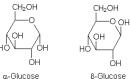
CARBOHYDRATES

Monosaccharides: cannot be hydrolyzed into simpler units, has C=O (carbonyl group)

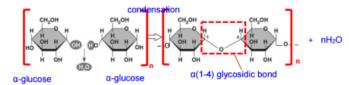
- \rightarrow Water soluble: relatively small, multiple -OH for H-bonding
- \rightarrow Aldose (C=O at end) vs Ketose (C=O at middle)
- \rightarrow Ring form vs Linear Chains (Cyclic ring = common, stable)

Disaccharides

Glucose + Lactose = Galactose Glucose + Glucose = Maltose Glucose + Fructose = Sucrose -Formation of **glycosidic bond**



through dehydration synthesis/condensation rxn



Polysaccharides:

Polymerisation = condensation of numerous monosaccharides

- \rightarrow Folded: Compact & ideal for storage
- \rightarrow Large & insoluble: does not affect ϕ of cell
- \rightarrow Structural: Cellulose, Chitin, Pectin
- \rightarrow Storage: Starch/Glycogen

Starch:

Structure: Amylose helices entangled w/ branched amylopectin

- \rightarrow compact helical arrangement = more glucose per unit
- \rightarrow branched arrangement = more hydrolysis sites
- \rightarrow large structural molecule = relatively insoluble
- : efficient hydrolysis + compact storage + insolubility

Cellulose:

- \rightarrow Inversion of alternate β -monomers
- \rightarrow formation of parallel chains of glucose w/ OH groups projecting from both sides of chains
- \rightarrow <u>crosslinkage</u> formation between neighbouring parallel chains

= microfibril formation

∴ high tensile strength + insolubility

Common Mistakes:

1. Monomer isomers \rightarrow arise from same linear monomer unit

	Cellulose	Starch
Monomer	β-glucose monomers	α-glucose monomers
Bond between monomer	Cellulose →β(1-4) glycosidic bond	$amylose \rightarrow \alpha(1-4)$ glycosidic bond $amylopectin \rightarrow \alpha(1-4) + \alpha(1-6)$ glycosidic bond
Orientation of monomer	Alternate glucose units are inverted w/ respect to each other	All glucose units in the chain have same orientation
Structure of each molecule	Long, linear unbranched straight chain	amylose → helical/coiled strand or amylopectin → coiled branched molecule
Bonds between molecules	Hydroxyl groups projecting outwards in both directions allow interchain hydrogen bonding	No interchain hydrogen bonding in starch
Branching	No branching	branching

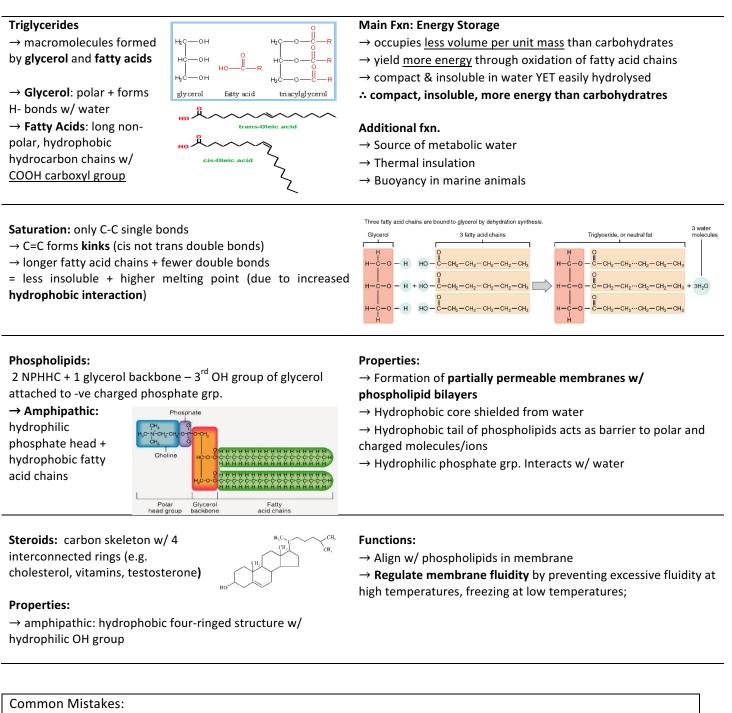
Glycogen

- \rightarrow More highly branched than starch, shorter chains
- \rightarrow Long-term storage molecule (less ease of hydrolysis)

