 Menstruación
- 👨 Mayores necesidades en las etapas de crecimiento

Función

El hierro es un componente esencial de la **hemoglobina**, la proteína encargada de llevar el oxígeno a todos los órganos y tejidos.

The diagram shows a cross-section of a blood vessel (Vaso sanguíneo) containing red blood cells (Globo rojo). A magnified view of a red blood cell shows the hemoglobin molecule (Molécula de hierro) with iron atoms (Fe) embedded in the heme groups.

Prevalencia

Grupo	Prevalencia
Población general:	30% puede sufrir ferropenia
Adolescentes:	9% ferropenia y 2% anemia
Niños de 1 a 2 años:	10% tiene déficit de hierro y 3% anemia

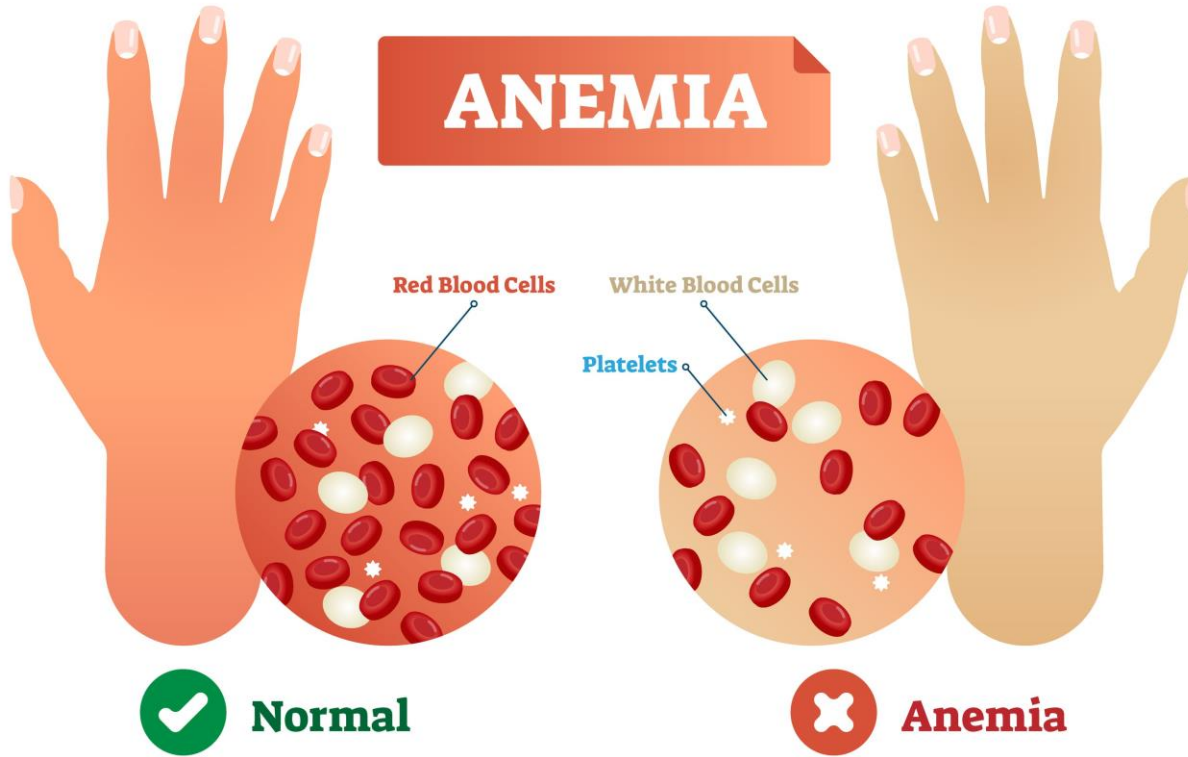
Prevención

- Evitar que los bebés tomen leche de vaca antes de los 12 meses y limitarla en el niño mayor a un máximo de 600 ml al día.
- Incorporar en la dieta alimentos ricos en hierro (yema de huevo, frutos secos, legumbres, carnes ricas...).

Tratamiento

- Administrar 4-6 mg/kg de hierro elemental dividido en tres tomas y acompañado de zumo de naranja para que la absorción sea mayor.
- Las inyecciones solo están indicadas en casos extremos ya que la respuesta es menos rápida que por vía oral.

ANEMIA



SYMPTOMS

Fatigue



Weakness



Pale or
Yellowish Skin



Irregular
Heartbeats



Shortness
of Breath



Dizziness or
Lightheadedness



Chest
Pain



Cold Hands
and Feet



Headache



Signs and symptoms of ferropenia

Fatigue and
Tiredness



Restless Leg
Syndrome

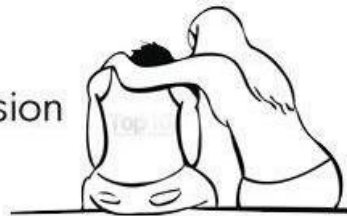


Shortness
of Breath

Frequent
Headaches



Depression



Increased
Sensitivity
to Cold



Hair Loss

Brittle Nails



Depending on its deficiency, it may occur:

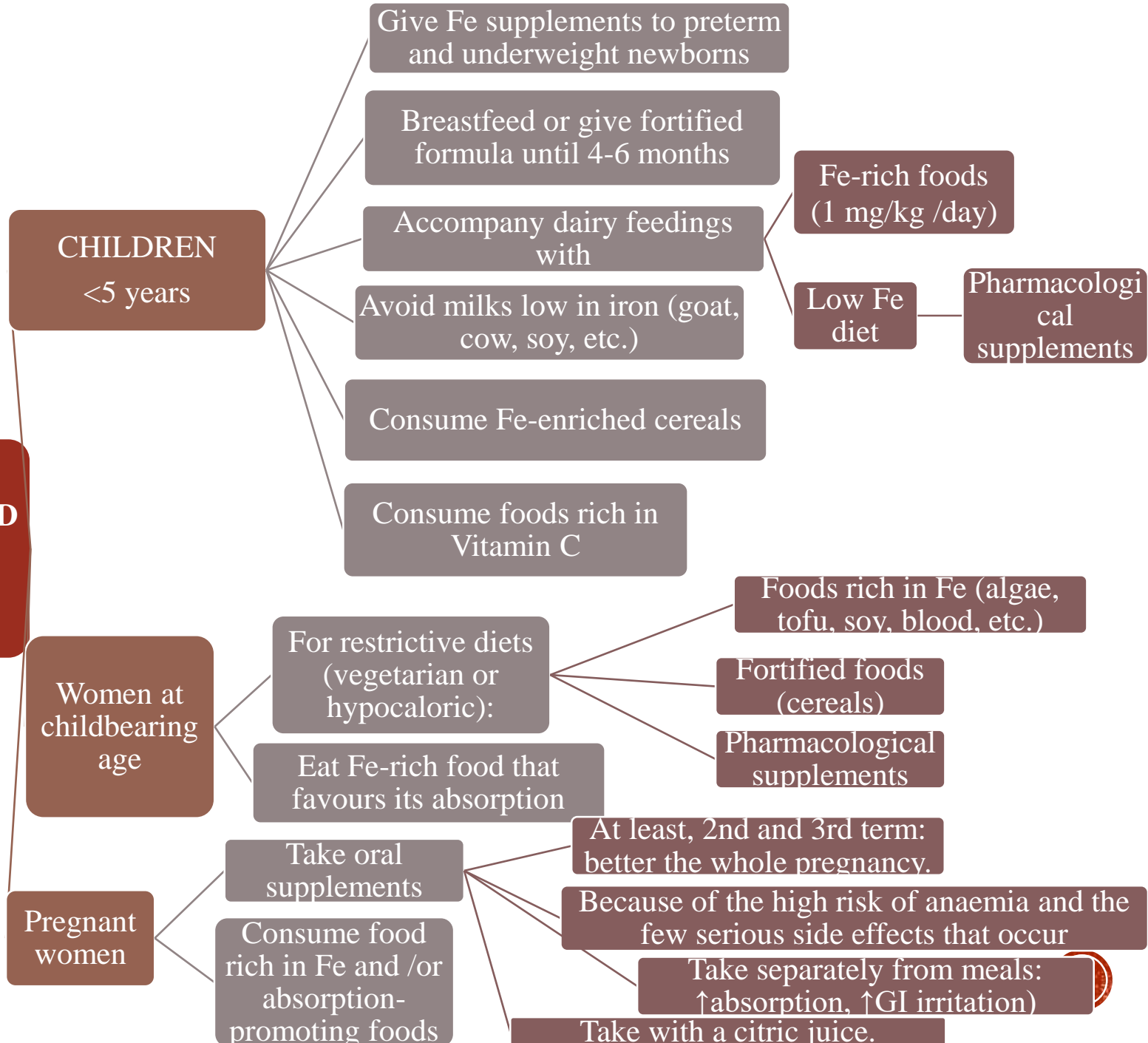
Iron deficiency, even without anaemia, leads to:

- The risk of prematurity and underweight in newborns.
- Delays in child development.
- A lack of concentration, poorer work performance and poorer intellectual performance.
- Weaker immune response to infection.

Iron deficiency may be due to:

- Insufficient contributions (very rich in vegetables, monotonous or low-calorie diets).
- Blood loss (menstruation, bleeding, gastrointestinal bleeding by ulcus, Crohn's disease, parasitosis, abuse of anti-inflammatory drugs, etc.).
- ↑Needs (children < 2 years, adolescence, pregnancy, lactation).
- ↓Absorption (gastrectomy, achlorhydria, malabsorption).
- Alterations in iron transport (transferrin deficit, though this is unusual).

TO PREVENT AND TREAT IRON DEFICIENCY



To increase the dietary intake of BIOAVAILABLE iron:

- Reinforce the consumption of:
 - Foods rich in iron or fortified with iron.
 - Foods with absorption activators of iron (foods rich in vitamin C).
- In iron-providing meals, decrease the consumption of:
 - Tea or coffee.
 - Dairy products.
 - Fibre (fibre-rich foods or supplements).

