PRINCIPALS OF RECOMMENDED NUTRITION

- Quantitative aspect
- Qualitative aspect
- Special components of diet
- Aesthetic aspect
- Socio-economic aspect

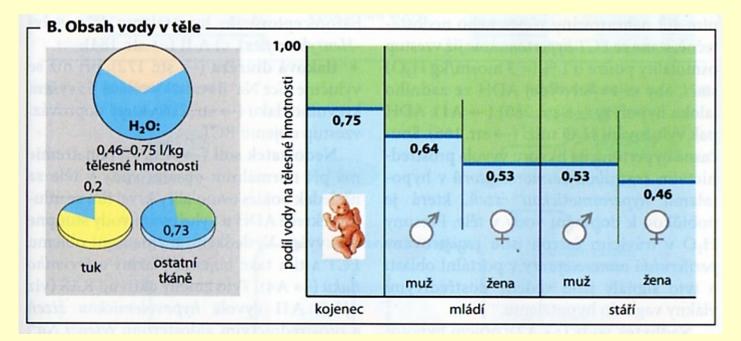
WATER, VITAMINS, MINERALS IN NUTRITION

WATER

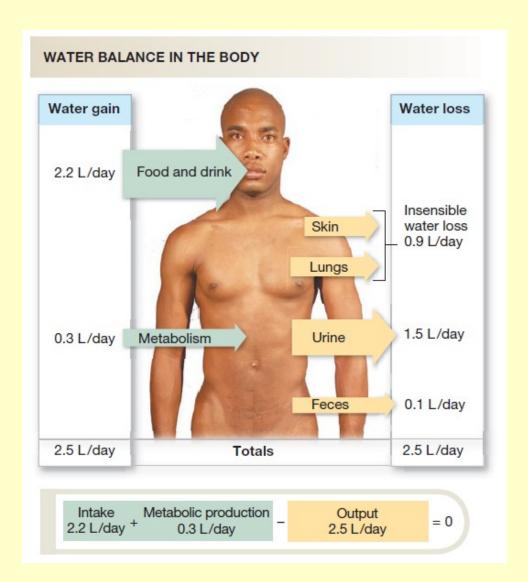
- 50-70% of body mass, newborns
- 2/3 intracellularly, 1/3 extracellularly
- metabolism
- compartmentalisation
- phylogenetic view

Water and its functions in the human body

- The transport medium, solvent, wetting and protection of the mucous membranes
- Age, sex, weight



Maintains Optimal And Keeps Mucosal Membranes Stable Heating & Cooling From Drying Out (Eyes, Mouth, etc.) Facilitates Blood Flow. Cellular Reproduction, Comprises At Least Movement & Life Itself 3/4 of Total Body Mass & Substance Supports The Efficient Removal of Toxins & Waste From Internal Organs Maintains Optimal Digestive Function & Elimination Permits the Absorption Primary Conduit For Delivering of Life-Essential All Body Fluids, Molecular Messages **Nutrients & Energy** And Especially Oxygen Delivery Without Water, Cells Cannot Grow, The Body Can Survive For Weeks Reproduce or Survive, and the Without Food, But Only **Entire Organism Dies** A Few Days Without Water

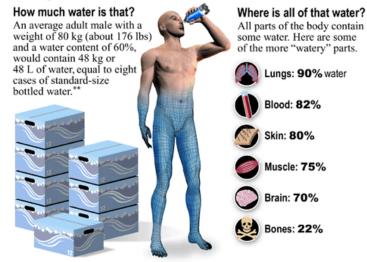


The water content in different tissues (male, 70 kg)

	% of water
blood	83%
muscle tissue	76%
skin	72%
bones	22%
fats	10%
tooth enamel	2%

