

source
RDA
function
deficiency
manifest

pattern

Vitamin - A.

Essay ✓

#1 pro-vitamin A
{ precursor of VA }
1 mark.

Beta carotene (plants)

→ sometimes 4 mark
then write pro vitamin of all
vitamin.

Sources.

- yellow and dark green vegetables and
fruits

↓
good source of carotene.

o Animal source.

↓
milk, butter, cream, egg yolk,

fisher liver oils.

RDA

{mark}

(recommended daily allowance)

Men

750 - 1000 $\mu\text{g/day}$

750 $\mu\text{g/day}$

women

400 - 600 $\mu\text{g/day}$

children

1000 $\mu\text{g/day}$

pregnancy

Retinoids

• compounds with vitamin A activity are collectively called as retinoids.

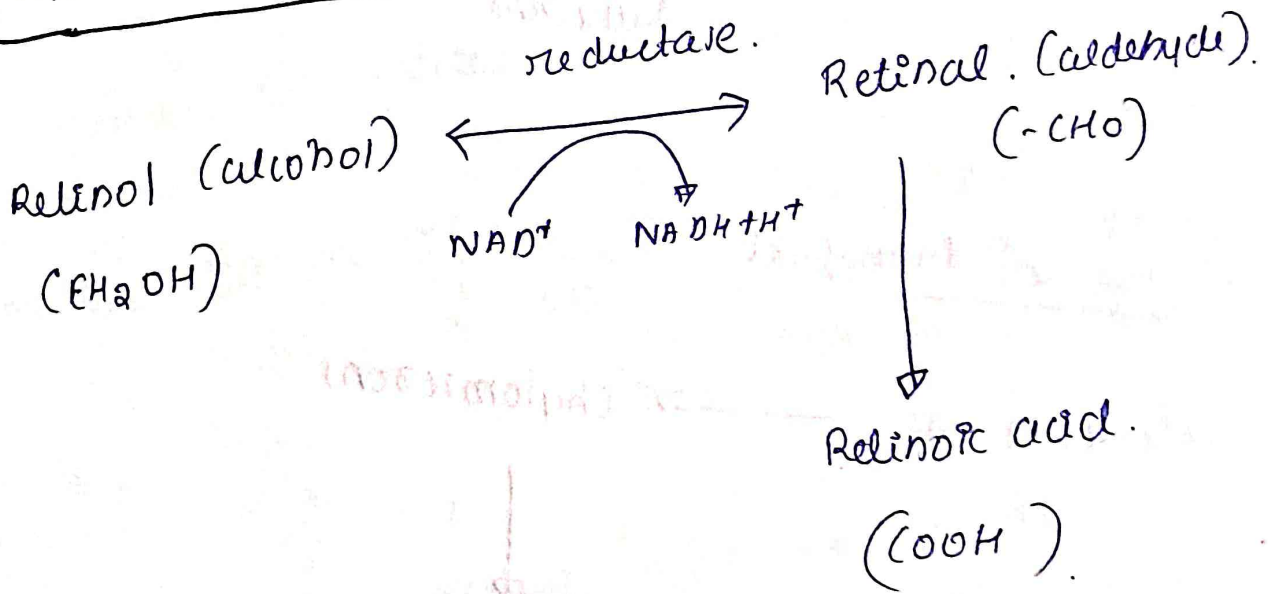
• all-trans retinal (vit A₁) → most common.

• 11 cis retinal → biologically active.

