PRINCIPALS OF RECOMMENDED NUTRITION

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

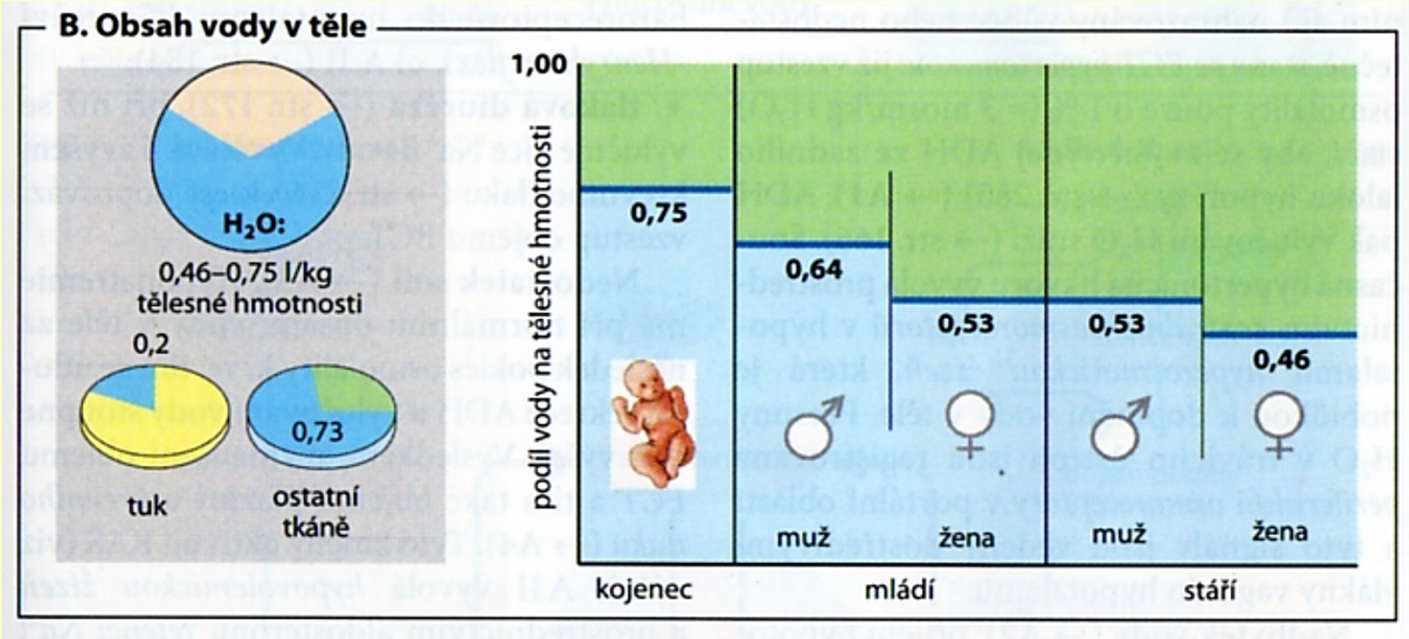
WATER, VITAMINS, MINERALS IN NUTRITION

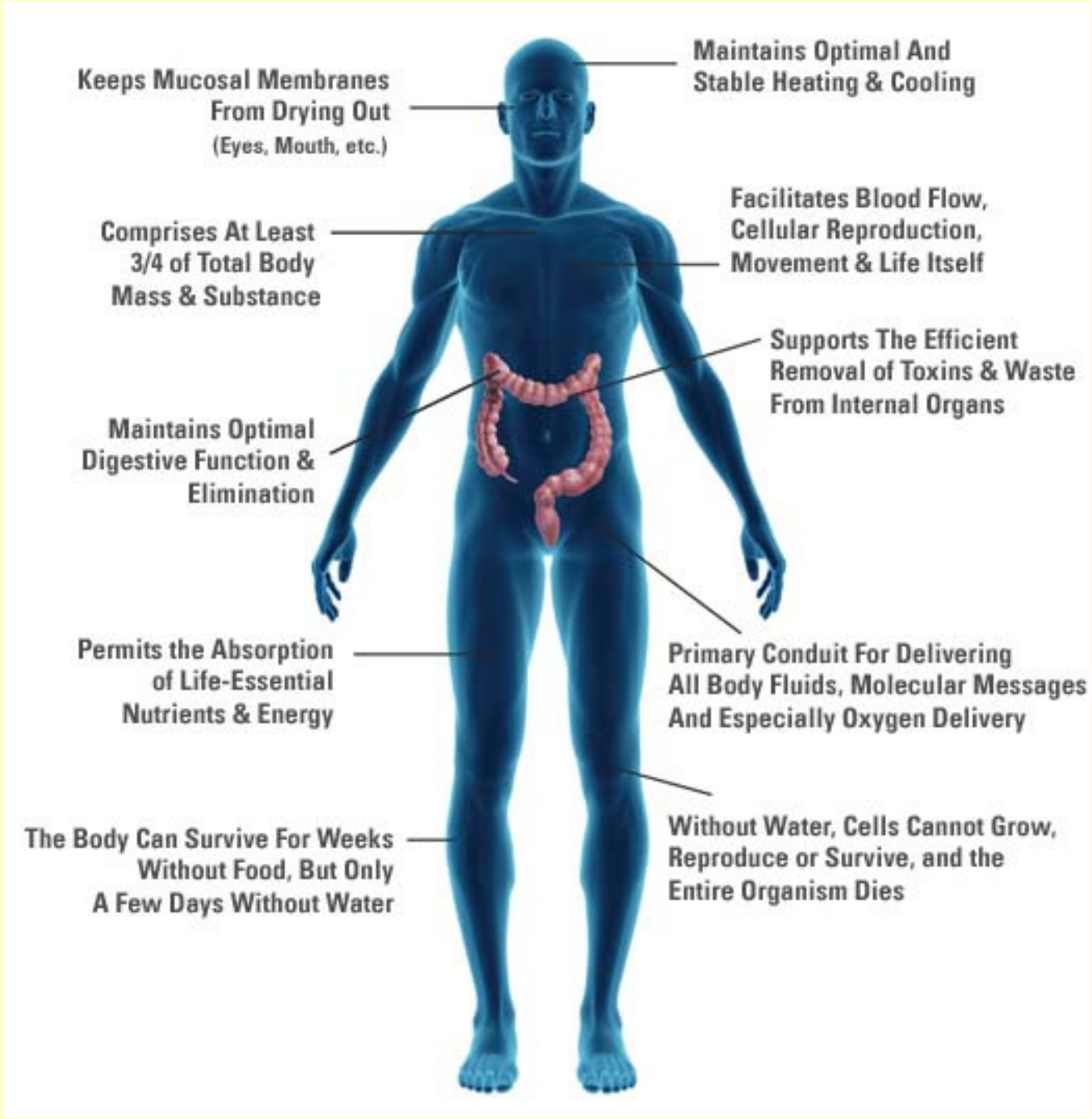
WATER

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

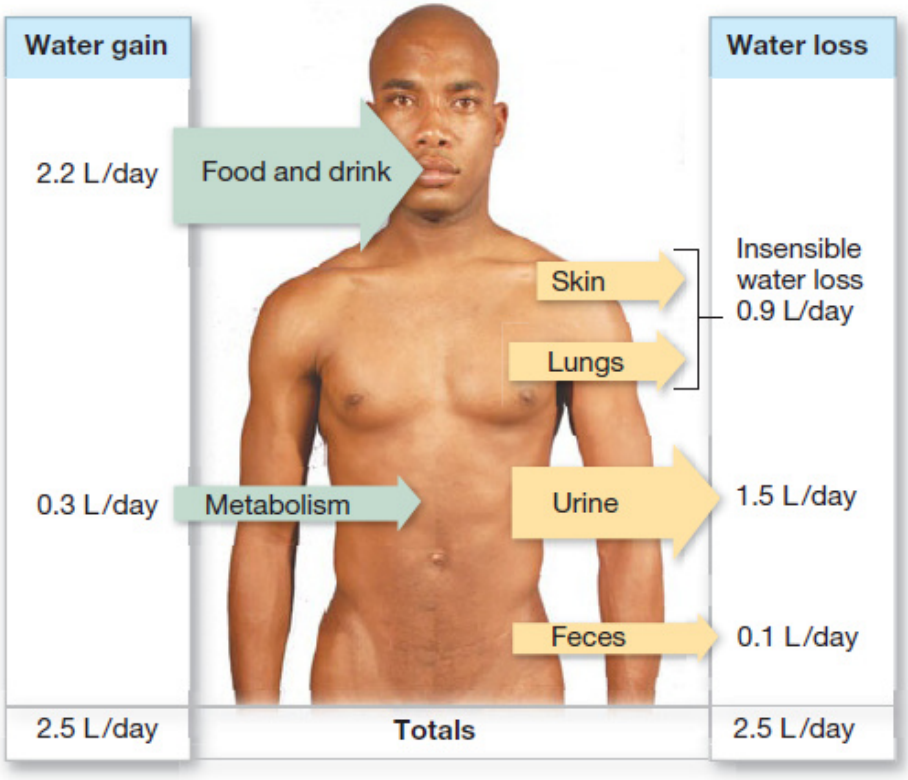
Water and its functions in the human body