Your very own body of water

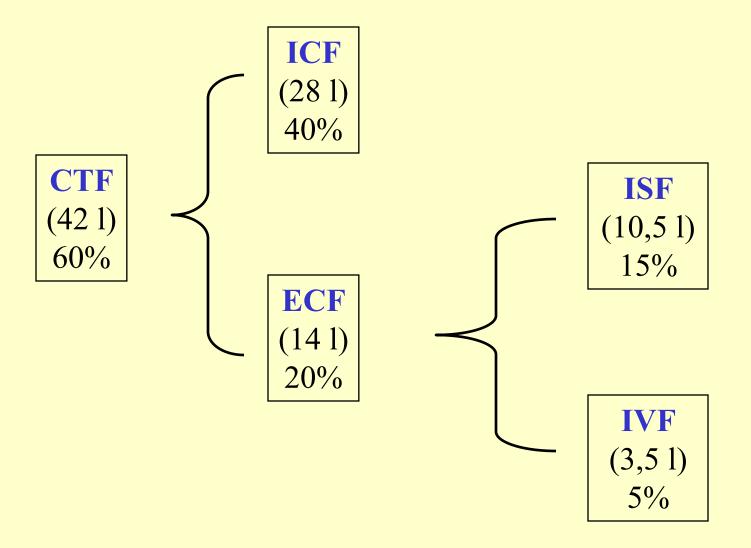
The average human body is composed of about 55% water. The average adult male is about 60% water, the average adult female about 50% water.*



* Muscle contains more water than fat does. Males generally have higher muscle content than females.

** 1 litre of water weighs 1 kilogram. A standard size container of bottled water is 500 mL.

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Clinical examination: evaluation of extracellular (plasmatic) levels of electrolytes (Na, K)

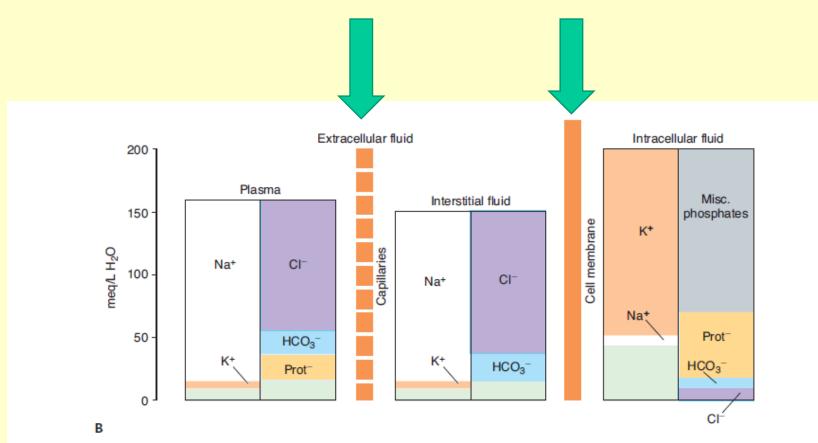
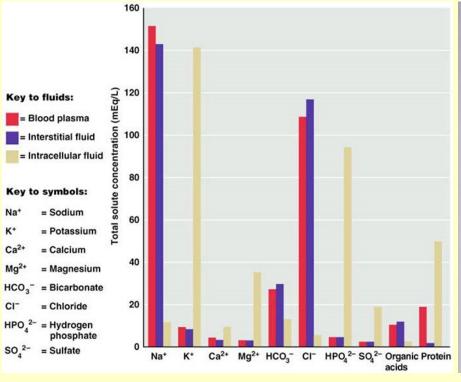
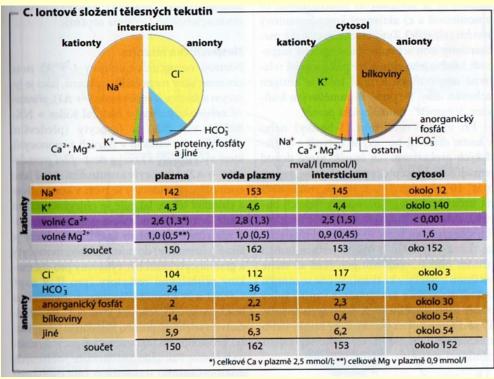
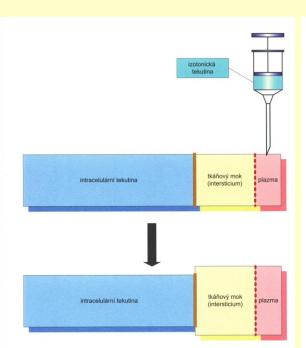


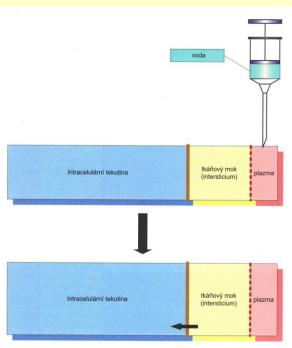
FIGURE 1–1 Organization of body fluids and electrolytes into compartments. **A)** Body fluids are divided into Intracellular and extracellular fluid compartments (ICF and ECF, respectively). Their contribution to percentage body weight (based on a healthy young adult male; slight variations exist with age and gender) emphasizes the dominance of fluid makeup of the body. Transcellular fluids, which constitute a very small percentage of total body fluids, are not shown. Arrows represent fluid movement between compartments. **B)** Electrolytes and proteins are unequally distributed among the body fluids. This uneven distribution is crucial to physiology. Prot⁻, protein, which tends to have a negative charge at physiologic pH.



