

PHYSIOLOGICAL FACTORS CONTROLLED

- core body temp
- metabolic wastes (eg. CO₂, urea)
- blood pH / glucose
- ψ of blood
- conc. of O₂ / CO₂ in blood

INTERNAL ENVIRONMENT

- for cells = tissue fluid
- ↳ cell's immediate environment

TEMPERATURE

- temp ↓ = metabolic reactions ↓
- temp ↑ = proteins & enzymes denatured

WATER POTENTIAL

- ψ ↓ = H₂O moves out of cells ∴ metabolic reactions stop / slow down
- ψ ↑ = H₂O enters cell ∴ swell / burst

GLUCOSE CONC

- ↓ = respiration slow / stop ∴ energy ↓
- ↑ = H₂O moves out of cells

HOMEOSTATIC CONTROL

3 interdependent components:

- receptor, control center, effector
- Use negative feedback control loop:
- Receptor senses environmental stimuli.
- sends information through nervous system to a central control (in brain / spinal cord.) → input
- ↳ aka control centre

uses:

- nervous system
- endocrine system (2 coordination systems)

→ +ve feedback ⇒ enhances / accelerates output created by an activated stimulus

- -ve feedback ⇒ reduces the output or activity of any organ / system back to its normal range of functioning.

THERMOREGULATION

- control of body temperature.
- involves nervous & endocrine system
- mammals generate heat & retain it within their bodies (not dependent on environment)
- heat released during respiration, much produced by liver (energy requirement ↑)
- heat produced absorbed by blood flowing through liver & distributed around rest of body.

Homeostasis

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Definition ⇒ Maintenance of constant internal environment in an organism

→ Control centre signals an effector to carry out an action ⇒ output.

→ These actions ⇒ corrective actions

↳ to correct / reverse the change & bring it back to set point

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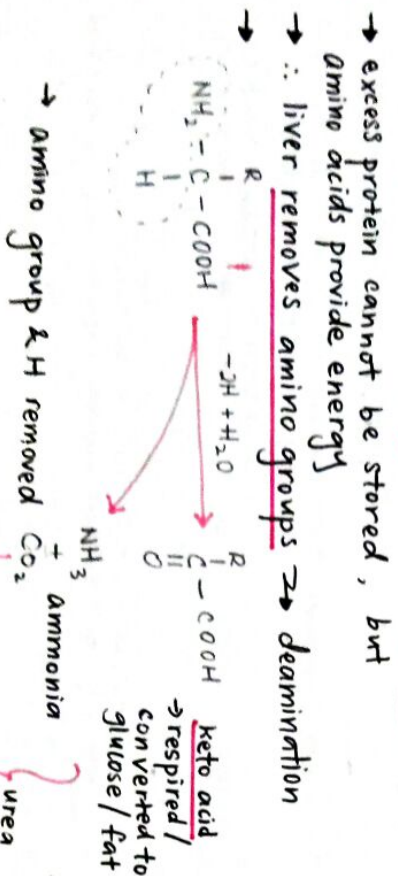
RESPONSES TO TEMP CHANGE

- Hypothalamus ⇒ central control for body temp.
- Thermoreceptor cells in hypothalamus monitor temperature of blood as it passes through the brain.
- Hypothalamus also receives info from skin receptors (monitor external temperature).

EFFECTOR	TEMP ↓	TEMP ↑
	Smooth muscles in arterioles in skin.	→ vasoconstriction → lumen of arterioles ↓ ∴ blood to capillaries ↓ ∴ less heat lost from blood
Sweat glands	→ Production of sweat ↓ ∴ heat loss by evaporation ↓	→ Production of sweat ↑ → H ₂ O has high latent heat of vapourisation
Erector pili muscles in skin	→ contract, erecting skin hairs ∴ trapping an insulating layer of air next to the skin	→ muscles relax, lowering skin hairs, reducing insulation.
Skeletal muscles	→ involuntary contraction generates heat which is absorbed by blood & carried around the body	→ no shivering