Compounds with Vitamin A activity. ✓

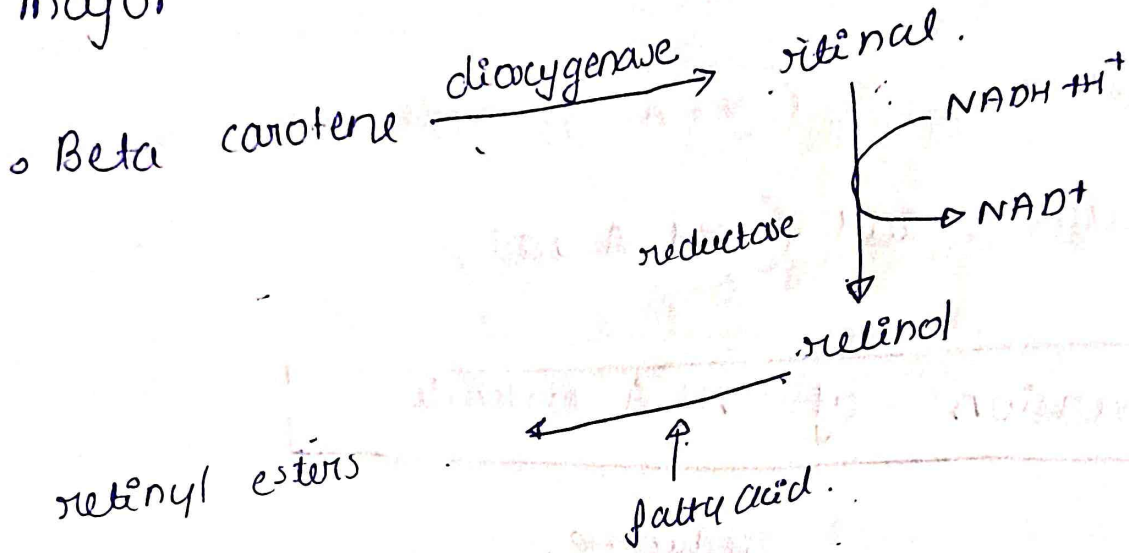
- retinol (vit A alcohol)
- retinal (vit A aldehyde)
- retinoic acid (vit A acid)

Interconversion of vit A molecules.

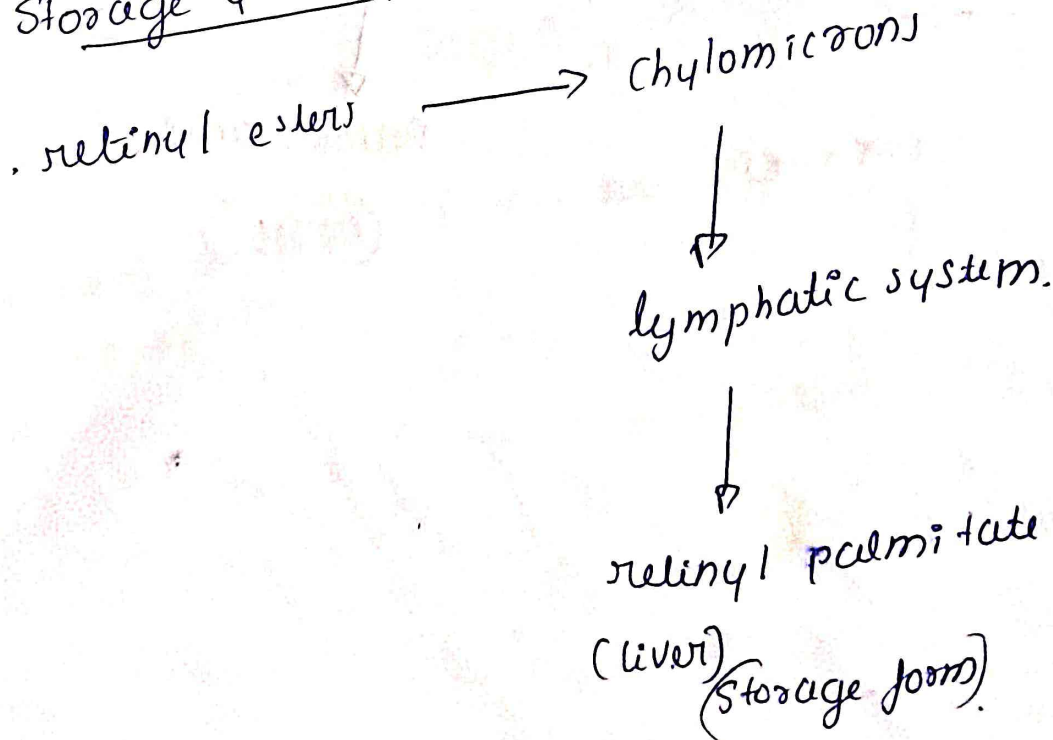


Absorption.