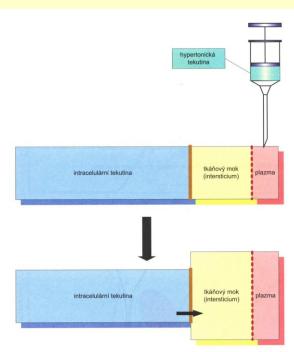




Obr. 8.42 Při příjmu izotonické tekutiny se tekutina rozprostře mezi intravaskulární a extravaskulární část extracelulárního kompartmentu, do intravaskulárního kompartmentu tekutina nepřechází, protože bariéra je pro ionty nepropustná, a voda nepřechází, protože obě strany bariéry jsou izotonické



Obr. 8.43 Při příjmu čisté vody se voda rozprostře do všech kompartmentů, aby vyrovnala jejich osmolaritu



Obr. 8.44 Při příjmu hypertonické tekutiny přechází voda do extracelulárního prostoru z prostoru intracelulárního, aby vyrovnala osmolaritu obou kompartmentů

t a b l e **5-1** Body Water and Body Fluid Compartments

Body Fluid Compartment	Fraction of TBW*	Markers Used to Measure Volume	Major Cations	Major Anions
TBW	1.0	Tritiated H ₂ 0 D ₂ 0 Antipyrene		
ECF	1/3	Sulfate Inulin Mannitol	Na ⁺	CI ⁻
Plasma	1/12 (1/4 of ECF)	RISA Evans blue	Na ⁺	CI ⁻ HCO ₃ - Plasma protein
Interstitial	1/4 (3/4 of ECF)	ECF-plasma volume (indirect)	Na ⁺	CI ⁻ HCO ₃ -
ICF	2/3	TBW-ECF (indirect)	K ⁺	Organic phosphates Protein

^{*}Total body water (TBW) is approximately 60% of total body weight, or 42 L in a 70-kg man. ECF = extracellular fluid; ICF = intracellular fluid; RISA = radioiodinated serum albumin.

HOMEOSTASIS

- •Izoionia concentration of ions
- •**Izotonia** osmotic concentration
- •Izohydria ratio between acids and bases
- •Izovolemia –ECL volume (volumoreceptors or baroreceptors,

RAS, ADH)

Izovolemia

Cause – result

Hypovolemia (dehydratation)

Complex disorders!

• Hypervolemia (hyperhydratation)

EXAMINATIONS AT HYDRATATION DISORDERS

- 1. Anamnesis diseases of kidneys, GIT, DM, DI, drugs, intake and output=balance, body mass changes, etc.
- 2. Laboratory examinations: electrolytes, blood osmolality, RBCC, total plasmatic proteins; Astrup examination

OBJECTIVE EXAMINATIONS

- 1. Skin changes
- 2. Body mass changes
- 3. Diuresis changes (oliguria, anuria, polyuria)
- 4. Respiration disorders (respiratory acidosis, alkalosis; secondary changes Kussmaul breathing)
- 5. CNS disorders (changes of reflexes, muscle tonus, paresthesias, changes of consciousness, coma)
- 6. Central venous pressure changes (filling of neck veins)
- Circulation changes: dehydratation tachycardia,
 hypotonia

CAUSES OF HYDRATATION DISORDERS

- 1. Disturbance of normal intake of water and ions
- 2. Disturbance of normal circulation of water and ionts between ECL and GIT
- 3. Disturbance of cell metabolism
- 4. Disturbance of loss of water and ions
- 5. Excessive loss of water (and ions) by skin

DEHYDRATATION

= decreased volume of body fluids accompanied by lack of sodium

HYPERTONIC DEHYDRATATION = loss of (only) water

Bigger lack of water than sodium. Disorders of intake and big losses.

Cell dehydratation.