Effector	TEMP ↓	TEMP ↑
Adrenal & Thyroid gland	<ul style="list-style-type: none"> Hypothalamus releases a hormone which activates the anterior pituitary gland to release thyroid stimulating hormone (TSH) TSH stimulates thyroid gland to secrete <u>Thyroxine</u> Thyroxine ↑ metabolic rate 	<ul style="list-style-type: none"> Hypothalamus reduces the release of TSH. ∴ <u>Thyroxine</u> ↓ from thyroid gland.
Behaviour	<ul style="list-style-type: none"> curling up warm clothing etc. 	<ul style="list-style-type: none"> resting w/ limbs spread out air conditioning etc.

DEAMINATION



- Ammonia solubility ↑ & toxic ∴ NH₃ converted to urea
- urea diffuses from liver cells into blood plasma.
- As blood passes through the kidneys, urea is filtered & excreted.

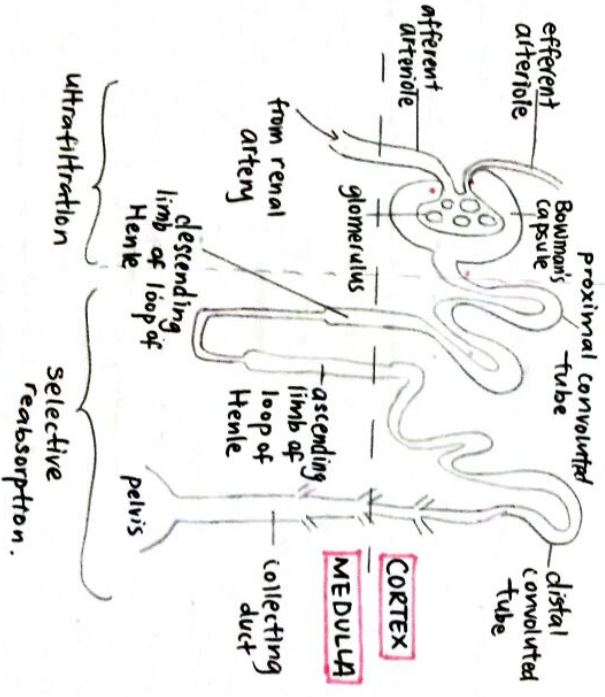
EXCRETION

- removal of unwanted products of metabolism.
- major excretory products
 - CO₂
 - urea
- produced by aerobic respiration
- CO₂ ↓, pH of blood ↓
- urea
- nitrogenous excretory product
- produced in liver
- other nitrogenous ex. pro. = creatinine, uric acid.

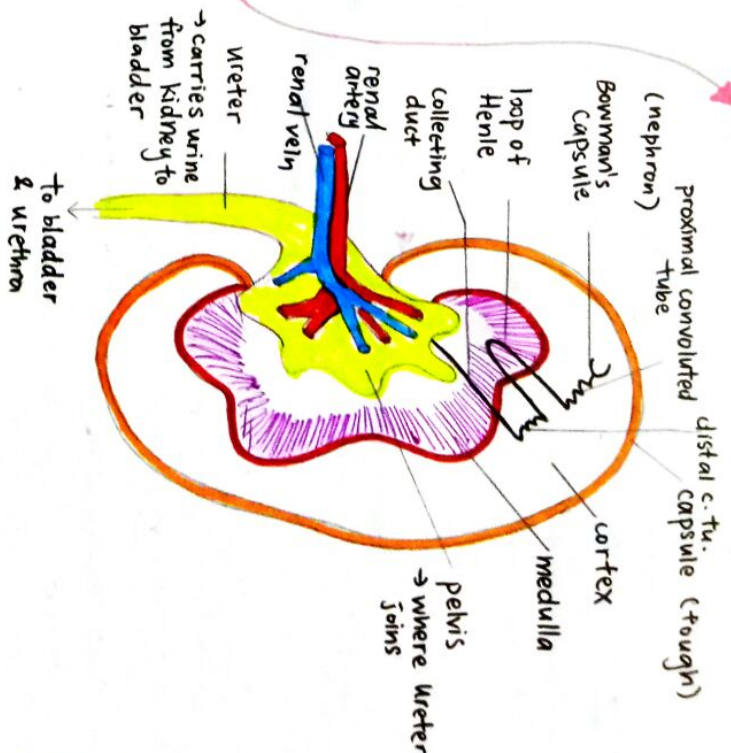
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STRUCTURE OF NEPHRON



