Vision

Phototransduction of light

By

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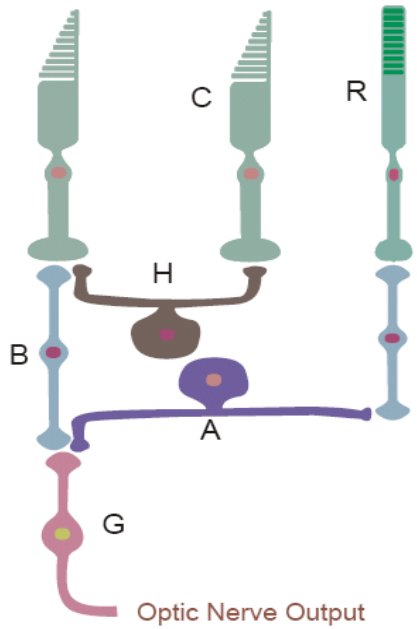
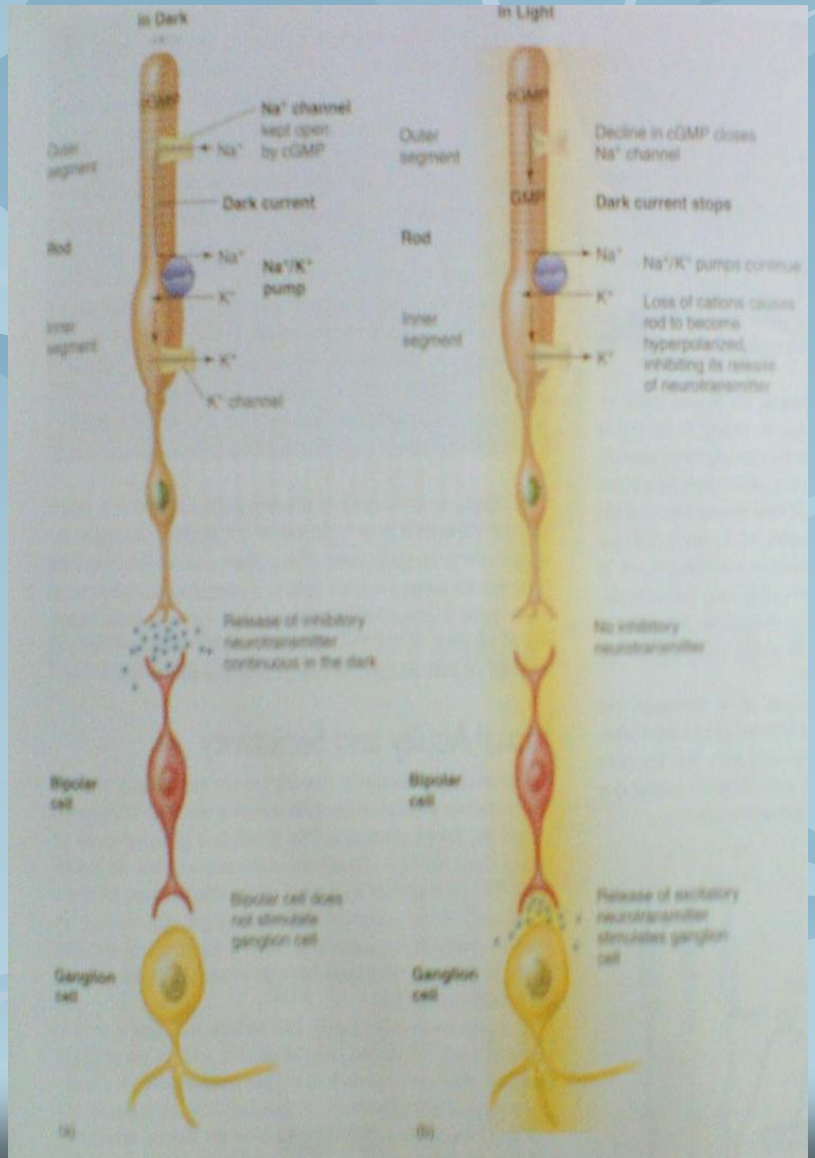
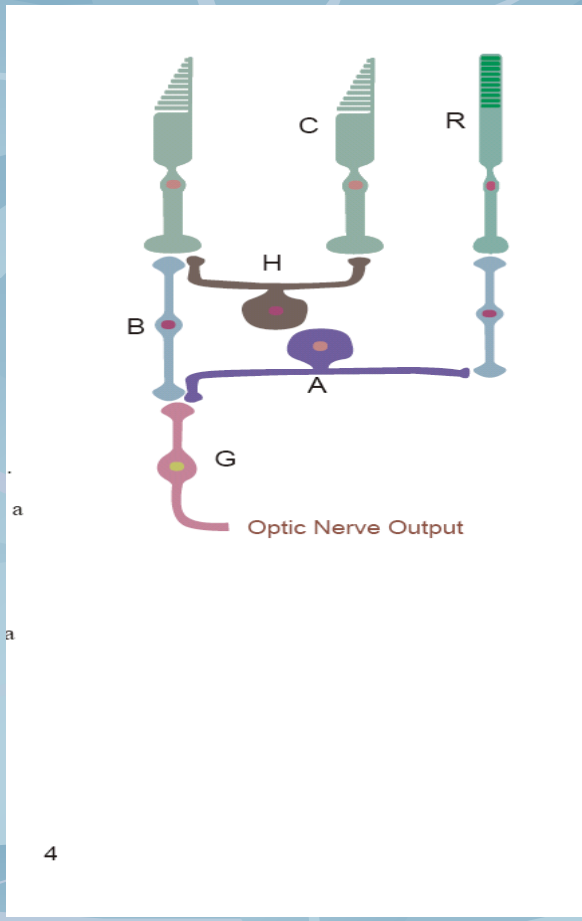
Shape of rodes& cones