	Cellulose	Starch
Function	structural support/high tensile strength (cell wall)	compact energy storage molecule
Reason	Many long straight chains interact/ crosslink via hydrogen bonding forming bundles of microfibrils	Long helical strands of α-glucose makes it compact allowing packing of many glucose units per unit volume

LIPIDS

Lipids \rightarrow non-polar, insoluble in water vs. <u>soluble in non-polar organic solvents</u>



- 1. Fatty acids: non-polar, insoluble and produces water when oxidized
- 2. Emulsion test: white precipitate \rightarrow fats present

PROTEINS

A Condensation Reaction Between Two Amino Acids

AA: aa

- \rightarrow Chiral C atom w/ H,
- \rightarrow Amine group (-NH₂)
- \rightarrow Carboxyl group (-COOH)
- \rightarrow Variable R domain

Dipeptides

 \rightarrow AA undergo

condensation rxn. to form polypeptides w/ **peptide bond**

 \rightarrow OH from COOH + H from NH2 = 1 H2O removed

R groups:

→ neutral AA = electrically neutral R groups

→ non-polar R groups (hydrophobic aa)/polar R groups (hydrophilic aa)

→ electrically-charged aa = acidic aa w/ COOH groups or basic aa w/ OH groups

Zwitterions:

 \rightarrow ionized aa accept and donate H+ from carboxyl/amine grp.

Polypeptides: aa polymers – condensation rxn: <u>peptide bond</u> between aa.

 \rightarrow N-terminus: end w/ free amino group

C-terminus: end w/ free carboxyl grp

Primary:

 \rightarrow <u>specific number + sequence</u> of AA in a polypeptide chain linked by <u>peptide bonds</u>

Secondary:

$\rightarrow \alpha$ -helix/ β -pleated sheet

 \rightarrow <u>hydrogen bonding</u> between -CO and -NH groups of polypeptide backbone

α-helix:

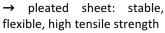
 \rightarrow single polypeptide wound into a helix

 \rightarrow H-bonds formed between -CO of one AA residue and -NH group of another AA residue (3.6 AA away)

→ R-groups project away from helix

β -pleated sheet:

→ H-bonds between CO group of one region and NH group of an adjacent region of a single polypeptide chain,



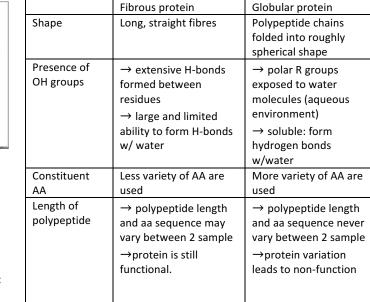
Tertiary:

→ <u>Bonds:</u> hydrophobic interaction, H-bonds, ionic bonds, disulphide bridges-: cysteine w/ sulfhydryl R group

- → Bond formation between <u>R groups of same polypeptide</u>
- → Extensively folding into specific 3D conformation,

Quaternary:

→ More than 1 polypeptides, same 4 types of interactions/bonds



→ +/- charge on same aa = buffer ability (aa = amphoteric, donate/accept H+)

Haemoglobin:

- → found in RBCs, acts as oxygen-carrier
- \rightarrow 2 α -globin, 2 β -globin subunits w/ 1
- prosthetic haem group each
- \rightarrow 1 haem group = 1 porphyrin ring + 1 Fe²⁺ ion

(binds to 1 O₂ to form oxyhaemaglobin)