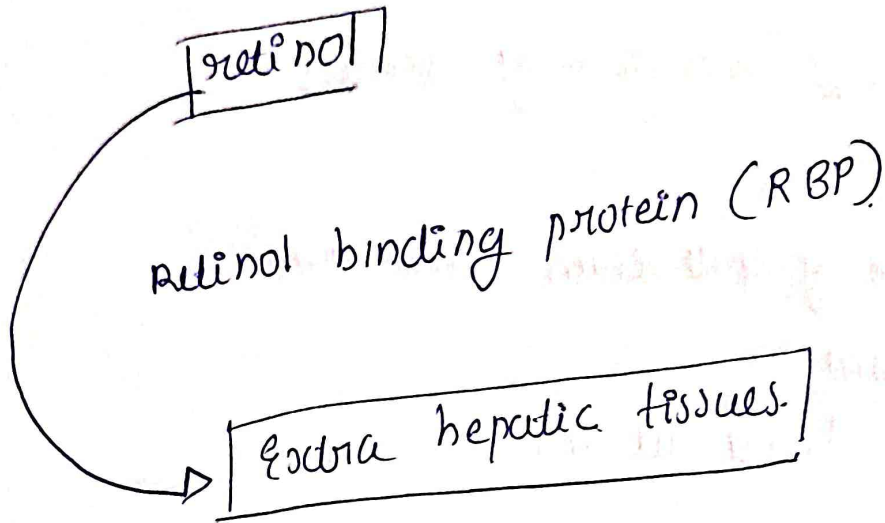
o major site - Intestine.



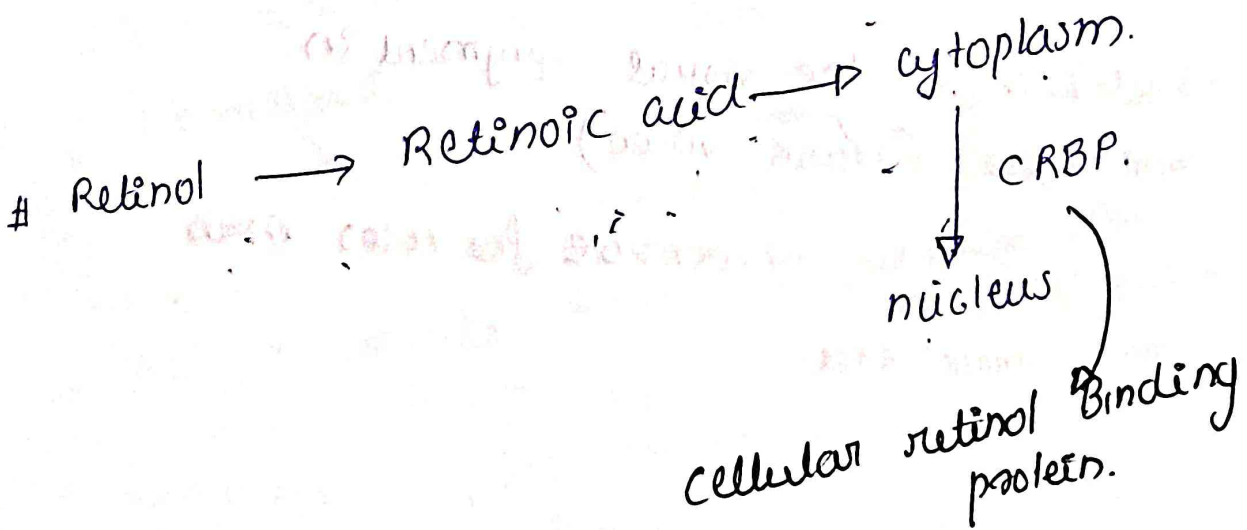
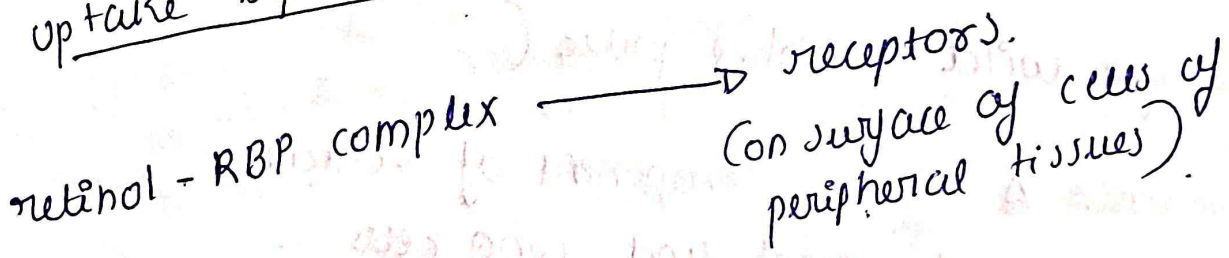
Storage & transport.