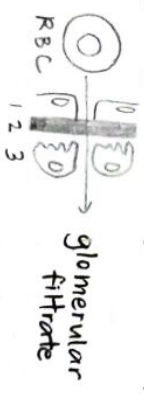
STRUCTURE OF THE KIDNEY



ULTRAFILTRATION

→ blood in glomerulus separated from lumen of Bowman's capsule by

- endothelium
 - ↳ one cell thick w/ pores (more than normal)
- basement membrane
 - ↳ made up of network of collagen & glycoproteins
- podocytes (of Bowman's capsule inner lining)
 - ↳ many finger-like projections w/ gaps between them



- Blood in glomerulus is at a relatively high pressure. ∴ efferent arteriole is narrower than afferent arteriole. ψ of blood plasma \uparrow . This forces molecules from blood into Bowman's capsule.
- Pores in endothelium & slits between podocytes let all molecules through.
- Basement membrane → filter.
- RBC, WBC, plasma proteins cannot pass
- ∴ glomerular filtrate = blood plasma w/out proteins

FACTORS AFFECTING GLOMERULAR FILTRATION RATE

- ψ in blood lower ∴
 - pressure \uparrow
- But solutes in blood plasma \uparrow than in glomerular filtrate. (ψ \uparrow in blood? No. \downarrow)
- overall, effect of diff in pressure outweighs the effect of diff in solute conc.

REABSORPTION IN PCT

- Some substances of glomerular filtrate need to be retained
 - H_2O
 - glucose
 - Na^+ , Ca^{2+}
 - vitamins & amino acids.
- ∴ taken back into blood through walls of PCT → selective reabsorption
- PCT made of single layer of cuboidal epithelial cells
 - microvilli to \uparrow SA

Hemostasis

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② → blood plasma rapidly removes absorbed substances

→ Reabsorbed:

- all glucose
- most Na^+ , Ca^{2+}
- all amino acids & vitamins
- most H_2O

↳ solutes in filtrate \downarrow ∴ ψ of filtrate \uparrow than in blood ∴

H_2O enters blood ∴ $\frac{1}{2}$ of the urea (small & easily pass through)

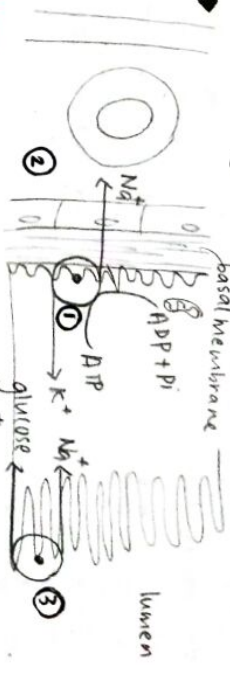
→ not reabsorbed:

- uric acid
- creatinine
- ↳ actively secreted out of cells

→ tight junctions hold adjacent cells tight ∴ so reabsorbed substances pass through cells.

→ many mitochondria to provide energy for Na^+ - K^+ pump proteins in outer membranes.

→ co-transporter proteins in membrane facing lumen.



① → Na^+ - K^+ pumps in basal membrane of PCT cells use ATP made by mitochondria.

→ $[Na^+]$ in cytoplasm \downarrow

→ basal membrane folded so SA \uparrow for many Na^+

③ → $[Na^+]$ in cyto \downarrow ∴ Na^+ diffuse into PCT

→ Na^+ can only enter through co-transporter protein.