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




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

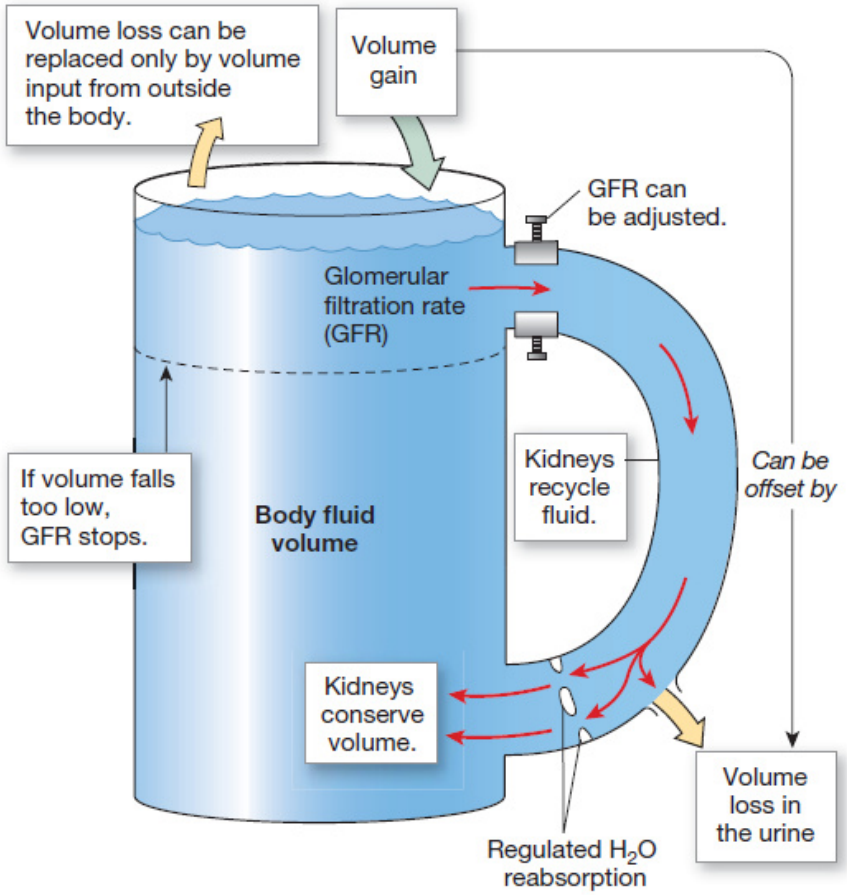
WATER BALANCE IN THE BODY

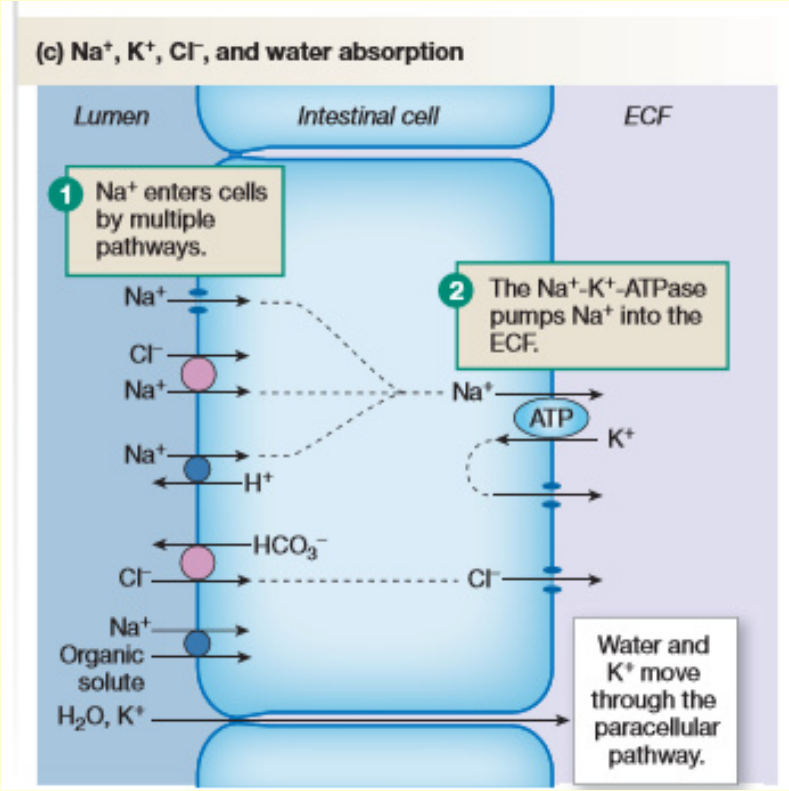


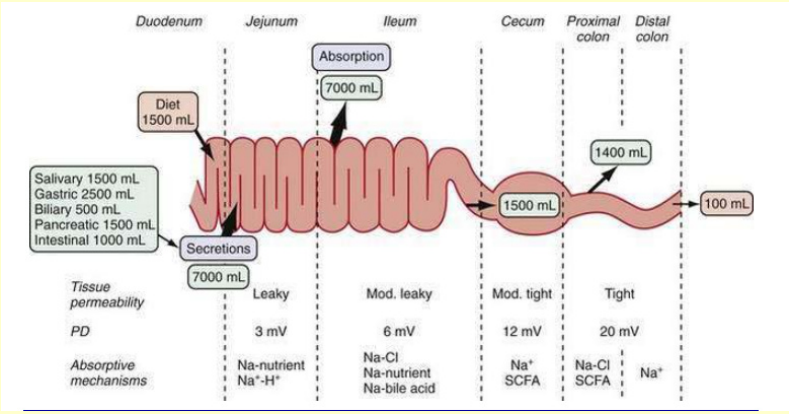
Intake		Metabolic production		Output	
2.2 L/day	+	0.3 L/day	–	2.5 L/day	= 0

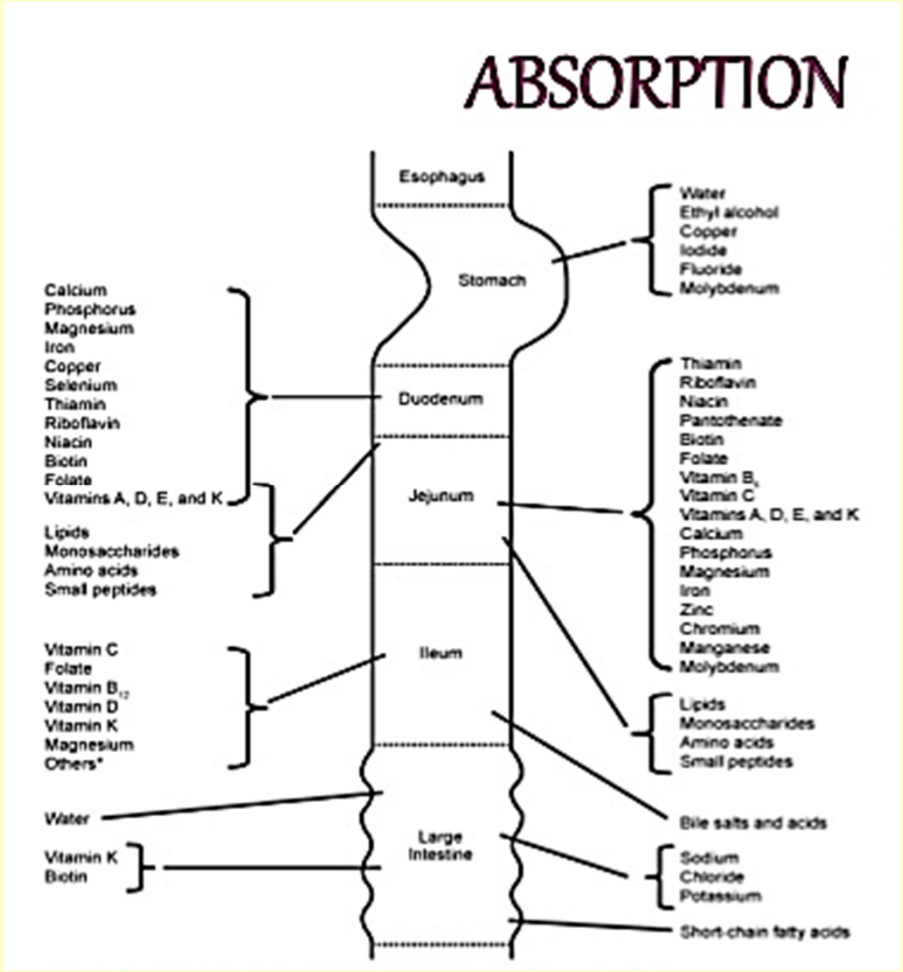
THE KIDNEYS CONSERVE VOLUME

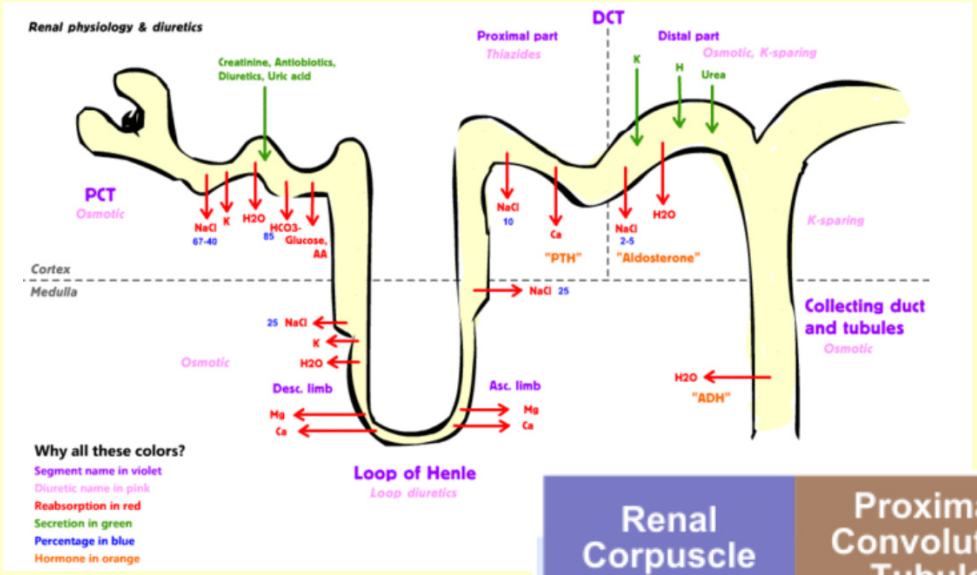
Kidneys cannot restore lost volume. They only conserve fluid.

