

Avitaminoses, trace elements, lack of nutrients

Disorders of nutrients in dental medicine

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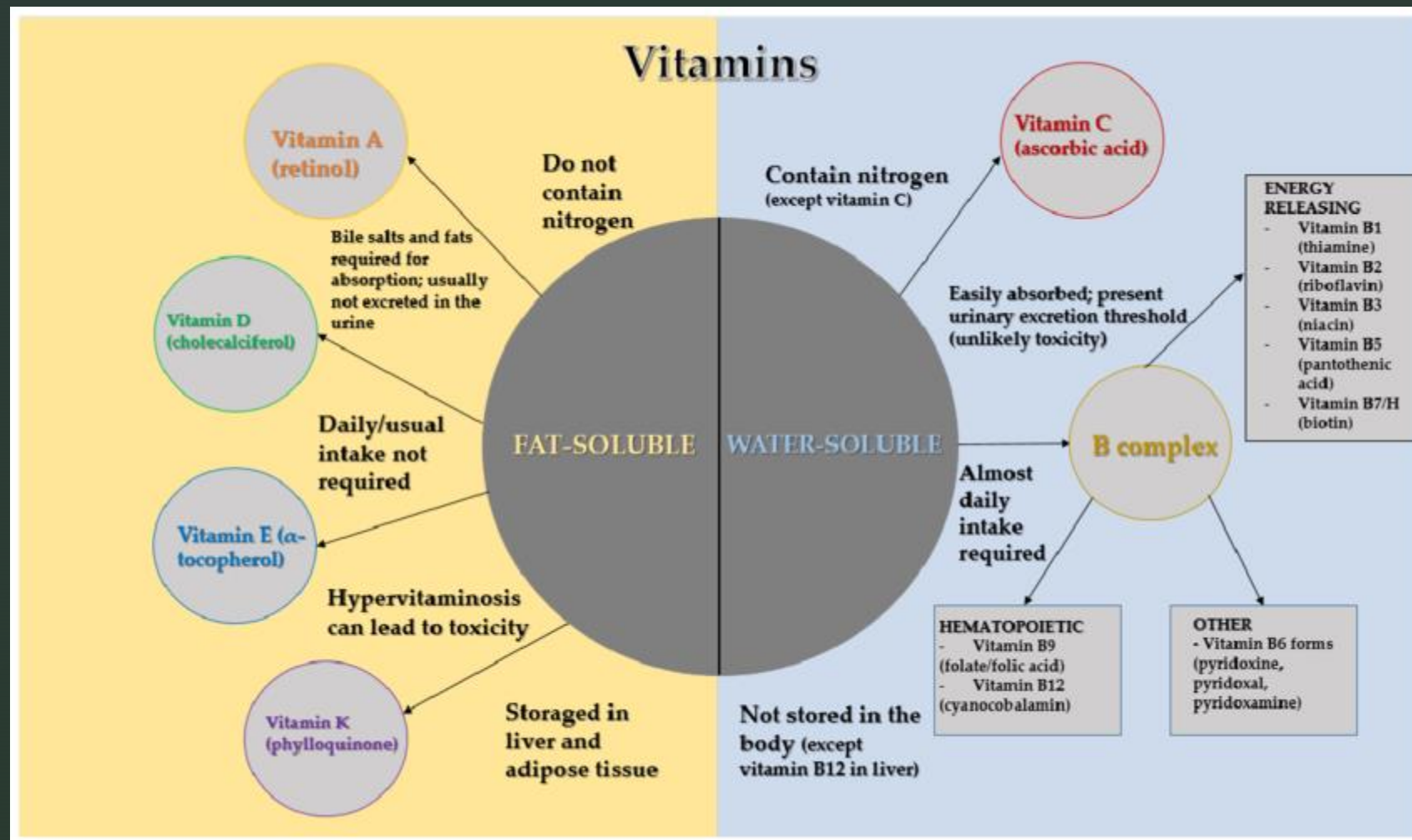
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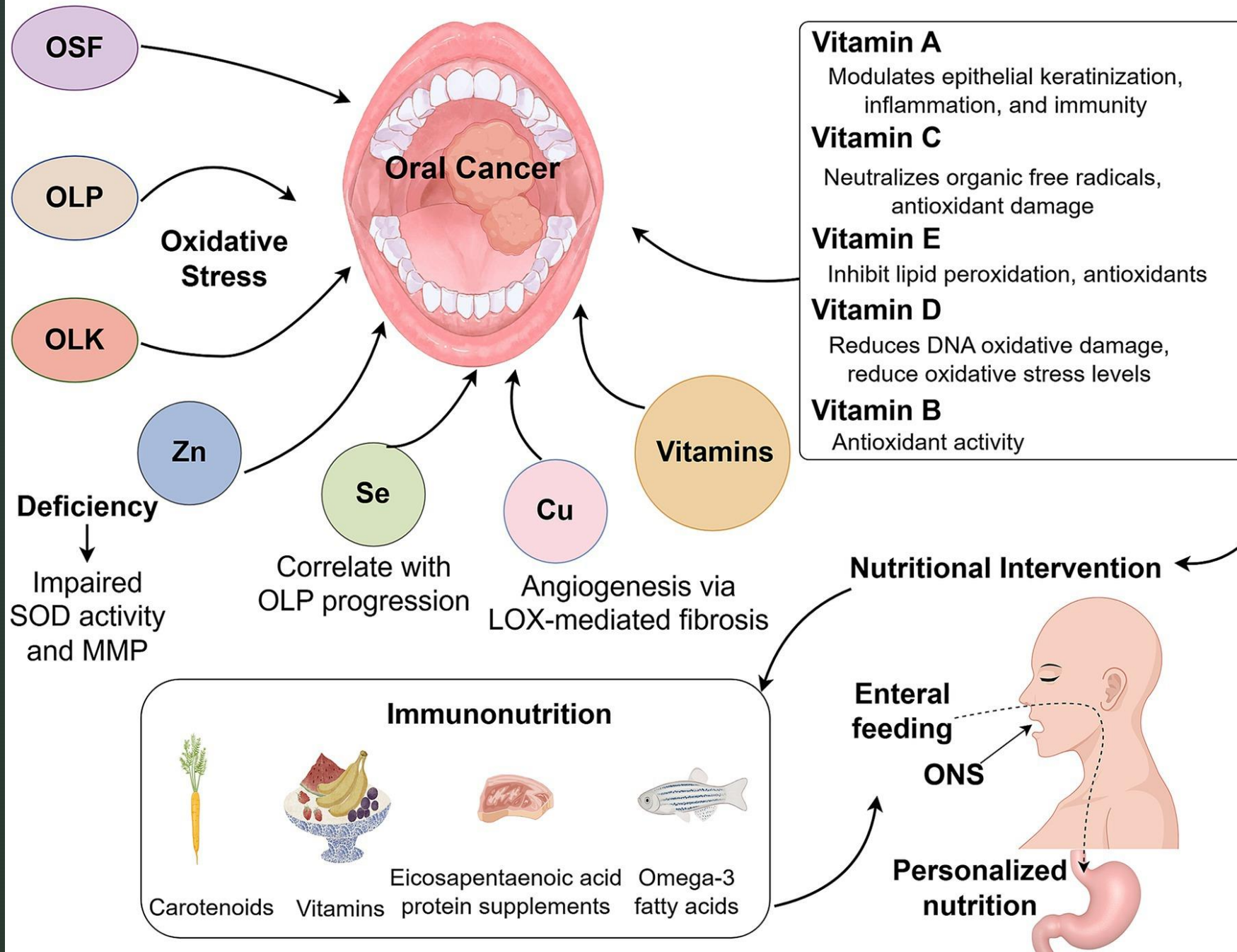
Vitamin deficiency and overdose



General characteristics of selected vitamin deficiencies

Deficiency	Systemic effects	Oral effects
Vitamin A	Night-blindness, xerophthalmia	Unconformed contribution to leukoplakia and cancer
Thiamin (B1)	Neuritis, cardiac failure	None
Riboflavin (B2)	Dermatitis	Angular stomatitis and glossitis
Nicotinamide (Niacin, B3)	Dermatitis, CNS disease (dementia), diarrhea	Glossitis, gingivitis, stomatitis
Cyanocobalamin (B12)	Pernicious anemia, Wernicke-Korsakoff syndrome	Glossitis, aphtae
Folic acid (B9)	Macrocytic anemia	Glossitis, aphtae
Ascorbic acid (C)	Scurvy (purpura, delayed wound healing, bone lesions in children)	Gingival swelling and bleeding
Vitamin D	Ricketts	Hypocalcification of teeth (severe rickets only)

Antioxidant Effects of Vitamins



Vitamin A deficiency

- Fat soluble vitamin
- Deficiency -> ↑children and maternal mortality
 - Increased needs during growth period
 - Typical in infants and pre-school aged children; fats malabsorption
- Incidence – 1-2.5 millions/year
- Systemic manifestations
 - Night blindness, xerophthalmia, keratomalacia, Bitot spots, follicular hyperkeratosis, prone to infections, nail fragility

Oral manifestation of vitamin A deficiency

- Pathomechanism
 - Columnar epithelium -> squamous and keratinised in rats (no evidence in humans)
- Manifestation
 - Epithelium -> keratotic changes, hyperkeratinisation
 - Dentin, enamel -> enamel hypoplasia (even amelogenesis imperfecta), ↑caries, periodontitis
 - Prone to fungal infections
 - ? Prone to cancer



Marked hyperkeratosis – the lesion cannot be scraped off

Vitamin D deficiency

- Two forms – ergocalciferol (D2) and cholecalciferol (D3)
 - D3 formed by UV (skin exposure) -> liver -> kidney
- Effects
 - Ca^{2+} and PO_4^{3-} regulation (blood, bones)
 - Intestinal Ca^{2+} absorption, bones remodelling
 - Immunotolerance -> dendritic cells -> $\uparrow T_{reg}$ cells
 - Tumour-suppressor, anti-inflammatory

Oral manifestation of vitamin D deficiency

- Pathomechanism
 - $\downarrow Ca^{2+}$ intestinal resorption -> hypocalcaemia -> secondary hyperparathyroidism -> PTH activating osteoclasts (RANK) -> bones mass reduction and Ca^{2+} release
 - \downarrow VDRE stimulation -> \downarrow amelogenins -> improper mineralisation
 - \downarrow cell differentiation -> improper bone formation
 - \downarrow cathelicidin (LL-37) expression -> \downarrow antimicrobial/endotoxin defence
- NOTE – may mimic *amelogenesis imperfecta**
 - A genetic disease (AMELX gene affected)

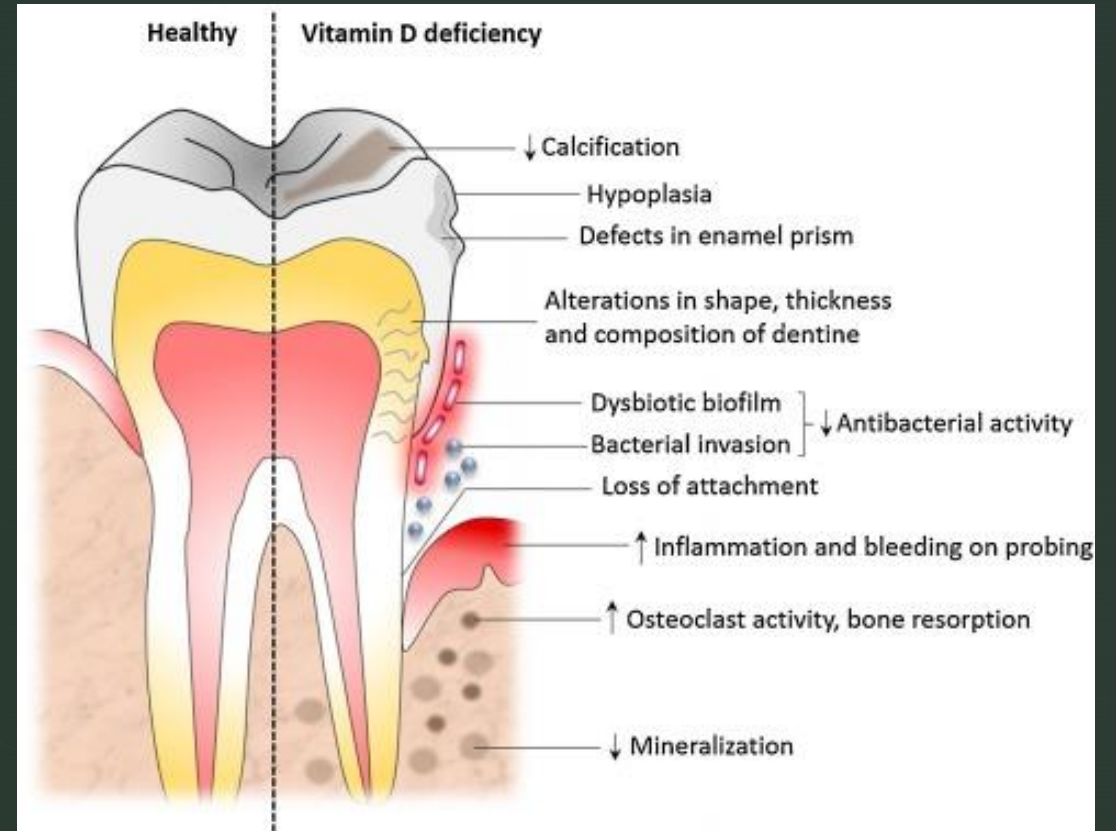
*some sources link vitamins A and D as an acquired forms of amelogenesis imperfecta, this is controversial highly