- **Receptors of vision (Rods&cones):-**
- 1- Outer segment (modified cilia) has disks full of photosensitive pigment (rhodopsin) react with light to initiate A.P
- -In cones is conical , small and contain 3 types of rhodopsin
- - in rods it is big, rode like and contain one type of rhodopsin
- -There are Na channels in the outer segment
- 2- Inner segment full of mitochondria (source of energy for Na-K pump), it is thick in cones
- There is Na-K pump in inner segment



Visual Receptors: Rods and Cones

Rods

-abundant in the periphery of the retina

-best for low light conditions

-see black/white and shades of gray

Cones

- abundant in & around fovea

- best for bright light conditions

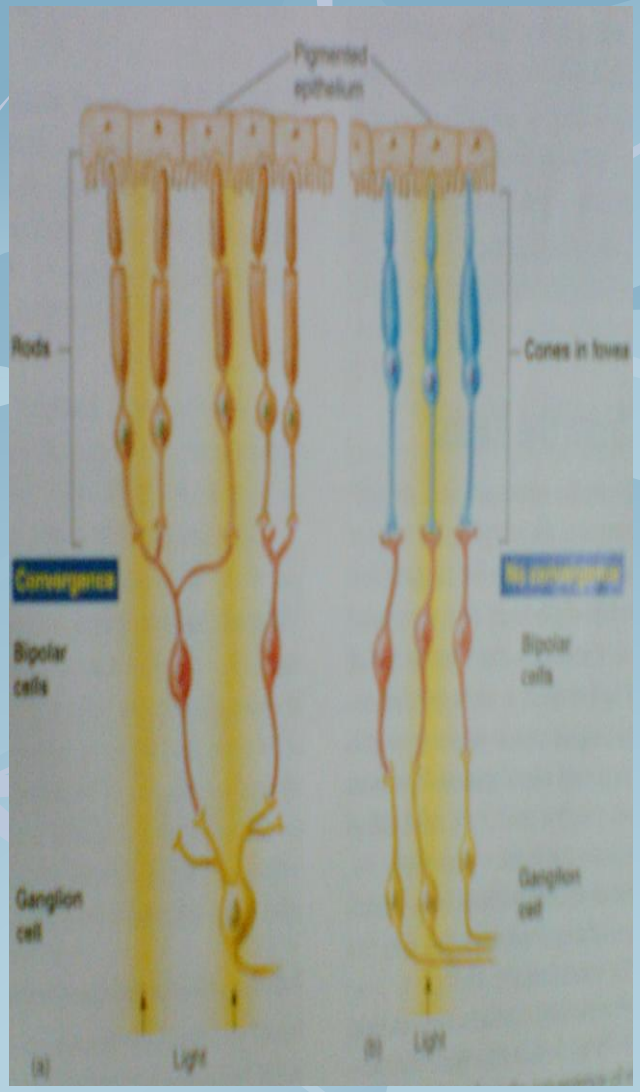
-see all colors

- **Convergence:-**
- **low convergence in cones / each foveal cone synapse with →one bipolar cell →one ganglion cell →single optic nerve fiber**
- **Value of low convergence //increases visual acuity → integrated information from small area of retina**

Disadvantage// decreases sensitivity to light i.e need high threshold of illumination to stimulate cones)

- 2- high convergence of rods/
- several rodes about 300 synapse with one bipolar cell& one ganglion cell
- -high convergence/// decreases visual acuity acuity = integrated information from large area of retina
- - but increases sensitivity to light i.e so low light threshold stimlate the rods)

- 3- 120 million rode& 6 million cone &1.2 million optic nerve fibers , so convergence is 105 receptor : 1 fiber.



peripheral rods

large spacing (lower density)

large convergence

G large ganglion cell

Integrates information from a large area of retina (3 deg)

large spacing and large convergence result in low acuity

foveal cones

high density

small convergence

G small ganglion cell

Integrates information from a small area of retina (.03 deg)

small spacing and low convergence result in high acuity.

By daylight, only the central fovea sees clearly & in color.
On a dark night, only the periphery sees, only in black & white, and with poor resolution.
The fovea is blind. only the periphery sees, only in black & white and with poor resolution.
The fovea is blind.

Genesis of photoreceptor potential

- -Rodes &cones potentials are graded, local potential (generator potential) propagated as A.P in ganglion cells.
- -Ganglion cell action potential (all or none A.P) transmitted to optic nerve.
- Rodes &cones & horizontal cells & Bipolar cell responses are depolarization at dark and hyperpolarization at light

- **- Cones respond to high levels of light intensity (illumination)**
- **-Rods respond to levels of light intensity (illumination) below threshold levels for cones, so rods are more sensitive**
- s