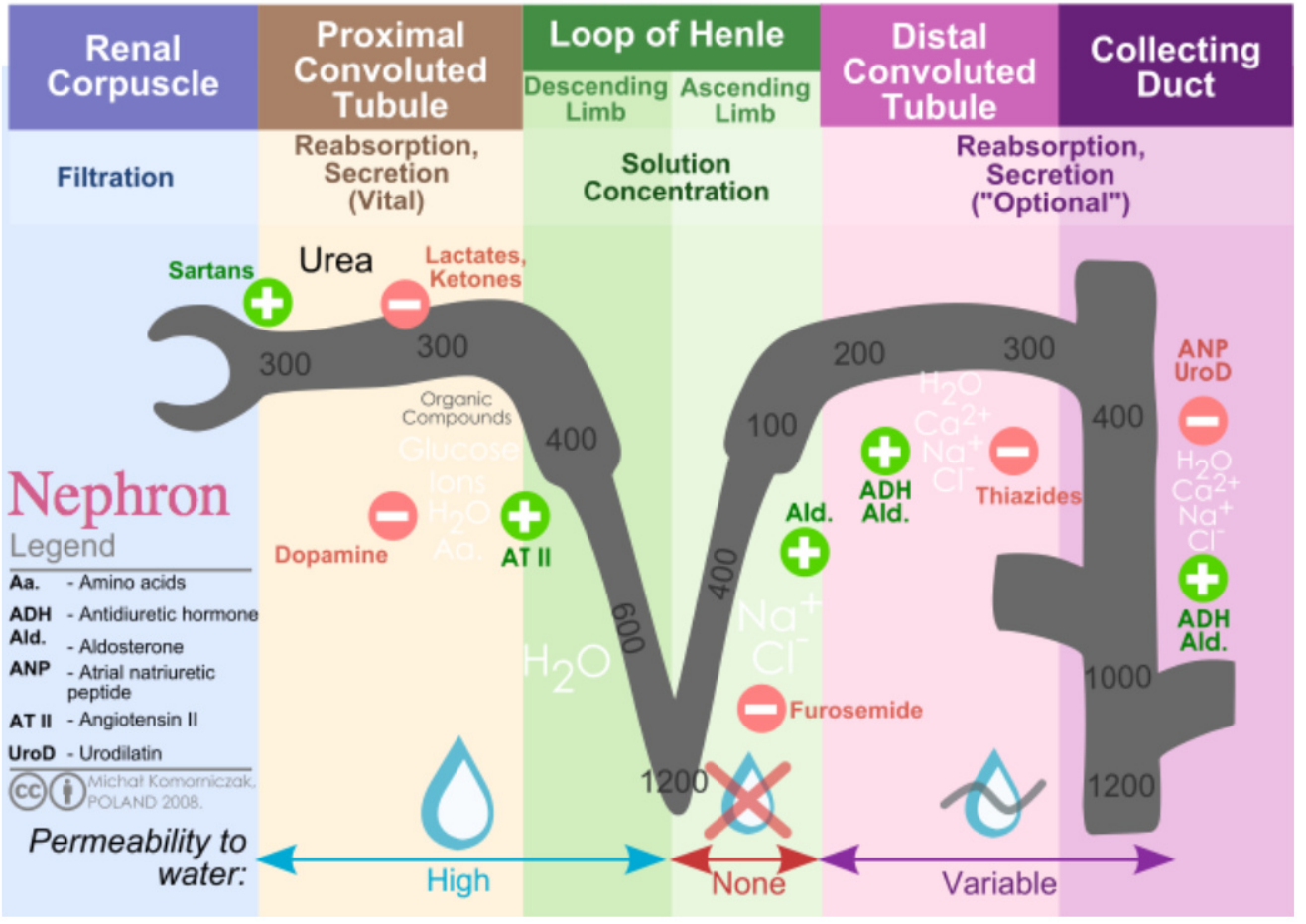



(c) Na+, K+, Cl−, and water absorption
Lumen
Intestinal cell
ECF
1 Na+ enters cells by multiple pathways.
2 The Na+-K+-ATPase pumps Na+ into the ECF.
Na+
Cl−
Na+
Na+
H+
HCO3−
Cl−
Na+
Organic solute
H2O, K+
Na+
ATP
K+
Cl−
Water and K+ move through the paracellular pathway.


Duodenum
Jejunum
Ileum
Cecum
Proximal colon
Distal colon
Absorption
7000 mL
Diet 1500 mL
Salivary 1500 mL
Gastric 2500 mL
Biliary 500 mL
Pancreatic 1500 mL
Intestinal 1000 mL
Secretions
7000 mL
1400 mL
1500 mL
100 mL
Tissue permeability
Leaky
Mod. leaky
Mod. tight
Tight
PD
3 mV
6 mV
12 mV
20 mV
Absorptive mechanisms
Na-nutrient Na+-H+
Na-Cl Na-nutrient Na-bile acid
Na+ SCFA
Na-Cl SCFA
Na+

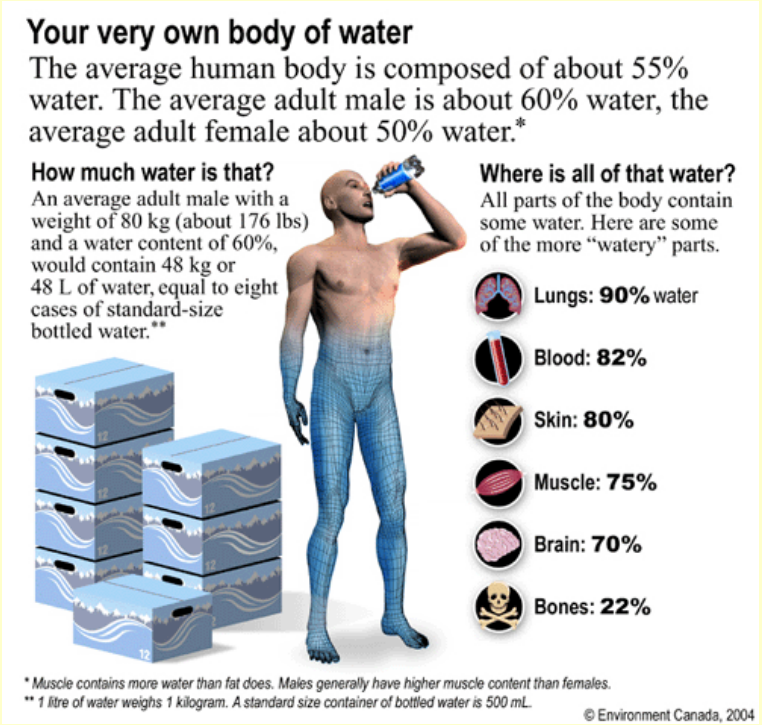

ABSORPTION
Esophagus
Stomach
Duodenum
Jejunum
Ileum
Large Intestine
Water
Ethyl alcohol
Copper
Iodide
Fluoride
Molybdenum
Calcium
Phosphorus
Magnesium
Iron
Copper
Selenium
Thiamin
Riboflavin
Niacin
Biotin
Folate
Vitamins A, D, E, and K
Lipids
Monosaccharides
Amino acids
Small peptides
Vitamin C
Folate
Vitamin B12
Vitamin D
Vitamin K
Magnesium
Others*
Water
Vitamin K
Biotin
Thiamin
Riboflavin
Niacin
Pantothenate
Biotin
Folate
Vitamin B6
Vitamin C
Vitamins A, D, E, and K
Calcium
Phosphorus
Magnesium
Iron
Zinc
Chromium
Manganese
Molybdenum
Lipids
Monosaccharides
Amino acids
Small peptides
Bile salts and acids
Sodium
Chloride
Potassium
Short-chain fatty acids

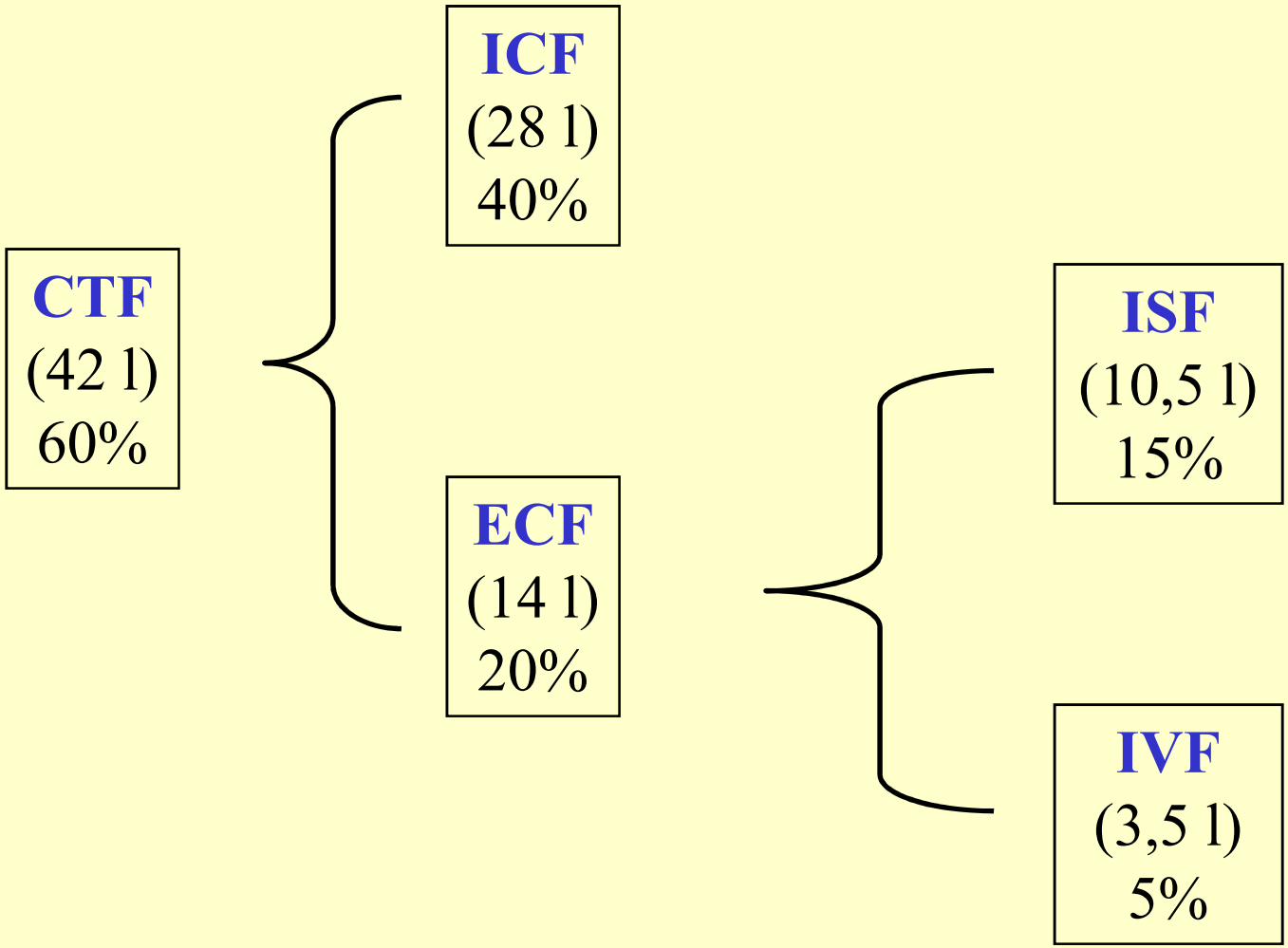

Renal physiology & diuretics
DCT
Proximal part
Thiazides
Distal part
Osmotic, K-sparing
Creatinine, Antiobiotics, Diuretics, Uric acid
K
H
Urea
PCT
Osmotic
NaCl 67-40
K
H2O
HCO3- 85
Glucose, AA
NaCl 10
Ca
"PTH"
NaCl 2-5
H2O
"Aldosterone"
K-sparing
Cortex
Medulla
NaCl 25
Collecting duct and tubules
Osmotic
25 NaCl
K
H2O
Osmotic
Desc. limb
Asc. limb
Mg
Ca
H2O
"ADH"
Why all these colors?
Segment name in violet
Diuretic name in pink
Reabsorption in red
Secretion in green
Percentage in blue
Hormone in orange
Loop of Henle
Loop diuretics