THE MONSEN MODEL

DISPONIBILIDAD Fe	CONTENIDOS
BAJA	<30 g carne y/o pescado y <25 mg vit. C
MEDIA	<30 g carne y/o pescado y 25-75 mg vit. C
ALTA	30-90 g carne y/o pescado y 25-75 mg vit. C >90 g carne y/o pescado >75 mg vit C



IRON OVERLOAD:

When Fe \uparrow it accumulates as hemosiderin, causing:

- tissue damage
- \uparrow oxidative potential
- \uparrow risk of chronic diseases related to oxidative stress (e.g. liver cancer, cardiovascular disease, and other metabolic disorders related to insulin resistance).

Overload may occur in cases of:

- repeated blood transfusions.
- chronic, excessive consumption of alcohol and Fe and/or excess vitamin C.
- toxic doses of Fe or high doses of parenteral Fe.
- **HEMOCHROMATOSIS**, a disease that is characterized by the hyper-absorption of dietary Fe and produces excessive deposits in the whole body.



IRON OVERLOAD → Symptoms of haemochromatosis:

Abnormal accumulation of iron in the liver
Excessive tissue ferritin levels
Elevated serum transferrin levels
Oxidation of LDL cholesterol
Cardiovascular complications

- **It affects 0.1% of the European population and 0.3-0.5% of the U.S.**
- **It's the genetic disorder most prevalent in people of Western Europe**
- **It affects more often men**
- **It usually manifests between 30 and 50 years of age**



TREATMENT OF HAEMOCHROMATOSIS:

OBJECTIVE

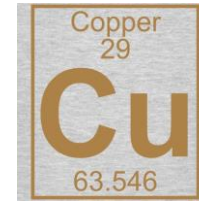
- To remove excess body Fe: extract 0.5 l of blood/week for 2 or 3 years until Fe deposits are exhausted.
- Then, to maintain normal levels of Fe using less frequent extractions.
- To provide supportive care for damaged organs.

DIET

- RESTRICT OR ELIMINATE:
 - The consumption of alcohol.
 - The consumption of supplements and vitamin C.
 - The consumption of foods rich in haeme iron.
 - Kitchen utensils made of iron.
 - The consumption of raw seafood (the seafood can be eaten cooked).
 - The consumption of processed foods fortified with Fe.



COPPER



- **Copper is necessary in trace amounts for red blood cell, collagen and energy production; neuron signalling; immunity; and iron metabolism.**
- **It is an essential micronutrient** (≈ 80 mg in adults) for growth, immunity, brain and bone development, erythropoiesis, glucose metabolism and cholesterol, etc.
- **It is a cofactor for several enzymes** (known as ‘Cu proenzymes’) involved in energy production, iron metabolism, neuropeptide activation, connective tissue synthesis, and neurotransmitter synthesis.

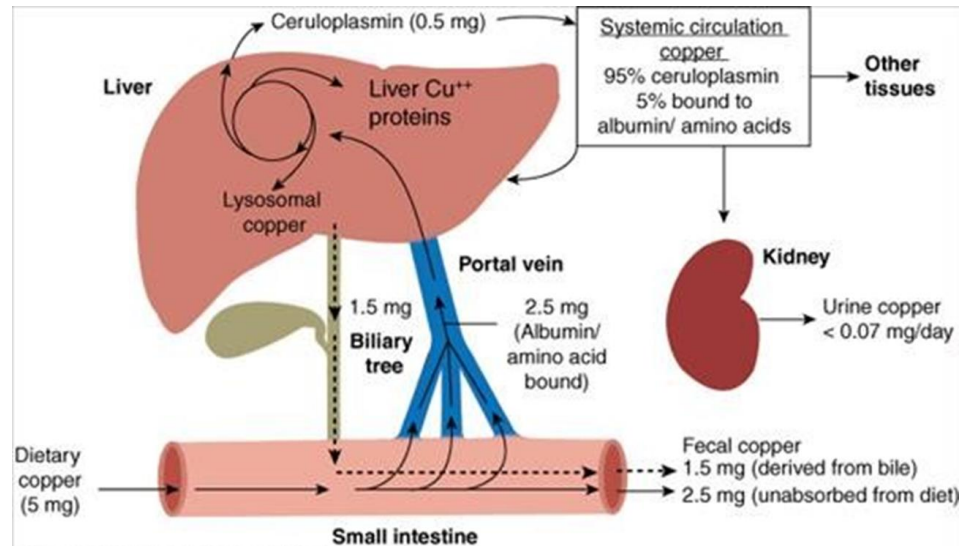


COPPER REQUIREMENTS

- The DRI is 2-3 mg/day (adults).

ABSORPTION

- Copper is absorbed in the duodenum (where it competes with Zn for metallothionein). It is transported attached to ceruloplasmin and albumin and deposited in small amounts in tissues, especially the liver and the brain, and is excreted in bile.



COPPER FOOD SOURCES

- Copper is well distributed in foods such as animal products (except milk), shellfish (e.g. oysters), organ meats (e.g. liver, kidney), muscle meats, chocolate, nuts, cereal grains, dried legumes and dried fruits.

Food	Content (mg)
Beef liver, fried, 3 oz	12.4
Oysters, 3 oz	3.63
Orange juice, 1 cup	0.11
Cashews, dry roasted, ¼ cup	0.61
Sunflower seeds, ¼ cup	0.59
Baking chocolate, 1 square	0.92
Mushrooms, cooked, 1 cup	0.79
Tropical trail mix, 1 cup	0.74
Beans, white, canned, 1 cup	0.61
Yogurt, 8 oz	0.03
Broccoli, raw, 1 cup	0.04
Peaches, canned, 1 cup	0.05
Milk chocolate, 1 oz	0.16
Milk, 2% fat, 1 cup	0.03
DRI Range	
0.2-1.3 mg/day, depending on age and gender	

From U.S. Department of Agriculture, National Nutrient Database



COPPER DEFICIENCY AND TOXICITY

Signs of deficiency: Copper deficiency is generally rare. Possible reasons for inadequate copper intake or deficiency include genetic disorders such as **Menkes disease** (in which copper absorption is faulty), malnutrition, prolonged parenteral nutrition, malabsorption and gastric bypass.

Toxicity: Copper toxicity is rare from dietary sources, except in the case of **Wilson's disease**. In this rare genetic condition, excessive amounts of copper accumulate in the body and can be fatal.



MENKES DISEASE

- ✓ **Menkes disease** affects copper levels in the body.
- ✓ It is characterized by sparse, kinky hair, a failure to gain weight and grow at the expected rate (i.e. a failure to thrive), and a deterioration in the nervous system.



❖ **Other signs and symptoms include:**

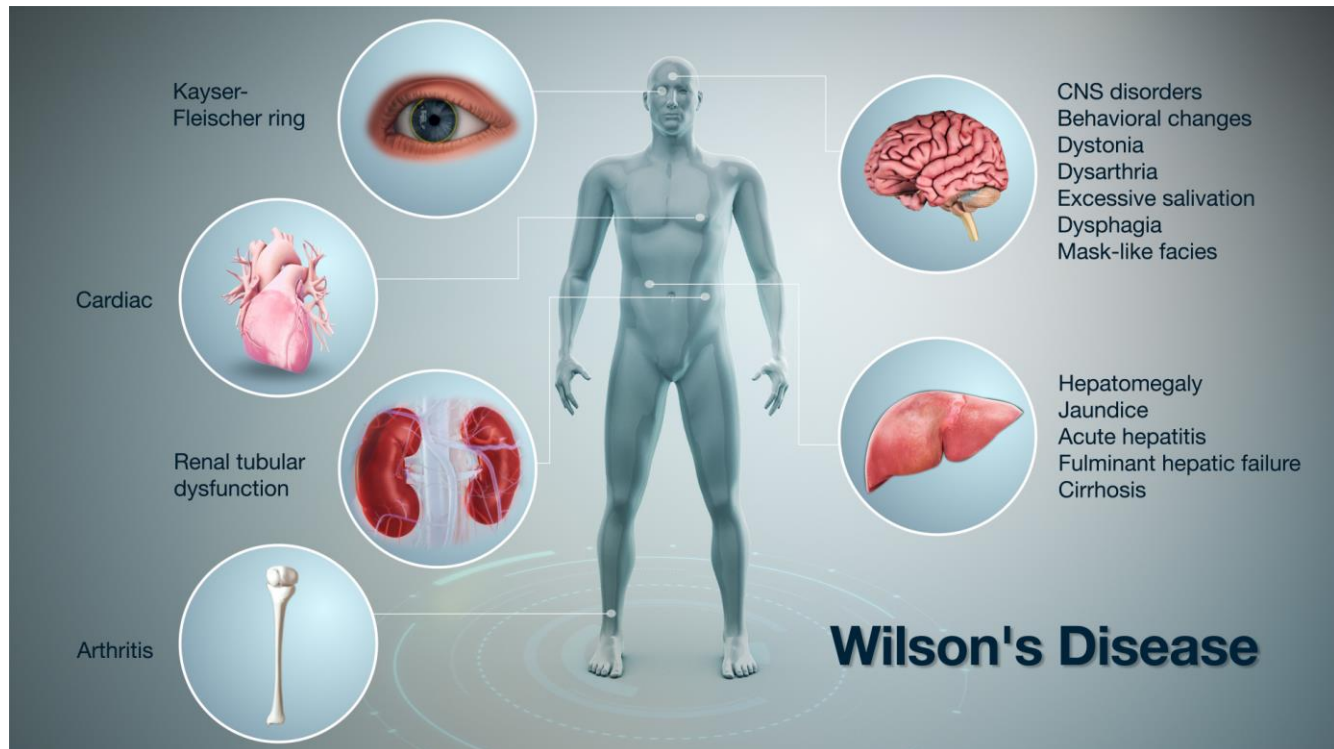
- A weak muscle tone (hypotonicity), sagging facial features, seizures, developmental delay, and intellectual disability.
- Children with Menkes syndrome typically begin to develop symptoms during infancy and often do not live past the age of three.

→ **Early treatment with copper may improve prognosis in some affected individuals.** In rare cases, symptoms begin in later childhood.



WILSON'S DISEASE

Wilson disease is a rare genetic **disorder** characterized by **excess copper stored in various body tissues**, particularly the liver, brain, and corneas of the eyes. It is a progressive **disease** which, if left untreated, may cause liver (hepatic) **disease**, central nervous system dysfunction, and death.