Oral manifestation of vitamin D deficiency

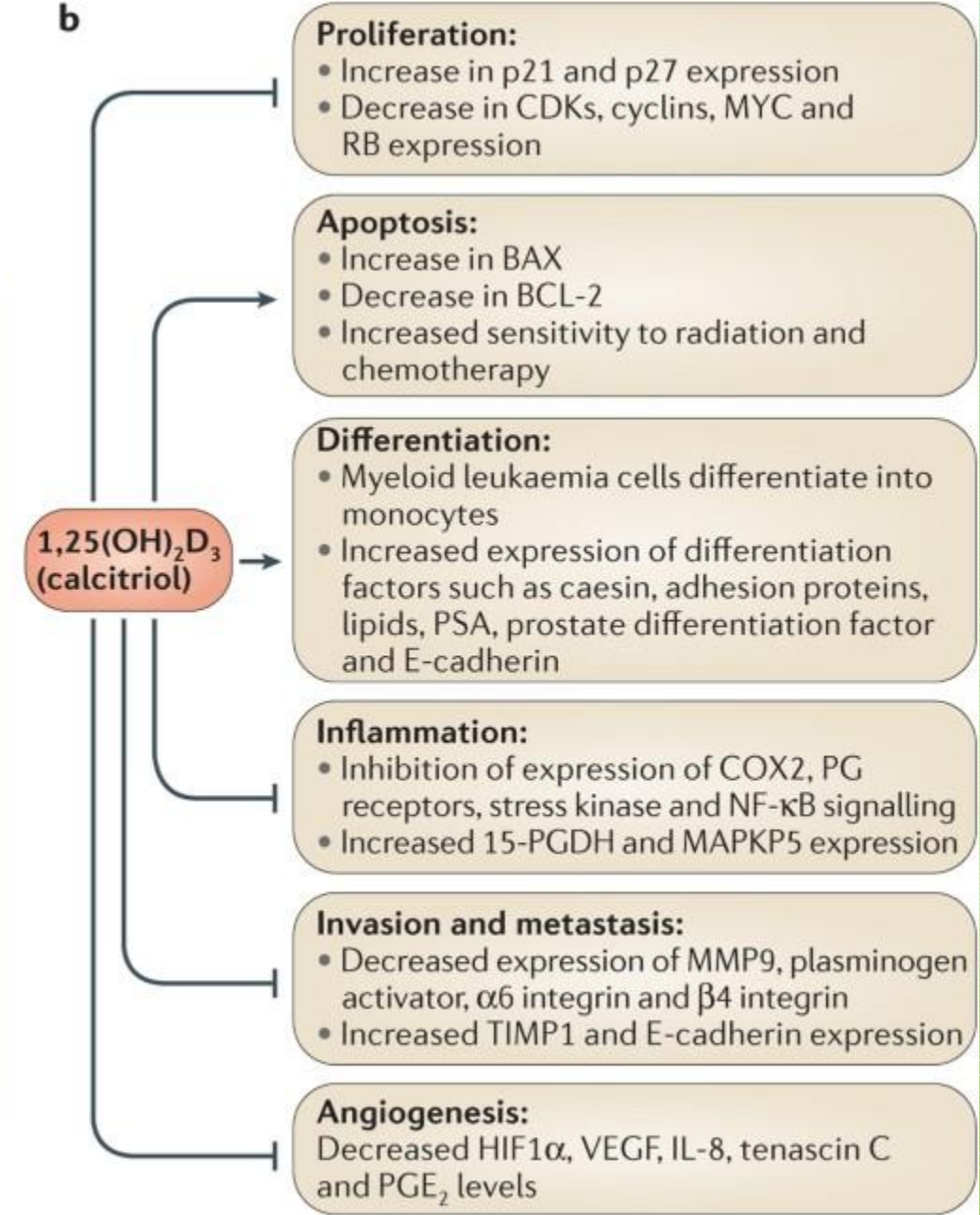
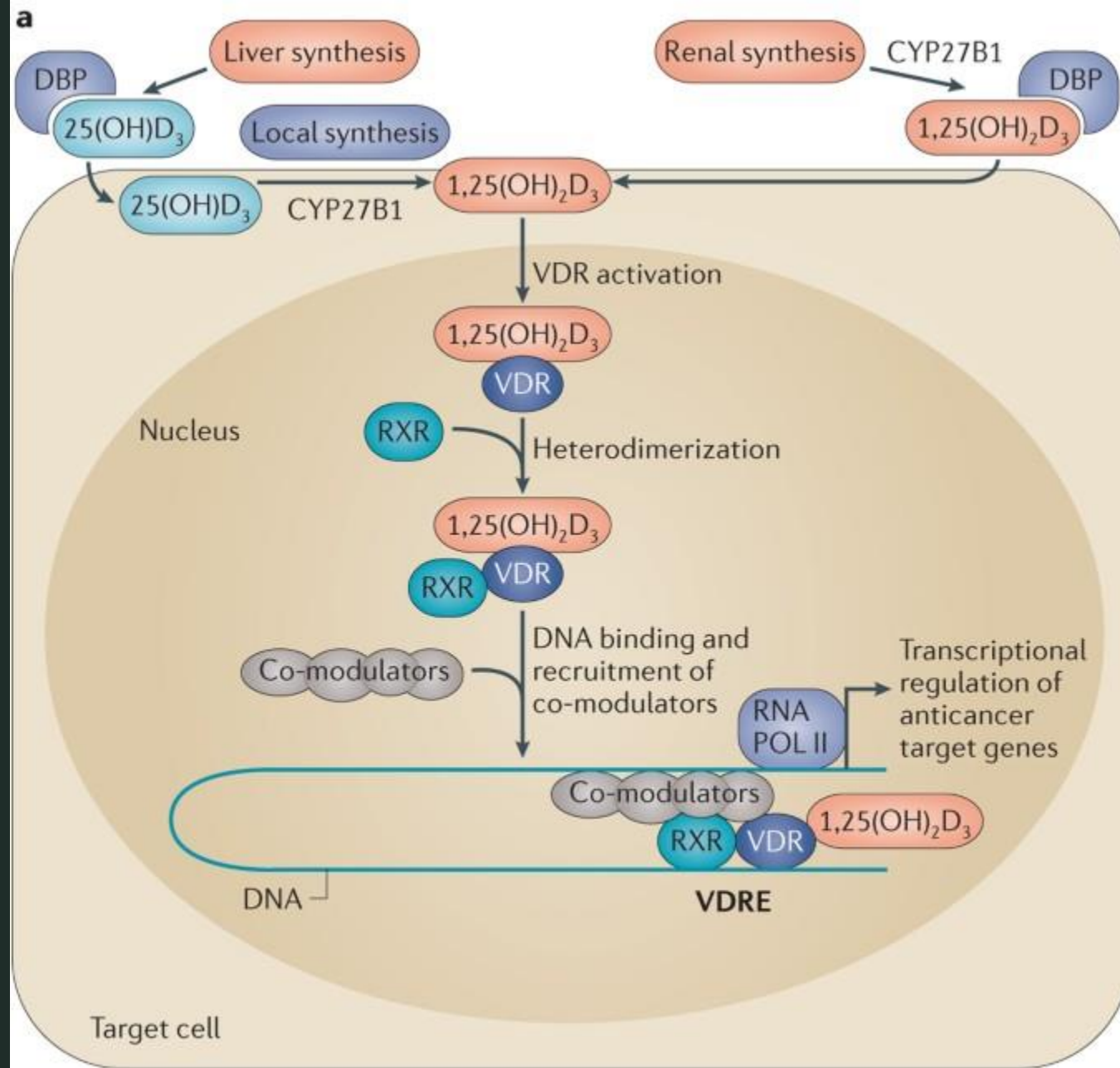
- Manifestation
 - Enamel hypoplasia, hypomineralisation
 - Dentin defects
 - Ectodermal dysplasia
 - Jawbone resorption
 - Increased caries incidence



Dentin and enamel hypoplasia



Summary effects of vitamin D effect in oral cavity



Vitamin E deficiency

- Risk groups for deficiency
 - Preterm infants, pregnant women, metabolic diseases
- Main role
 - Membranous antioxidant, anti-inflammatory
- Manifestation
 - Neurologic manifestation, immune system disorders, haemolytic anaemia
 - NO ORAL MANIFESTATION
 - *(alleviate mucositis due to oncology treatment)*



Mucositis and xerostomia in patient treated for oropharyngeal cancer

Vitamin K deficiency

- Risk groups for deficiency
 - Infants, home births, rejection of vitamin K prophylaxis
- Main role
 - Coagulation cascade -> Factors II, VII, IX and XI; anticoagulant proteins C and S
 - γ -carboxylation -> Ca^{2+} binding -> phospholipid membranes interaction
- Manifestation
 - Bleeding – mucosal, submucosal (may be fatal in some organs)



Water soluble vitamins – B group

- Members
 - B1 (thiamine), B2 (riboflavin), B3 (niacin), B5 (pantothenic acid), B7 (biotin/vitamin H), B9 (folate), B12 ((cyano)cobalamin)
- Complete avitaminosis rare
 - Endangered groups – alcoholics, malabsorption (e.g. Crohn's disease), imbalanced diet vegans
- Roles
 - Energy production, cell metabolism, brain and neural function, red blood cells formation, cytotoxic immunity

Vitamin B1

- Function
 - Energy production, nerve impulses transmission, myelin sheath formation and maintenance
- Manifestation
 - Tingling, paraesthesia
 - Wernicke-Korsakoff syndrome (Wernicke encephalopathy – acute, Korsakoff psychosis - chronic)
- Oral symptoms
 - „Burning mouth“ – mouth sores, hyperesthesia



GABA
TELEPSYCHIATRY

PSYCHIATRIC SYMPTOMS OF VITAMIN B1 DEFICIENCY



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https://gabapsychiatrist.com/wp-content/uploads/2024/07/Psychiatric-symptoms-of-Vitamin-B1-deficiency_portrait.webp



www.gabapsychiatrist.com



+1(833)312-4222

Wernicke-Korsakoff syndrome (alcohol-associated neurological disorder)

COAT RACK

Wernicke's encephalopathy (acute phase) clinical features:

Confusion

Ophthalmoplegia

Ataxia

Thiamine is an important aspect of Tx

Korsakoff's psychosis (chronic phase) characteristic findings:

Retrograde amnesia (recall of some old memories)

Anterograde amnesia (ability to form new memories)

Confabulation

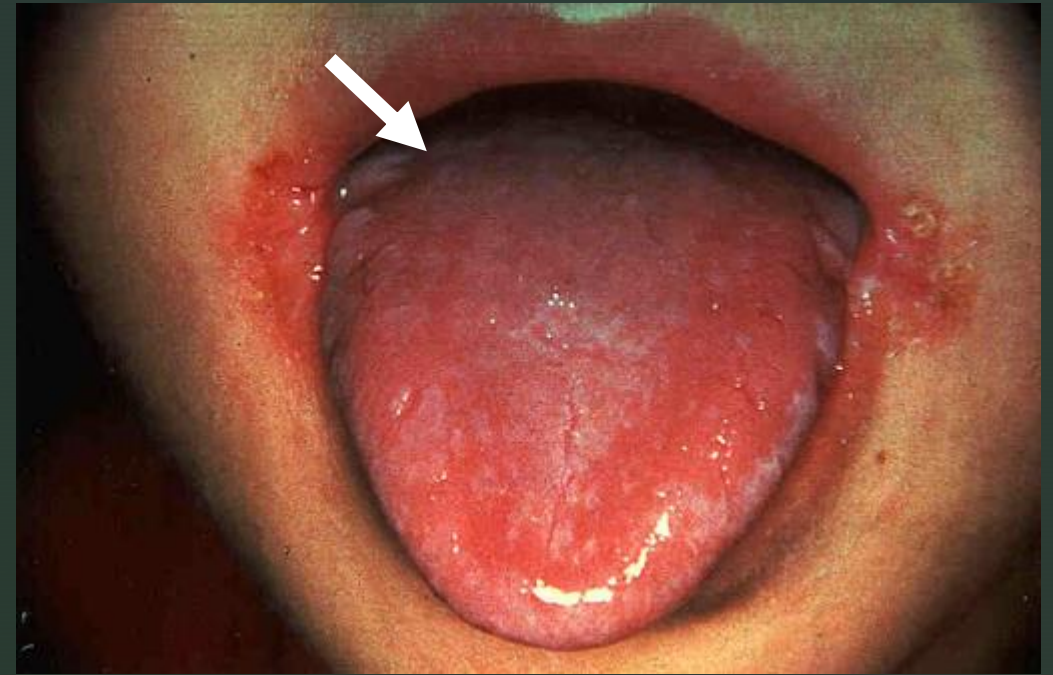
Korsakoff's psychosis



rnpedia.com/mnemonics

Vitamin B2 (riboflavin) deficiency

- Role
 - Energy metabolism
- Oral manifestations
 - Recurrent aphthous stomatitis
 - Glossitis
 - Angular cheilitis
- *NOTE – symptoms from B1 and B2 deficiency may overlap*



Upper left - Angular cheilitis
Upper right – glossitis (tongue inflammation)
Lower right – sore eyes

<https://cdn.ncbi.nlm.nih.gov/pmc/blobs/51cd/11049216/da2643874cba/dentistry-12-00109-g006.jpg>

<https://lh5.googleusercontent.com/proxy/51sldPX59n9wpsWEdxywv9hPIKjy5Lm2moz8YMphnoEt960p5WSYCBiQuAdxtahyvZKrqWfva2q6gfgjx2B7icE5ryb517ViBkbUfQNuGDIGt4RZLgAyInemRN4Yn3XeOQ>

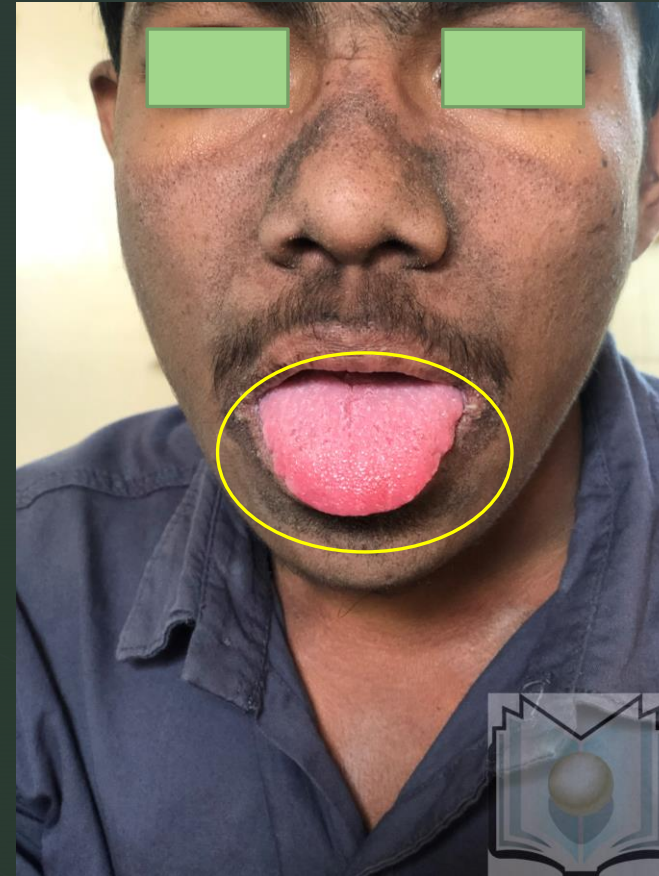
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Vitamin B3 deficiency

- Role
 - Lipoproteins and cholesterol metabolism - ↓LDL, ↑HDL
 - Neural and brain function
- Manifestation
 - Pellagra – dermatitis, diarrhoea, dementia
- Oral manifestation
 - Recurrent aphthous stomatitis, glossitis, dry mouth
 - Foul breath -> bacterial overgrowth -> caries (?)