Renal Corpuscle
Proximal Convoluted Tubule
Loop of Henle
Descending Limb
Ascending Limb
Distal Convoluted Tubule
Collecting Duct
Filtration
Reabsorption, Secretion (Vital)
Solution Concentration
Reabsorption, Secretion ("Optional")
Sartans
Urea
Lactates, Ketones
300
300
Organic Compounds
Glucose
Ions
Aa.
400
Dopamine
AT II
600
200
100
400
Ald.
300
400
ADH
Ald.
Thiazides
ANP
UroD
ADH
Ald.
1000
1200
1200
Furosemide
Nephron
Legend
Aa. - Amino acids
ADH - Antidiuretic hormone
Ald. - Aldosterone
ANP - Atrial natriuretic peptide
AT II - Angiotensin II
UroD - Urodilatin
Michał Komorniczak, POLAND 2008.
Permeability to water:
High
None
Variable

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%





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

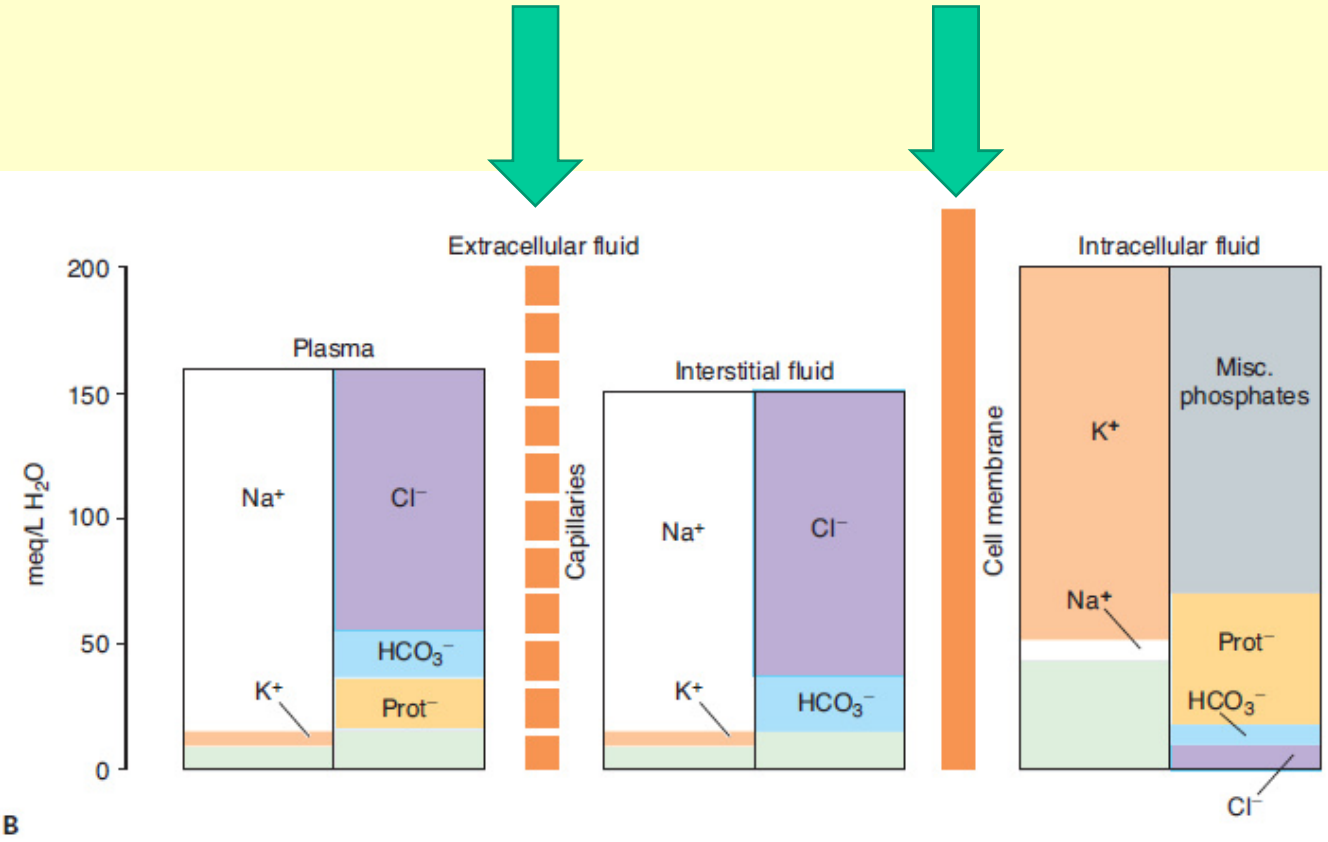
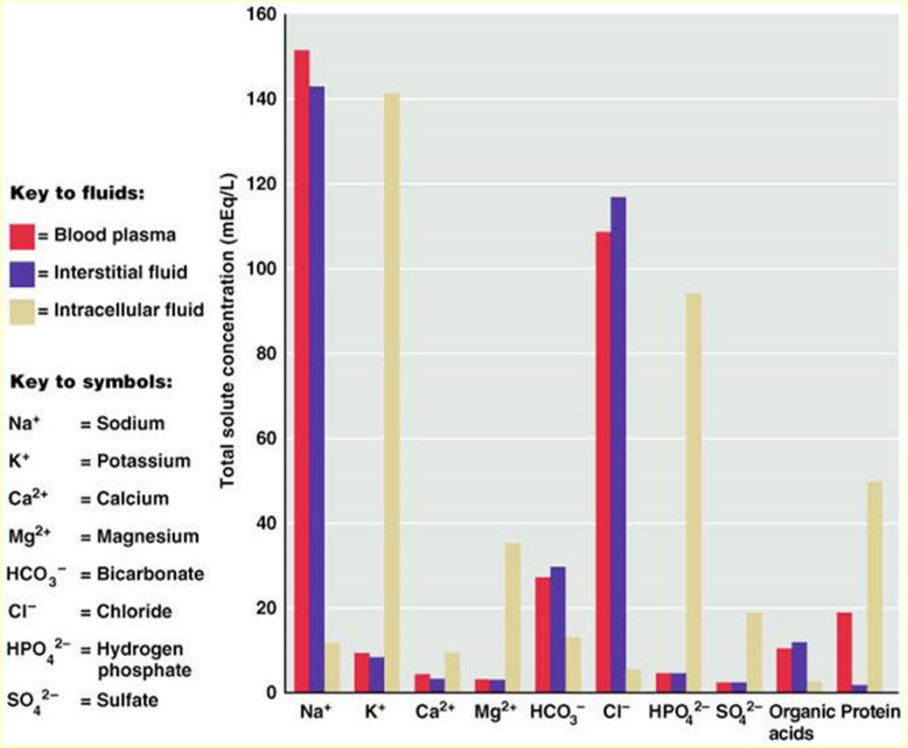
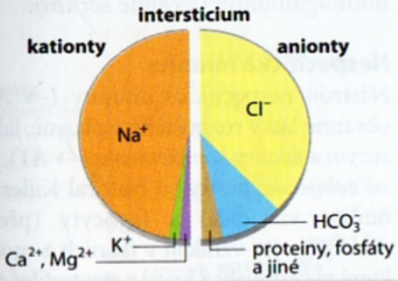
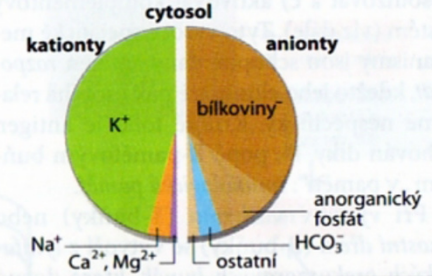


FIGURE 1–1 **Organization of body fluids and electrolytes into compartments. A)** Body fluids are divided into Intracellular and extracellular fluid compartments (ICF and ECF, respectively). Their contribution to percentage body weight (based on a healthy young adult male; slight variations exist with 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.



C. Iontové složení tělesných tekutin

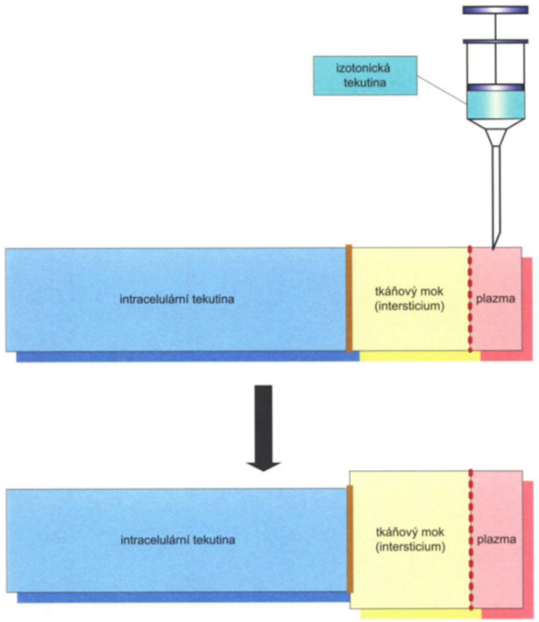




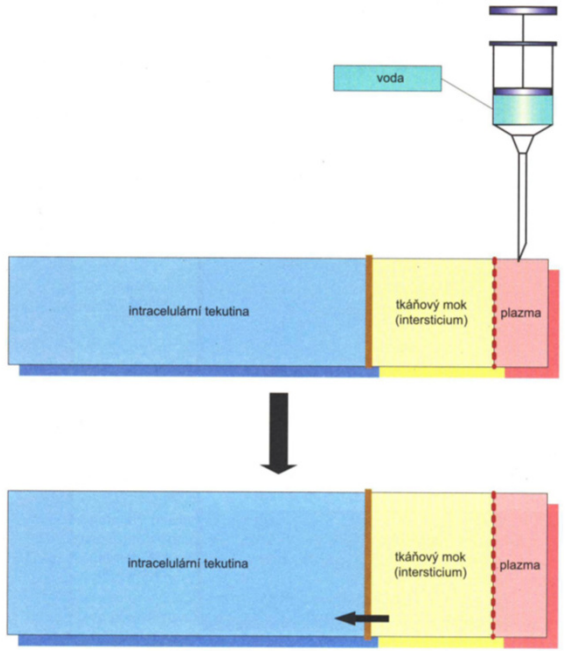
mval/l (mmol/l)

	iont	plazma	voda plazmy	intersticium	cytosol
kationty	Na^+	142	153	145	okolo 12
	K^+	4,3	4,6	4,4	okolo 140
	volné Ca^{2+}	2,6 (1,3*)	2,8 (1,3)	2,5 (1,5)	< 0,001
	volné Mg^{2+}	1,0 (0,5**)	1,0 (0,5)	0,9 (0,45)	1,6
	součet	150	162	153	oko 152
anionty	Cl^-	104	112	117	okolo 3
	HCO_3^-	24	36	27	10
	anorganický fosfát	2	2,2	2,3	okolo 30
	bílkoviny	14	15	0,4	okolo 54
	jiné	5,9	6,3	6,2	okolo 54
	součet	150	162	153	okolo 152