WILSON'S DISEASE:

- An inborn error of metabolism prevents Cu from being eliminated through the bile.
- Symptoms, which usually appear in late adolescence, may include:
 - Jaundice, intestinal inflammation, blood vomiting and abdominal pain (liver is the first organ affected and, in half of the patients, the only one).
 - Tremors and difficulty in walking, speaking and swallowing.
- Symptoms of mental illness, including homicidal or suicidal behavior, depression and aggression.
- Women may have menstrual irregularities, lack of periods, infertility or multiple spontaneous abortions.
- A green corneal ring (Cu deposits in the cornea).
- Affected renal tubules.

Regardless of how it begins, Wilson's disease is always fatal if not diagnosed and treated with:

- D-penicillin (indefinitely even without symptoms).
- Zn salts block (Cu absorption).
- Dietary restriction of foods rich in Cu, e.g. seafood, dried fruits, liver.



Enjoy your lunch!

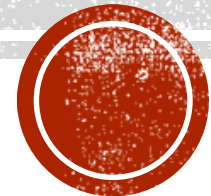


Thank You
For Your Attention...



UNIT 3.2 DIET FOR OVERWEIGHT AND OBESITY.

NUTRITIONAL GOALS. INDICATIONS AND ADVERSE EFFECTS. EDUCATION AND PRACTICAL RECOMMENDATIONS FOR WEIGHT CONTROL. TYPES OF LOW-CALORIE DIETS.



UNIT 3.2
Prof. Pilar Vila-Donat

ACCORDING TO WHO...



“Obesity is one of the greatest public health challenges of the 21st century. Its prevalence has tripled in many countries of the WHO European Region since the 1980s, and the numbers of those affected continue to rise”

“Obesity has reached epidemic proportions globally, with at least 2.8 million people dying each year as a result of being overweight or obese”

<https://www.who.int/news-room/facts-in-pictures/detail/6-facts-on-obesity>



Will the COVID-19 pandemic worsen the obesity epidemic?

- **Deaths due to Covid-19 have reached 1.8 million globally in one year (2020).**



- **Deaths due to obesity have reached at least 2.8 million globally each year.**



→Obesity affects a large percentage of the Spanish population.

→Overweight is more common among men than women, whereas obesity is more prevalent among women.

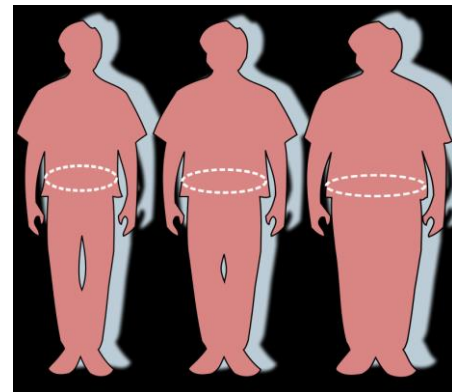
→In Spain, one in two adults aged 25–60 years has a higher Body Mass Index (BMI) than is recommended (BMI \geq 25 kg/m²), and 14.5% of Spanish adults are obese (BMI \geq 30 kg/m²).



<http://www.seedo.es/>



- ❖ **Obesity** is a medical condition whereby excess body fat has accumulated to an extent that it may have a negative effect on a person's health.
- ❖ A person is generally considered obese when their body mass index (**BMI**), obtained by dividing their weight by the square of their height, is **over 30 kg/m²**. A person whose BMI is in the **25–30 kg/m²** range is defined as **overweight**.



BMI is defined as a person's weight in kilograms divided by the square of their height in metres (kg/m²).

$$\text{BMI} = \frac{\text{weight in kg}}{(\text{height in m})^2}$$



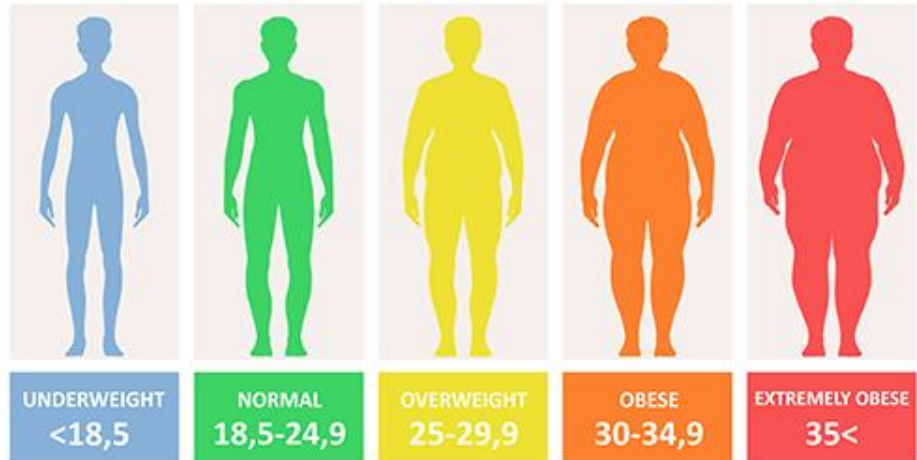
BMI (body mass index), which is based on a person's height and weight, is an inaccurate measure of **body fat content; it **does not** take into account **muscle mass, bone density, overall body composition, or racial and sex differences.****





BMI	Weight status
Below 18.5	Underweight
18.5-24.9	Normal weight
25.0-29.9	Overweight
30.0-34.9	Obesity class I
35.0-39.9	Obesity class II
Above 40	Obesity class III

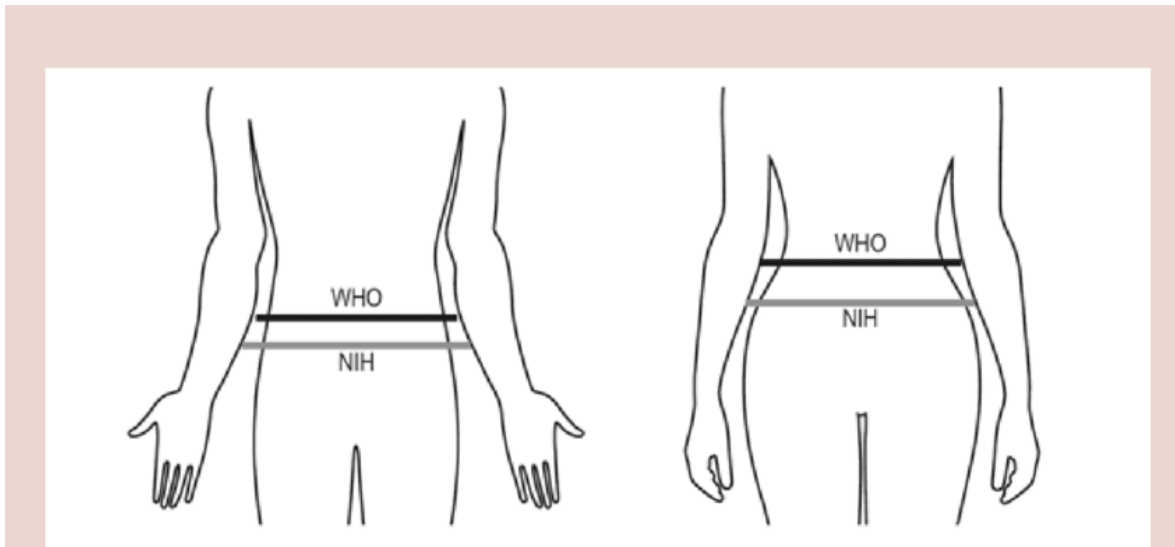
BODY MASS INDEX



WAIST CIRCUMFERENCE (WC)

- ✓ **Waist circumference is an assessment tool that can complement BMI to assess disease risk.**
- ✓ **Excess fat located in the upper abdominal region (visceral fat) is associated with a higher risk than fat located in other areas. Abdominal fatness is an independent risk factor (even when BMI is not increased) and is a predictor of comorbidities and mortality.**
- ✓ **Waist circumference is a significant predictor of a range of health problems including hypertension, coronary heart disease (CHD), and type-II diabetes.**





- The **risk of metabolic complications** increases for:
 - **Men** with a waist circumference > 102 cm
 - **Women** with a waist circumference > 88 cm.





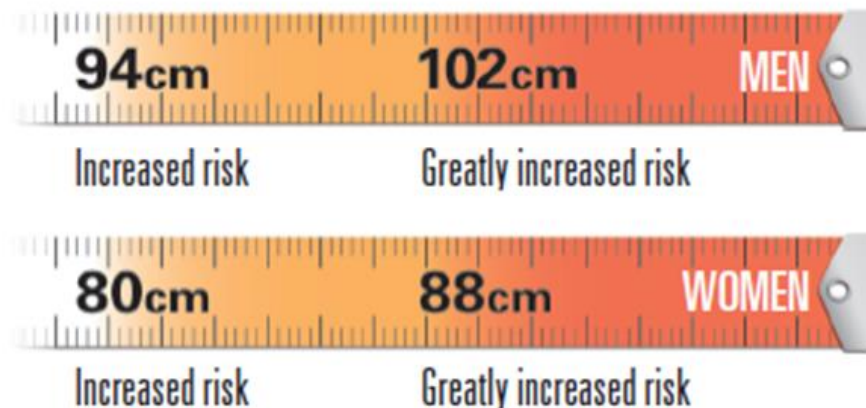
Gynoid or android obesity



	Body mass index ^a		
Men and women	18.5–24.9 kg/m ²	25–29.9 kg/m ²	≥30 kg/m ²
Classification	Normal weight	Overweight	Obese
Risk of co-morbidities	Low	Increased	High
	Waist circumference ^b		
Men	<94 cm	94–101.9 cm	≥102 cm
Women	<80 cm	80–87.9 cm	≥88 cm
Classification	Normal fat distribution	Moderate central fat accumulation	High central fat accumulation
Risk of co-morbidities	Low	Increased	High