→ water soluble: hydrophilic amino acid side chains of subunits are on external surface

Cooperative Binding:

 \rightarrow Subunits move w/ respect to each other

 \rightarrow formation of 1 oxyhaem increases affinity for other subunits by inducing conformational change in shape of subunits

Sickle Cell:

 \rightarrow 6th aa changed from glutamic acid (hydrophilic) to valine (hydrophobic)

- \rightarrow 3D conformation of polypeptide changes \rightarrow rod-shaped Hb
- \rightarrow Hb aggregation occurs at low O2 concentrations causes RBC
- to get sickle shape \rightarrow higher chance of lysis, capillary damage

Collagen:

 \rightarrow <u>Tropocollagen</u>: Fibrous structural protein w/ 3 polypeptide chains wound together

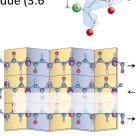
→ <u>H-bonds</u>: between AA of adjacent polypeptide chains between NH, CO groups of peptide backbone (high tensile strength)

 \rightarrow <u>H-bonds</u>: between OH groups of hydroxyproline (insoluble in water)

→ small glycine residues = tight/compact triple helical structure VS. bulky, inflexible proline and hydroxyproline = rigid molecule.

→ covalent cross-links form between lysine residues at the C and N ends of adjacent/parallel tropocollagen molecules; collagen fibrils lie in bundle up to form collagen fibres;





ENZYMES

Enzymes:

- globular proteins, chemically unaltered reusable 1. catalysts that increase rate of rxn.
- 2. lowers activation energy by forming ES-complex

Active site:

 \rightarrow Enzyme 1⁰ structure \Rightarrow substrate-specific 3D conformation with specific region of substrate binding \rightarrow substrate and active site have complementary conformation and charge

- 1. Catalytic AA \Rightarrow catalyse conversion between substrate and enzyme's active site
- 2. <u>Contact AA</u> \Rightarrow bind reversibly w/ weak H-bonds + ionic bonds

Models of Action:

1. Lock and Key Hypothesis - enzyme is the lock and substrate is the key

→ catalytic R-groups already in position to act on bonds

2. Induced fit hypothesis: substrate induces a change in shape in enzyme

 \rightarrow catalytic R-groups at active site brought into precise orientation

Mode of Action:

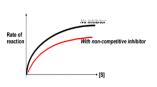
→ Enzymes lowers the activation energy barrier

- 1. **P**roximity \Rightarrow substrates aligned next to each other in active site for rxn. to occur
- 2. **O**rientation \Rightarrow substrate oriented such that its bonds are exposed to attack
- 3. Strain \Rightarrow strains bonds to be broken / distorts the substrate and reduces E_a to achieve transition state
- 4. Provide a favourable MicroEnvironment
- 5. Direct Catalysis \Rightarrow R-groups of AA residues in active site participate in direct catalysis - e.g. Acid-base catalysis

Competitive Inhibitor: similar charge and shape/ conformation to the substrate

- inhibitor binds to enzyme active site reversibly (w/ weak, non-covalent bonds) and block substrate binding.

At high [substrate], inhibitor fx. Is negligible the substrate since molecules can effectively out-compete the inhibitor molecules for active sites;



Non-competitive inhibitor: binds to allosteric site (another site on enzyme not active site) either permanently or reversibly (depends on inhibitor)

- Alters shape/conformation of the specific enzyme active site = substrate cannot bind in correct orientation - Rate of rxn. decreases with \uparrow [inhibitor] = cannot be neutralized by \uparrow [substrate]

Temperature:

 \rightarrow Increase in temperature = \uparrow KE of enzyme and substrate molecules

 \rightarrow \uparrow frequency of effective collision between substrate and enzyme = more ES complex;

 \rightarrow \uparrow no. of molecules w/ energy to overcome activation energy barrier

 \rightarrow Rxn. rate increases with temperature up to the optimum temperature;

Optimal Temperature:

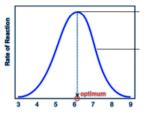
 \rightarrow maximum no. of effective molecular collisions reached hence max rate of rxn.; =

 \rightarrow post-optimal thermal agitation breaks H-bonds, ionic bonds and other weak interactions that stabilizes conformation = enzyme denaturation

pH:

 \rightarrow Optimal pH \Rightarrow rate of rxn. is at maximum

1. Excess [H+]/[OH-] ions affect ionisation of R groups of AA (COO- -> COOH and excess -OH-= NH3 + becoming -NH2)



 \rightarrow Disruption of ionic/H-bonds: stabilize specific conformation of active site = denaturation of enzyme

→ pH: change specific charge of R groups of catalytic residues in the active site (affect temporary binding between enzyme and substrate = no enzyme-substrate complex formed)

[Substrate]: low conc.: active sites of the enzymes readily available to catalyse rxn. ([substrate] = limiting factor)

- [Substrate] increases = rate of rxn. increases as more active sites occupied by substrates

- At higher [substrate], saturation of active sites = [enzyme] = limiting factor

(Similar for [enzyme] – more active sites available)

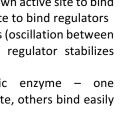
- K_m (Michaelis constant) = conc. of substrate at $\frac{1}{2} V_{max}$

Allosteric Regulation: allosteric enzymes regulated by inhibitors/activators

- 2+ subunits - each w/ own active site to bind substrates + allosteric site to bind regulators - 2 conformational states (oscillation between active/inactive forms = regulator stabilizes respective form)

cooperativity: allosteric enzyme - one subunit binds to substrate, others bind easily to substrate too

feedback inhibition: end- product inhibition (end-product binds to enzyme early in pathway) = accumulation of end-product = more binding to allosteric site = altered conformation of specific active site = substrate unable to bind = lower rate of rxn.



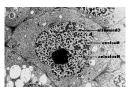
CELL STRUCTURE

Overview: Eukaryotic cell structure – cell surface membrane, nucleus, cytoplasm, cell wall (depends)

1) Nucleus: largest organelle - 5µm diameter, spherical

- surrounded by **double membrane (nuclear envelope)** = 2

phospholipid bilayers w/ numerous nuclear pores (regulate passage of substances into/out of nucleus)



- contains chromatin/ chromosomes: hereditary

material (DNA) – thin elongated chromatin threads (not in mitosis) = DNA complex + histone proteins VS. **condensed** thicker, shorter chromosomes (before mitosis)

- **chromatin:** most = loosely coiled, diffuse **euchromatin** (unstained) vs. some = tightly coiled, dark stained heterochromatin

- <u>fxn.: contains hereditary material + regulates protein</u> <u>synthesis (thus controls entire cell)</u>

2) Nucleolus: one or more in 1 nucleus = contains DNA, rRNA + proteins

- fxn.: site of ribosomal RNA synthesis (to form ribosomes) + assembly site of rRNA + ribosomal proteins into subunits

3) Ribosomes = large subunit + small subunit (each subunit = rRNA + proteins)

- ribosomal subunits assembled in nucleolus -> exported out of nucleus into cytoplasm

 ribosomes = freely floating in cytosol (free ribosomes)/ attached to outer surface of rough endoplasmic reticulum (bound ribosomes)

(70S ribosome – prokaryotes, chloroplasts, mitochondria vs 80S ribosome – eukaryotes)

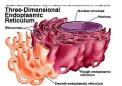
- fxn.: site of protein synthesis - translation of mRNA

4) Cell Surface Membrane* - refer to CSM notes

5) Nuclear Envelope – double membrane perforated by nuclear pores (formed by a protein pore complex)

fxn.: allow regulating passage of substance

6) Endoplasmic Reticulum: network of membranous tubules (cisternae) w/ single membrane separating cytosol from cisternal space