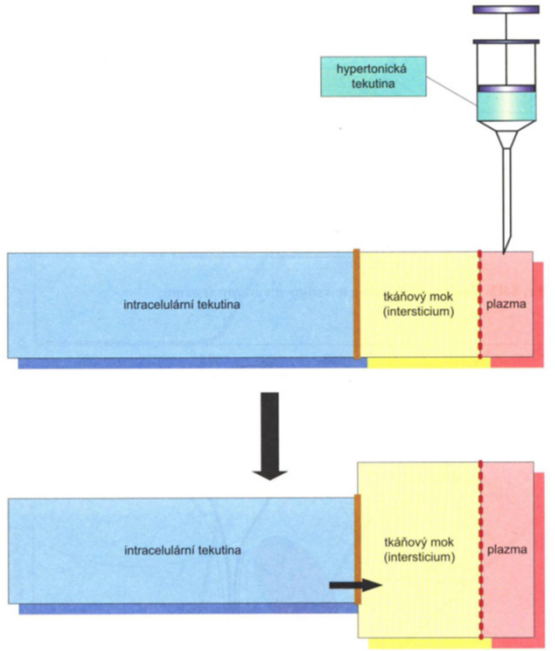
*) celkové Ca v plazmě 2,5 mmol/l; **) celkové Mg v plazmě 0,9 mmol/l



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*

table 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_2O D_2O Antipyrene		
ECF	1/3	Sulfate Inulin Mannitol	Na^+	Cl^- HCO_3^-
Plasma	1/12 (1/4 of ECF)	RISA Evans blue	Na^+	Cl^- HCO_3^- Plasma protein
Interstitial	1/4 (3/4 of ECF)	ECF–plasma volume (indirect)	Na^+	Cl^- 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
- **Hypovolemia (dehydratation)**
- Hypervolemia (hyperhydratation)

Cause – result

Complex disorders!

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)
7. 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.

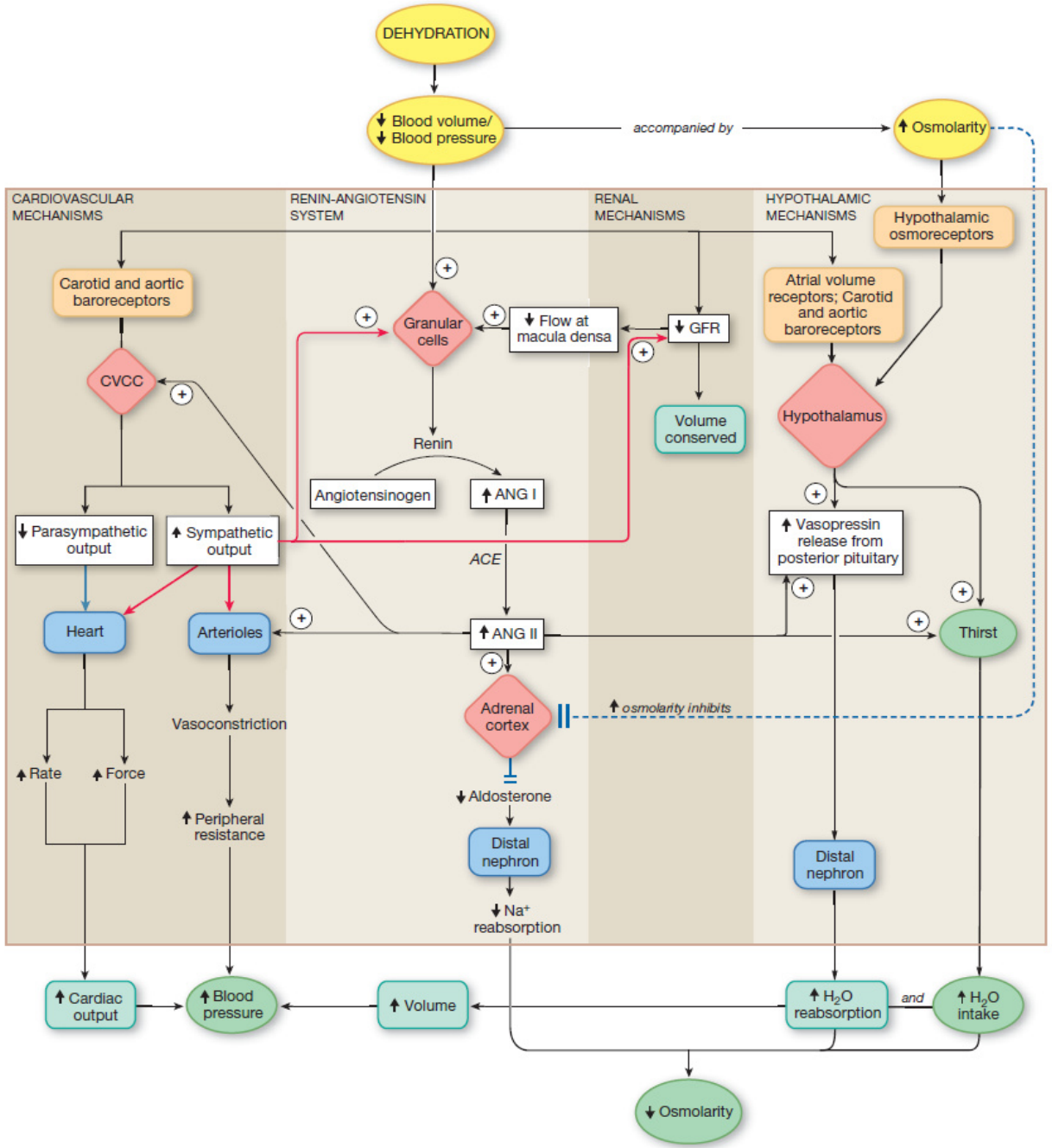
IZOTONIC DEHYDRATATION = isonatremic

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



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.

table 5-2 Changes in Volume and Osmolarity of Body Fluids

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

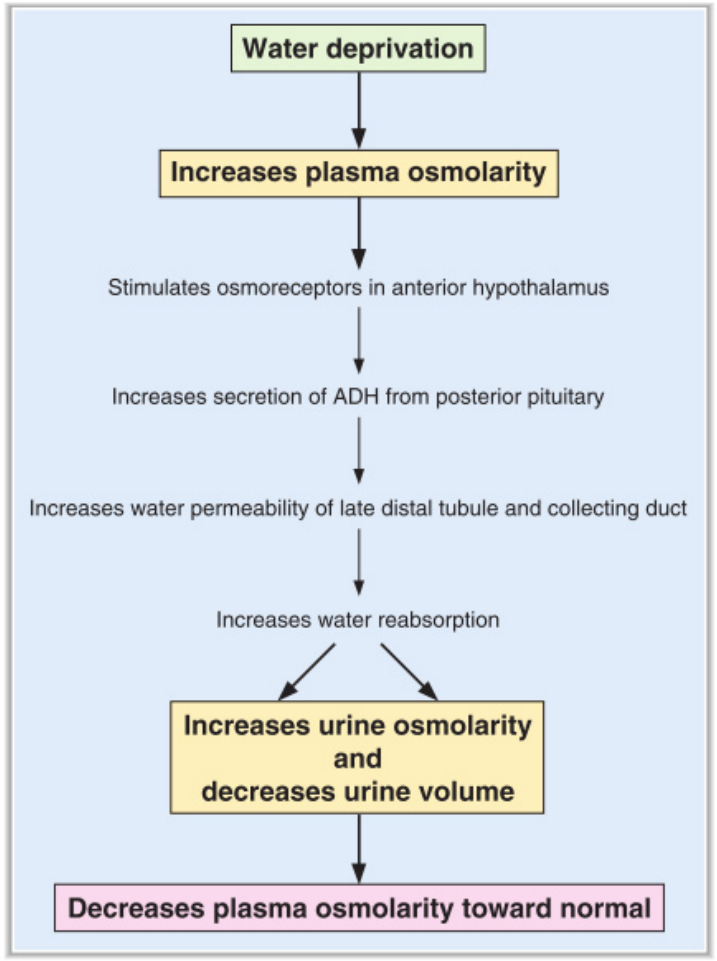


FIGURE 5-14 Responses to water deprivation. ADH = antidiuretic hormone.

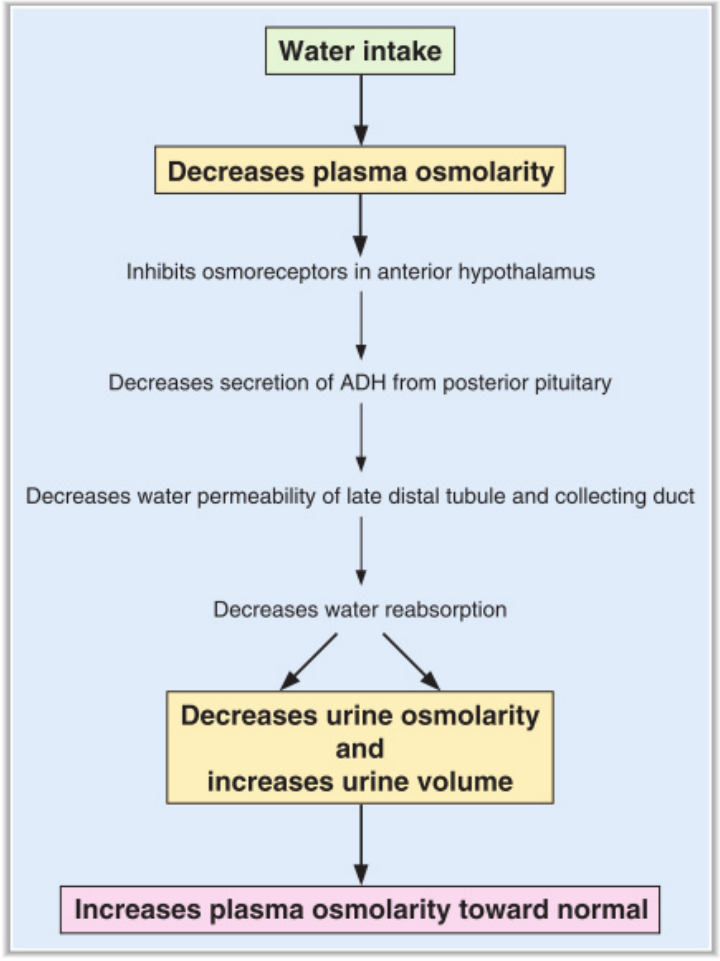


FIGURE 5-15 Responses to water intake. ADH = antidiuretic hormone.