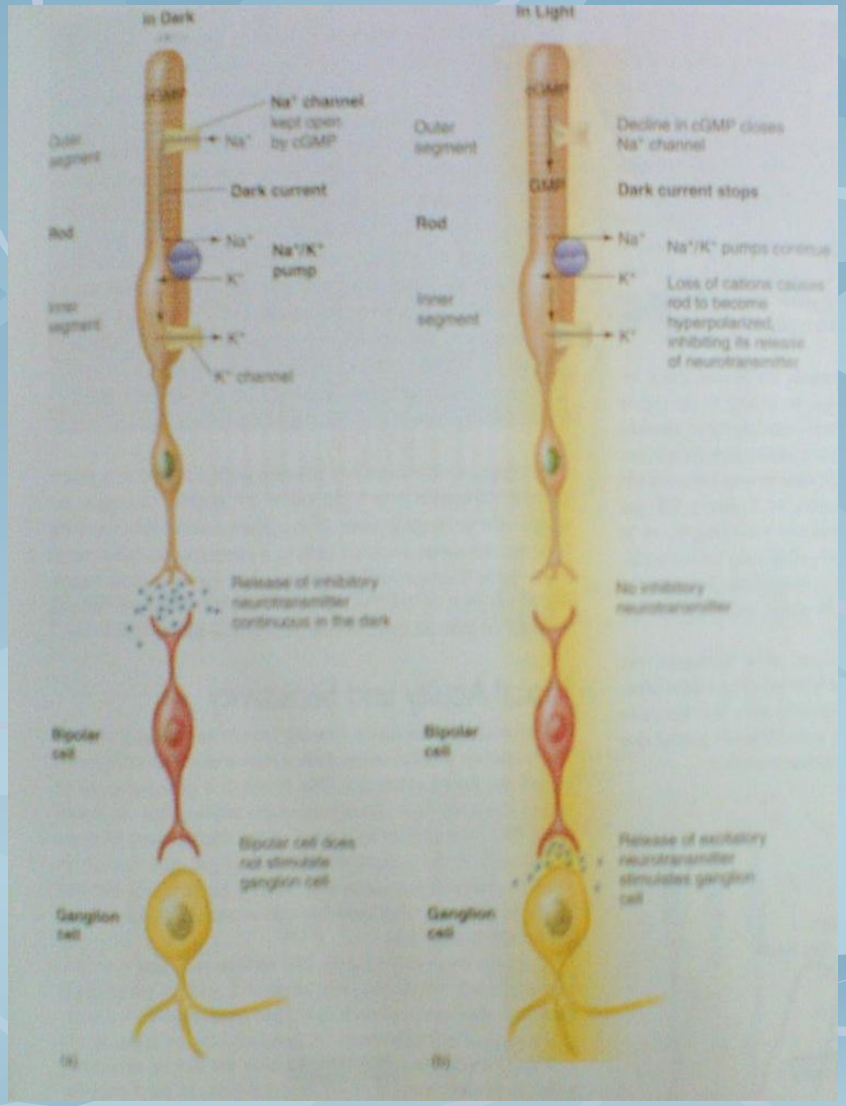
- **Photosensitive compounds:-**
- 1- In cones it is rhodopsin formed of :-
- Opsin protein + retinene 1 (retinal = aldhyde form of Vit A) = visual purple
- 2-There are 3 types of rhodopsin in 3 types of cones each respond to a certain wave length of light
- 3-In Rods its rhodopsin formed of /
- Scotopsin protein + retinene 1
- It is stored in disks of rods at outer segment
- -It forms (90% of its protein)
- -At dark rhodopsin is in 11-cisretinal form (inactive) but light sensitive form which increase sensitivity of rods to light

- Ionic basis of photoreceptor potential at dark
- -In dark Na channels in rods outer segment are open
- -Na-K pump in inner segment pump Na
- -Na flow from inner to outer segmet (called Na current) ⟶ Depolarization flow to synaptic endings ⟶ steady release of neurotransmitter at synapses with bipolar cells ⟶ which get depolarization potential ⟶ganglion cells

ELECTROPHYSIOLOGY OF VISION (PHOTOTRANSDUCTION)

- **A-At Dark (scotopic vision, dimlight vision):-**

- **1-Rhodopsin in 11-cisretinal (inactive form-light sensitive form which increase sensitivity of rods to light)**
- **2- (5 –GMP) in the c-GMP form**
- **c-GMP at c-GMP gated Na channels, it bound to proteins at Na channel membrane & keep them open) → opening of Na channels at outer segment → allow Na influx- → depolarization.**
- **3- Dark current (Na current):- At the inner segment Na pumped by Na- K pump to outside & re-entered through Na channels (at outer segment) .**


In Dark
cGMP
Na+ channel kept open by cGMP
Na+
Outer segment
Dark current
Rod
Na+
Na+/K+ pump
K+
Inner segment
K+
K+ channel
Release of inhibitory neurotransmitter continuous in the dark
Bipolar cell
Bipolar cell does not stimulate ganglion cell
Ganglion cell
(a)
In Light
cGMP
Decline in cGMP closes Na+ channel
Outer segment
GMP
Dark current stops
Rod
Na+
Na+/K+ pumps continue
K+
Loss of cations causes rod to become hyperpolarized, inhibiting its release of neurotransmitter
Inner segment
K+
No inhibitory neurotransmitter
Bipolar cell
Release of excitatory neurotransmitter stimulates ganglion cell
Ganglion cell
(b)

4- A wave of depolarization spread to synaptic terminals.