Thirst. Decreased skin turgor. CNS symptoms.

Hydratation.

IZOTONIC DEHYDRATATION = isonatremic

Causes – bleeding, diuretics, "blind spaces"

Hypovolemic syndrome: decreased diuresis, symptoms of dehydratation.

HYPOTONIC DEHYDRATATION

Always bigger deficiency of sodium than water.

Cell hyperhydratation.

Losses by GIT, kidneys.

Hypovolemic syndrome, CNS symptoms.

HOMEOSTATIC COMPENSATION FOR SEVERE DEHYDRATION DEHYDRATION ◆ Blood volume/ accompanied by Osmolarity ◆ Blood pressure CARDIOVASCULAR RENIN-ANGIOTENSIN RENAL HYPOTHALAMIC MECHANISMS SYSTEM MECHANISMS MECHANISMS Hypothalamic osmoreceptors + Atrial volume Carotid and aortic receptors; Carotid baroreceptors and aortic ♦ Flow at Granular **♦** GFR baroreceptors macula densa cells CVCC Ŧ Hypothalamus Volume conserved Renin Angiotensinogen **↑** ANG I (+) ↑ Vasopressin Parasympathetic ♠ Sympathetic release from output output posterior pituitary ACE $_{ullet}$ \oplus **(+)** (+) Heart Arterioles **↑** ANG II Thirst ⊕∤ osmolarity inhibits Adrenal Vasoconstriction cortex **▲** Force ▲ Rate **♦** Aldosterone ♠ Peripheral resistance Distal Distal nephron nephron ♦ Na⁺ reabsorption **♦** Blood ↑ H₂O † H₂O ↑ Cardiac and ↑ Volume pressure reabsorption intake output **♦** Osmolarity

HYPERHYDRATATION

= increased volume of extracellular fluid

HYPOTONIC HYPERHYDRATATION – water intoxication

Cell hyperhydratation. Decreased osmolality.

Excessive intake of liquids (dialysed patient, patient with kidney disorders), hyperproduction of ADH

IZOTONIC HYPERHYDRATATION

Increased volume of ECF. Osmolality stabile.

Heart failure, nefrotic syndrome, liver cirrhosis.

Oedemas and water withholding in serose cavities.

HYPERTONIC HYPERHYDRATATION = hypernatremic

Rare. Increase of ECF caused by sodium abundance. Osmolality increases.

Primary hyperaldosteronism.

Туре	Key Examples	ECF Volume	ICF Volume	ECF Osmolarity	Hct and Serum [Na+]
Isosmotic volume expansion	Isotonic NaCl infusion	1	No change	No change	↓ Hct –[Na+]
Isosmotic volume contraction	Diarrhea	\	No change	No change	↑ Hct –[Na+]
Hyperosmotic volume expansion	High NaCl intake	1	\downarrow	↑	↓ Hct ↑ [Na+]
Hyperosmotic volume contraction	Sweating Fever Diabetes insipidus	1	↓	↑	–Hct ↑ [Na+]
Hyposmotic volume expansion	SIADH	1	↑	\	–Hct ↓[Na+]
Hyposmotic volume contraction	Adrenal insufficiency	\	↑	\downarrow	↑ Hct ↓[Na+]

 $⁻⁼ no\ change;\ ECF = extracellular\ fluid;\ Hct = hematocrit;\ ICF = intracellular\ fluid;\ SIADH = syndrome\ of\ inappropriate\ antidiuretic\ hormone.$

SIADH = syndrome of inappropriate antidiuretic hormone secretion)

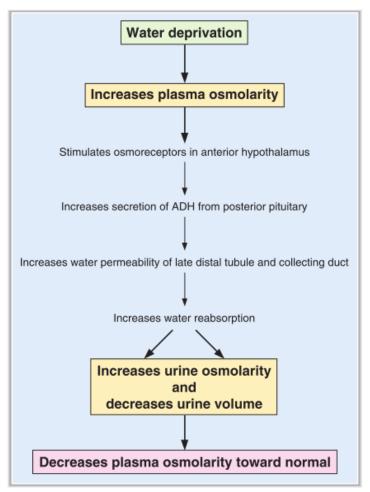


FIGURE 5-14 Responses to water deprivation. ADH = antidiuretic hormone.