Release from liver.



uptake by tissues.

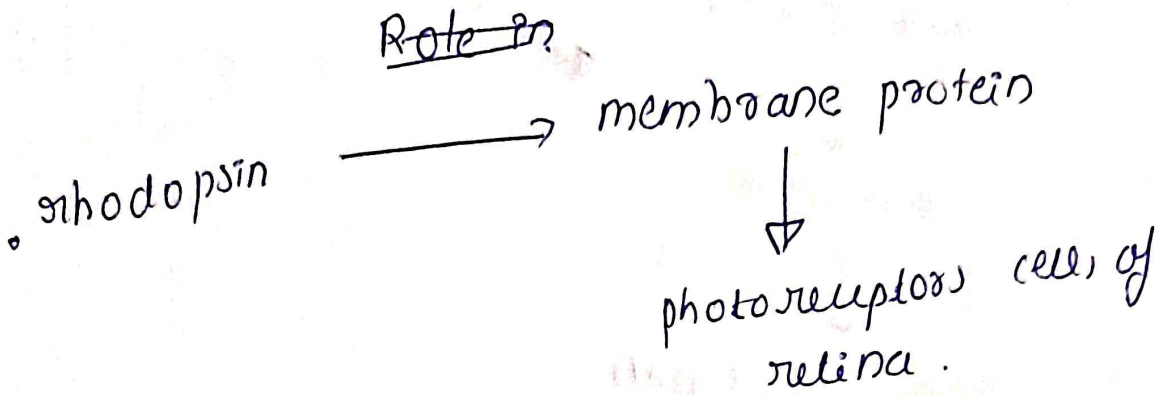


Functions of vitamin A.

- In vision
- growth & differentiation of tissues.
- reproduction
- maintenance of epithelium and skin
- anti-oxidant
- preventing heart attacks.

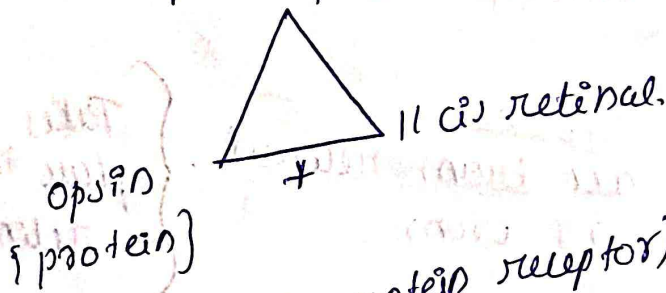
Role in vision.

- George wild Nobel prize (
- vitamin A is a component of visual pigments of rod and cone cells.
- Rhodopsin is the visual pigment in rod cells (dark vision)
- Conopsin is responsible for color vision in cone cells.



• vision in dim light.

Rhodopsin = opsin + 11 cis retinal.



• when rhodopsin is exposed to light, series of isomerization takes place.