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**

SOLUBLE

- in water: diffusion, D, J; **vit.B_{12}** - I
- in lipids: deficient absorption in disorders of lipids absorption (pancreatic enzymes or bile missing)

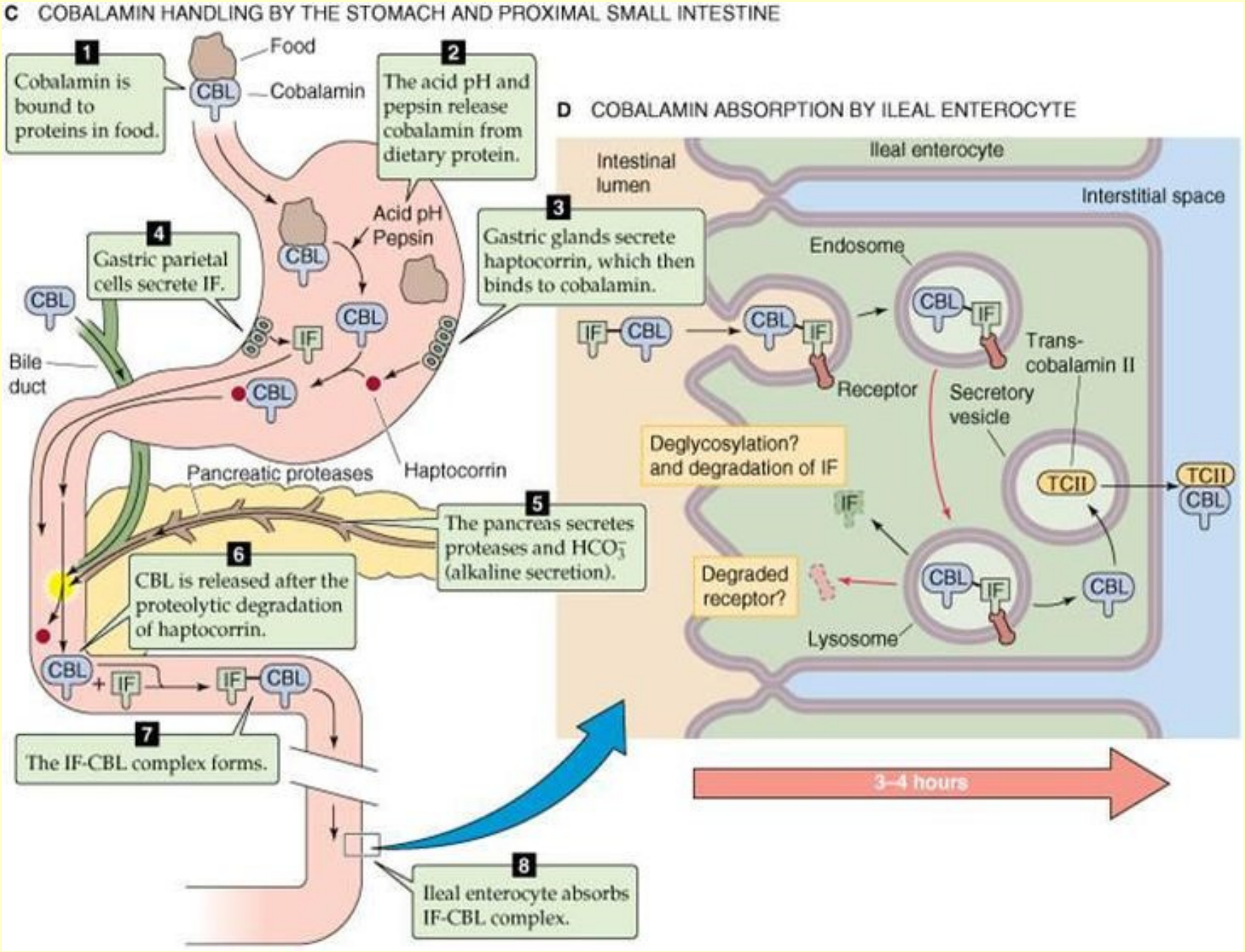
Vitamin	Species	Place of absorption	Transport mechanism	Maximal absorption capacity in humans / day	Daily dose
C	Humans, guinea pig	Ileum	Active	>5000mg	<50mg
Biotin	Hamster	Small intestine	Active	?	?
Cholin	Guinea pig, hamster	Small intestine	Facilitated diffusion	?	?
Folic acid (pteroylglutamate)	Rat	Jejunum	Facilitated diffusion	> 1000µg (dose)	100-200µg
Folic acid (5-methyltetrahydrofolate)	Rat	Jejunum	Diffusion	> 1000µg (dose)	100-200µg
Nicotinic acid	Rat	Jejunum	Facilitated diffusion	?	10-20mg
Pantothenic acid		Small intestine	?	?	(?)10mg
B_6 (pyridoxine)	Rat, hamster	Small intestine	Diffusion	> 50mg (dose)	1-2mg
B_2 (riboflavin)	Humans, rat	Jejunum	Facilitated diffusion	10-12mg (dose)	1-2mg
B_1 (thiamine)	Rat	Jejunum	Active	8-14mg	Approx. 1mg
B_{12}	**Humans, rat, hamster**	**Distal ileum**	**Active**	**6-9µg**	**3-7µg**

VITAMIN B_{12}

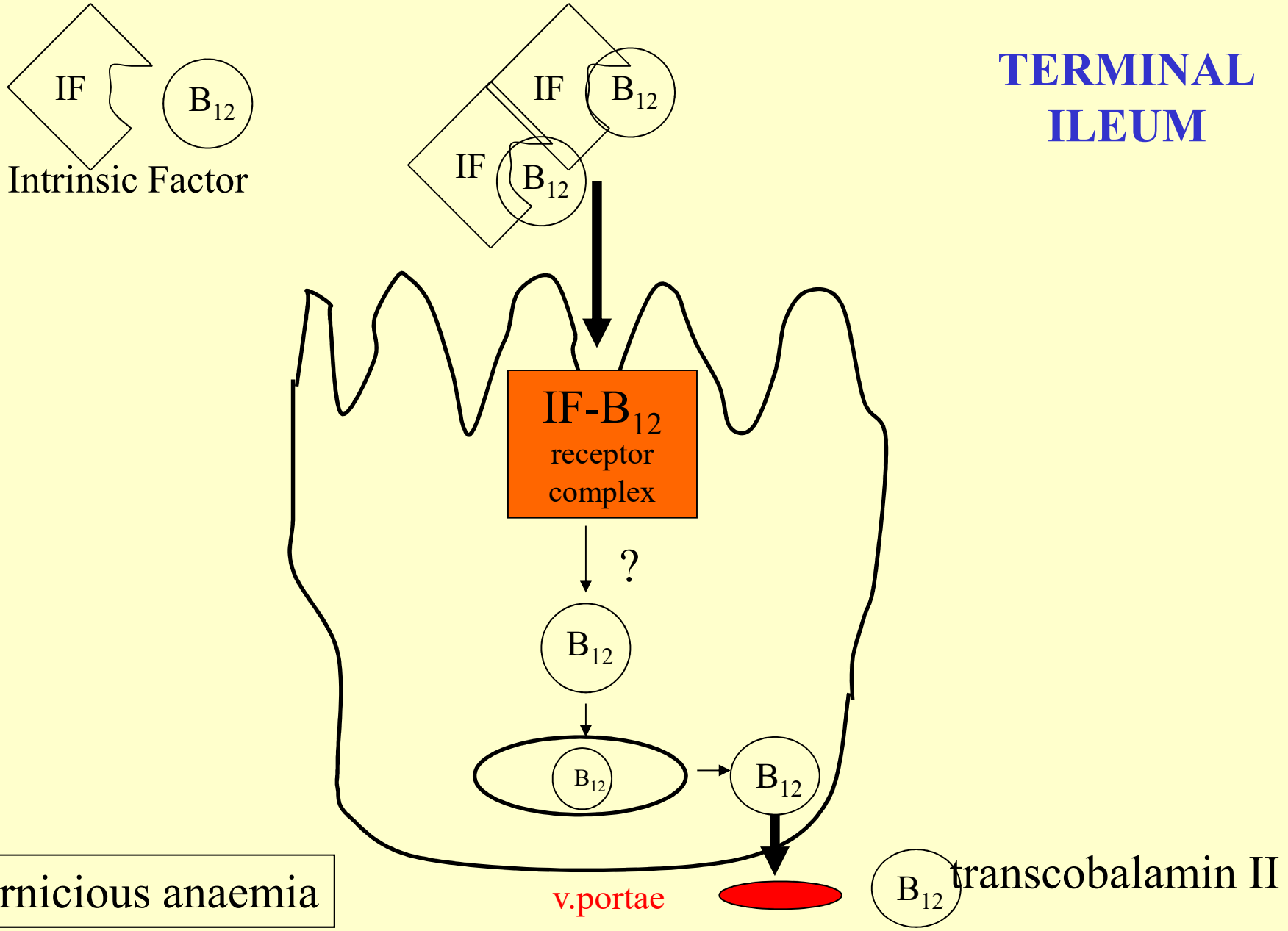
- Daily dose is close to absorption capacity
- Synthesised by bacteria in colon – BUT there is not absorption mechanism
- Store in liver (2-5mg)
- In bile 0,5-5μg / day, reabsorbed
- Daily loss – 0,1% of stores ➞ stores will last for 3-6 years