5- Synaptic mediators are continuously (steadily) released (mainly glutamate + Ach + dopamine + GABA.)

6- Response in bipolar cells(depolarization) → ganglion cells- → AP in optic nerve- → vision at dark.

NB/

1-at dark rhodopsin is inactive (cis-retinal needs light for its activation) / inactive rhodopsin is essential for depolarization

- its inactivation keeps Na channels open& Na current occurs,this is the causative factor for depolarization.

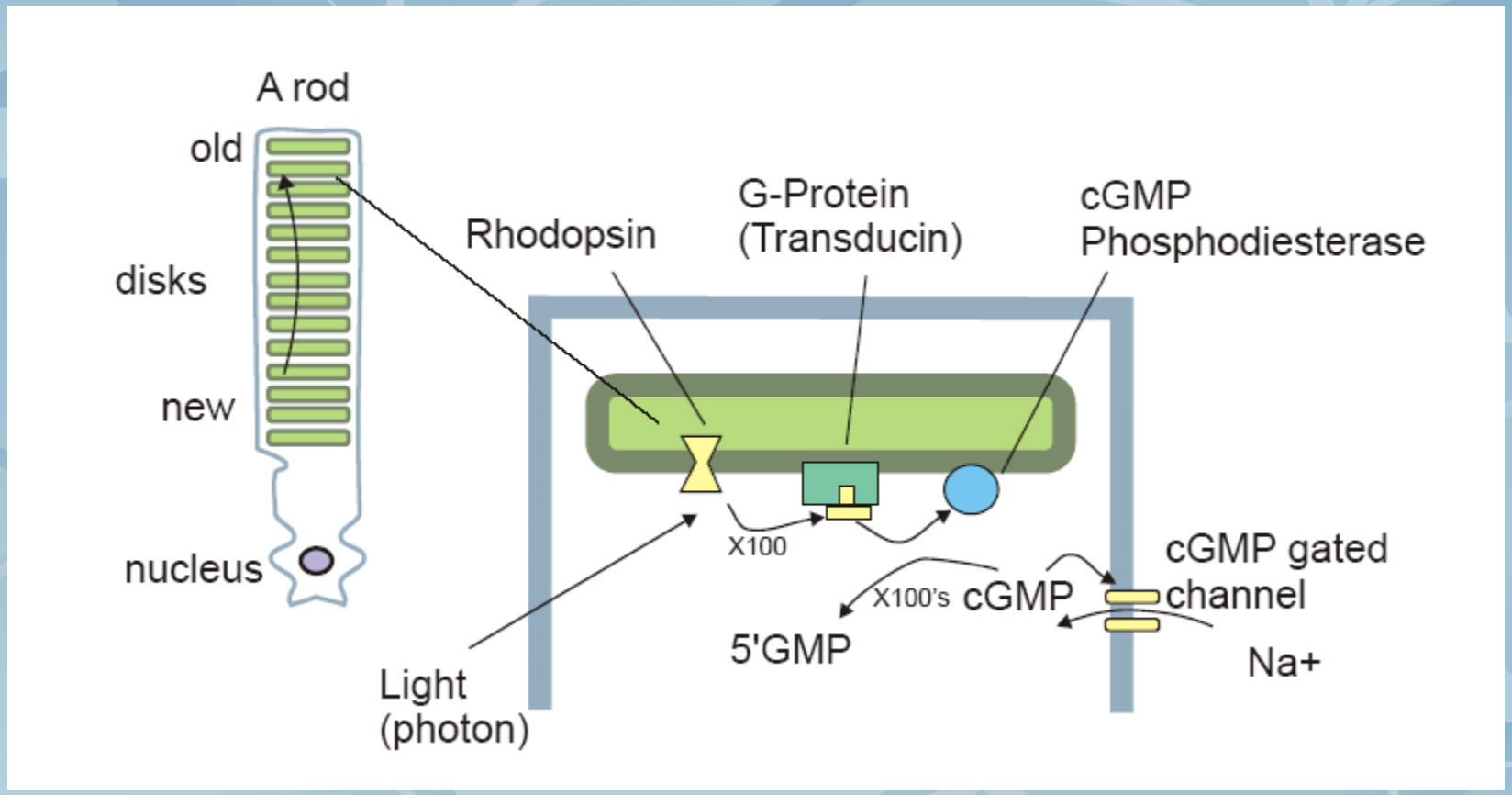
2-at dark rhodopsin is regenerated from retinine + scotopsin