Pellagra
- „gloves, socks, necklace“ style of lesions



Pale tongue

Vitamin B5 (pantothenic acid) and vitamin B6 (pyridoxine)

- Vitamin B5
 - Role
 - Energy metabolism, cholesterol and hormones synthesis, coenzyme A synthesis
 - Manifestation
 - Headache, nausea, muscle cramps, paraesthesia
 - ORAL – xerosis (dry mucosa), cheilitis
- Vitamin B6
 - Role
 - Haemoglobin synthesis, amino acid metabolism, protein synthesis
 - Manifestation (ORAL)
 - Angular cheilitis, glossitis
 - Recurrent aphthous stomatitis
 - Halitosis
 - Fungal infections (decreased immunity)
 - Pale mucosa (possible anaemia)

SOURCES



SUNFLOWER SEEDS
1,34mg(100g)



GARLIC
1,23mg(100g)



SALMON
0,8mg(100g)



BEANS
0,9mg(100g)



HAZELNUT
0,7mg(100g)



SESAME
0,79mg(100g)



WHEAT
0,6mg(100g)



BELL PEPPER
0,52mg(100g)

PYRIDOXINE

B6

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DAILY NORM



2 MG

B6

+

B2

+

Zn

BENEFICIAL FEATURES



**REDUCES TOXICOSIS
DURING PREGNANCY**



**PREVENTS
SKIN DISORDERS**



**REDUCES
NERVOUS
DISORDERS**



**REDUCES NIGHT
MUSCLE SPASMS**

DEFICIENCY



LOSS OF APPETITE



**DRY SKIN
AROUND THE EYES**



FATIGUE



**THE APPEARANCE
OF KIDNEY STONES**



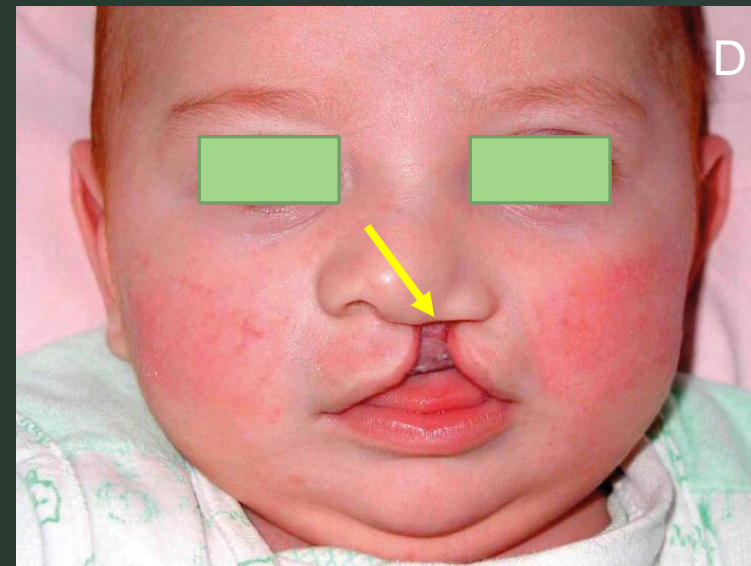
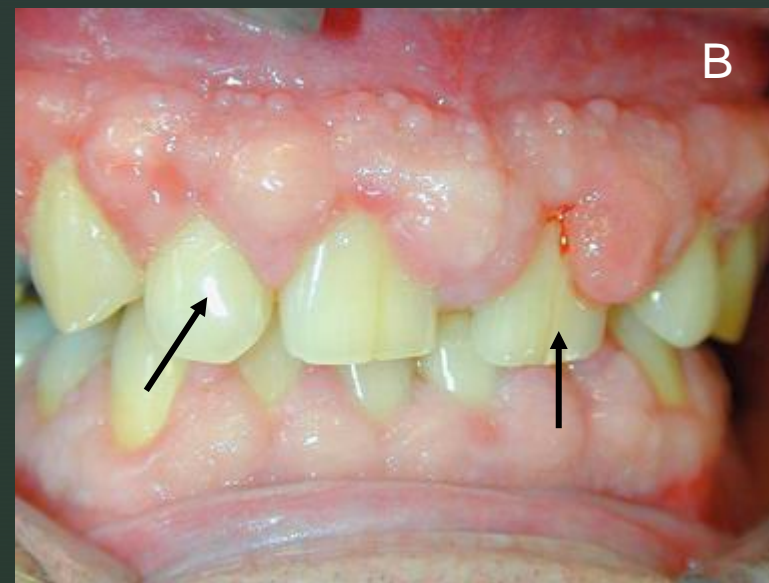
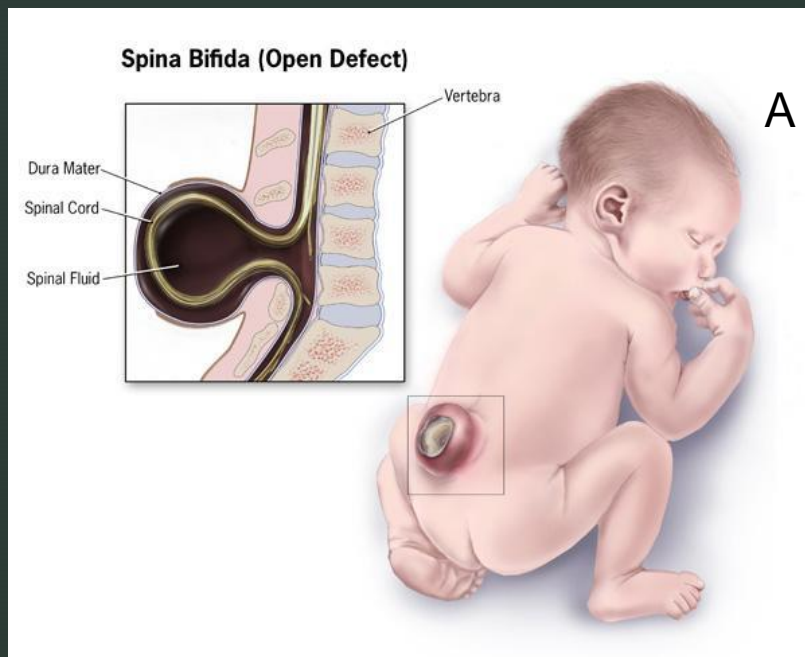
**SORES IN THE
CORNERS
OF THE MOUTH**



FOCAL HAIR LOSS

Vitamin B9 deficiency

- Role
 - DNA synthesis, tissue growth, cells function, neural tube development
- Manifestation
 - Megaloblastic anaemia, spina bifida occulta
- Oral symptoms
 - Gingivitis, angular cheilitis, glossitis
 - Cleft lip (unilateral mainly)



A – spina bifida occulta; B – gingivitis (swollen and bleeding gums)
C – recurrent aphthae, D – unilateral lip cleft

Vitamin B12 deficiency

- Role
 - Nerve cell function, mood-regulating neurotransmitters balance
 - DNA replication
 - ↓homocysteine level -> ↓cardiovascular risk
- Manifestation
 - Pernicious anaemia
 - Disrupted DNA synthesis -> less frequent and faulty divisions -> megaloblastic anaemia
 - Neural and cognitive defects, psychosis possible
 - Impaired myelin synthesis

Oral manifestations of vitamin B12 deficiency

- Anaemia
 - Mucosal pallor
 - Anaemic gingiva
 - Poor oxygen delivery suspected
- Dry mouth
- Glossitis („strawberry tongue“)
- Taste perception alteration



Exfoliative
glossitis



Vitamin C deficiency

- Role
 - Collagen synthesis
 - Collagen hydroxylation -> important for tissue repair, wound healing
 - Carnitine & norepinephrine biosynthesis, tyrosine metabolism
 - Peptide hormones amidation
 - Antioxidant (vitamin E synergy)
 - Cell signalling, epigenetics
 - Bacterial infections prevention

Vitamin C deficiency manifestation (scurvy) – oral cavity

- Gingival hypertrophy
 - Inability to produce procollagen -> tissue and vessels fragility -> prone to bleeding and inflammation -> overgrowth (cytokines stimulation)
- Deep dental pockets
- Bruising easily -> possible petechiae
- Delayed wound healing
- (?) Oral infections containment impaired



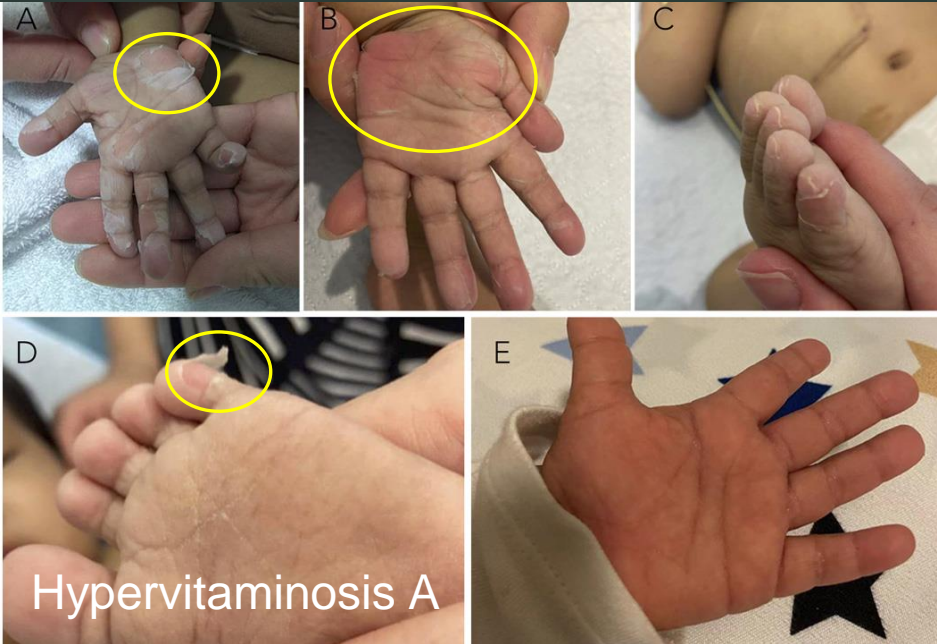
Ascorbic
acid therapy



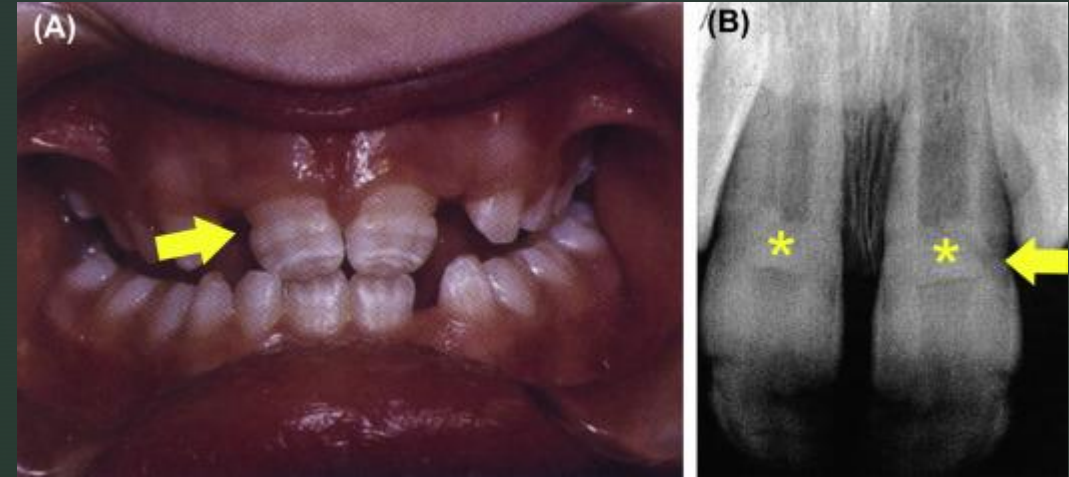


Vitamins overdose (rare)