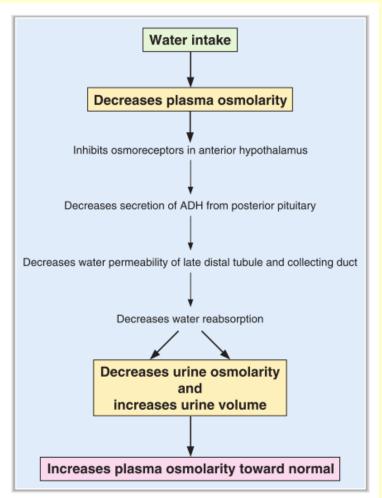


FIGURE 5-15 Responses to water intake. ADH = antidiuretic hormone.

VITAMINS

= all organic compounds of diet, necessary for life, health and growth; NO source of energy

HYPOVITAMINOSIS (AVITAMINOSIS) HYPERVITAMINOSIS

- 1. Decrease supply in diet
- 2. Food intake disorders
- 3. Absorption disorders
- 4. Increased consumption
- 5. Store organ diseases

1. Increased supply in diet – usually **iatrogenic**

✓ in water: diffusion, D, J; vit.B₁₂ - I

SOLUBLE

in lipids: deficient absorption in disorders of lipids absorption (pancreatic enzymes or bile missing)

Vitamin	Place of absorption	Transport mechanism	Maximal absorption capacity in humans / day	Daily dose
С	Ileum	Active	>5000mg	<50mg
Biotin	Small intestine	Active	?	?
Cholin	Small intestine	Facilitated diffusion	?	?
Folic acid (pteroylglutamate)	Jejunum	Facilitated diffusion	> 1000μg (dose)	100-200μg
Folic acid (5- methyltetrahydrofolate)	Jejunum	Diffusion	> 1000μg (dose)	100-200μg
Nicotinic acid	Jejunum	Facilitated diffusion	?	10-20mg
Pantothenic acid	Small intestine	?	?	(?)10mg
B ₆ (pyridoxine)	Small intestine	Diffusion	> 50mg (dose)	1-2mg
B ₂ (riboflavin)	Jejunum	Facilitated diffusion	10-12mg (dose)	1-2mg
B ₁ (thiamine)	Jejunum	Active	8-14mg	Approx. 1mg
B ₁₂	Distal ileum	Active	6-9µg	3-7μg

VITAMIN B₁₂

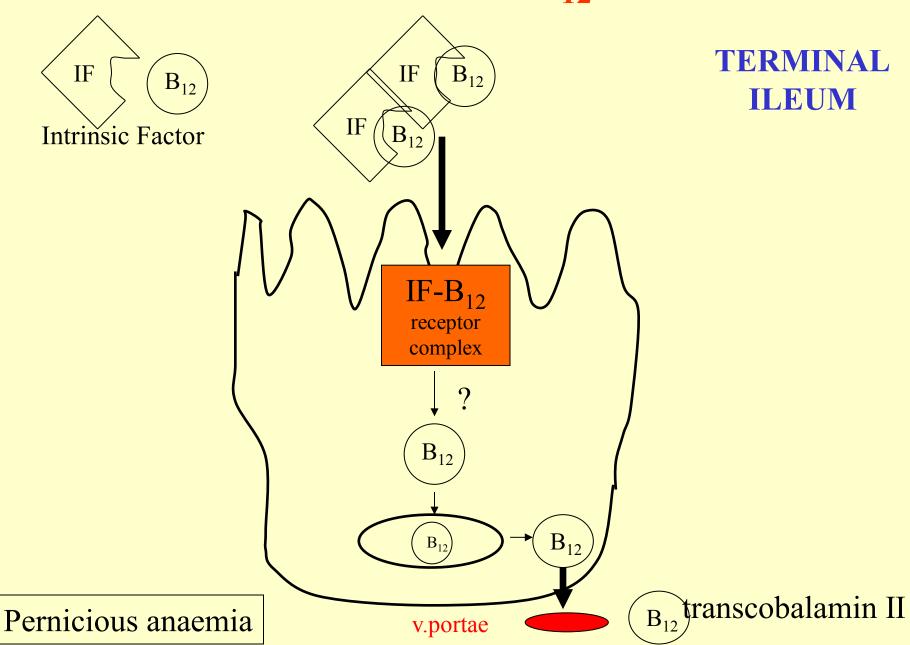
- Daily dose is close to absorption capacity
- •Synthesised by bacteria in colon BUT there is not absorption mechanism
- •Store in liver (2-5mg)
- •In bile 0,5-5μg / day, reabsorbed
- •Daily loss -0.1% of stores \longrightarrow stores will last for 3-6 years