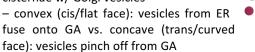
- Rough ER: continuous w/ outer

membrane of nuclear envelope – flattened, interconnected cisternae + **bound ribosomes** that stud outer surface of membrane

-Translation of mRNA to protein/ protein synthesis by bound ribosomes + glycosylation/modification and transportation of proteins

-Smooth ER: Tubular membrane bound sacs called cisternae without bound ribosomes

 <u>Lipid and steroid synthesis + carbohydrate synthesis +</u> <u>detoxification;</u> 7) Golgi Apparatus: single membranebound w/ saucer-like membranous cisternae w/ Golgi vesicles





- <u>fxn.: glycosylation of proteins/lipids</u>, <u>modification of</u> glycoprotein and glycolipids, formation of lysosomes, production of polysaccharides, sorts and targets materials for <u>secretion</u>

8) Lysosomes: single membrane-bound w/ hydrolytic enzymes – optimal pH (acidic)

- storage vesicle to keep enzymes apart & prevent destruction of the rest of the cell

- **Digestion of materials**: digestive enzymes mix w/ contents of vesicles = useful products released into cytosol

- Autolysis/self-rupture: self-destruction of cell by release of lysosomal contents within cell

- **Autophagy:** destruction of worn out organelles, digested products returned to cytosol for re-use

9) Mitochondrion: double membranebound: outer membrane = **smooth**, inner membrane – highly infolded, numerous **cristae**



- intermembrane space: between the outer and inner membrane vs. matrix: inside inner membrane

outer and inner membrane vs. matrix: inside inner memb – 70S ribosomes, circular DNA

- cristae: large surface area – enzyme attachment fxn. OP fxn.: site of cellular respiration – generate ATP vs. Krebs cycle

matrix vs. oxidative phosphorylation – cristae
 10) Chloroplast: lens-shaped (2μm Outer membrane

- 5μm) + double membrane w/ intermembrane space + interconnected sacs = thylakoids (inside chloroplast)



- stacks of thylakoids = **grana** Chloroplast diagram (chrlorophyll + enzymes on thylakoid membrane)

 - fluid outside thylakoid = stroma (w/ circular DNA, ribosomes, enzymes, starch grains.

- <u>fxn.: site of photosynthesis</u>

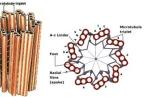
11) Microtubules – hollow rods (25nm diameter) = **tubulin** wall + each tubulin – dimer w/ 2 subunits

- help maintain cell shape (cytoskeleton) / intracellular transport/ chromosome movement in ell division/ strucutrual component of centrioles, cilia and flagella

x2

12) Centrioles – 9 triplets of microtubules (rod-like

- microtubules structure)
- found in pairs, perpendicular to each other
- organize spindle fibres during cell division
- anchorage for cilia and flagella



CELL MEMBRANE

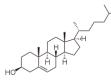
Phospholipid Bilayer: Around 7.5nm/10⁻⁹m thick – present in both prokaryotic/eukaryotic cell.

Phospholipid: A phospholipid = a glycerol molecule + phosphate (PO4-) group = hydrophilic region, 2 hydrocarbon tails / chains = hydrophobic region.

form bilayers in aqueous medium (or micelle)
 held together by weak hydrophobic interactions/van der Waals forces + contribute to membrane fluidity

Cholesterol: four ring structure, hydrophobic yet **amphipathic** – hydrophilic OH group (fxn.: regulates membrane fluidity)

 prevents membrane from being overly fluid at warmer temperatures (cholesterol's phospholipids' lateral movement)



rigidity restricts

- prevents membrane from being overly firm at lower temperatures (cholesterol prevents the close packing of phospholipids and hence prevents its solidification/ crystallization)

Proteins: determine membrane's specific functions **Integral/intrinsic protein:** amphipathic – hydrophilic polar/charged R groups + non-polar R groups

- hydrophobic regions lie in hydrophobic core of bilayer vs hydrophilic region (exposed to aqueous medium)