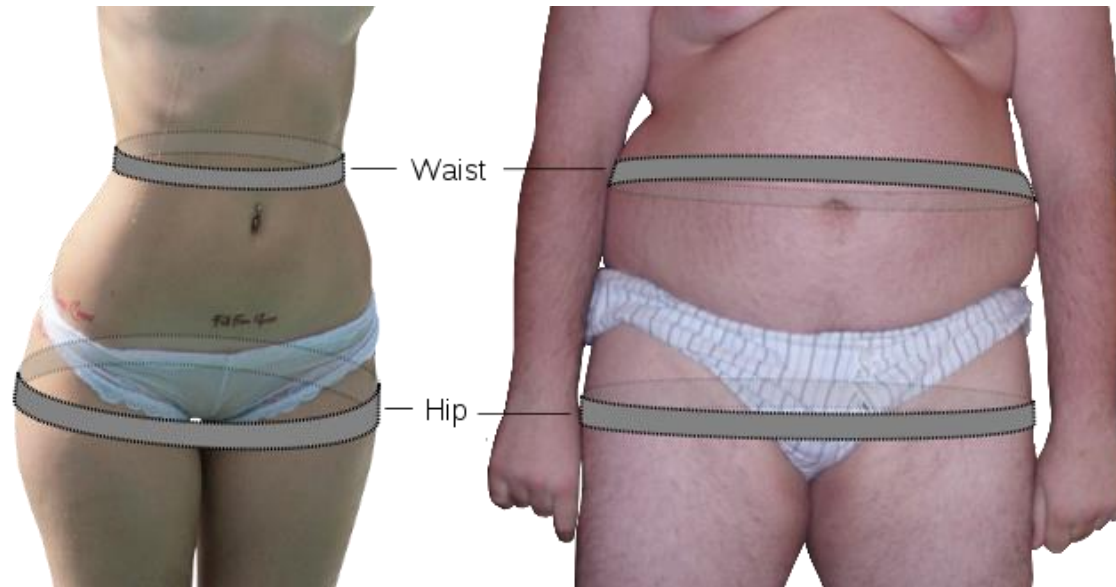
^aWHO.¹²

^bLean et al.⁴



WAIST-HIP RATIO (WHR)

The **waist-hip ratio** or **waist-to-hip ratio** (WHR) is the dimensionless **ratio** of the circumference of the **waist** to the circumference of the **hips**. It is calculated as **waist** measurement divided by **hip** measurement (W/H). For example, a person with a 30" (76 cm) **waist** and 38" (97 cm) **hips** has a **waist-hip ratio** of roughly 0.78.



$$\text{WHR} = \frac{\text{Waist Circumference}}{\text{Hip Circumference}}$$

Female	Male	Health Risk
0.80 or lower	0.95 or lower	Low health risk
0.81 to 0.84	0.96 to 1.0	Moderate risk
0.85 or higher	1.0 or higher	High risk



- **Body mass index (BMI), waist circumference (WC), and waist-hip ratio (WHR)** are three measures used to estimate **body fat content**.
- **BMI** is easy to calculate but does not differentiate between lean and fat mass. **WC** correlates strongly with **BMI** when predicting **cardiovascular and metabolic risk**.
- The clinical utility of **WHR** has receded in recent years due to its weaker association with **cardiovascular and metabolic risk factors** in comparison with **other clinical adiposity measures**.



Common health consequences of overweight and obesity

Increased BMI is a major risk factor for non-communicable diseases such as:

- Cardiovascular diseases (mainly heart disease and strokes).
- Diabetes.
- Musculoskeletal disorders (especially osteoarthritis, a highly disabling degenerative disease of the joints).
- Some cancers (including those of the endometrium, breast, ovary, prostate, liver, gallbladder, kidney, and colon).



→ **Childhood obesity** is associated with a higher risk of obesity, premature death and disability in adulthood. As well as increased future risks, obese children experience **breathing difficulties, increased risk of fractures, hypertension, early markers of cardiovascular disease, insulin resistance and psychological effects.**



Consequences of obesity

Metabolic disorders: gout, cholelithiasis, type-2 diabetes, hyperlipidemia and arteriosclerosis.

Respiratory system disorders: chronic hypoventilation, obstructive sleep apnea.

Cardiovascular effects: hypertension, coronary disease, impaired venous return (lower-limb edema), varicose veins, thrombophlebitis.

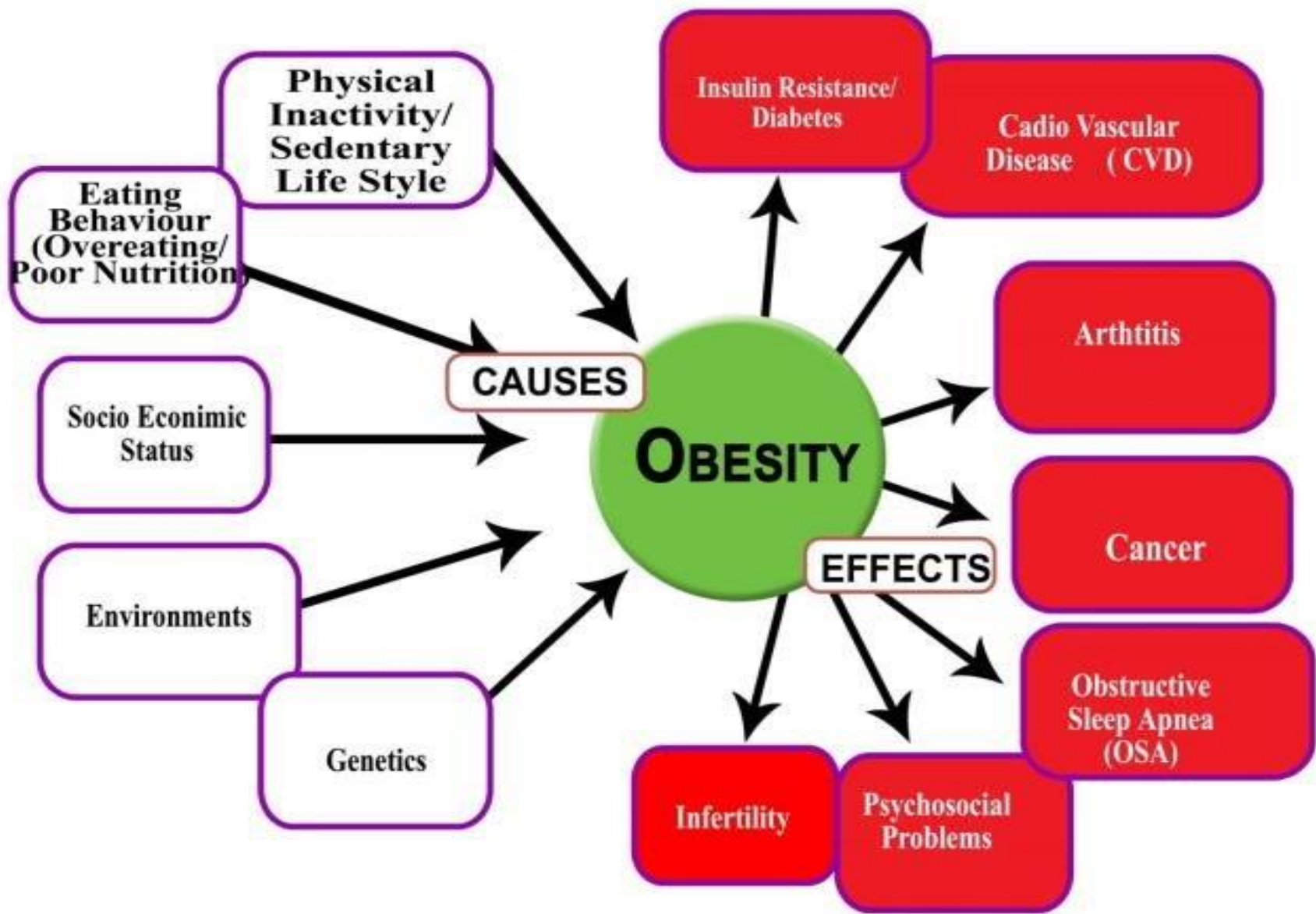
Mechanical impact: hiatal hernia, poor hygiene (redundant skinfolds and limited motility), intertrigo, musculoskeletal stress.

Endocrine effects

gonadal dysfunction (increased conversion of androgens to estrogens in peripheral adipose); in males, erectile dysfunction and oligospermia; in women, menstrual alterations, amenorrhea and decreased fertility.

Psychopathological aftermath





TREATMENT MUST BE INDIVIDUALIZED



❖ Comprehensive clinical assessment:

- ✓ physical examination and laboratory analysis
- ✓ clinical, dietetic and weight history (previous anti-Obesity treatment)
- ✓ physical activity (work and leisure)
- ✓ psycho-social characteristics
- ✓ comorbidity (diabetes mellitus, hypertension, dyslipidemia, sleep apnea)
- ✓ risk factors (smoking, central obesity, menopause, obesity family history)

❖ Identification of organic causes and/or stable habits

- ✓ conditions that precede the act of eating (hunger, boredom, tension, depression, seeing others eating, offers and invitations, hours, places, situations, etc.)
- ✓ intake habits (speed, quantity, quality, frequency, etc.)

→ **This will establish specific objectives and reinforce behaviours that specifically combat the patient's problems.**



HYPOCALORIC DIET: OBJECTIVES

A hypocaloric diet is always the first step in treatment.

- a) Decrease body fat while respecting lean mass.**
- b) Maintain long-term weight loss and prevent future gain.**
- c) Improve functional capacity and quality of life.**
- d) Re-educate eating behaviours and lifestyle.**



Hypocaloric diets should be balanced and nutritionally complete and should force the body to oxidize its reserves of fat.

They should contain a good **supply of fibre**, as satiety modulates digestion/absorption and improves peristalsis.

They should be accompanied by **regular physical exercise**.

They should not be adhered to indefinitely as this leads to negative nitrogen balance and a loss of efficacy.

They are **difficult to establish**, since they are based on a **difficult balance**. They should:

- be balanced and **nutritionally complete**.
- contain sufficient amounts of all **essential nutrients**.
- be **sufficiently hypocaloric** to enforce the consumption of fat reserves, despite the body's immediate metabolic adjustments.

The most critical nutrient is **PROTEIN** since the patient is intended to lose fat while maintaining a **positive nitrogen balance**.

The **carbohydrate intake:**

- Should therefore be **enough** to avoid degrading amino acids to synthesize glucose (gluconeogenesis).

The **PROTEIN intake:**

- Should be 15–25%* distributed across **several shots** (less weight is lost, but it is loss more fat and less protein)
- Should be of **high biological quality:**
- Should contain enough essential amino acids
 - To have a right protein turnover
 - To have a minimum amount of protein that is converted to energy (nitrogen overload of liver and kidneys)



***These requirements are provided by 1,300-1,900 kcal diets. These amounts are a long way from the 800-1,200 kcal needed to lose weight.**



Why do very restrictive diets not work? They are 'unhealthy' because they lose a lot of lean mass.