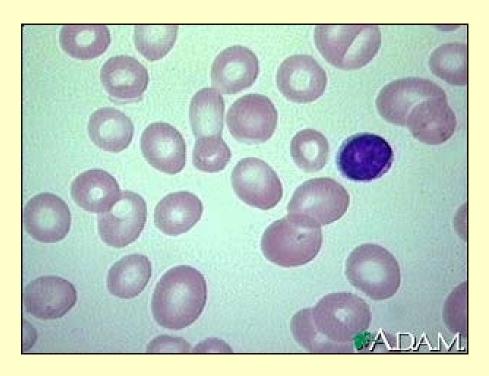
ABSORPTION

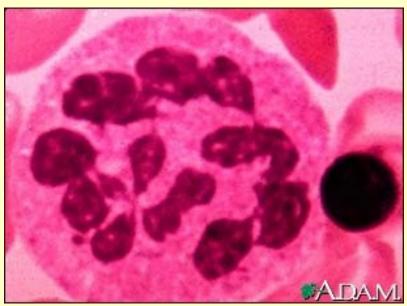
- 1. Gastric phase: B₁₂ is bound to proteins, low pH and pepsin release it; bound to glycoproteins R-proteins (saliva, gastric juice), almost pH-undependable; intrinsic factor (IF) parietal cells of gastric mucosa; most of vitamin bound to R-proteins
- 2. Intestinal phase: pancreatic proteases, cleavage of $R-B_{12}$, bound to IF (resistant to pancreatic proteases)

COBALAMIN HANDLING BY THE STOMACH AND PROXIMAL SMALL INTESTINE Food Cobalamin is The acid pH and CBL Cobalamin bound to pepsin release D COBALAMIN ABSORPTION BY ILEAL ENTEROCYTE cobalamin from proteins in food. lleal enterocyte dietary protein. Intestinal lumen Interstitial space Acid pH Gastric glands secrete Pepsin Endosome CBL Gastric parietal haptocorrin, which then cells secrete IF. binds to cobalamin. (CBL) CBL CBL IF-CBL 学师 Trans-Bile cobalamin II duct • CBL Receptor Secretory vesicle Deglycosylation? and degradation of IF Haptocorrin Pancreatic proteases TCIL TCIL CBL The pancreas secretes proteases and HCO1 (alkaline secretion). Degraded CBL CBL is released after the CBL receptor? proteolytic degradation of haptocorrin. Lysosome CBL)+ IF IF-CBL The IF-CBL complex forms. 3-4 hours Ileal enterocyte absorbs IF-CBL complex.

ABSORPTION OF B₁₂ VITAMIN







HYPOVITAMINOSES

Folic acid – disorders of embryo development (clefts)

B₁₂ – pernicious anaemia

C – scurvy (scorbutus)

D – rickets (rhachitis, English disease, English sickness)

E – fertility problems

K - haemorrhage

HYPERVITAMINOSES

A – teratogenic effects

D – kidney failure

K – anaemia, GIT disorders

B₆ – peripheral polyneuropathy

BERI-BERI (B₁)

"The first clinical descriptions of beriberi were by Dutch physicians, Bontius (1642) and Nicolaas Tulp (1652). Tulp treated a young Dutchman who was brought back to Holland from the East Indies suffering from what the natives of the Indies called beriberi or "the lameness." Tulp's description of beriberi was a detailed one, but he had no clues that it was a dietary deficiency disease. This discovery came more than two hundred years later. Nicholaas Tulp (1593-1674) is best remembered as the central figure in Rembrandt's famous painting, "The Anatomy Lesson" (1632).

