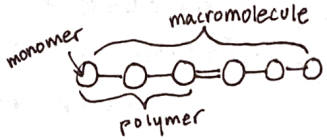
BIOL101 TEST GUIDE (#1)

LO1 - Life & Science

- LO: Describe elements of research design and how they impact conclusions
  - → sample size, randomization, double blind, strengths & weaknesses
  - → causation ∅ causation
- LO: Formulate testable hypothesis & design controlled exp
- LO: Distinguish science from unjustified claims

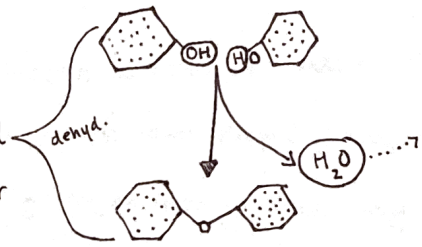
LO2 - Macromolecules

- LO: name & explain 5 major themes of biology
- LO: classify polysaccharides based off their structure/function in plants/animals & describe how formed
  - → 4 major macromolecules (biological polymers) in cell:
    1. Carbohydrates (monosaccharide + monosaccharide → polysaccharide + h_2o)
    2. Lipids
    3. Proteins
    4. Nucleic Acids



4 polysaccharides:
1. Starch: storage + plants
2. glycogen: storage + animals
3. cellulose: structure + plants
4. chitin: structure + ½ animals

  - → Hydrolysis & Dehydration Reactions
    - Hydrolysis: breaks polymers into monomers by adding H_2O
    - Dehydration reaction: links monomers together, H_2O released

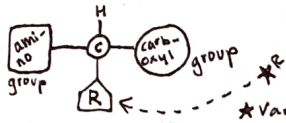


- LO: define lipids & explain their functions & properties in polar/nonpolar
  - → Lipids = nonpolar & hydrophobic
    - → 4 important types of lipids: fats, phospholipids, cholesterol, and steroids
  1. Fats (monomer = glycerol & fatty acids) (aka a triglyceride bc "3" & "glycerol")
     - → unsaturated fats/triglycerides: hydrocarbon chain has 1+ double bond (can't pack tight ∴ liquid)
     - → saturated fats/triglycerides: no double bonds (can pack easily ∴ solid)
  2. Phospholipids (hydrophilic head (glycerol + phosphate mol) + 2 hydrophobic tails)
     - → "amphiphilic" molecule

..... form phospholipid bilayer .....

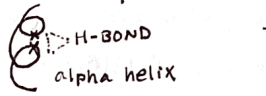
- LO: draw protein structure & depict the consequence of mutations on normal structure & function
  - → Proteins' monomers are amino acids. Amino acid + amino acid = dipeptide
  - → 1. Primary structure in proteins = just the sequence of the amino acids in order

x = amino acid. ☆ (covalent) PEPTIDE bonds b/w amino group of 1 amino acid & carboxyl of another



★ R groups are what make amino acids VARY
★ Variation is key

  - → 2. Secondary structure in proteins = H-bonds b/w amino group of 1 amino acid & carboxyl of another



  - → 3. Tertiary structure in proteins = overall 3-D shape of polypeptide due to R-group interactions b/w amino acids (↓ H-bonds or covalent)
  - → 4. Quaternary structure: (NOT ALL HAVE) → R-group interactions b/w multiple polypeptide chains

(interactions b/w R-groups CAUSE folds)

  - → One mutated amino acid can affect/ruin the entire polypeptide's structure & ∴ function
    - → One incorrect/misplaced amino acid can make protein deffective bc specific R-groups & orders cause each protein's unique folds & functions
- LO: identify how human body uses macromolecules from food
  - → Eat for 2 reasons:
    1. Raw organic ~~molecules~~ materials for building our own macromolecules
    2. Energy for cellular work