DIET		Cal/day	Adverse effects
LCCD	Low-calorie complet diet	1300-1900	
LCD	Low-calorie diet	800-1200	Water and proteins are lost. REE and TEE are decreased.
VLCD	Very low-calorie diet (before bariatric surgery). Must be clinically supervised.	450-800	The patient feels tired and reduces their physical activity. There is an increased tendency to depression, bulimia or binge eating.



VLCDs

- ❑ **Very low-calorie diets** are clinically supervised **diets** that involve **eating** roughly **800 calories** a day or fewer (450-800 Kcal).
- ❑ They are sometimes considered for obese and severely obese patients managing diabetes or preparing for surgery or fertility treatment.
- ❑ VLCDs use **meal** replacements such as drinks, shakes, and prepared food bars instead of **meals** for at least two **meals** a day.



LCD

Lose fat mass

Energy balance is negative

Dietary allowances < total energy expenditure



Losing 1 kg of fat requires ‘burning’ or avoiding the intake of 7,200 kcal (800 g x 9 kcal/g), which is counter-balanced because:

→ The body adapts to new circumstances, such as decreasing resting energy expenditure (REE) and the thermic effect of food (TEF) and minimizing total energy expenditure (TEE) to 1000-1200 (kcal /day).

→ Fat, water and proteins are lost with fewer kcal, so **muscle mass is decreased**. All the above leads to **fatigue, decreased physical activity**, lower REE and lower TEE.

CHARACTERISTICS OF MODERATE HYPOCALORIC DIETS

Moderate weight reduction

Losing 5-10 kg/year (225-450 g/week) reduces mortality by 25%.

Increased physical activity

Helps to maintain lean mass and increase expense due to physical exercise and Total Energy Expenditure (TEE).

Can be achieved with just 1 of hour of moderately intense activity per day (e.g. walking).

Moderately hypocaloric satiating diets (decrease TEE by 500-600 kcal):

15-25% proteins of high biological value produce a satiating effect, a positive nitrogen balance and increased thermogenesis of food.

45-55% carbohydrates

25-35% fat

Supplement with micronutrients should be considered (since it is difficult to cover a person's daily needs with diets <2000 kcal and almost impossible with diets <1500).

Patients should drink a lot of water (it increases satiety).

20-40 g fibre: to increase chewing time, produce early satiation, delay gastric emptying and digestion, and decrease fat absorption.

Dietary Recommendations

Re-educate/change lifestyle to avoid regaining the lost weight:

- ✓ Never go shopping before meals.
- ✓ Buy only what you will use and read the labels.
- ✓ Cook the right amounts, use simple techniques, and eat off small plates.
- ✓ Bring food to the table on the plate and only what will be consumed.
- ✓ Don't eat alone; try to chat while eating.
- ✓ Drink plenty of liquids (water and/or light refreshments).
- ✓ Don't do any other activity (such as watching TV) while eating.
- ✓ Chew carefully and rest between bites.
- ✓ Leave the table immediately you've finished eating.
- ✓ Go to parties knowing what you can eat and what you can drink. Try to convince your family to do the same.



Low fat/low CH diets

Low-carbohydrate diet (LCHD)

<20 to 60 g/day (less than 20% of total caloric intake); increase the proportion of fats or proteins by the same amounts.

A carbohydrate restriction < 20 g is potentially a ketogenic diet.

LCHD produces more weight loss in the short term (6 months).

> 1 year, LCHD produces a similar weight loss to that achieved with LFD.

> 1 year, LCHD produces a greater HDL increase and triglyceride decrease than LFD.

> 1 year, LFD produces a greater decrease in LDL cholesterol than LCHD.

> 1 year, LCHD produces more adverse effects than LFD.

The higher late mortality observed in LCHD may be further increased by eating fats of animal origin.

Recommendations:

- To lose weight it is useful to reduce the proportion of carbohydrates and increase the proportion of fats.
- To control LDL cholesterol it is useful to follow an effective LFD.
- HDL cholesterol and triglyceride levels are controlled better with a DBHC.
- A LCHD should not contain a high percentage of animal fat.

Fibre-enriched diets

- ✓ There is insufficient evidence that fibre-enriched diets or diets rich in whole grains promote weight loss.
- ✓ Glucomannan supplements may have a **discreet effect via a satiating mechanism.**
- ✓ Fibre supplements other than glucomannan **can help minimally to lose weight.**
- ✓ Diets enriched or supplemented with glucomannan, P. ovata and β -glucans **decrease LDL cholesterol.**

Recommendations:

- Fibre supplements (mainly glucomannan) can increase the effectiveness of a hypocaloric diet.
- Obese patients with lipid abnormalities can benefit from diets enriched in fibre or fibre supplements (mainly glucomannan).

Hyper-proteic diets (HPD)

- ✓ HPD can induce greater weight loss in the short term (<6 m.) than a conventional carbohydrate-rich diet, but not in the long term (>12 m.).
- ✓ There are insufficient data to establish the effectiveness of HPDs in maintaining weight after an initial loss phase with another type of diet.
- ✓ HPDs promote the preservation of lean mass better than carbohydrate-rich diets.
- ✓ HPDs may increase the risk of all causes of cardiovascular mortality in the very long term, mainly if the proteins are of animal origin.

Recommendations:

- When treating obesity, it is not recommended to make changes in the proportion of diet proteins.
- To ensure that lean mass is maintained or increased, it is effective to increase the protein content of low-calorie diets above 1.05 g/kg.
- If an HPD is prescribed, the intake of animal protein should be limited so as not to increase the risk of mortality in the very long term.

❖ Meal replacements for LCD and VLCD

- Substituting one or more meals with commercial preparations may **facilitate adherence to a low-calorie diet**, thus promoting weight loss and loss of weight maintenance.
- **The beneficial effect is greater** when LCD and VLCD are used in a structured context that includes **guidelines for exercise, education and modification of eating habits**.
- **No significant adverse effects associated** with the use of food substitutes have been reported in the context of LCDs.

Recommendations:

- **Replacing some meals with commercial substitutes in the context of low-calorie diets may be useful for weight loss and weight maintenance in obese or overweight adults.**



Evidence-based nutritional recommendations for the prevention and treatment of overweight and obesity in adults (FESNAD-SEEDO consensus document). The role of diet in obesity treatment (III/III) → Nutrición Hospitalaria

Table III

Conditions which must be met by the dietary treatment of obesity

- It decreases body fat while preserving maximum lean mass.
- It is achievable for a prolonged period of time.
- It is effective in the long term, in other words, maintaining lost weight.
- It prevents future weight gain.
- It involves dietary education which eradicates errors and unsuitable eating habits.
- It reduces the cardiovascular risk factors associated with obesity (arterial hypertension, dyslipidaemia, pre-diabetes or diabetes mellitus).
- It results in improvements in other comorbidities associated with overweight (sleep apnoea, osteoarthritis, neoplastic risk, etc.).
- It induces psychosomatic improvement, with self-esteem being recovered.
- It increases functional capacity and quality of life.

Table V

Recommendations on macronutrient distribution for the treatment of obesity¹¹

<i>Energy</i>	<i>500-600 kcal deficit/day on the baseline estimates obtained through equations or on normal intake</i>
Carbohydrates	45-55%
Proteins	15-25%
Total fats	25-35%
Saturated	< 7%
Monounsaturated	15-20%
Polyunsaturated	< 7%
Trans fatty acids	< 2%
Fibre	20-40 g

is considered to be a diet which results in a calorie deficit of between 500 and 1 000 kcal/day, with a total

RECOMMENDATIONS

- ✓ An energy deficit of 500–1,000 kcal/day in the energy requirements of the obese adult patient is enough to induce an 8% weight loss in the first 6 months of treatment.
- ✓ Restricting the size of portions and/or the diet's energy density are effective strategies for reducing the weight of obese patients through dietary management.
- ✓ Reducing the proportion of carbohydrates and increasing the proportion of fats does not bolster the effect of diet on weight loss.

- ✓ A low-fat diet is effective in controlling LDL cholesterol levels in obese patients. HDL cholesterol and triglyceride levels, on the other hand, are better controlled by a low-carbohydrate diet.
- ✓ Low-carbohydrate diets should not contain a high proportion of fats of animal origin.
- ✓ Dietary fibre (especially glucomannan) supplements may increase a diet's efficacy in achieving weight loss in obese patients.
- ✓ Obese patients with hypercholesterolemia may benefit from diets enriched or supplemented with dietary fibre (especially glucomannan supplements).

- ✓ In the dietary management of obesity, it is not recommended to change the diet's proportion of protein.
- ✓ To maintain or increase lean body mass during the administration of a hypocaloric diet, increasing the diet's protein content to levels above 1.05 g/kg is effective.
- ✓ When a hyper-proteic diet is prescribed, the protein fraction of animal origin should be restricted to prevent an increased risk of mortality in the very long term.
- ✓ In the context of hypocaloric diets, replacing or substituting some meals with meal replacement preparations may be useful for achieving or maintaining weight loss in obese or overweight adults.

- ✓ VLCDDs may be used for the dietary management of patients with obesity provided they are accompanied by specific clinical indications and close and strict medical follow-up.
- ✓ VLCDDs should not be used with patients who do not satisfy the established medical indications and requirements.
- ✓ VLCDDs may be necessary in the preoperative preparation of bariatric surgery in patients with hepatic steatosis and increased surgical risk. They must always be administered under close medical control and with due consideration for any adverse effects that may arise.
- ✓ VLCDDs with commercial preparations may be necessary in the immediate period following bariatric surgery to help the patient achieve an adequate protein intake.

- ✓ FESNAD (Spanish Federation of Nutrition, Food and Dietetic Associations)
- ✓ SEEDO (Spanish Association for the Study of Obesity)
- ✓ <file:///C:/Users/M%C2%AA Pilar/Downloads/ConsensFESNADIII-III.pdf>

Thank You
For Your Attention...



Unit 3.4 Dyslipidemic diet.