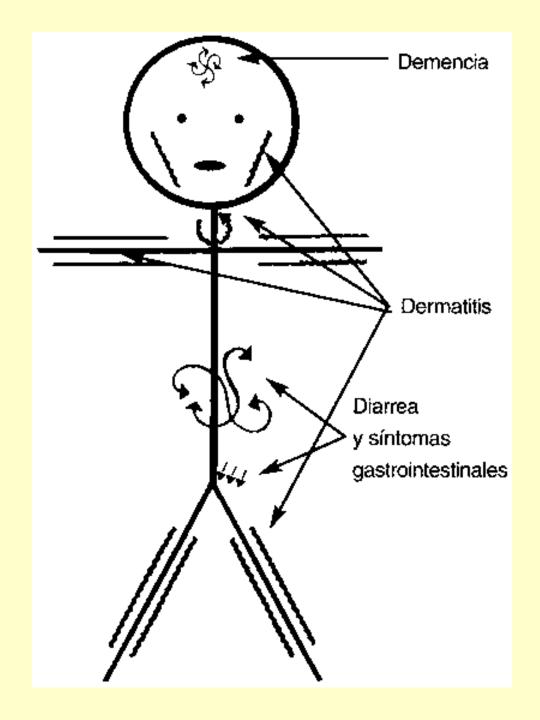


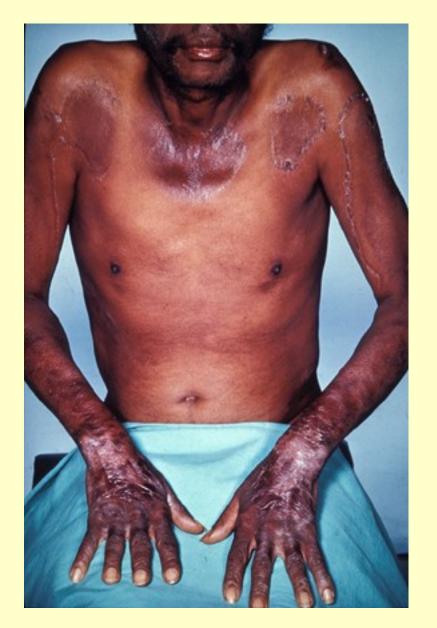
I.UGDUNI BATAVORUM apud GEORGIUM WISHOFF.

M.D.CC XXXVIIII

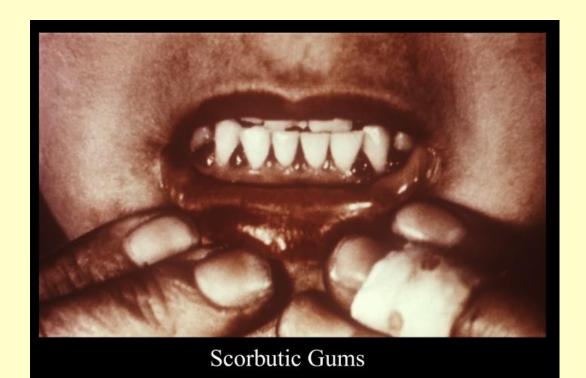
PELAGRA

(3 D disease) (niacin)





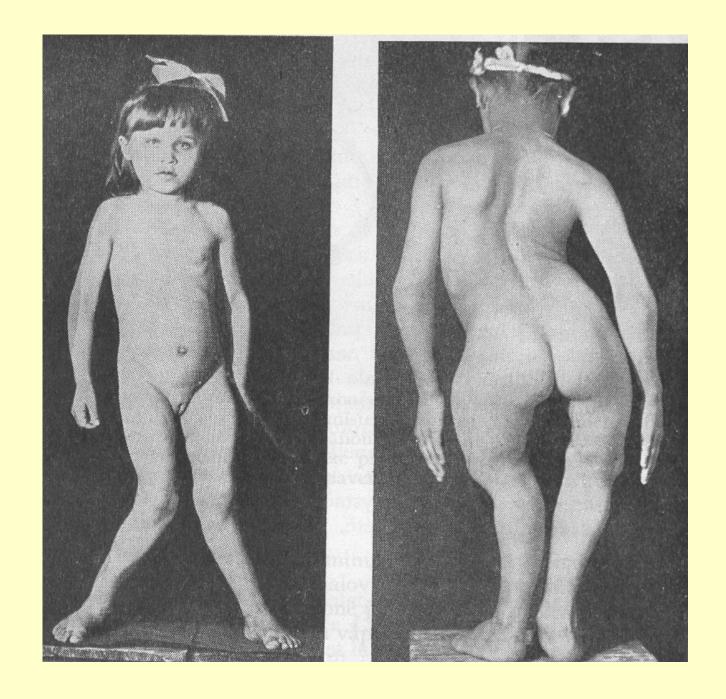




SCURVY



RICKETS



Mineral	Daily need (dose)
Na	3,0 g
K	1,0 g
C1	3,5 g
Ca	1,2 g
P	1,2 g
Fe	18,0 mg
J	150,0 μg
Mg	0,4 g
Со	?
Cu	?
Mn	?
Zn	15 mg