• rhodopsin releases ~~all-trans~~ ^{11-cis} retinal & opsin

• This will activate G-protein, ~~transduction~~ transduction.

• Transduction begins nerve impulse transmitted by optic nerve to brain.

Series of Isomerizations,

Single photon

rhodopsin

Batho - rhodopsin

meta - rhodopsin 1

meta - rhodopsin 2

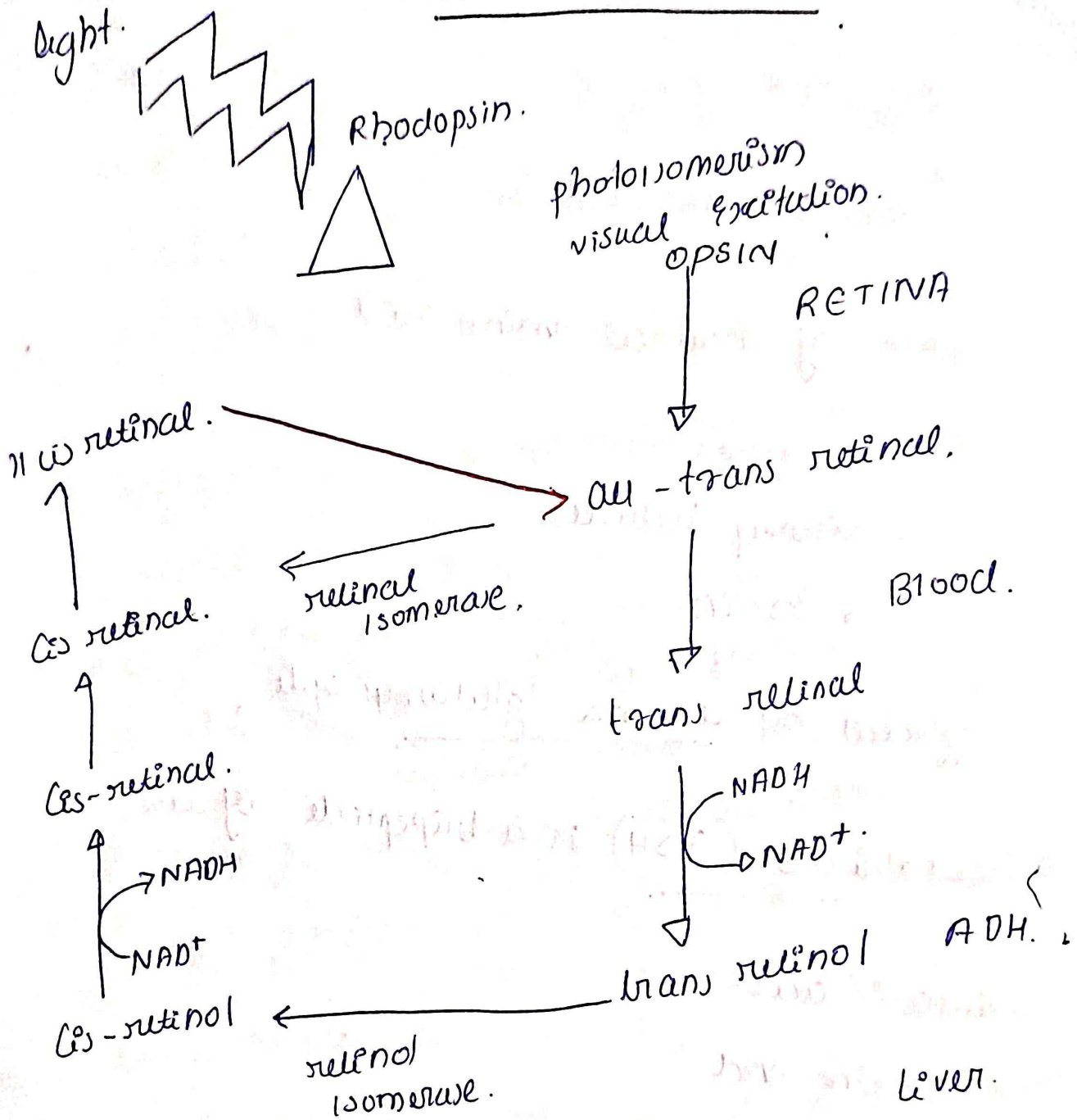
opsin + ^{11-cis} all ~~trans~~ retinal.
all trans

{ Takes place in retina }

vision

isomerisation steps

WAD'S VISUAL CYCLE.



regeneration of rhodopsin

ADH
→ alcohol dehydrogenase

Fig.

visual pigments - G protein coupled receptors.