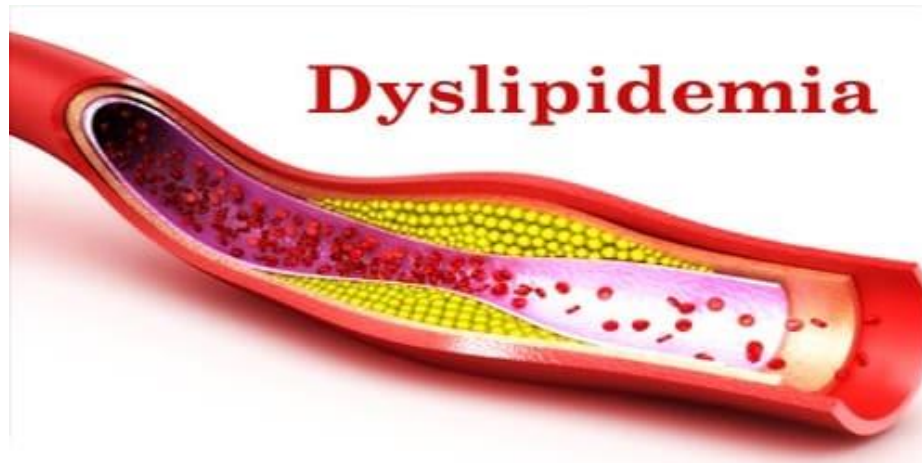
Types, treatment, options, associated pathologies. Influence of dietary components on lipid profile. General and specific recommendations.



UNIT 3.4
Prof. Pilar Vila-Donat

❖ What is dyslipidemia?

Dyslipidemia is defined as an elevated total or low-density lipoprotein (LDL) cholesterol level or a low high-density lipoprotein (HDL) cholesterol level. It is an important risk factor for coronary heart disease (CHD) and stroke.



- ✓ **Dietary fat-intake and elevated blood lipids are considered risk factors for dyslipidemia and the development of atherosclerosis and diabetes.**
- ✓ **Elevated cholesterol and lipoproteins are the main factors that cause dyslipidemia.**
- ✓ **Elevated lipids are known as triglycerides (TC), lipoproteins (LDL), apolipoproteins (apo-B), and TG and low HDL, and apo A-I in the blood.**



Lipid disorders are classified by their dyslipidemia profile:

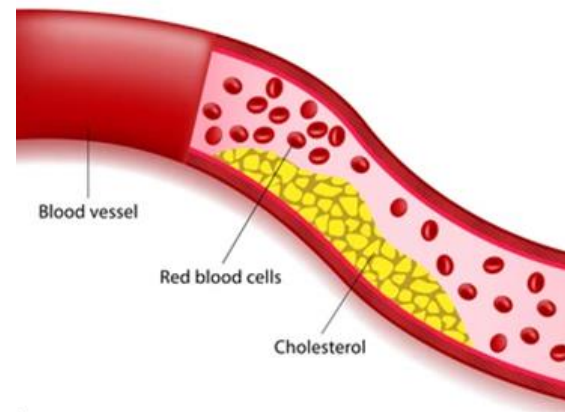
- ✓ Elevated LDL
- ✓ Elevated LDL accompanied by high triglyceride (TG)
- ✓ Elevated TG
- ✓ Low HDL
- ✓ Elevated LDL accompanied by low HDL
- ✓ Inherited lipoprotein disorders that are often presented in young patients at high risk of future CVD include familial hypercholesterolemia (FH), familial hypercholesterolemia combined with hyperlipidemia (FCHL), familial hypoalphalipoproteinemia, apolipoprotein A-I mutations, lecithin cholesterol acyl transferase (LCAT) deficiency, and hyper-TG associated with lipoprotein lipase deficiency.



Lipids	Values	Level
Total Cholesterol (TC)	< 200	Ideal
	200-239	Borderline
	≥ 240	High
LDL-cholesterol (LDL-c)	< 100	Ideal
	100-129	Desirable
	130-159	Borderline
	≥ 190	Very High
HDL-cholesterol (HDL-c)	< 40	Low
	> 60	High
	< 150	Ideal
Triglycerides (TAG)	150-200	Borderline
	201-499	High
	≥ 500	Very High



- Approximately 20% of adults have total cholesterol > 250 mg/dl.
- 50-69% of middle-aged adults have total cholesterol > 200 mg/dl.
- One out of four patients in Primary Care consultations are diagnosed with dyslipidemia.
- Most dyslipidemic patients receive treatment for this condition (73% drugs, 69% diet, 7% no treatment) but only one third of patients achieve adequate control.



shutterstock.com · 1198491688



CAUSAS DISLIPEMIA

NO MODIFICABLES

1. GENÉTICAS O HEREDITARIAS:

5% de la población general

2. EMBARAZO

MODIFICABLES

1. FACTORES DIETÉTICOS:

Consumo de alimentos ricos en AGS, colesterol, azúcares simples, AG trans, alcohol excesivo.

2. SEDENTARISMO

3. ESTRÉS

4. OBESIDAD O SOBREPESO

5. TABACO

- **ENFERMEDADES** (Las más frecuentes: DM mal controlada, hipotiroidismo, IRC, hepatopatías)
- **FÁRMACOS**: Progestágenos, estrógenos, corticoides, *diuréticos*, *beta-bloqueantes*, Ciclosporina, Tacrolimo, Isotretinoína, Inhibidores de la proteasa, anti-retrovirales, fluconazol, algunos IBP, *vitamina D*

CAUSES → Lifestyle, genetics, disorders such as low thyroid hormone levels or kidney disease, drugs, or a combination of the above.



Tabla I. Dislipemia. Niveles de lípidos en plasma sanguíneo considerados como patológicos.

Colesterol total	≥ 200 mg/dl
Triglicéridos	≥ 200 mg/dl
HDL-colesterol	< 40 mg/dl
LDL-colesterol	≥ 130 mg/dl



The latest joint European guide on CVD prevention recommends the use of the **SCORE system** because it is based on data series derived from large and representative European cohorts.

SCORE Risk Charts

The European cardiovascular disease risk assessment model **Systematic Coronary Risk Evaluation (SCORE)**: these high and low cardiovascular risk charts are based on gender, age, total cholesterol, systolic blood pressure and smoking status. They also comprise relative risk charts, qualifiers and instructions.



<https://www.escardio.org/static-file/Escardio/Subspecialty/EACPR/Documents/score-charts.pdf>



Advantages of SCORE

- ✓ It is based on a large data set that has been thoroughly tested with European data.
- ✓ It operates with hard, reproducible endpoints (CVD death).
- ✓ The risk of death by CHD or stroke can be derived separately.
- ✓ It enables the development of an electronic interactive version of the risk chart.
- ✓ The SCORE risk function can be calibrated to each country's national mortality statistics.

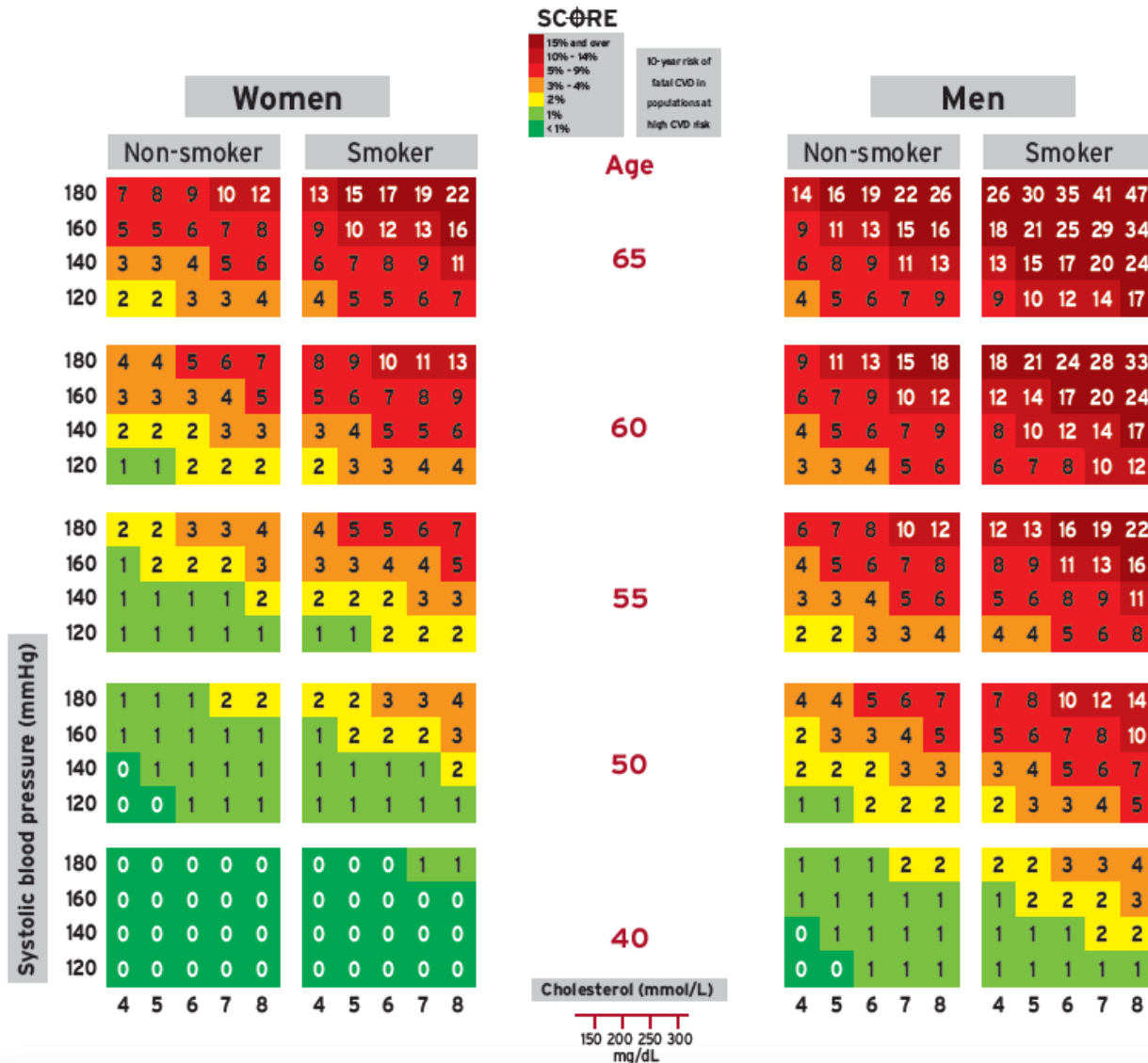
The SCORE database combines results from:

- ✓ 12 European cohort studies.
- ✓ 250,000 patient data sets.
- ✓ 3 million person-years of observation.
- ✓ 7,000 fatal CV events.