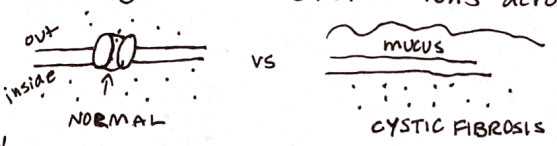
LO3 - A Tour of Cell

- LO: predict structures of the prokaryotic cell that would be antibody targets
  - Prokaryotes = Bacteria & Archae, Eukaryotes = Animals, Plants, Fungi
  - Prokaryotes: no membrane enclosed organelles
  - The best antibiotic targets would be organelles that prokaryotes have but eukaryotes don't
    - cell wall, other stuff
  - some antibiotics actually work to break down *structure* of target (ex: destroy cell wall)
  - others work to destroy *information pathways* so the cell can't replicate DNA or make proteins @ ribosome
- LO: describe how a protein is synthesized and exported from a cell & how disease can be caused when this goes awry
  - DNA is "blue-print" for protein synthesis/expression. Folding is the final touch. Transport vesicle brings protein to Golgi Apparatus (kinda like sorting mechanism). Ribosomes (beginning) is what makes them
  - made in RER: protein will be exported elsewhere... made in cytosol: keep in cytosol
  - insulin = example of a protein secreted from cell
- LO: explain how insulin-producing cells are like dysfunctional factories when a person is diabetic
  - Beta pancreas cells = insulin factories (Type 1 diabetes = no beta pancreas cells)
    - insulin in ~~cells~~ blood → receptor binds → glucose transporters do their work
    - glucose taken out of blood & glycogen created
  - Alpha pancreas cells = glucagon releasers
    - liver cells break down glycogen stores into glucose & released back into blood

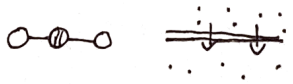
Pancreas
- a-cells ..... → create insulin .... → stimulates glucose receptors .... → glucose level drop & glycogen to liver
- B-cells ..... → create glucagon .... → stimulates break down of glycogen into glucose .... → glucose to blood

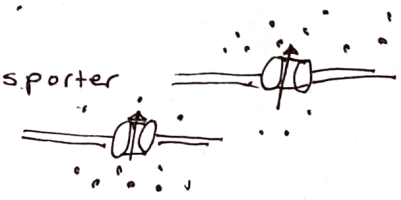
LO4: Structure + Function of Membranes

- LO: interpret experiments about protein production and make conclusions about why protein production is impaired in cystic fibrosis
  - Cystic Fibrosis: we all need protein CFTR in plasma membrane to allow chloride ions across membrane (w/ CF: ✱PROBLEM IN PRIMARY STRUCTURE ∴ FOLDS WRONG)
    - Without it, mucus builds up in airways
    - lungs clogged w/ mucos: trouble breathing & pain
    - CF PATIENTS: CFTR is produced but not on surface of lung cells, where needed



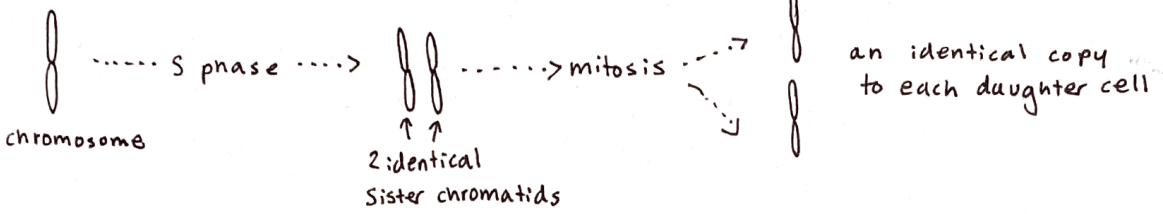
- LO: Categorize molecules that do/do not cross membranes freely
  - CAN: nonpolar small molecules, water (slowly)
  - CAN'T: ions or large polar
    - ACTIVE TRANSPORT: against gradient, protein transporter
    - FACILITATED D: along gradient, w/ transport protein:
    - SIMPLE: along gradient, no help
  - water: osmosis & aquaporins





BIO TEST ~ review sheet

- **L9:**
  - Sexual vs Asexual reproduction (sexual = variety)
  - MITOSIS: cells producing identical daughter cells
    - → things a cell must do before dividing:
      1. grow in size (increase ATP & protein production)
      2. duplicate DNA so each daughter cell gets full content
      3. double organelles
      4. ensure each daughter gets ½ (equal division)
    - → interphase → mitosis or meiosis (cells spend most time here. G1 grow & increase organelles, S duplicate DNA, G2 grow more)