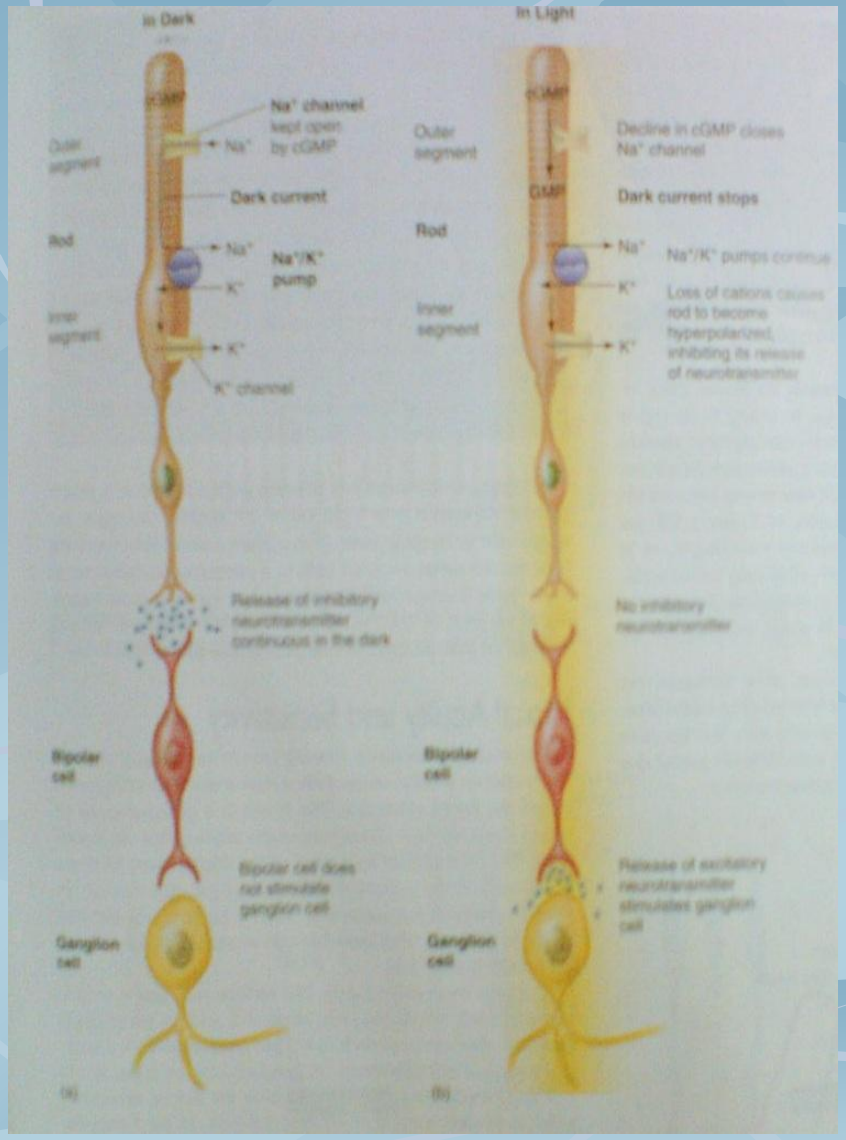
B-Incident light (PHOTOPIC VISION)