SCORE - European High Risk Chart

10 year risk of fatal CVD in high risk regions of Europe by gender, age, systolic blood pressure, total cholesterol and smoking status



How do I use the SCORE charts to assess CVD risk in asymptomatic persons?

1. Use the **low risk charts** in Andorra, Austria, Belgium*, Cyprus, Denmark, Finland, France, Germany, Greece*, Iceland, Ireland, Israel, Italy, Luxembourg, Malta, Monaco, The Netherlands*, Norway, Portugal, San Marino, Slovenia, Spain*, Sweden*, Switzerland and the United Kingdom.

Use the **high risk charts** in other European countries. Of these, some are at **very high risk** and the charts may underestimate risk in these. These include Albania, Algeria, Armenia, Azerbaijan, Belarus, Bulgaria, Egypt, Georgia, Kazakhstan, Kyrgyzstan, Latvia, FYR Macedonia, Moldova, Russian Federation, Syrian Arab Republic, Tajikistan, Turkmenistan, Ukraine and Uzbekistan.

*Updated, recalibrated charts are now available for Belgium, Germany, Greece, The Netherlands, Spain, Sweden and Poland.

2. Find the cell nearest to the person's age, cholesterol and BP values, bearing in mind that risk will be higher as the person approaches the next age, cholesterol or BP category.

3. Check the qualifiers

4. Establish the total 10 year risk for fatal CVD.

Relative Risk Charts

Note that a low total cardiovascular risk in a young person may conceal a high relative risk; this may be explained to the person by using the relative risk chart. As the person ages, a high relative risk will translate into a high total risk. More intensive lifestyle advice will be needed in such persons. This chart refers to relative risk, not percentage risk, so that a person in the top right corner is at 12 times higher risk than a person in the bottom left corner.

Another approach to explaining risk to younger persons is to use cardiovascular risk age. For example, in the high risk chart, a 40 year old male hypertensive smoker has a risk of 4%, which is the same as a 65 year old with no risk factors, so that his risk age is 65. This can be reduced by reducing his risk factors.

Systolic Blood Pressure (mmHg)	Non-Smoker					Smoker				
	4	5	6	7	8	4	5	6	7	8
180	3	3	4	5	6	6	7	8	10	12
160	2	3	3	4	4	4	5	6	7	8
140	1	2	2	2	3	3	3	4	5	6
120	1	1	1	2	2	2	2	3	3	4

Cholesterol (mmol/L)

©ESC 2018

Risk estimation using SCORE: Qualifiers

• The charts should be used in the light of the clinician's knowledge and judgement, especially with regard to local conditions.

• As with all risk estimation systems, risk will be over-estimated in countries with a falling CVD mortality rate, and under estimated if it is rising.

• At any given age, risk appears lower for women than men. However, inspection of the charts shows that their risk is merely deferred by 10 years, with a 60 year old woman resembling a 50 year old man in terms of risk.

• Risk may be higher than indicated in the chart in:

- Sedentary or obese subjects, especially those with central obesity
- Those with a strong family history of premature CVD
- Socially deprived individuals and those from some ethnic minorities
- Individuals with diabetes- the SCORE charts should only be used in those with type 1 diabetes without target-organ damage; Other diabetic subjects are already at high to very high risk.
- Those with low HDL cholesterol* or increased triglyceride, fibrinogen, apoB, Lp(a) levels and perhaps increased high-sensitivity CRP.
- Asymptomatic subjects with evidence of pre-clinical atherosclerosis, for example plaque on ultrasonography.
- Those with moderate to severe chronic kidney disease (GFR <60 mL/min/1.73 m²)

*Note that HDL cholesterol impacts on risk in both sexes, at all ages, and at all level of risk. This effect can be estimated using the electronic version of SCORE, HeartScore, which has been updated to include HDL cholesterol level.

Visit www.heartscore.org
For the interactive version of the SCORE risk charts

Source: European Guidelines on CVD Prevention in Clinical Practice 2016
Eur J Prev Cardiol. 2016 Jul;23(11):NP1-NP96. doi:10.1177/2047487316653709



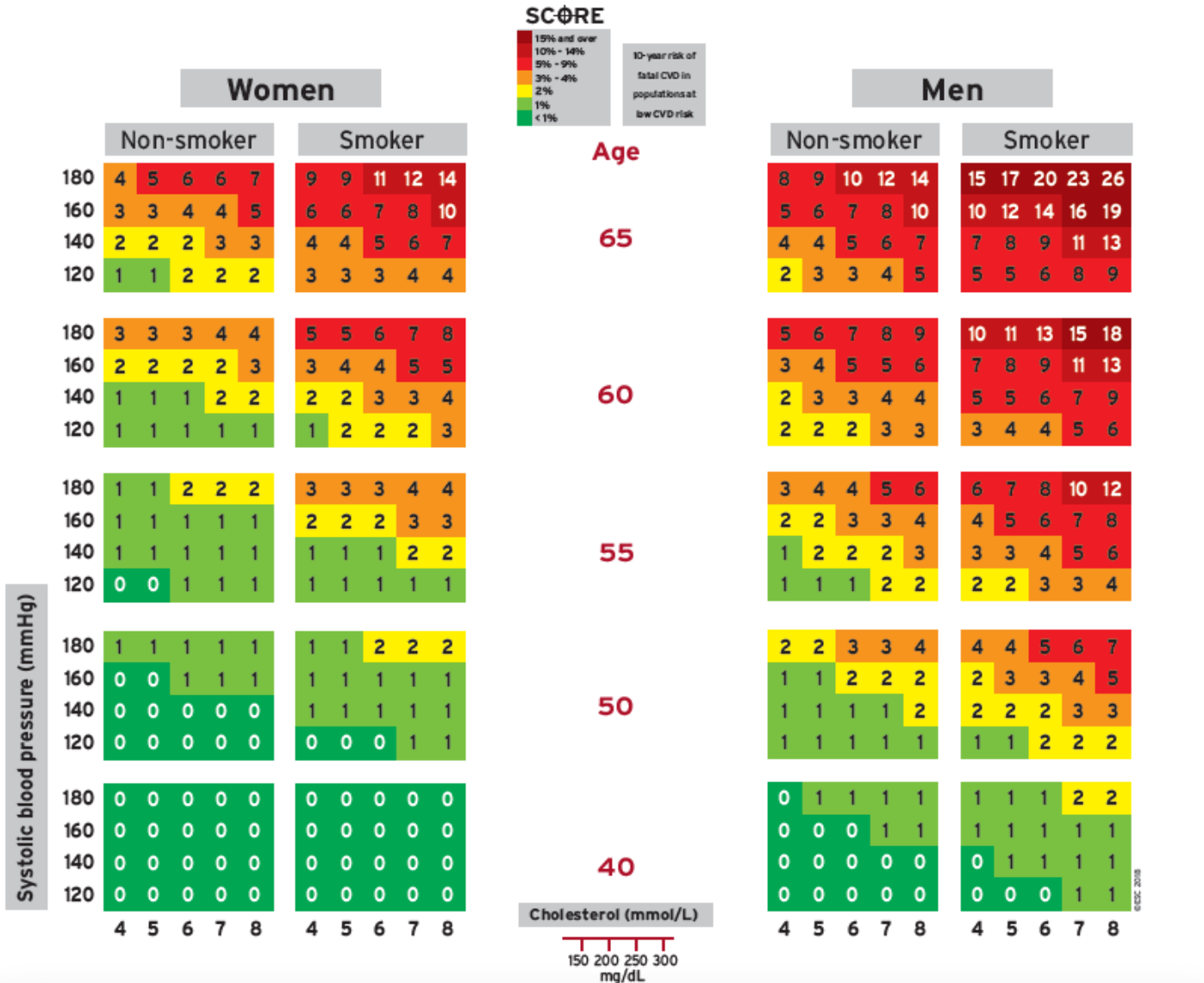
EAPC
European Association
of Preventive Cardiology

European Society of Cardiology



SCORE - European Low Risk Chart

10 year risk of fatal CVD in low risk regions of Europe by gender, age, systolic blood pressure, total cholesterol and smoking status



How do I use the SCORE charts to assess CVD risk in asymptomatic persons?

1. Use the **low risk charts** in Andorra, Austria, Belgium*, Cyprus, Denmark, Finland, France, Germany, Greece*, Iceland, Ireland, Israel, Italy, Luxembourg, Malta, Monaco, The Netherlands*, Norway, Portugal, San Marino, Slovenia, Spain*, Sweden*, Switzerland and the United Kingdom.

Use the **high risk charts** in other European countries. Of these, some are at **very high risk** and the charts may underestimate risk in these. These include Albania, Algeria, Armenia, Azerbaijan, Belarus, Bulgaria, Egypt, Georgia, Kazakhstan, Kyrgyzstan, Latvia, FYR Macedonia, Moldova, Russian Federation, Syrian Arab Republic, Tajikistan, Turkmenistan, Ukraine and Uzbekistan.

*Updated, recalibrated charts are now available for Belgium, Germany, Greece, The Netherlands, Spain, Sweden and Poland.

2. Find the cell nearest to the person's age, cholesterol and BP values, bearing in mind that risk will be higher as the person approaches the next age, cholesterol or BP category.

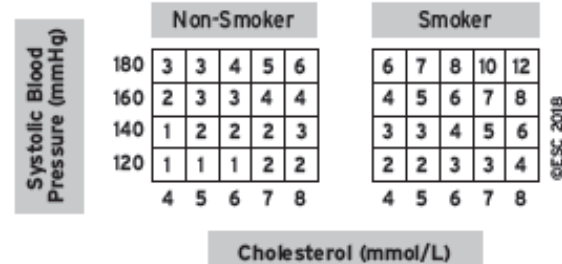
3. Check the qualifiers

4. Establish the total 10 year risk for fatal CVD.

Relative Risk Charts

Note that a low total cardiovascular risk in a young person may conceal a high relative risk; this may be explained to the person by using the relative risk chart. As the person ages, a high relative risk will translate into a high total risk. More intensive lifestyle advice will be needed in such persons. This chart refers to relative risk, not percentage risk, so that a person in the top right corner is at 12 times higher risk than a person in the bottom left corner.