Vitamin	Acute	Chronic
A	Headache, loss of consciousness Nausea, vomiting Irritability Fever, skin rash	Dry patchy skin Dry cracking lips Brittle nails Hair loss Movement stiffness (bones alteration)
D	Tremor (extremities) Dehydration Subcutaneous bleeding Congenital bone defects (pregnancy)	Pancreatic cancer risk Cardiovascular morbidity Fractures Enamel hypoplasia Dentine pulp calcification
E	Increased bleeding, impaired immunity, delayed wound healing Hypertension, angina pectoris, atherosclerosis Fatigue, intestinal cramps, diarrhea	
K	Hemolytic anemia (vit. K3 only)	
C	Nerves excitability Skin rash, itching	
B group	Various neural symptomatology mostly	



Hypervitaminosis A



Hypervitaminosis D

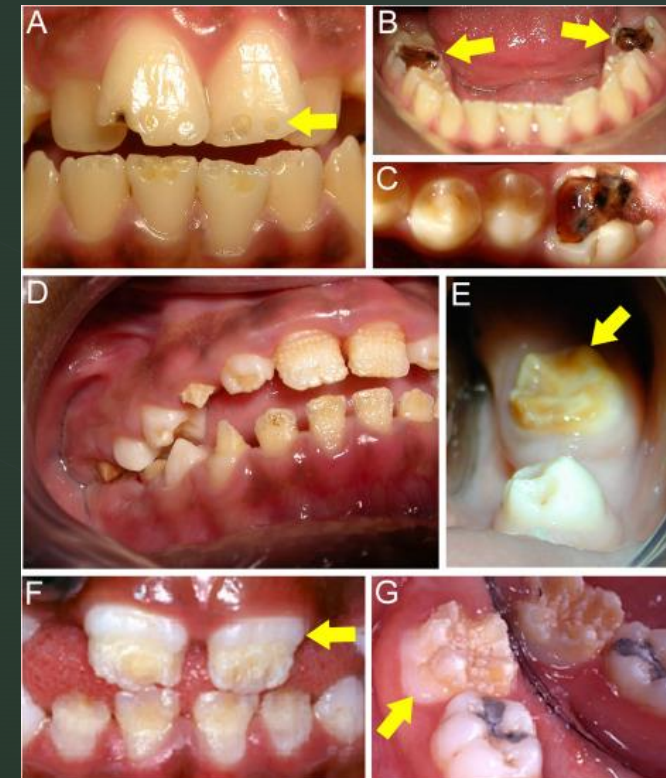
Vitamin C Toxicity
Maximum limit of vitamin C intake - 2000 mg/day
iCliniq
The Virtual Hospital

Symptoms of Vitamin C Toxicity

- Diarrhea
- Abdominal cramps
- Nausea and vomiting
- Headache
- Insomnia
- Fever

Severe toxicity causes

- Bone fractures
- Kidney stones
- Bleeding through the nose
- Muscle pain
- Seizures



<https://s3.amazonaws.com/images.icliniq.com/infographics/Vitamin+C+Toxicity+%282%29.jpg>

<https://ars.els-cdn.com/content/image/3-s2.0-B978012809965000029X-f29-03-9780128099650.jpg>

https://www.mja.com.au/sites/default/files/issues/217_11/mja2.51776.jpg

<https://ars.els-cdn.com/content/image/3-s2.0-B9780323913867000131-f24-02-9780323913867.jpg>

A burning question appears...

Vitamin K3 overdose may be manifested with jaundice?

TRUE

FALSE

Haemolytic anaemia
leads to serum bilirubin
elevation
(liver capacity reached)



Trace elements disorders

Iron

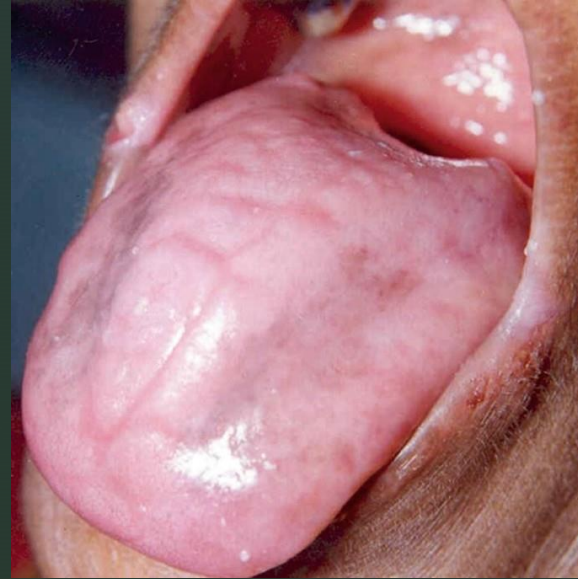
- Role
 - Part of proteins and enzymes
 - Haemoglobin, myoglobin
 - Cytochrome a-c, p450; cytochrome c reductase, catalases, peroxidases, xanthine oxidases, tryptophan pyrrolase, succinate dehydrogenase, glucose-6-phosphate dehydrogenase, and choline dehydrogenase
 - Energy metabolism
 - DNA, RNA, collagen, antibody synthesis

Oral manifestation of iron deficiency

1. Iron deficiency anaemia (microcytic hypochromic)
 - Pathomechanism – poor oxygen delivery, unable to contain infections -> subclinical chronic inflammation
 - Angular cheilitis
 - Atrophic glossitis
 - Mucosal atrophy, candida infections
 - Mucosal pallor
 - Stomatitis
 - Plummer-Vinson syndrome (Paterson-Kelly sy./sideropenic dysphagia)



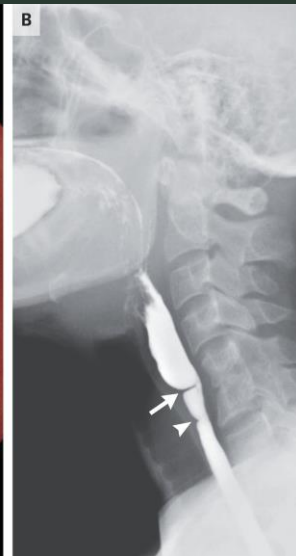
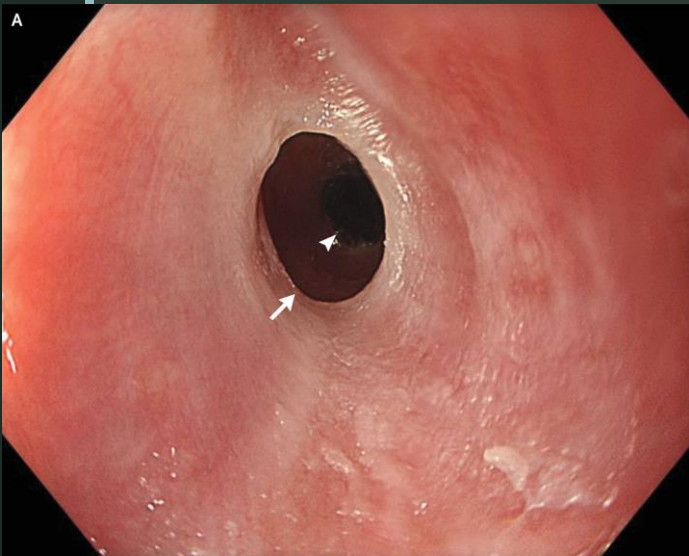
Angular cheilitis



Mucosal pallor



Median rhomboid glossitis



Plummer-Vinson syndrome

- Triad – dysphagia, iron deficiency anaemia, oesophageal webs
- Women slightly more affected
- Autoimmune part contribution
- Possible malignant transformation – squamous cell carcinoma of oesophagus



<https://www.ccjm.org/content/ccjom/85/8/581/F1.large.jpg>

https://media.springernature.com/lw685/springer-static/image/chp%3A10.1007%2F978-3-319-55387-0_4/MediaObjects/394201_1_En_4_Fig2_HTML.jpg

<https://www.researchgate.net/publication/51632598/figure/fig1/AS:202503011475475@1425291888626/The-appearance-of-median-rhomboid-glossitis-MRG.png>

https://www.nejm.org/cms/10.1056/NEJMicm2309721/asset/2bca29b1-3e0b-44bd-be1b-843072096cab/assets/images/large/nejmicm2309721_f1.jpg

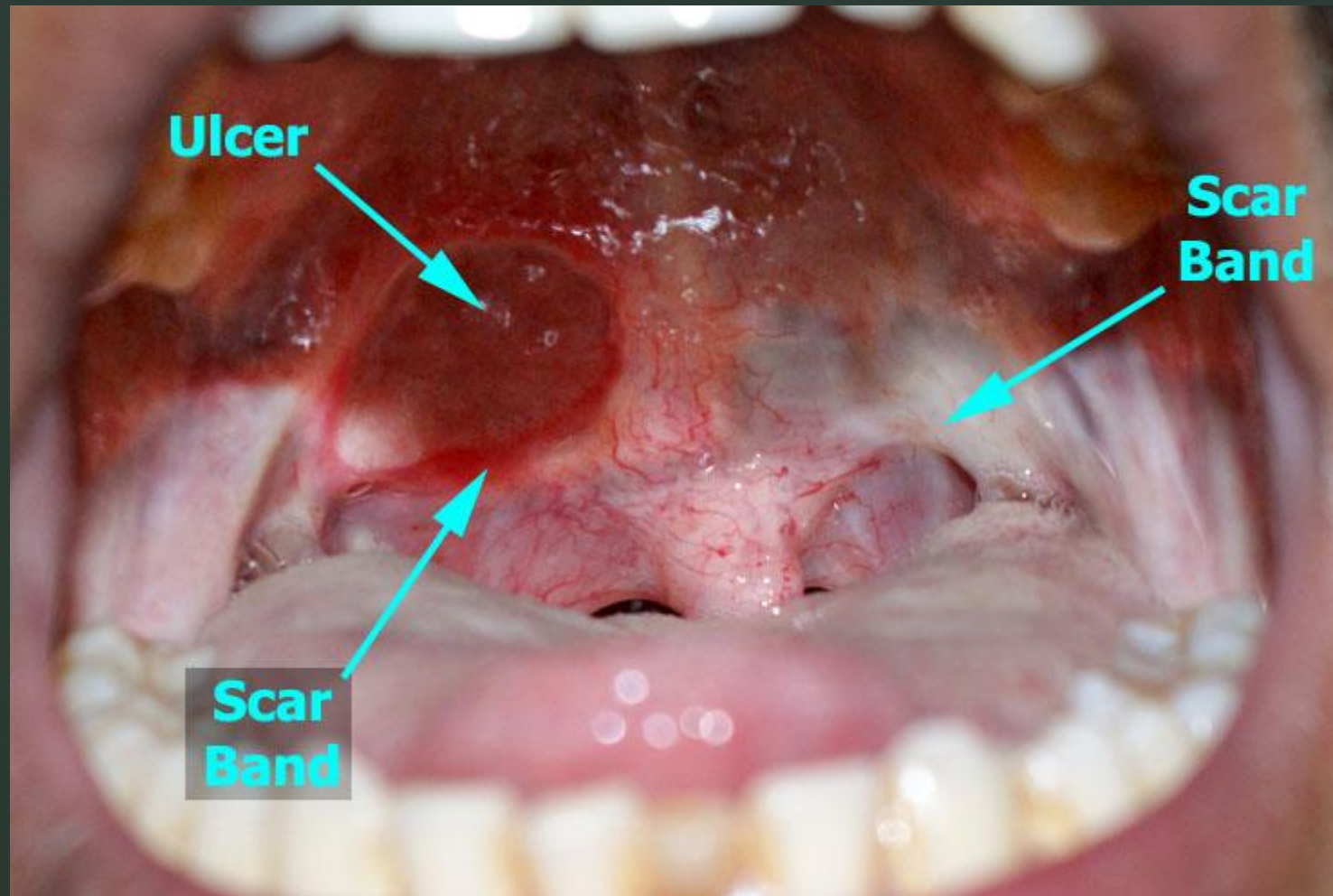
Oral manifestations of iron deficiency

2. Premalignant lesions

- \downarrow serum Fe^{2+} , \uparrow Fe^{2+} -binding capacity
- Promotion of oral submucous fibrosis (1% risk)
- Pathomechanism – poor oxygen delivery, constant inflammation, additional stimulation of cell division (e.g. arecoline from areca nuts)
 - Similar mechanism estimated during smoking – carcinogens, ROS

3. Tumour progression

- Similar mechanisms suspected



Oral submucous fibrosis

Copper

- Role – important part of various enzymes
 - Cytochrome c oxidase – energy production
 - Superoxide dismutase – antioxidant defence (superoxide to hydrogen peroxide)
 - Lysyl oxidase – collagen and elastin production
 - Ceruloplasmin – Fe oxidation -> haemoglobin synthesis
 - Tyrosinase – melanin production
 - Phospholipid synthesis -> myelin production
 - Thyroxin production
 - Free copper – oxidative stress

Oral manifestation of copper deficiency

1. Anaemia and defective keratinisation
2. Frequent inflammation and prolonged healing, protracted infections
 - Neutropenia observed, granulocytes maturation disorders
3. Osteoporosis (mostly jawbone and skull)
 - Lysyl oxidase and ascorbate oxidase impaired -> improper bone matrix synthesis