- **- Light- ⟶ Conformational change of photopigment retinine-1 in rhodopsin (11-cisretinal form changed to ⟶**
- **all-trans isomer called metarhodopsin II which is an active rhodopsin) ⟶Activation of G – protein (transducin) ⟶ activation of phosphodiestrase enzyme ⟶ conversion of c-GMP to 5- GMP ⟶ Decreased intracellular c-GMP ⟶ closure of Na channels in outer segment .**
- **-but still Na pump out of inner segment ⟶ Hyperpolarization of photoreceptors (-70 ~ -80)**

- **Hyperpolarization → Decreased release of synaptic transmitter → Response in bipolar cells (hyperpolarization) (this cause decreased release of inhibitory synaptic transmitter) → Generator potential in amakrine cells & ganglion cells (depolarize) → AP → optic nerve → optic pathway**

- **NB/**
- **-these reactions occur in both rods &cones but in rods occur at low illumination as in dimlight & in cones at high illumination.**
- **- in cones 4 times faster**


A rod
old
disks
new
nucleus
Rhodopsin
G-Protein
(Transducin)
cGMP
Phosphodiesterase
X100
X100's cGMP
5'GMP
cGMP gated
channel
Na+
Light
(photon)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Inner segment
K+
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Release of excitatory neurotransmitter stimulates ganglion cell
Ganglion cell
(b)

- **Synaptic mediators in retina:-**
- **Ach, glutamate, dopamine, serotonine,GABA, substance P,somatomedin, VIP, enkephalins, glucagons,neurotensin.**
- **In dark:- all transmitters are continuously (steadily) released by depolarization of rods depolarize bipolar cell →generator potential → AP in ganglion cells**
- **In light:- hyperpolarization of the receptors decrease inhibitory transmitter release →→ depolarize amacrine cell →generator potential → AP in ganglion cells.**

*-metarhodopsin II (in rods&cones)decompose by light into:-

- Retinine 1 + scotopsin

-Retinine 1 + scotopcin at dark → vit A + scotopsin → rhodopsin regeneration

- then decompose by light.

* NYCTALOPIA:- (night blindness)

-- Vitamine A deficiency cause rods , cones & retinal degeneration & loss of rods

-- R / vit A if receptors are well.

Dark adaptation:-

-
- -When a person moves from lighted environment → a dimly lighted environment, the retina becomes more sensitive to light & the person will see at dark (accustomed to dark) in about 20 min.(only gross features but no details or colors) .
- - Rhodopsin in darkness is essential for depolarization of rodes to see in dark
- (Na channels to open & for dark adaptation)
- -Once light enters the eye metarhodopsin from rhodopsin initiates cycle of events for light vision.

- **Dark adaptation has 2 components:-**

- **1- rapid (about 5 minutes) drop in visual threshold .**
- **Fast dark adaptation of cones , only in fovea**
- **-sensitivity of cones to light increase to see at that time.**

- **2- less rapid (till 20 min) drop in visual threshold .**
- **- dark adaptation of rodes in the peripheral retina**
- **- sensitivity of rodes to light increase, in 1 min increase 10 folds**
- **(rodes increase their sensitivity to light by convergence 300:1 ganglion cell , so summation at ganglion cells potential will increase sensitivity of rods to light)**

- **N.B (20 min for dark adaptation are for regeneration of rhodopsin → increase sensitivity of rodes to light → a drop in visual threshold**

- **Q- Why radiologists & aircraft pilots wear red goggles in bright light?**
- **A- Light wavelength of the red (at end of spectrum) stimulate the cones & stimulates rods to some extent, so red goggles for rods act as dimlight, so with it rods are adapted to darkness & form large amounts of rhodopsin while the person in bright light & when person enter dark places he can see well & not remain 20 mint.**

- **2-Light adaptation:-**
- **-When light switched on again, the rodes are knocked out of action (they stop sending AP at high levels of light) & cones start to function to adjust & adapt to the level of brightness in 5 min this is called Light adaptation**