↳ brings in glucose / amino acid at same time

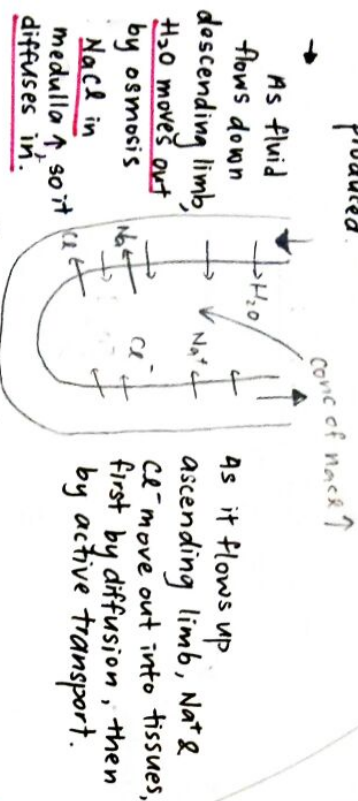
→ passive movement of Na^+ into cell provides energy to move glucose molecules even against conc. grad.

↳ secondary indirect active transport

→ 64% of H_2O NOT reabsorbed & enters loop of Henle

REABSORPTION IN LOOP OF HENLE

- Function \Rightarrow build up a high conc of Na^+ & Cl^- in tissues of medulla
- ↳ allows highly conc. urine to be produced.



By the time fluid reaches bottom, ψ ↓ than at top.

- This creates ψ in tissues of medulla.
- Descending limb permeable to H_2O & Na^+ & Cl^-
- Ascending limb only permeable to Na^+ , Cl^- , urea
- Two limbs running side by side with fluid flowing in opposite directions
- ↳ enables max conc of solutes to build up inside & outside at bottom of loop
- ↳ Counter-current effect.
- Ascending loop a loop of Henle permeable to urea \therefore urea also conc. in medulla tissue
- ↳ H_2O can move out until ψ of urine same as medulla.
- ↳ controlled by ADH
- Urea concentrated in tissue fluid in medulla

REABSORPTION IN DCT & CD

- Fluid lost H_2O & Na^+ in loop of Henle
- \therefore conc of urea \uparrow
- in DCT & CD Na^+ actively pumped out into tissue fluid
- K^+ actively transported into tubule
- H_2O moves out \therefore medulla ψ ↓, \therefore conc. of urea \uparrow more.
- urine formed.

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OSMOREGULATION

- ↳ control of ψ of body fluids
- osmoreceptors \rightarrow specialised sensory neurones in hypothalamus
- when ψ below set point, nerve impulses sent to posterior pituitary gland.
- ADH secreted
- ↳ stimulates reabsorption of H_2O

EFFECTS OF ADH

- Acts on cell surface membranes of collecting ducts cells
- makes them more permeable to water.
- ADH molecules bind to receptor proteins on cell surface membranes.
- enzyme (phosphorylase) activated in the cell
- Cells contain ready-made vesicles surrounded by membrane containing aquaporins (water-permeable channels).
- Vesicles move to CSM & fuse w/ it.
- Permeability to H_2O \uparrow , \therefore H_2O can move through aquaporins out of tubule, into tissue fluid of medulla.
- Volume of urine ↓, conc \uparrow
- when ψ of blood \uparrow , osmoregulators not stimulated
- ADH not secreted.
- Aquaporins move out of CSM back into cyto as part of vesicles
- permeability \downarrow , vol of urine \uparrow , conc \downarrow

CONTROL OF BLOOD GLUCOSE

- If glucose conc too low \rightarrow not enough for respiration
- If glucose conc too high \rightarrow effects ψ
- Carried out by hormones secreted by endocrine tissue in pancreas
- The cells \rightarrow islets of Langerhans