Oral manifestation of copper overdose

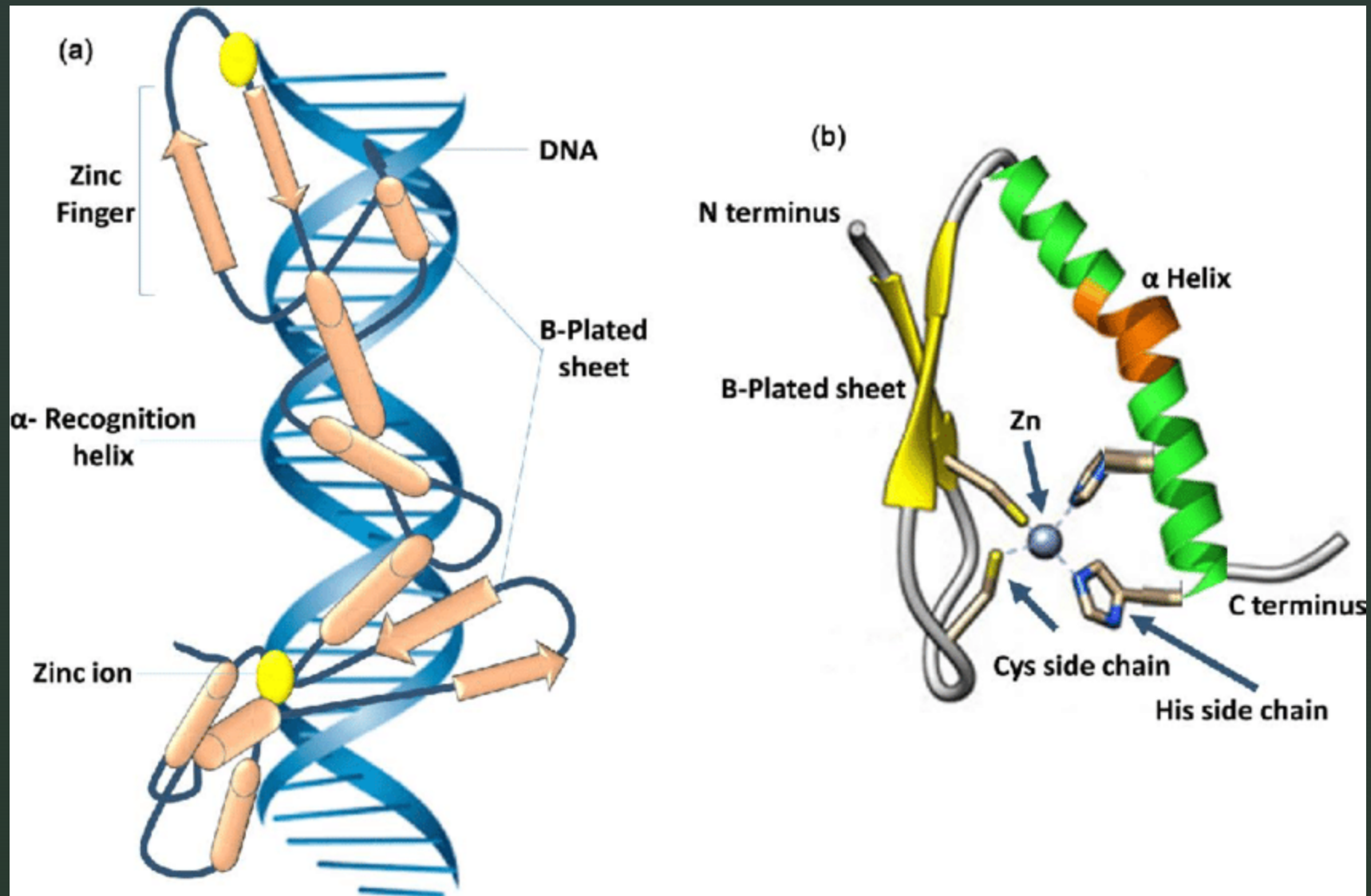
4. Premalignant and malignant lesions
 - Oral leucoplakia, oral submucous fibrosis
 - Increased risk of squamous cell carcinoma
 - Prominent in India and consumers of areca nuts
 - Serum copper levels were significantly higher compared to non-consumers
 - Salivary concentration seems to be decisive
5. (?) Cariogenic properties



Acute intoxication by copper sulfate (pond algaecide) – intake for 2 days (to treat „black tongue“, advised by friends), discoloration for 2 months due to copper + poor hygiene
Treatment – 1 month prednisone, 1 week fluconazole (BOTH INEFFECTIVE); chelation (EFFECTIVE)

Zinc

- Role
 - Catalytic activity of enzymes
 - Immune function, wound healing
 - Zinc fingers -> protein synthesis, DNA synthesis, cell division
 - Taste and smell perception
 - Growth and development of an organism
 - Antioxidant defence
 - Antimicrobial properties



Oral manifestations of zinc deficiency

1. Cariogenicity (disputed)
 - Plaque control, malodour control, calculus formation cessation
2. Taste disturbances
 - Impaired perception and neural transmission
3. Parakeratosis and loss of filiform papillae
4. Premalignant and malignant lesions
 - Oral leucoplakia and squamous cell carcinoma
 - Lack of zinc cannot counter free copper action

Oral manifestations of zinc deficiency and overdose

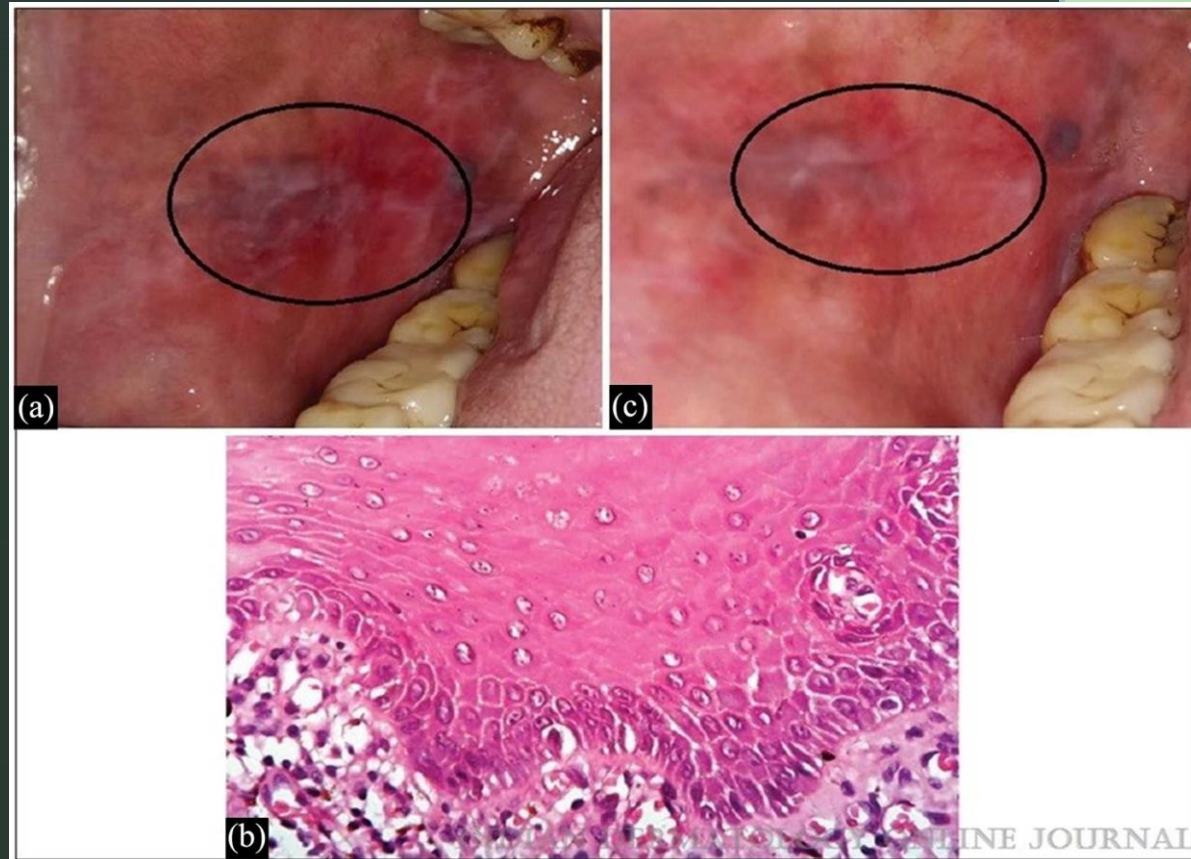
1. Superoxide dismutase (Cu-Zn)
 - Anti-carcinogenic properties -> impaired in zinc deficiency
 2. Impaired inactivation of copper-related enzymes
 - ↑lysyl oxidase -> ↑collagen cross-links -> malignant transformation factor
 - Zinc counters mucosal copper absorption (physiologically protective)
-
1. (OVERDOSE) - Oral submucous fibrosis
 - Serum zinc increased with drop of iron concentration
 - Carcinogenesis disputed



© Jere Mammino, DO

Acrodermatitis enteropathica
(zinc deficiency, congenital, autosomal
recessive)

https://www.aocd.org/resource/resmgr/ddb2_high/Acrodermatitis_enteropath_1_.jpg
<https://html.scrip.org/file/1111758-rld21.jpeg?20240724104417>



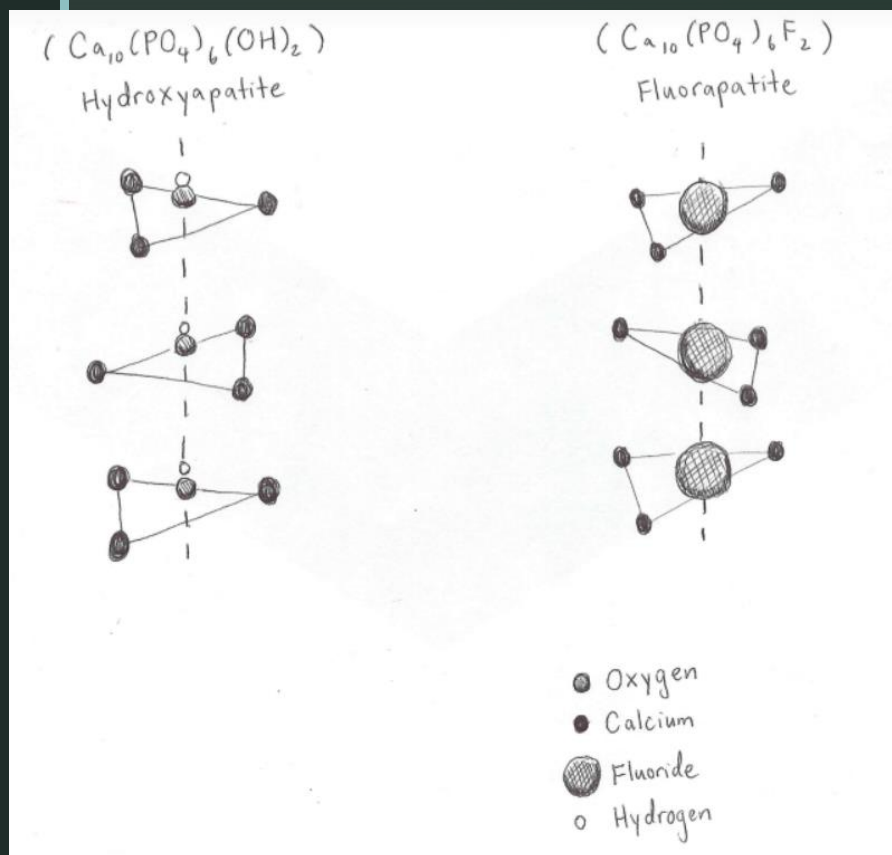
Lichen planus improvement – a) before
zinc supplementation, b) after, with
reduction of erosive patterns (c)

Cobalt, chromium, molybdenum, selenium

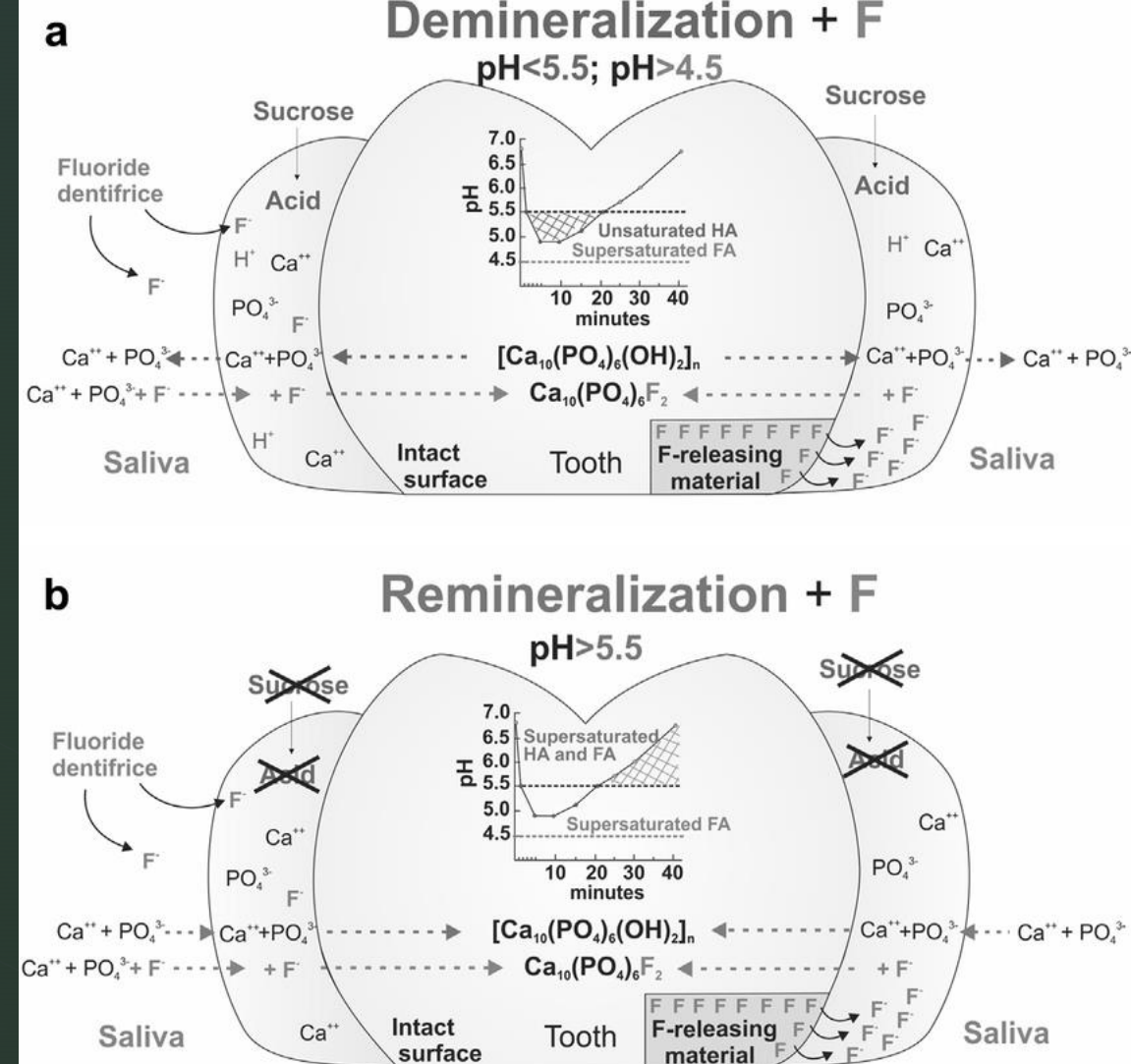
- Cobalt deficiency -> see vitamin B12
- Cobalt, chromium, nickel exposure -> lichen planus, lichenoid reaction
- Chromium deficiency -> hyperglycaemic effect
 - Intensifies diabetic syndrome manifestation
- Selenium deficiency -> possible premalignant and malignant transformation
 - ↓glutathione peroxidase levels
- Vanadium, boron, molybdenum -> possibly cariostatic (inconclusive)