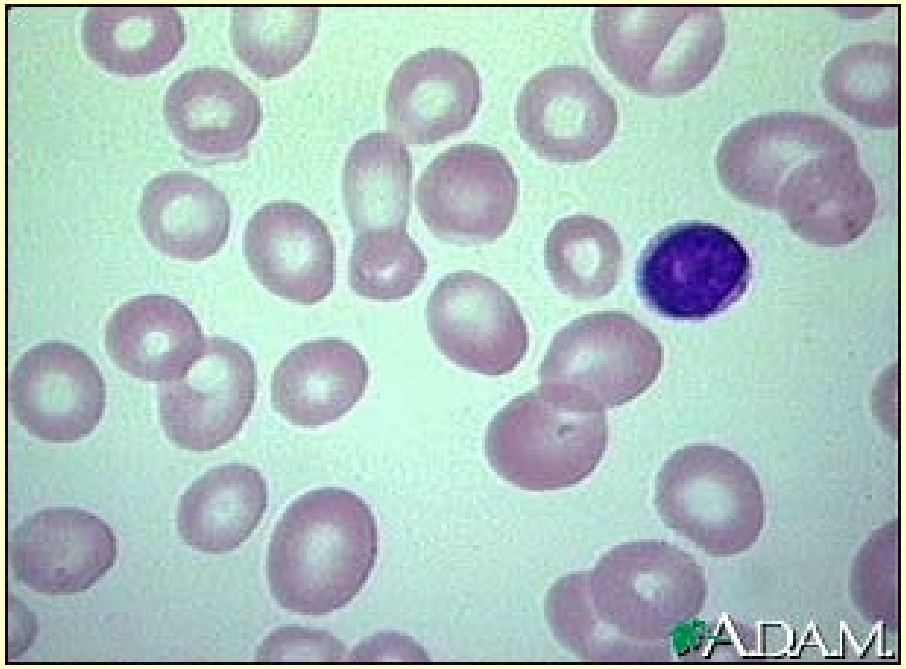
ABSORPTION

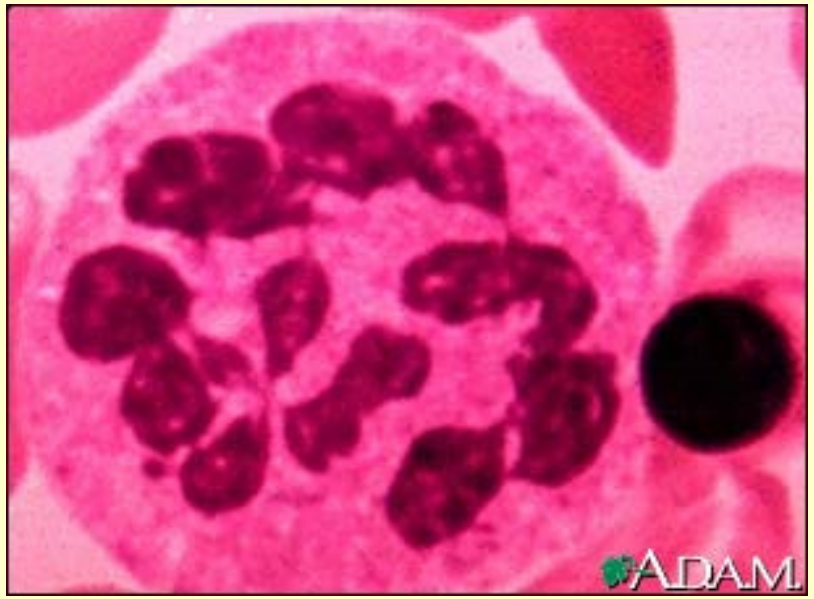
1. **Gastric phase**: B_{12} is bound to proteins, low pH and pepsin release it; bound to glycoproteins – **R-proteins** (saliva, gastric juice), almost pH-undependable; intrinsic factor (**IF**) – parietal cells of gastric mucosa; most of vitamin bound to R-proteins
2. **Intestinal phase:** pancreatic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


C COBALAMIN HANDLING BY THE STOMACH AND PROXIMAL SMALL INTESTINE
1
Cobalamin is bound to proteins in food.
Food
CBL
Cobalamin
2
The acid pH and pepsin release cobalamin from dietary protein.
Acid pH
Pepsin
3
Gastric glands secrete haptocorrin, which then binds to cobalamin.
4
Gastric parietal cells secrete IF.
IF
Bile duct
Haptocorrin
Pancreatic proteases
5
The pancreas secretes proteases and HCO_3^- (alkaline secretion).
6
CBL is released after the proteolytic degradation of haptocorrin.
7
The IF-CBL complex forms.
8
Ileal enterocyte absorbs IF-CBL complex.
D COBALAMIN ABSORPTION BY ILEAL ENTEROCYTE
Intestinal lumen
Ileal enterocyte
Interstitial space
Endosome
Receptor
Trans-cobalamin II
Secretory vesicle
Deglycosylation? and degradation of IF
TCII
Degraded receptor?
Lysosome
3–4 hours

ABSORPTION OF B_{12} VITAMIN




A.D.A.M.


A.D.A.M.

HYPOVITAMINOSES

Folic acid – disorders of embryo development (clefts)
B_{12} – pernicious anaemia
C – scurvy (scorbutus)
D – rickets (rhachitis, English disease, English sickness)
E – fertility problems
K - haemorrhage

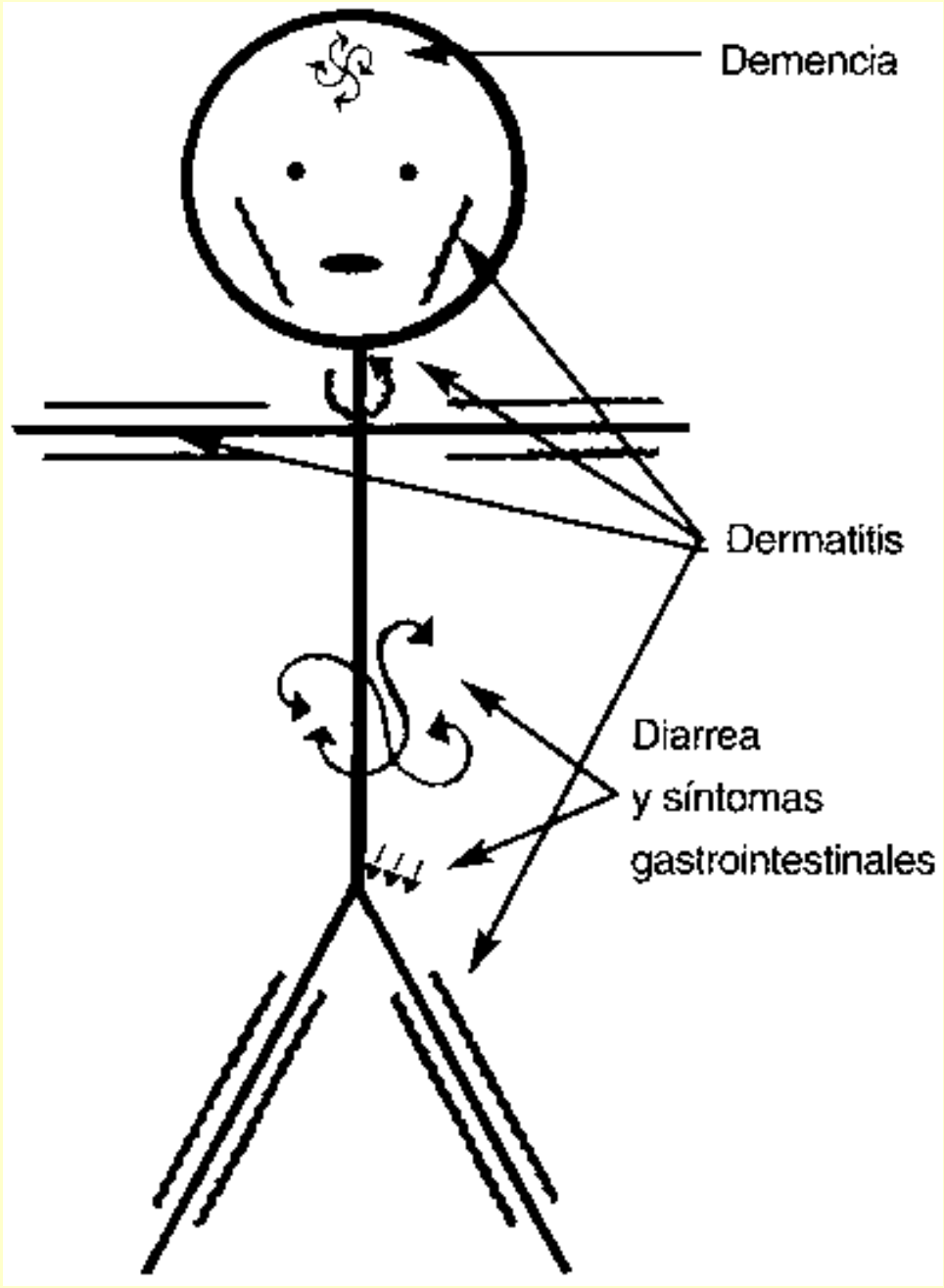
HYPERVITAMINOSES

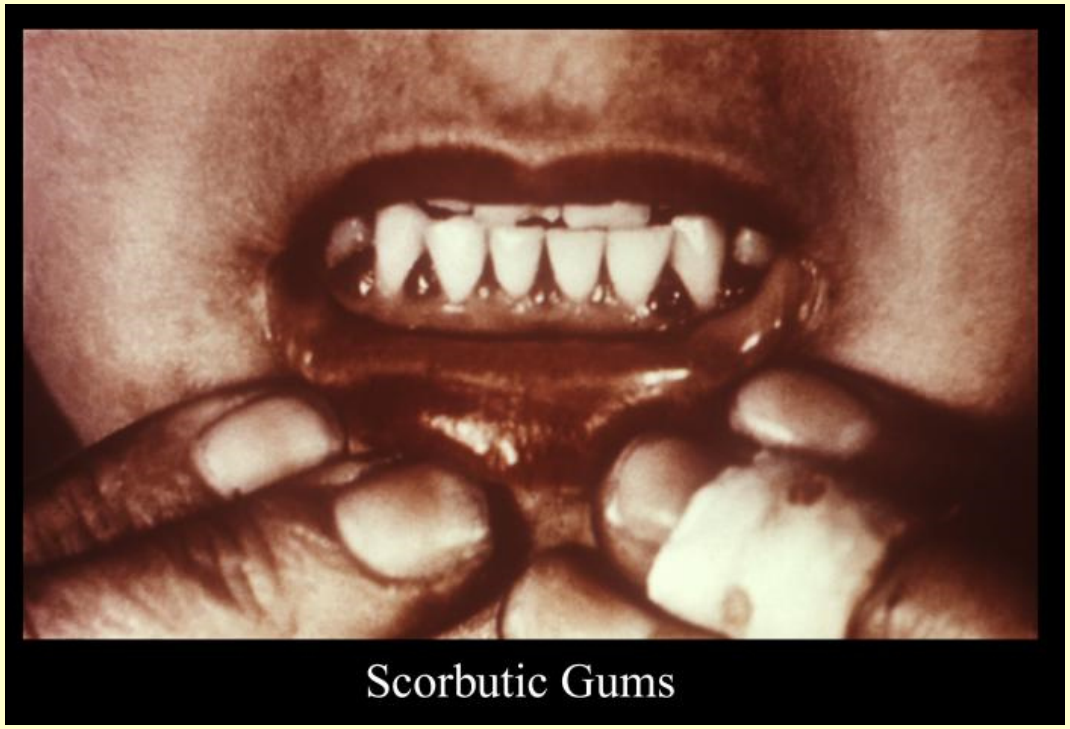
A – teratogenic effects
D – kidney failure
K – anaemia, GIT disorders
B_6 – peripheral polyneuropathy

BERI-BERI (B_1)

"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).