→ Islets → α cells - secrete glucagon
β cells - secrete insulin

→ α & β cells → receptors & central control

HIGH BLOOD GLUCOSE

- Glucose can only enter cell through GLUT
- Muscle cells - GLUT 4
- Brain cells - GLUT 1
- Liver cells - GLUT 2
- As blood flows through pancreas, α & β cells detect increase in glucose conc.
- α stop secreting glucagon, β secrete insulin
- Insulin receptors in liver, muscle, adipose
- Insulin stimulate cells to increase absorption rate of glucose from blood
- ↳ convert glucose → glycogen.
- When insulin molecules bind to receptors on muscle cells, vesicles w/ GLUT 4 proteins move to csm & fuse → glucose enter cell
- Insulin activates enzyme glucokinase
- ↳ phosphorylates glucose
- ↳ traps glucose inside cells
- Insulin activates phosphofructokinase & glycogen synthase
- ↳ add glucose molecules to glycogen.

LOW BLOOD GLUCOSE

- α cells secrete glucagon, β stop secreting insulin.
- Insulin ↓, rate of uptake of glucose ↓, not 0.
- muscle cells no receptors ∴ no respond to glucagon.
- Glucagon binds to receptor molecules on liver cells, G-protein activated.
- G-protein activates enzyme within membrane that catalyses ATP → cyclic AMP → second messenger

Homeostasis

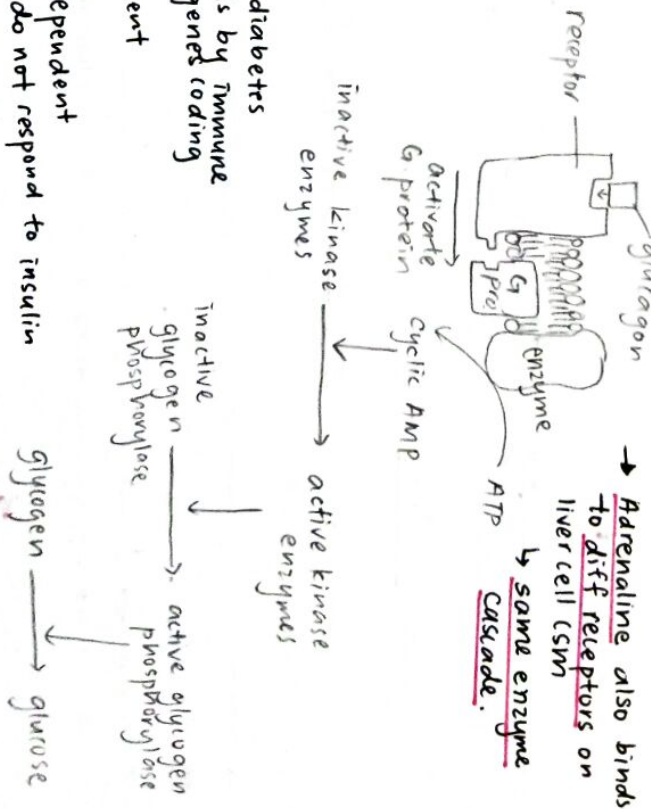
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DIABETES MELLITUS

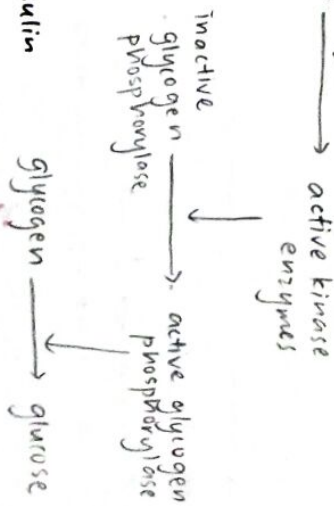
diabetes

- Type I**
 - juvenile-onset diabetes
 - attack of β cells by immune system / lack genes coding for insulin
 - insulin-dependent
- Type II**
 - non-insulin dependent
 - liver & muscle do not respond to insulin