PROPHASE

- chromatin coils to form distinct chromosomes
- nuclear envelope starts dissolving

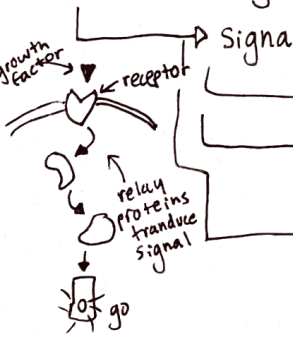
(check rest on diagram)

TELAPHASE

- animal cells = cleavage furrow
- plant cells not flexible to just stretch into cleavage.
  - → so: membraneous vesicles containing cell wall material collect @ middle & fuse to form membraneous disk

- G_1 & G_2 serve as checkpoints → if something went wrong in S phase, cell dies @ G2 checkpoint.
  - → if didn't die @ G2 checkpoint, the daughter cell would only have ½ genetic content needed & not function right

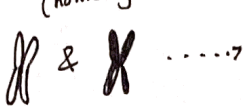
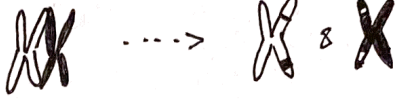
  - Cells knowing when to divide:
    - → Signals to YES DIVIDE: growth factors / stimulatory signals
      - → cells that need to divide have a receptor for this specific growth factor / hormone
      - → issues that could lead to cancer: • abnormal relay protein, active all time, w/o signal
    - → signals to STOP DIVISION: same as stimulatory but inhibitive & "STOP" instead
      - → issues to lead to cancer: • absent inhibitory signal or receptor
        • absent / non-functional relay protein



  - Cancer Cells
    - → not only grow on top of eachother, but migrate & don't stay in 1 place. Form in primary tumor, then move to other parts of body.
      - → Metastasis: tumor cells moved into blood cells to invade completely diff body areas
    - → keep growing despite their lack of free space (are not density-dependent inhibited)
    - → no anchorage dependence — grow whether or not in contact w/ suitable surface
  - BCRA-1 gene: when mutated, puts woman @ increased risk for breast/ovarian cancer
    - → pre-existing cells?

• **L10:** (only writing down stuff I really don't know)

- Meiosis vs. Mitosis
  - Meiosis:
    1. homologs *pair* & crossing over occurs (before metaphase I)
    2. offspring not identical 2 parent
    3. sex cells use this
    4. genetic content = halved
  - Mitosis:
    1. homologs don't pair
    2. offspring identical
    3. somatic cells use this
    4. genetic content (=) to parent cell

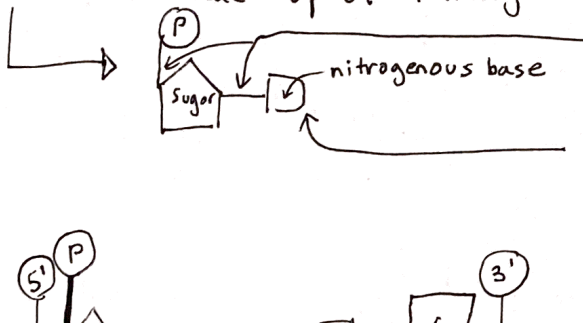
- 2 key stages for variation in Meiosis:
  1. crossing over (tetrads!) (homologous)  ("dancing") 
  2. Independent orientation (during metaphase!)
     - Its *RANDOM* which chromosomes go into which temp. daughter cell after metaphase I. Random how they split after lining up
- total number of chromosomes that meiosis can produce in gametes = 2^n = variation! (combinations?)

• **L11:** (~~didn't look @ outline yet~~) (none)

• **L12:** (none)

• **L13:**

- Nucleotides → made up of 1. nitrogenous base (A, T, C, G for DNA) 2. deoxyribose for DNA 3. phosph. group

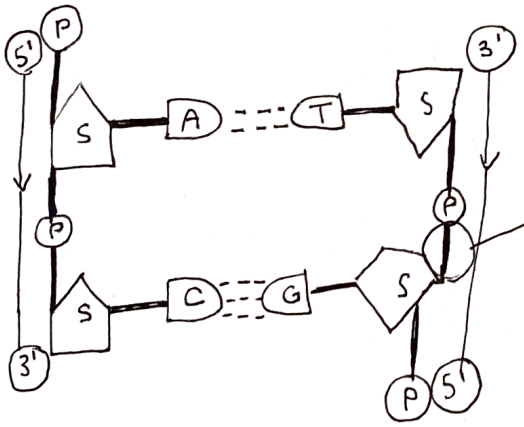


★covalent bonds

Polynucleotides are held together by hydrogen bonds!