Fluorine

- Role
 - Most negatively charged element -> most reactive
 - Fluorapatite formation -> acid resistant
 - Fluorohydroxyapatite has both some OH-groups and some substituted by fluorine
 - Prevention in childhood has protective effects also on permanent dentition
 - Prevention of cariogenic bacteria growth
 - Promoting remineralisation -> countering bacterial acid effect
 - Osteoblastic activity promotion
 - Synergy with calcium



Fluorapatite – OH-groups fully substituted by fluorine



Ca-hydroxyapatite loss compensated with fluorapatite creation (enhanced then with Ca-hydroxyapatite recreation during pH compensation)

Oral manifestation of fluorine deficiency and overdose

- Deficiency
 - Poor remineralisation of teeth
 - Increased caries occurrence
 - -> ↑enamel damage by bacterial acids
 - ↑plaque formation -> ↑cariogenic bacterial behaviour
- Overdose (fluorosis)
 - Brown stains and marks -> cosmetical
 - No increased caries risk
 - Severe may lead to permanent tooth damage



Severe fluorosis with mottled enamel – region with high natural occurrence of fluorine

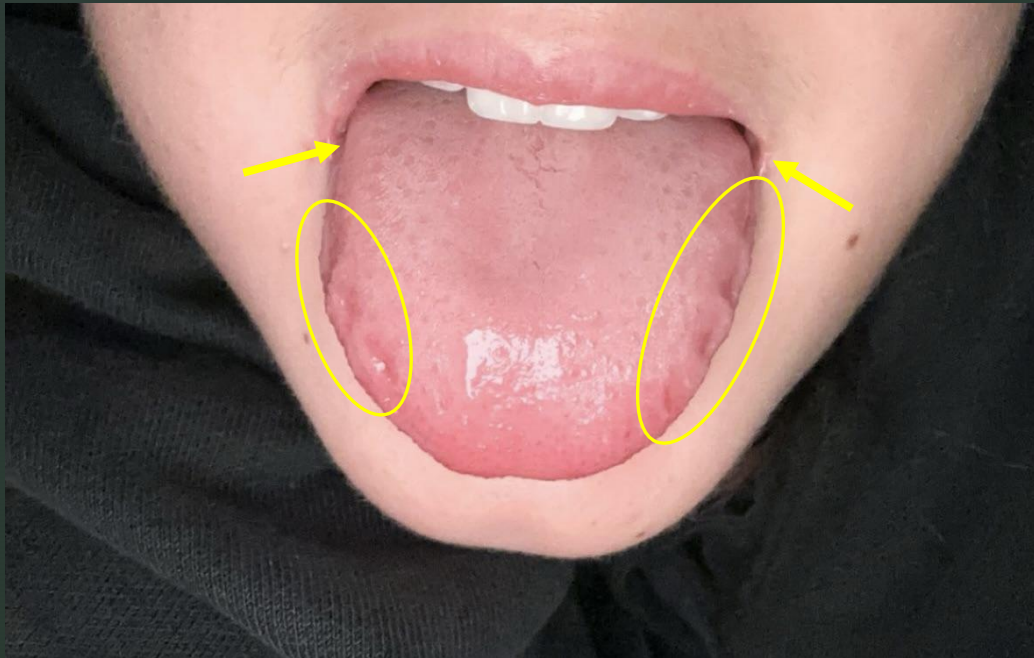
Iodine

- Role
 - Thyroid hormone synthesis
 - Immunological function and antioxidant defence
 - Mostly in primitive life forms
 - High concentration in thymus
 - Salivary glands prevent peroxidation by Na^+/I^- symporter

Oral manifestations of iodine deficiency and overdose

- Deficiency
 - Hypothyroidism -> deposition of glycosaminoglycans
 - Lips thickening
 - Macroglossia
 - Delayed teeth eruption
- Overdose
 - Bleeding ulcerations (GIT)
 - Hyperthyroidism – brown pigmentation – buccal mucosa, gingiva, palate, tongue
 - ↑melanin production (unknown means)

<https://pocketdentistry.com/hyperthyroidism/>
<https://i.redd.it/7a8s2k7loo6c1.jpeg>



Low thyroid function – „tooth“ prints on tongue
and no space between tongue and lips
(Hashimoto's thyroiditis on the picture)



Hyperthyroidism – pitted enamel, gingival
staining and tooth surface loss

Cariogenic versus cariostatic elements

Element	Mechanism of action	Element	Mechanism of action
Fluorine	↑enamel resistance Incipient lesions remineralisation	Selenium	(?) Dentin structure changes in mandibular condyles
Molybdaenum	(?) similar/synergy to fluorine (Hungary, New Zealand)	Cadmium	(?) Countering fluorine action
Vanadium	(?) limited proofs in hamsters, limited evidence in men	Lead	Ca^{2+} and PO_4^{3-} ions substitution Hypercalcemia, hyperphosphatemia induction
Strontium	↑content in younger teeth	Manganese	↑caries incidence in Mn-rich areas ↑young boys affected more
Lithium	(?) indirect, unclear	Copper	(?) unclear
		Zinc	(?) controversial



Malnutrition, imbalanced diet

Disorders of nutrients



Protein-energy malnutrition (PEM)

- Definition
 - One or more macronutrients are insufficiently supplemented by the diet
 - Can be selective (e.g. just proteins) or total
 - Lack of one nutrient may be connected with excess of another (e.g. kwashiorkor - ↓proteins, ↑sugars and fats)

Systemic manifestations of PEM

- Decrease in body weight (even up to 60 – 80 % of normal weight)
- Reduced metabolic rate, bradycardia
- Thin, dry skin, wrinkles, loss of pigmentation (?), delayed wound healing
- Prone to infections
- Delayed psychomotor development, impaired cognitive functions, apathy, lethargy
- Hypothermia or feeling constantly cold
- Oral health issues

KWASHIORKOR VS MARASMUS

- In preschool children (1-5 years of age)
- Due to low protein intake
- Mild growth retardation
- Mild reduction in body weight
- Protruding abdomen and subcutaneous fat reserved
- Ribs not very prominent
- Poor appetite
- Enlarged fatty liver
- Oedema present
- Moonfacies
- Sparse hair
- Flaky paint-like skin
- Lethargic
- Requires adequate amount of protein



Kwashiorkor

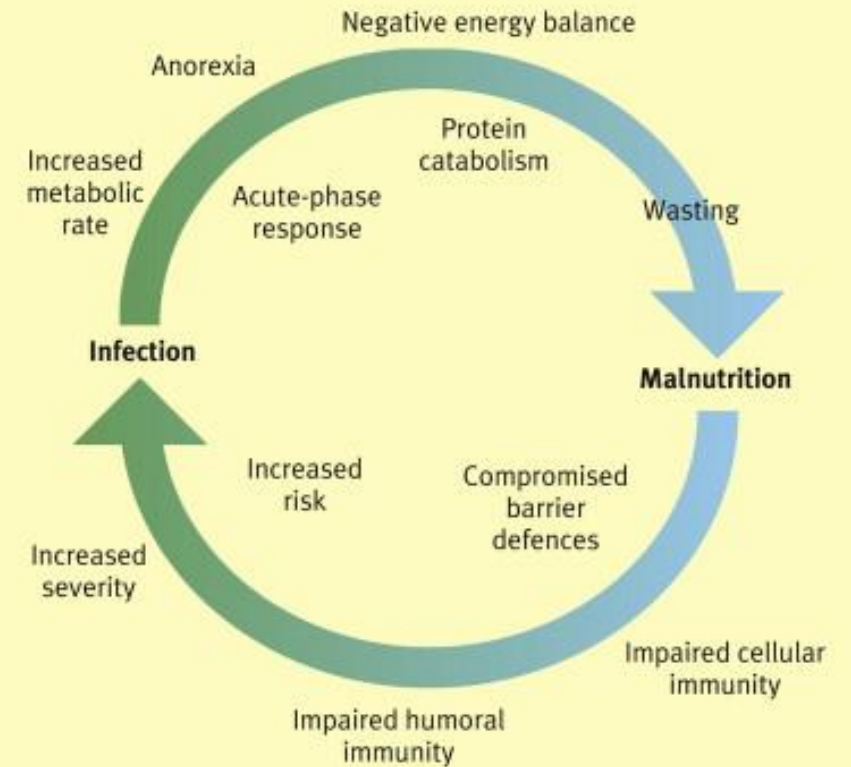
- In weakened infants (<1 year old)
- Due to low calorie intake
- Severe growth retardation
- Severe reduction in body weight
- Shrunken abdomen and subcutaneous fat not preserved
- Prominent ribs
- Voracious feeder
- No fatty liver
- Oedema not present
- An old man like face
- No hair changes noted
- Dry and wrinkled skin
- Alert but irritable
- Requires adequate amount of protein, fat and carbohydrate