- transmembrane protein (spans entire membrane) vs unilateral protein (halfway across membrane)

Peripheral/extrinsic protein: not embedded, loosely attached to membrane surface/ integral protein (weak ionic/ H-bonds)

- attached to fibres of **ECM** (exterior of membrane) vs. held by cytoskeleton (cytoplasmic side of membrane)

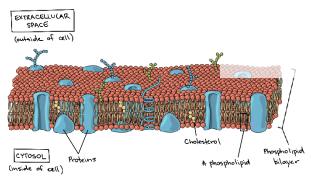
Carbohydrates: glycolipids and glycoproteins = carbohydrates bound to proteins

- carbohydrates project out of cell into ECM

- fxn.: markers for cell-cell recognition (e.g. distinguish cells as 'self'/'non-self' = immune system)

- cell-cell recognition = cell adhesion = cells attached to one another to form tissues, organs;

- receptor for pathogens to bind to host cells and enter cell - found in myelin sheath = electrical insulation of nerve cell



Fluid Mosaic Model: <u>fluid state</u> = lateral movement of Plipids, proteins within layer (weak Hphobic interactions) + <u>mosaic</u> = random arrangement of proteins embedded amongst Plipids

Membrane Function:

1) Regulate movement of substances across membrane

 selectively permeable membrane: hydrophobic core = ions, polar/large molecules need transport proteins

= non-polar molecules diffuse through hydrophobic core

2) Compartmentalization

- formation of unique environments for specialized activities such as enzyme reactions in lysosomes

- establishment of proton gradients within specialized organelles such as mitochondria and chloroplasts (ETC)

- storage of food (e.g. starch in amyloplasts)

3) Protein Localization

- localization of proteins of related function together so that sequential biochemical processes are facilitated

- (e.g. enzymes and proteins are grouped together into PS II and I on chloroplast thylakoid membrane)

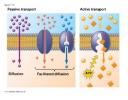
4) Increased Surface Area

- Increase surface area for chemical reactions such as the highly folded cristae of mitochondria increase surface area for insertion of electron transport carriers and ATP synthase complexes for oxidative phosphorylation to take place

Transport Across Membrane

- Simple Diffusion: non-polar molecules + water direct movement down conc. gradient w/o ATP + proteins
- relies on conc. gradient only = ends in dynamic equilibrium
- rate factors: molecular size (smaller

particle = faster diffusion), <u>solubility in</u> <u>lipid bilayer</u> (non-polar particles = faster diffusion), <u>concentration</u> gradient (steeper gradient = faster diffusion, <u>kinetic energy</u> (higher temperature = faster diffusion), <u>surface area of</u>



<u>membrane</u> (more SA = faster diffusion), <u>distance</u> (shorter distance = faster diffusion)

 Facilitated Diffusion: simple diffusion (no ATP, conc. gradient, passive) BUT w/ transport proteins

Channel Protein: transmembrane, w/ hydrophilic pore = ions/charged molecules pass through membrane (may be gated)
Carrier Protein: transmembrane = molecule binds to protein, change channel shape, allow molecule to pass (e.g. glucose)

3) Osmosis: movement of water mol. From region of higher WP to lower WP through selectively permeable membrane (simple diffusion – transient pore/facilitate diff – aquaporin)

4) Active Transport:

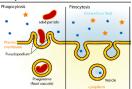
- Needed when polar, charged molecules or ions are to be transported (against a concentration gradient across the membrane requiring ATP) + needs carrier proteins / pumps and is also solute-specific

5) Bulk Transport: active transport w/o protein channels

- Endocytosis: infolding/extension of CSM = vesicle (carrier)

- Phagocytosis: pseudopodia formation = engulfing **solid** particle = formation of vesicle

 Pinocytosis: invagination of membrane -> liquids taken up
 Exocytosis:



macromolecules in vesicles = vesicle fuses w/ membrane = contents released into cytosol

Rohith | More free notes at tick.ninja