PELAGRA
(3 D disease)
(niacin)

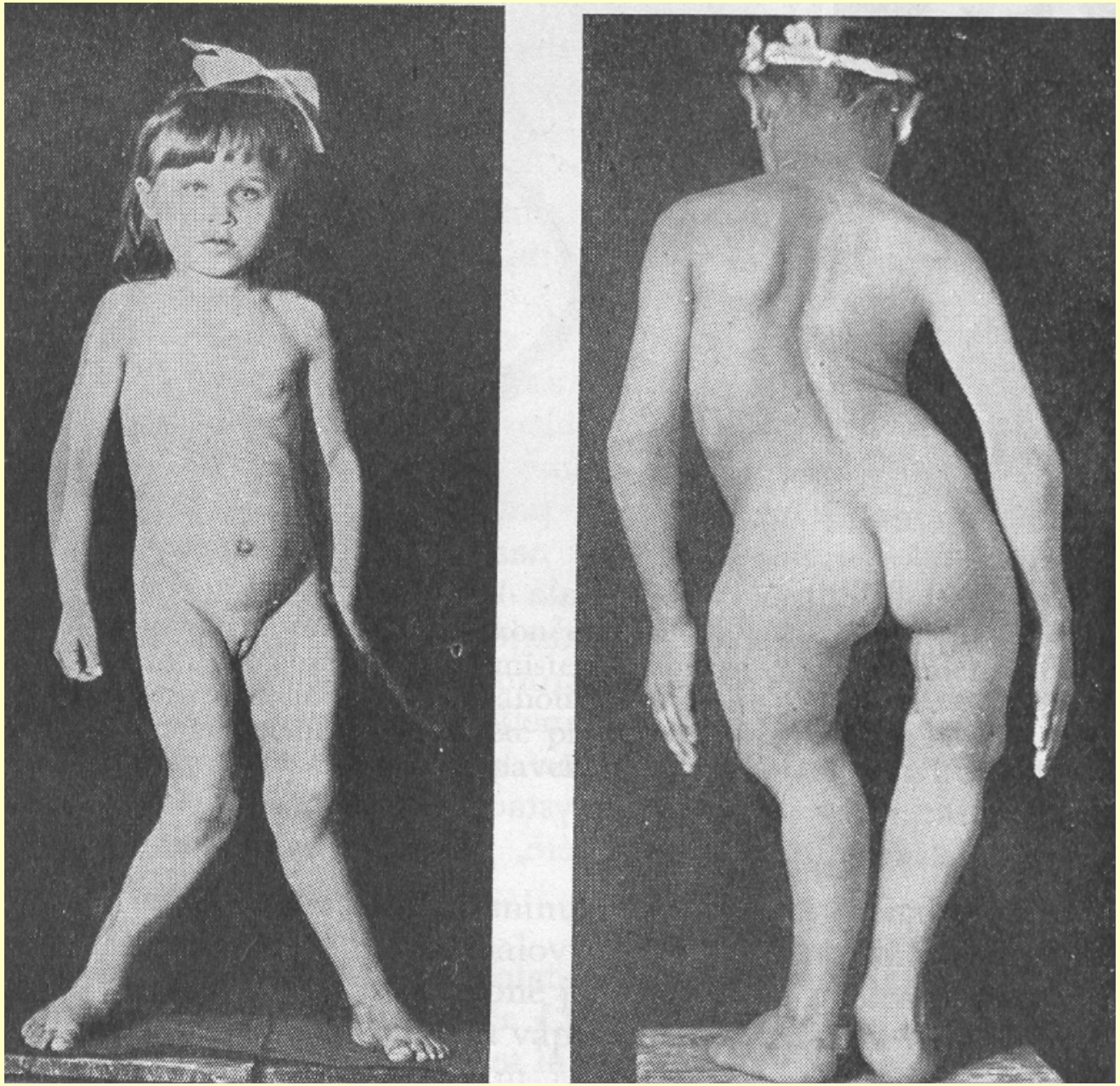




Scorbutic Gums

SCURVY

RICKETS



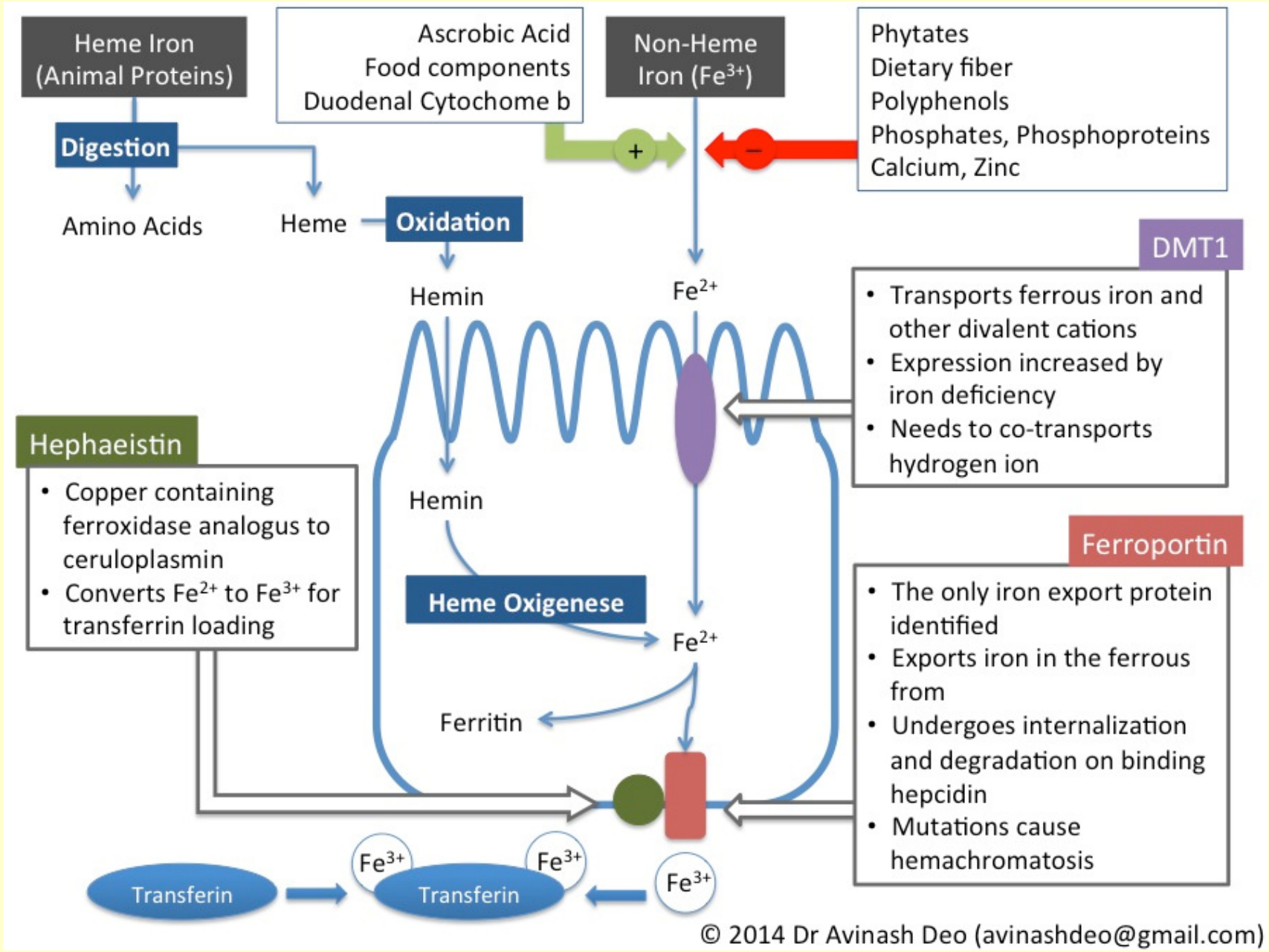
Mineral	Daily need (dose)
Na	3,0 g
K	1,0 g
Cl	3,5 g
Ca	1,2 g
P	1,2 g
Fe	18,0 mg
J	150,0 μg
Mg	0,4 g
Co	?
Cu	?
Mn	?
Zn	15 mg

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.B_{12}; 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)


Heme Iron (Animal Proteins)
Digestion
Amino Acids
Heme
Oxidation
Hemin
Ascrobic Acid
Food components
Duodenal Cytochome b
+
Non-Heme Iron (Fe^{3+})
−
Phytates
Dietary fiber
Polyphenols
Phosphates, Phosphoproteins
Calcium, Zinc
Fe^{2+}
DMT1
• Transports ferrous iron and other divalent cations
• Expression increased by iron deficiency
• Needs to co-transports hydrogen ion
Hephaeistin
• Copper containing ferroxidase analogus to ceruloplasmin
• Converts Fe^{2+} to Fe^{3+} for transferrin loading
Hemin
Heme Oxigenese
Fe^{2+}
Ferritin
Ferroportin
• The only iron export protein identified
• Exports iron in the ferrous from
• Undergoes internalization and degradation on binding hepcidin
• Mutations cause hemachromatosis
Fe^{3+}
Fe^{3+}
Fe^{3+}
Transferin
Transferin
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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