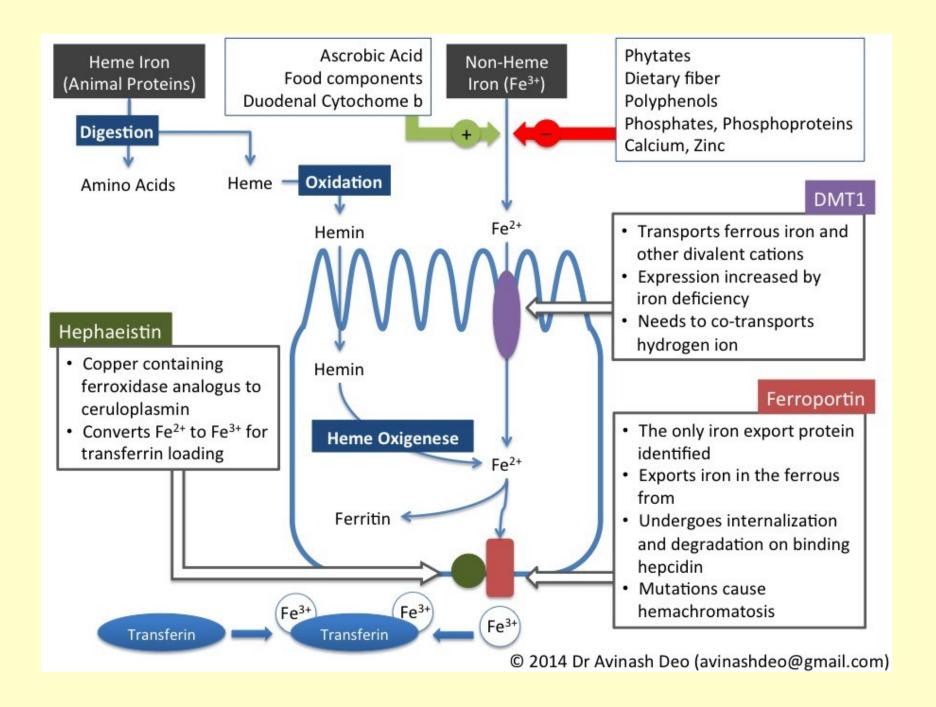
Coenzyme of metabolic reactions of saccharides; deficiency – increased irritability of CNS, peripheral vasodilatation, arrhythmias; excess – suppresses electrical activity of CNS and skeletal muscle

Part of enzymes (carboanhydrase in erythrocytes, lactatedehydrogenase, peptidases)

MINERALS AND TRACE ELEMENTS

- 1. Arsenic
- 2. Chrome experimental deficiency, glucose oral test is of diabetic character
- 3. Cobalt part of enzymes, vit.B₁₂; poisoning by cobalt (beer), cobalt cardiomyopathy
- 4. Copper impairment of cytochromoxidase (experiment), melanoma increase of radiosensitivity when copper is depleted; vessel wall damage
- 5. Fluorine
- 6. Iodine
- 7. Iron
- 8. Manganese catalyses similar reactions as Mg, stored in mitochondria, β1-globulintransmanganin
- 9. Molybdenum in xantinoxidase and flavoproteins, defficiency in humans???
- 10. Nickell
- 11. Selenium antioxidant, in diet bound to proteins (alcoholism, liver cirrhosis)
- 12. Silicon
- 13. Vanadium
- 14. Zinc part of metalloenzymes, proteosynthesis (ribosomes); deficiency-Middle East (parasites, fytates in diet); testes atrophy, immune disorders; in DM 50% of stores Zn (insulin stored in pancreas together with Zn)





Iron: Factors Affecting Absorption

Physical State (bioavailability)	heme > Fe ²⁺ > Fe ³⁺
Inhibitors	phytates, tannins, soil/clay (pica), laundry starch, iron overload, antacids
Competitors	lead, cobalt, strontium, manganese, zinc
Facilitators	ascorbate, citrate, amino acids, iron deficiency, stomach acid, high altitude, exercise, pregnancy