Another approach to explaining risk to younger persons is to use cardiovascular risk age. For example, in the high risk chart, a 40 year old male hypertensive smoker has a risk of 4%, which is the same as a 65 year old with no risk factors, so that his risk age is 65. This can be reduced by reducing his risk factors.



Risk estimation using SCORE: Qualifiers

• The charts should be used in the light of the clinician's knowledge and judgement, especially with regard to local conditions.

• As with all risk estimation systems, risk will be over-estimated in countries with a falling CVD mortality rate, and under estimated if it is rising.

• At any given age, risk appears lower for women than men. However, inspection of the charts shows that their risk is merely deferred by 10 years, with a 60 year old woman resembling a 50 year old man in terms of risk.

• Risk may be higher than indicated in the chart in:

- Sedentary or obese subjects, especially those with central obesity
- Those with a strong family history of premature CVD
- Socially deprived individuals and those from some ethnic minorities
- Individuals with diabetes- the SCORE charts should only be used in those with type 1 diabetes without target-organ damage; Other diabetic subjects are already at high to very high risk
- Those with low HDL cholesterol* or increased triglyceride, fibrinogen, apoB, Lp(a) levels and perhaps increased high-sensitivity CRP.
- Asymptomatic subjects with evidence of pre-clinical atherosclerosis, for example plaque on ultrasonography.
- Those with moderate to severe chronic kidney disease (GFR <60 mL/min/1.73 m²)

*Note that HDL cholesterol impacts on risk in both sexes, at all ages, and at all level of risk. This effect can be estimated using the electronic version of SCORE, HeartScore, which has been updated to include HDL cholesterol level.

Visit www.heartscore.org
For the interactive version of the SCORE risk charts

Source: European Guidelines on CVD Prevention in Clinical Practice 2016
Eur J Prev Cardiol. 2016 Jul;23(11):NP1-NP96. doi:10.1177/2047487316653709



EAPC
European Association
of Preventive Cardiology

 European Society of Cardiology



TREATMENT OF DYSLIPIDEMIA

The first step is the **PHASE I DIET**, which is intended to reduce the contributions of cholesterol and saturated fat.

If, after 3 months, the target is not reached, the **PHASE 2 DIET** (strict) is administered for the next three months.

If, after 6 months of diet, the patient has not reached the target, **DRUG TREATMENT** is considered.



EFFECT OF DIET ON PLASMA LIPIDS

	LDL-Cholesterol	HDL-Cholesterol	VLDL-Cholesterol
Unfavourable			
SFA	↑	-	-
MUFA <i>trans</i>	↑	↓	-
Cholesterol	↑	↓	-
CH without fibre	↑	↓	-
Beneficial			
MUFA <i>cis</i>	↓	↑	-
Soluble fiber	↓	-	-
Beneficial with possibles unfavourable effects			
AGP n-6	↓	↓	-
AGP n-3	↑	-	↓

Omega-6 to Omega-3 Ratio
 Disease Prevention
 2-4 to 1
 Western Diet
 15-16.7 to 1



Nutrition therapy for dyslipidemia

- ✓ National guidelines indicate that patients with elevated LDL cholesterol should consume **less than 7% of calories from saturated fat and less than 200 mg of cholesterol**.
- ✓ **Trans fatty acids should also be limited.**
- ✓ The incorporation of **functional foods**, such as **stanol-containing** margarine, soy products, and soluble fibre-rich cereals and vegetables, can provide further benefit.
- ✓ In addition to weight loss and **physical activity**, individuals with hypertriglyceridemia benefit from a **diet that is moderate in fat and CH** rather than a low-fat diet.
- ✓ Including **monounsaturated or omega-3 fatty acids** lowers serum triglycerides.
- ✓ Many of the dietary strategies for minimizing serum lipids also contribute to glycemic control in patients with diabetes mellitus.



<https://link.springer.com/content/pdf/10.1007/s11892-003-0084-z.pdf>



Table 2. Approximate LDL cholesterol reduction achievable by dietary modification

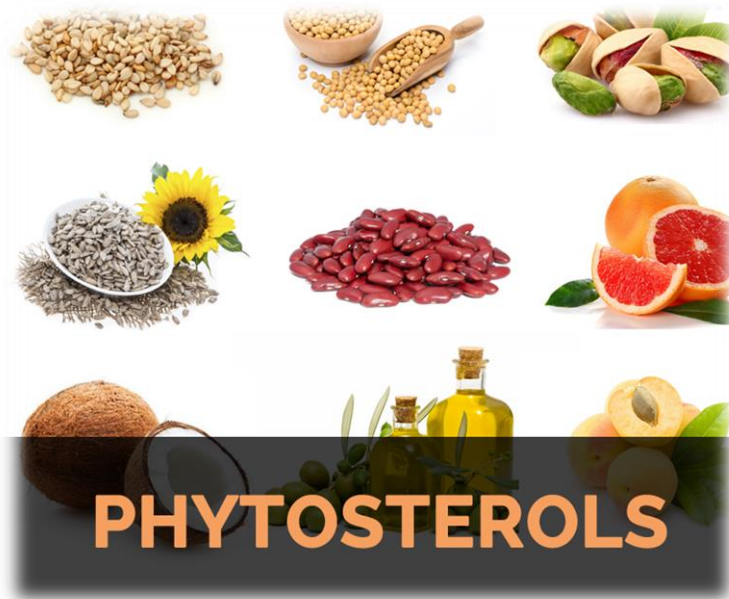
Dietary component	Dietary change	Approximate LDL reduction, %
Saturated fat	< 7% of calories	8–10
Dietary cholesterol	< 200 mg/d	3–5
Weight reduction	Lose 10 lb	5–8
Soluble fiber	5–10 g/d	3–5
Plant sterol or stanol esters	2 g/d	6–15
Cumulative estimate	—	20–30

LDL—low-density lipoprotein.
(Adapted from the National Cholesterol Education Program, Third Adult Treatment Panel; http://www.nhlbi.nih.gov/guidelines/cholesterol/atp3_rpt.htm [V-32].)

<https://link.springer.com/content/pdf/10.1007/s11892-003-0084-z.pdf>



- ✓ Plant sterols are found in plants **vegetables, fruits, wheat germ, whole grains, beans, sunflower seeds, and many vegetable oils.**
- ✓ Plant sterols **can help lower your LDL-C** or ‘bad’ cholesterol. A high LDL-C can increase your risk of heart disease.



RECOMMENDATIONS (USA) FOR DYSLIPIDEMIA IN THE GENERAL POPULATION:

- <30% of total energy as fat
- <10% of total energy of SFA → ↑ chol t, HDL-col.
- <10% of total energy as PUFA → ↓ chol t,
↑oxidative potential
- 10-15% of total energy as MUFA (oleic acid) → ↑ HDL- col, ↓col
total when replaced
- Cholesterol <300 mg/day or, better, 100 mg/1000 kcal (in diets of
2500 kcal)



RECOMMENDED DAILY INTAKE IN DYSLIPIDEMIC DIETS

Up to 50 g of oil, preferably
extra virgin olive oil.

2-6 rations of starch (integral
derivatives are better).

Take care with products
made with unknown fats.

2-3 servings of milk and
low-fat dairy products.

2-4 servings of all
kinds of fruit in any form.
Use nuts to replace other
fatty foods.

2-4 servings of vegetables
(raw and cooked).

- 2 servings of protein foods of animal origin.
- Exceptionally, red meat without visible fat, duck, goose, sausages, fatty meats and viscera.
 - Moderate consumption of fat-free pork and lean meats, crustaceans, cephalopods, roe and egg yolks (no restrictions on the egg white).

Limit alcohol to 30 g/day
and avoid it if the patient
is overweight and/or
suffers from
hypertriglyceridemia.

VERY MODERATE DIETARY RESTRICTIONS

Children and adolescents:

- Guide them towards a healthier lifestyle.
- Do not create a negative attitude to food.

The elderly:

To promote compliance with prescriptions and treatments, these should be adapted to the habits and circumstances of each patient. Dietary history should be taken to ascertain information about the patient's:

- Physical activity
- Weight
- Smoking habits
- Alcohol intake (<30g/day or eliminate)

If HBP (high blood pressure), decrease foods that are high in salt and Na.

If glucose intolerance, reduce sugar and calories.

Encourage the habitual consumption of different foods.

groups.



REMEMBER!

Foods that lower cholesterol:

- ✓ Oats
- ✓ Barley and other whole grains
- ✓ Beans
- ✓ Nuts
- ✓ Vegetable oils
- ✓ Apples, grapes, strawberries, citrus fruits
- ✓ Foods fortified with sterols and stanols.



FOODS THAT WILL LOWER
CHOLESTEROL

**Fatty fish
(salmon, tuna)**



Olive Oil



Whole grains



Berries



Avocados



Beans



Nuts



**Dark Chocolate
(in moderation)**



Spinach



**Red wine
(in moderation)**



Green and black teas



Soy



Citrus fruit



Vegetables



Remember → Daily exercise !!



DIET

EXERCISE



References

- Nutrición y dietética clínica. Jordi Salas-Salvadó, Anna Bonada i Sanjaume, Roser Trallero Casañas, M^a Engracia Saló i Solà, Rosa Burgos Peláez. Elsevier Masson. 2008.
- Tratado de Nutrición. Gil Hernández, A. (ed). 2005
- OMS. Dieta, nutrición y prevención de enfermedades crónicas. Informe de una Consulta Mixta de Expertos OMS/FAO. Organización Mundial de la Salud. Ginebra. 2003
- Nutrición y Dietoterapia de Krause. Mahan, L.D. and Escott-Stump, S. (10^a Edición). McGraw-Hill Interamericana, México. 2000
- NCEP report. Implications of Recent Clinical Trials for the National Cholesterol Education Program Adult Treatment Panel III Guidelines. Circulation. 2004;110:227-239.
- Tratamiento de las dislipemias en atención primaria. M.A. Fernández Calvo, O. Fernández Calvo, G. Charlín Pato. FAP, 4 (3) 2006.
- <http://www.san.gva.es/docs/dac/guiasap013dislipemias.pdf>
- <http://www.bedca.net/>



Thank You
For Your Attention...



YOU ARE
AWESOME!

SEE YOU
SOON

GOOD LUCK!



Pilar.vila@uv.es