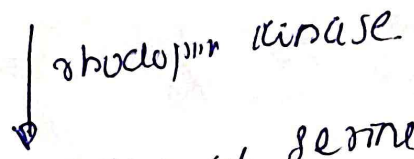
transducers - G proteins in retina.

Locking of 11-cis retinal with opsin.

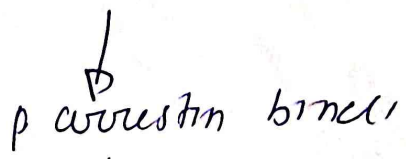


critical for pigment's catalytic activity.

activated rhodopsin.



phosphorylation of serine residue in rhodopsin



inactivation

Activated rhodopsin.

↓ rhodopsin kinase.

phosphorylation of serine residue in rhodopsin.

↓
β-arrestin binds.

↓
Inactivated rhodopsin.

Dark adaptation time.

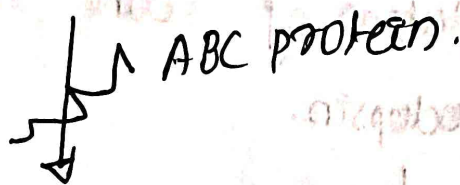
• Time required for re-synthesis of rhodopsin, when a person shifts suddenly from bright to dim light area.

• In increase in vit A deficiency.

Stargardt's disease.

all from retinal ABC protein.

all trans retinal from retinal epithelium.



Lives

Symptoms: macular degeneration + Blindness.

Deficiency manifestation

• Ocular manifestations.

• Siles and macular membrane lesions.

X → X } end of role of vitamin A in vision.

Ocular manifestation

- Night blindness (Nyctalopia)

Nyctalopia.

- xerophthalmia

all trans retinal
from. retinal.

- Bitot's spot

↓ ABC prot.

- keratomalacia.

- keratomalacia.

↓
• reduced vision in
dim light.

Nyctalopia (Night blindness).

- vision diminished in dim light ✓

- dark adaptation time increased ✓

- deficiency of vit A (It's retinal) ✓

xerophthalmia.

- conjunctiva becomes dry, thick & wrinkled.

- Cornea keratinized

- Normal transparency is lost.

- Cornea becomes glazy & lusterless due to
keratinisation of corneal epithelium.

Bitot's spot

- Increased thickness of conjunctiva in some areas.



- hoaryish - white biangular plaques in conjunctiva.

- These Three are completely reversible on supplementation of vitamin A.

Keratomalacia

- softening of cornea.
- degeneration of corneal epithelium.
- Corneal epithelium gets vascularised.
- Bacterial infections lead to corneal ulceration.
- pyorrhea of cornea.
- Total blindness.

{ Not reversible }

Causes of vitamin deficiency.

• Decreased intake of Vitamin A.

• Defective absorption { obstructive jaundice }

• reduced synthesis of RBP (cirrhosis of liver)

• Severe malnutrition - amino acids not available for RBP synthesis

• RBP excreted in urine - chronic nephrosis.

Hypervitaminosis A.

• Anorexia

• headache, irritability

• vomiting and drowsiness.

• Peeling of skin

• swelling over long bones (bony exostoses)

• Enlargement of liver in children

INTRACRANIAL TENSION

Eskimos - refrain from eating liver
of polar bear due to its high
vit A content.

cellular death -

higher conc. of retinol releases lysosomal enzymes
leading to cellular death.

H