Marasmus

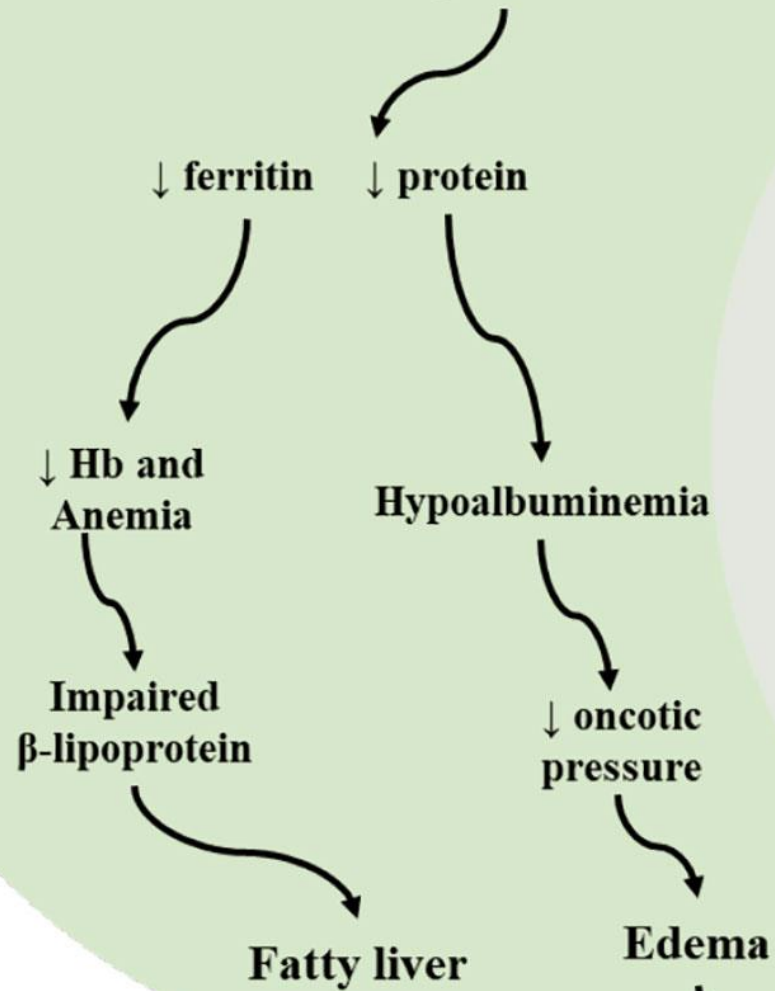


The vicious cycle of infection and malnutrition



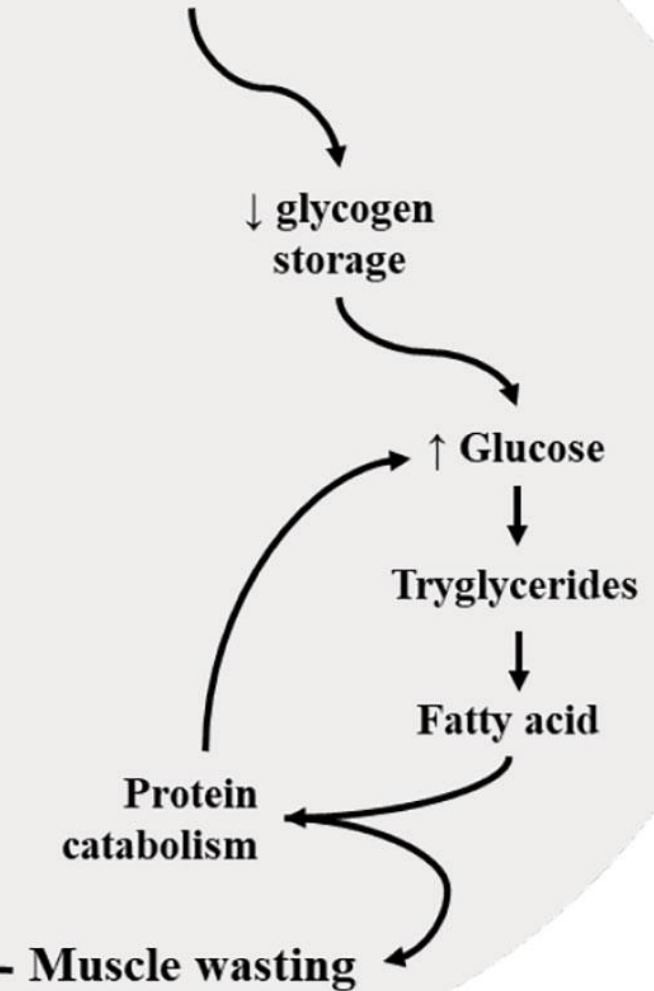
Kwashiorkor

Low dietary protein



Marasmus

Low dietary energy/calories



Marasmic-kwashiorkor

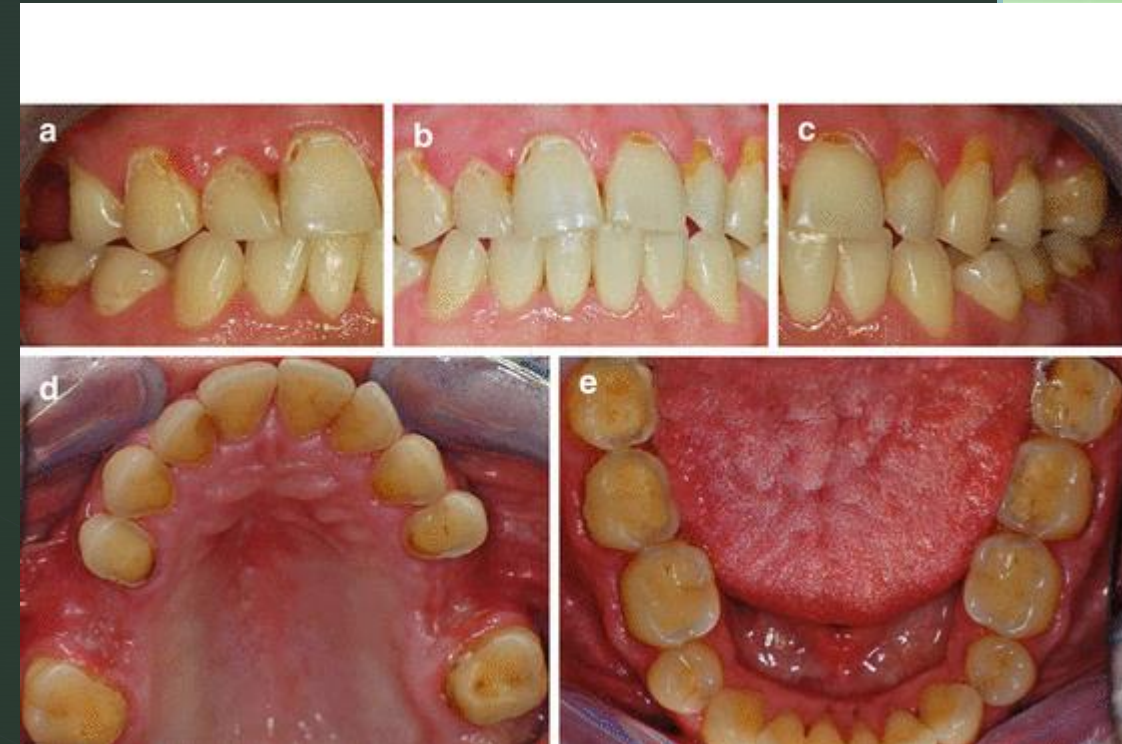
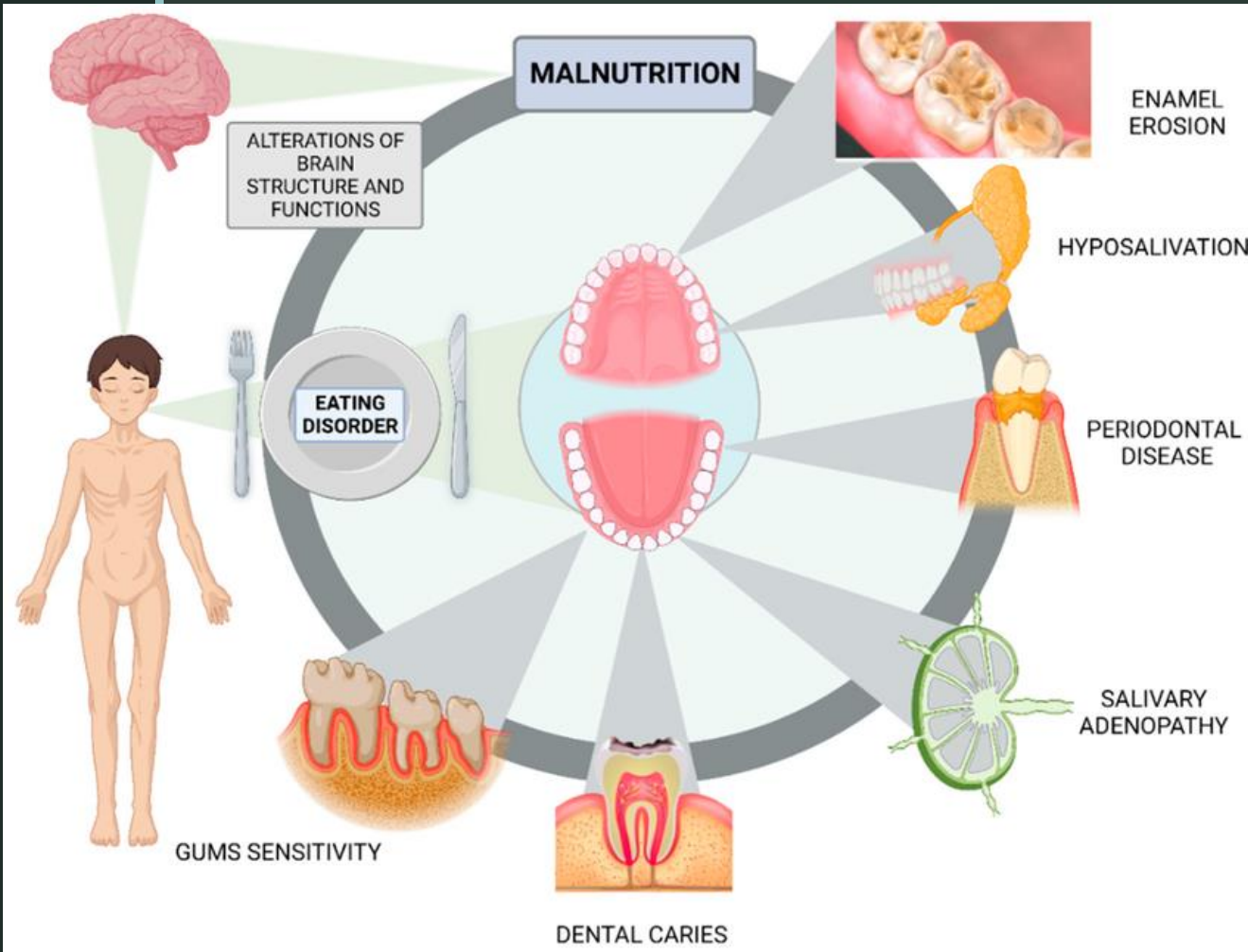


Oral health manifestations of PEM

1. Tooth defects
2. Salivary gland hypofunction
3. Increased risk of oral diseases
4. Oral tissue inflammation

Oral health manifestations of PEM – Tooth defects

- Delayed eruption of teeth
- Enamel hypoplasia
 - ↓vitamin A and vitamin D -> improper enamel formation
 - ↓proteins -> insufficient organic matrix -> ↓remineralisation, ↑demineralisation
 - ↓vitamin D -> hypocalcaemia
- Teeth may be smaller
- Tooth loss possible



Enamel erosion – „chalky“ teeth

Oral health manifestations of PEM – Salivary glands hypofunction

- Salivary production is energetically demanding
- ↓salivary flow -> xerostomia
- ↓proteins content -> ↓buffering capacity, ↓antimicrobial properties
- Salivary glands degeneration and/or atrophy
 - PEM + vitamin deficiencies (mostly A)
- Impaired taste perception

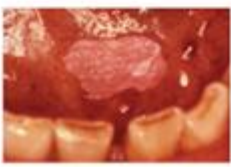
DRY MOUTH / XEROSTOMIA



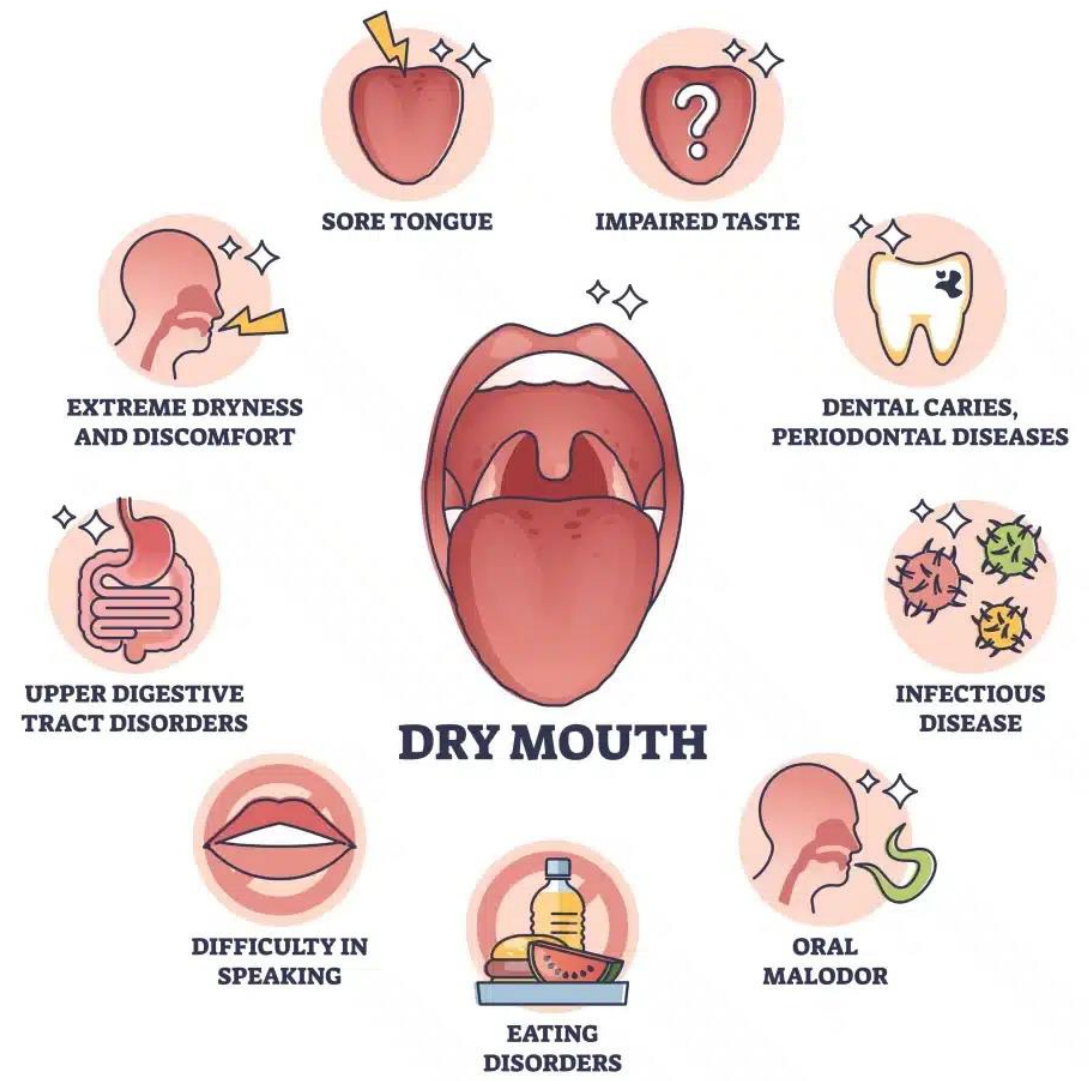
INCREASE OF PATHOGENIC BACTERIA
IN A PATHOGENIC BIOFILM



ORAL LESIONS



DENTAL CARIES



Oral health manifestations of PEM – Increased risk of oral diseases

- Impaired immunity
 - Unable to contain infections
 - ↑cariogenic/non-cariogenic bacteria ratio
- Reduced salivary defence
 - ↑plaque formation -> ↑cariogenic „behaviour“ of bacteria
- Delayed wound healing
 - PEM, ↓zinc, ↓vitamins -> ↓lack of macrophages (vitamin A) -> improper collagen synthesis, granulation tissue formation disorders
 - ↓containment of „non-sterile“ conditions -> ↑complications
 - Wound dehiscence, gingivitis, periodontitis, abscesses, bacteriemia, sepsis