- After meal, glucose conc remains high.
- kidney cannot reabsorb all glucose ∴ remains in urine.
- loss of H₂O & salts.
- uptake of glucose into cells is slow (no GLUT)
- cells metabolise fat & proteins ∴ build up of keto-acids
- ↳ blood pH ↓
- Between meals, glucose conc ↓ ∴ no glycogen stored



- cyclic AMP binds to kinase enzymes within cyto that activates other enzymes
- ↳ phosphorylation
- glycogen phosphorylase activated
- ↳ breakdown glycogen → glucose.
- ↳ glucose diffuse out through GLUT 2
- Glucose also made from amino acids & lipids → gluconeogenesis
- Adrenaline also binds to diff receptors on liver cell csm
- ↳ same enzyme cascade.



URINE ANALYSIS

- Presence of glucose & ketones → diabetes
 - ↳ blood glucose ↑, ∴ not fully reabsorbed from PCT
- Presence of protein → kidney problem / high bp
 - ↳ most protein too large to be filtered
 - ↳ even if filtered, reabsorbed by endocytosis in PCT, broken down into amino acids & absorbed by blood.
- DIP STICKS
 - contain enzymes glucose oxidase & peroxidase
 - ↳ immobilised on small pad at one end of stick.
 - glucose $\xrightarrow{\text{glucose oxidase}}$ gluconolactone + H₂O₂
 - H₂O₂ + chromogen $\xrightarrow{\text{peroxidase}}$ darker compound. (colourless)
 - resulting colour matched against colour chart
 - glucose ↓, darkness of colour ↑
 - Problem → do not indicate current blood glucose conc.
 - ↳ only while the time urine was collecting in bladder.
- BIOSENSOR
 - Pad impregnated with glucose oxidase.
 - glucose $\xrightarrow{\text{glucose oxidase}}$ gluconolactone
 - ↳ At the same time electric current generated & amplified
 - glucose ↑, current ↑, reading ↑

HOMEOSTASIS IN PLANTS

- Stomata control entry of CO₂ into leaf. → open & close even in constant light
- Open in response to:
 - ↳ light intensity
 - ↳ CO₂ conc ↓ in air spaces in leaf
 - ↳ when open CO₂ ↑ but H₂O ↓ (transpiration)
- Close in response to:
 - ↳ darkness
 - ↳ CO₂ conc ↑ in air spaces
 - ↳ humidity ↓
 - ↳ temp ↑
 - ↳ H₂O ↓
 - ↳ when close, CO₂ ↓ but H₂O ↑

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CLOSING OF STOMATA

- H⁺ pump stops & K⁺ leave guard cell & enter neighbouring cells
- H₂O leaves down ↓ grad. ∴ guard cells become flaccid.
- stomata close
- only when conserving H₂O (transpiration important)

OPENING OF STOMATA

- ATP-powered proton pumps in guard cell cm actively transport H⁺ out.
- H⁺ conc. inside ↓ ∴ K⁺ channels open & K⁺ move in.
 - ↳ ∴ cell -vely charged & has low conc. of K⁺
- ↳ diffuse down electrochemical grad.
- Extra K⁺ lower ↓ ∴ H₂O enters through aquaporins
- Guard cells become turgid ∴ stomata opens



- Ends of guard cells are joined, thin outer walls bend more readily.
- ∴ Guard cells curve & opening

ABSCISIC ACID (ABA)

- stress hormone
- Guard cells have ABA receptors
- ABA inhibits H⁺ pumps ∴ H⁺ not pumped out
- ABA stimulates movement of Ca²⁺ into cyto through cm & tonoplast
- Ca²⁺ acts as second messenger → activates channel protein to open ∴ allow -ve charge ions to leave guard cell
- Stimulates opening of K⁺ out of cell
- stimulates closure of channel proteins that allow K⁺ to enter
 - ↳ Cr²⁺ in cell ↓ ∴ ↓
- H₂O leaves by osmosis, guard cell flaccid, stomata close