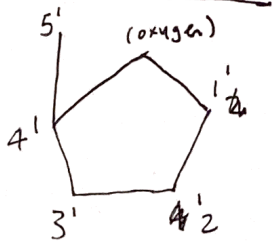
— = covalent bond

--- = hydrogen bond



★Phosphodiester bond = covalent bond connecting sugar of 1 ~~phosphate~~ nucleotide group to phosphate group of a dif one.

DEOXYRIBOSE



- #s = carbons
- 5' bonds to own phos. group
- 3' binds to other phos. group

- Diff. about somatic cells.
  - All have DNA, but diff body cells are still diff because they some genes in each are "turned on" or transcribed heavily whereas others aren't. Genes they express are different, ∴ so are the proteins.

BIO TEST 3

L15 - IMMUNITY

- Homeostasis: ≠ flat line, NO SET POINT, has ebbs & flows
  - ex: hypothalamus keeping our body @ healthy temp
  - change in a variable triggers a response which counteracts change = neg feedback
  - ex: caffeine = NEGATIVE FEEDBACK on sleep homeostasis, bc it is a competitive inhibitor to andenosine, & blocks that "sleepiness factor" from making us tired

INNATE IMMUNITY

- @ birth, FASTER THAN ADAPTIVE, nonspecific, no memory
- ex: acidic environment & phagocytes in uterus killing sperm
- faster bc always ready

2 TYPES OF CELLS

- white blood cells (natural killer cells)
- PHAGOCYTES (also white blood cells)
  - a) NEUTROPHILS:
  - b) MACROPHAGES: ("big eaters")

INFLAMMATORY RESPONSE

(innate internal)

- caused by a release of HISTAMINE & other chemicals
  - histamine causes blood vessels to dialate & b leaky
  - necessary so fluid can easily pass 2 affected area
  - also causes an elevated white blood cell count
  - symptom = fever, red, swelling

EXTERNAL INNATE: skin, an acidic environment (vag), secretions, mucus, membranes, hair

⇩ if breached

INTERNAL INNATE: phagocytic cells, natural killer cells, defensive proteins, inflammatory response!

= complement system proteins "punch holes"
↳ signalling proteins attract more phagocytes

LYMPHATIC SYSTEM INVOLVES BOTH:
↳ "body's" custodian

Diff Section:
VACCINES & STDs:
↳ refer to GRQs & google & class outline

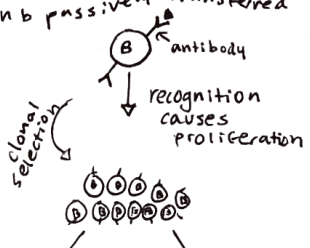
ADAPTIVE IMMUNITY

- Develops over lifetime, slower, specific responses, memory
- LOOK @ ALL FIGURES IN CLASS OUTLINES THAT ARE WELL-DETAILED!
- only vertebrates

Carried out by LYMPHOCYTES (T & B cells) (also white blood cells)

HUMORAL RESPONSE

- pathogens in body fluids
- B cells
- can b passively transferred



PLASMA CELLS aka EFFECTOR B CELLS

- PRODUCE ANTIBODIES w/ their big ER's & secrete antibodies to go out & bind more antigens

(last 5-6 days)

MEMORY CELLS

- surveillance cells
- last decades
- now can multiply even quicker

CELL-MEDIATED RESPONSE

- infected body cells
- T cells

(check detailed diagram on class outline!)

- cytotoxic T cell recognizes abnormalities on an infected cell.
- self-nonself complex, cytotoxic T cell BINDS to that cell.
- releases proteins that punch holes in cell

★ANTIBODIES DO NOT KILL CELLS → just FLAG DOWN other immune cells which do killing (ex: phagocytes)