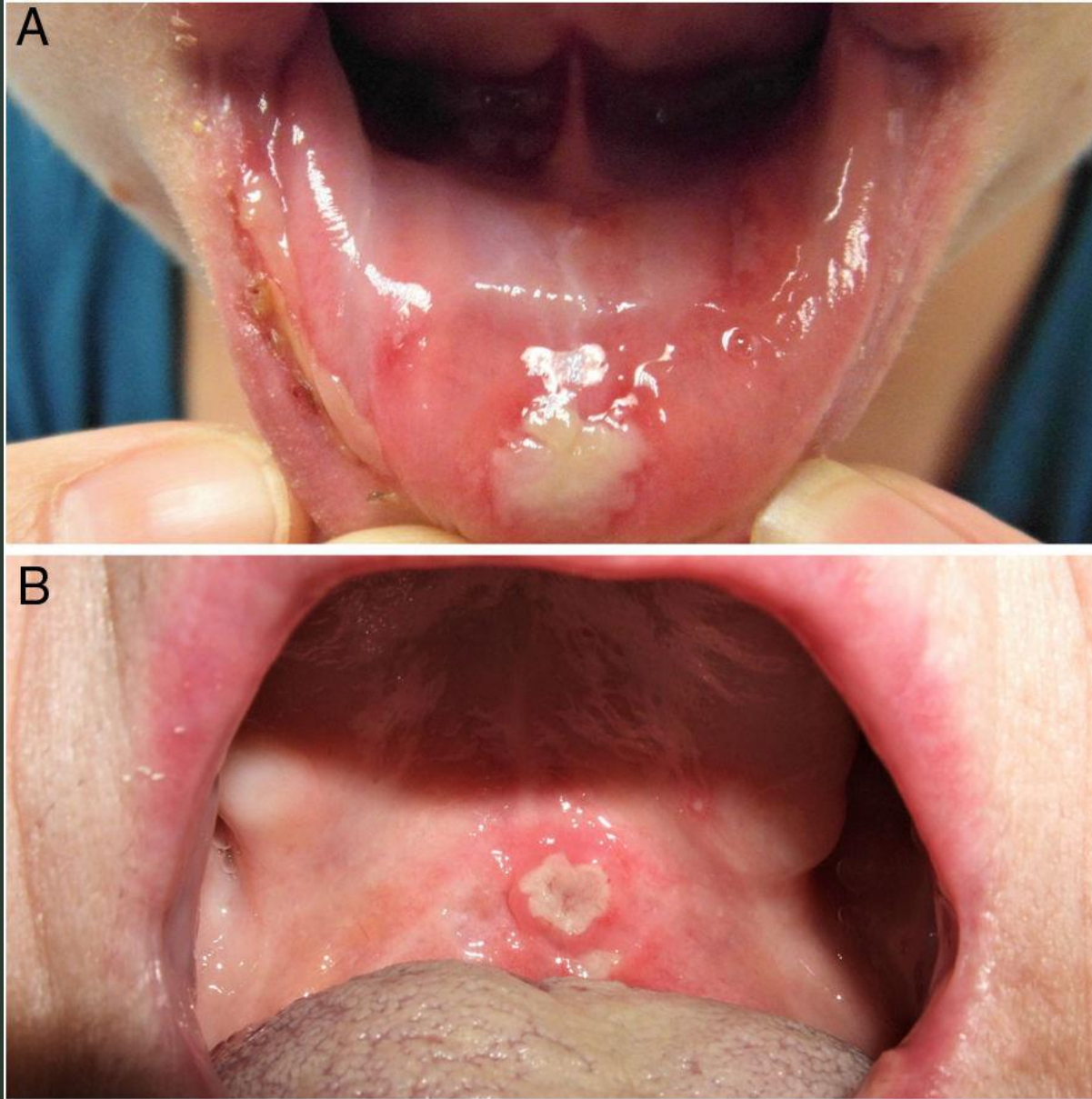
Wound dehiscence
Corticosteroids use, alcoholism,
thiamine deficiency



Chronic infection -> pus and fistula
Multiple diseases (mouth floor ca., Crohn's
disease, axonal polyneuropathy, ethanol abuse,
depression) led to secondary undernutrition

Oral health manifestations of PEM – Oral tissue inflammation

- ↓iron, folic acid, vitamin B12 -> recurrent aphthous stomatitis
- Repeated gingival inflammation -> transition to chronicity
 - Ligaments inflammation -> alveolar bone inflammation



Recurrent aphthous stomatitis

Oral health manifestations of PEM – Oral tissue inflammation

- Necrotising gingivitis
 - Malnutrition + poor oral hygiene
 - *Prevotella intermedia*, *Fusobacterium spp.*, *Bacteroides spp.*, *Treponema spp.*, etc.
 - Manifestations -> severe gum pain, profuse gum bleeding after minimal or no irritation, „punched out“ interdental papillae (ulcerated with dead tissue)
 - Foul breath, metallic taste
 - Fatigue, fever, cervical lymphadenopathy are very rare (compared to herpetic stomatitis)
 - Noma (*cancrum oris*) as a possible fatal complication
 - Rapidly-progressing gangrenous infection of mouth and face in debilitated children



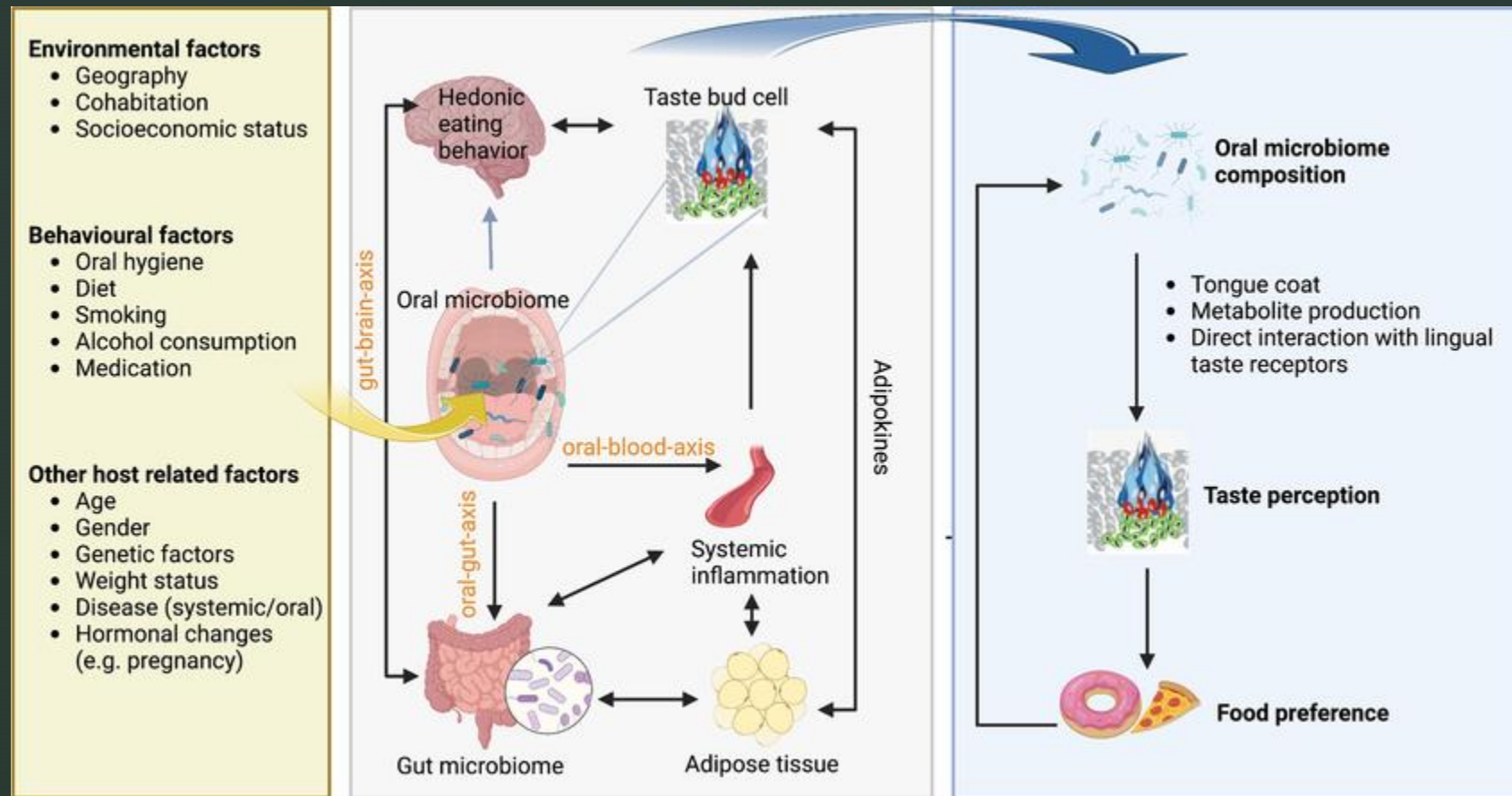
Acute necrotising ulcerative gingivitis



Noma (*cancrum oris*)

Overnutrition and obesity

- Overnutrition or increased caloric intake may lead to fat tissue accumulation
- Obesity and metabolic syndrome development
 - ↓insulin sensitivity (insulin resistance)
 - Low-grade systemic inflammation (below threshold of chronic inflammation)
- Oral and systemic health influence each other



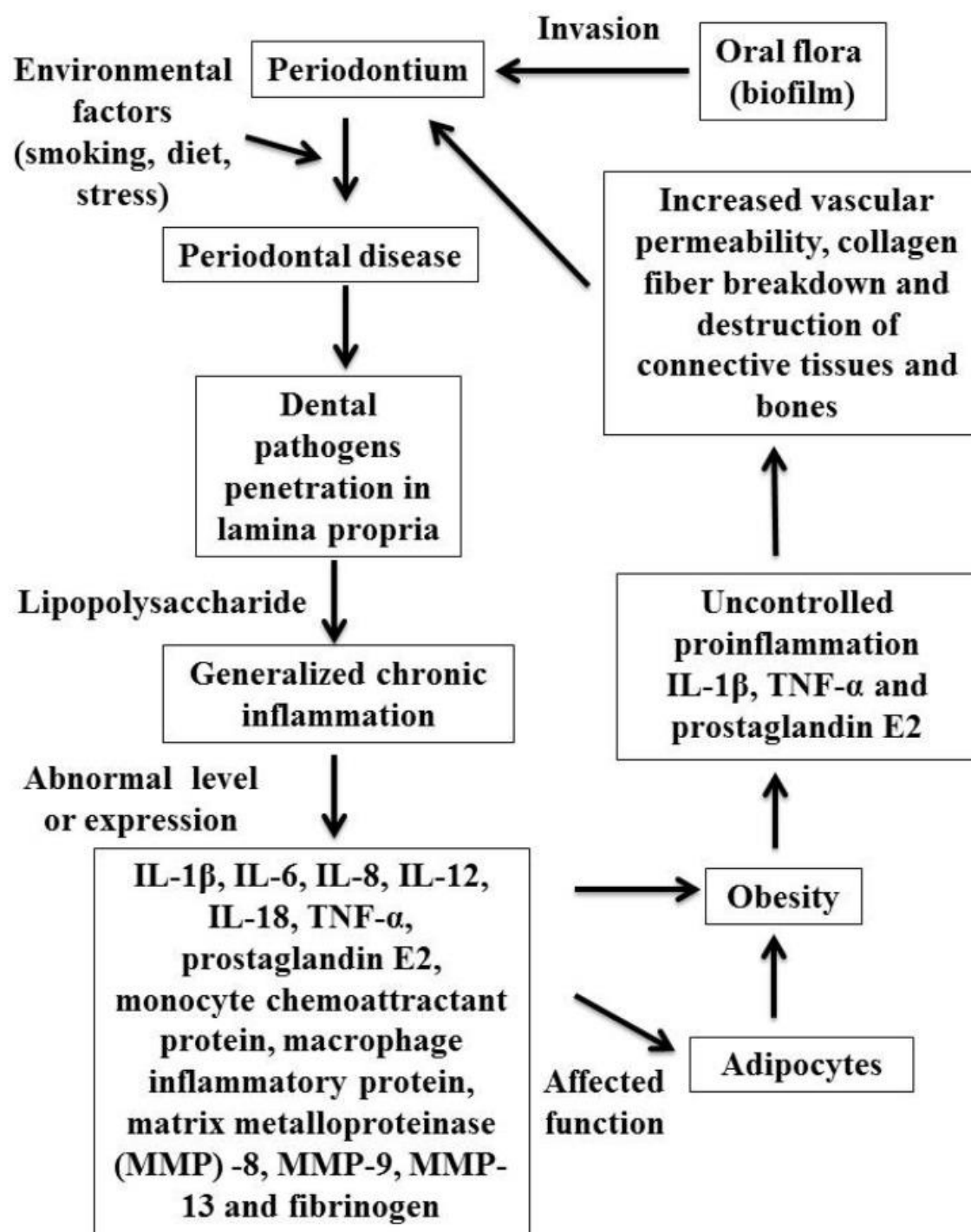
„Reprogramming“ of oral microbiota -> preference of „non-healthy“ food (dietary induced)

Oral manifestations of overnutrition and obesity

- Many findings controversial
 - Related to dietary habits more than to obesity directly
- Increased caries incidence
 - Less frequent eating, high content of sugars, snacking more than one time per day
 - \uparrow BMI \rightarrow \downarrow tooth brushing (≤ 1 time per day)
 - \uparrow BMI \rightarrow \downarrow salivary flow
- No significant relation between obesity and tooth loss

Oral manifestations of overnutrition and obesity – periodontal disease

- Obesity -> metabolic syndrome
 - Often combined with lack of micronutrients at expense of excessive sugar and saturated fats
- 1. Bacterial flora invasion
- 2. Pro-inflammatory cytokines production -> e.g. TNF- α , IL-1 β and IL-6
- 3. Adipokines secretion alteration -> pro-inflammatory adipokines prevail (e.g. leptin, resistin) and cytokines (PGE₂)
- 4. Vascular permeability increase, collagen fibers breakdown



Thank you for your attention!

