

Review for Final Part 2

①

I. a. DNA replication:

1. helicase (enzyme) unwinds a section of DNA, breaking H-bonds.
2. At each replication fork, DNA polymerase makes a complementary strand to each parent strand. It takes incoming nucleotide triphosphates, breaks off 2 phosphates, and forms a new phosphodiester bond to connect each new nucleotide to the growing chain. Only the base complementary to opposite base on the template strand will fit and get incorporated.

At each replication fork, one new DNA strand is made continuously (the "leading strand"). The other is made in short segments, because DNA polymerase can only travel from the 3' to 5' end of template DNA. The short segments are later attached to each other by DNA ligase. Eventually, all DNA is copied.

b. RNA Transcription:

1. DNA unwinds (probably via helicase)
2. RNA polymerase synthesizes a strand of RNA complementary to the DNA template strand. ~~Each~~ incoming nucleotide triphosphate has ~~the~~ 2 phosphates removed and a new phosphodiester bond is formed to attach it to the rest of the growing chain. When a "stop" sequence is reached, the pieces disassemble: RNA, DNA, RNA polymerase separate
3. mRNA is processed. introns (non-coding sections) are cut out (by enzymes) and the exons (coding sections) are spliced together.

C. Protein translation:

1. Initiation: mRNA binds to small subunit of ribosome (in cytoplasm of cell). "start" codon on mRNA goes in the "P-site" on Ribosome. Activated tRNA w/ complementary anticodon comes to P-site and H-bonds to start codon. Large subunit of ribosome attaches.

(2)

1c continued. 2. Elongation - charged tRNA w/ anticodon complementary to next codon comes in to A-site (adjacent to P site). It H-bonds to codon. The amino acid attached to the first tRNA in the P site is removed from that tRNA and attached to the aa on the 2nd tRNA (new peptide bond is formed). Ribosome moves down by 3 bases. First (uncharged) tRNA comes off. 2nd tRNA w/ 2 aa's attached is now in P-site. A site is empty. New charged tRNA comes in. Cycle repeats many times.

3. Termination - "stop" codon is reached. An enzyme called releasing factor cuts the polypeptide from the last tRNA. All pieces (mRNA, protein, ribosome subunits, tRNA) separate.

1 d. e⁻ transport chain and ATP production

NADH and FADH₂ go to ~~a~~ series proteins on the inner mitochondrial membrane, and are oxidized to NAD⁺ or FAD. The e⁻ they lose are transferred in many steps between proteins and mobile e⁻ carriers. At ~~p~~ enzyme complexes I, III, and IV, as e⁻ are transferred, the energy released is used to pump protons (H⁺) into the intermembrane space. This creates a proton gradient (different concentration of H⁺ on each side of membrane). The final acceptor of e⁻ is O₂.

The proton gradient is a high-energy state. H⁺ "want" to have equal concentrations on each side of membrane. The only way for H⁺ to get back to the other side is to go through a protein called ATP synthase. The energy released by H⁺ going from higher to lower concentration is used to synthesize ATP from ADP + P_i.

2. mRNA - carries genetic info from DNA to ribosome, where proteins are synthesized.

rRNA - major component of ribosomes, the site of protein synthesis.
tRNA - adapter molecules that deliver the appropriate aa to the growing protein. Interpret genetic code.

(3)

3. In a substitution mutation, only one amino acid is changed.
 (unless it's a silent mutation - then no aa's are different.)
 In a frameshift mutation, many aa's are changed. ~~at~~ The entire sequence after the insertion or deletion will give a totally different amino acid sequence. The resulting protein is totally different from the original.

4. oxidoreductase would catalyze step 6 of glycolysis, steps 3, 4, 6, and 8 of citric acid cycle (and others -)

Ligase - ? maybe none. But ligases would catalyze aa's \rightarrow protein or nucleotides \rightarrow DNA or RNA

lyase - Step 7 of CAC (add H₂O to double bond), Step 2 of β -ox

isomerase - Step 2 of glycolysis, Step 5 of glycolysis, Step 2 of CAC

transferase - maybe step 1 and 3 of glycolysis - Phosphate transferred

hydrolase - maybe step 1 of CAC - acetyl group removed from

CoA

5. Carb. digestion starts in mouth w/ salivary amylase starting to hydrolyze starches. (others don't start in mouth)

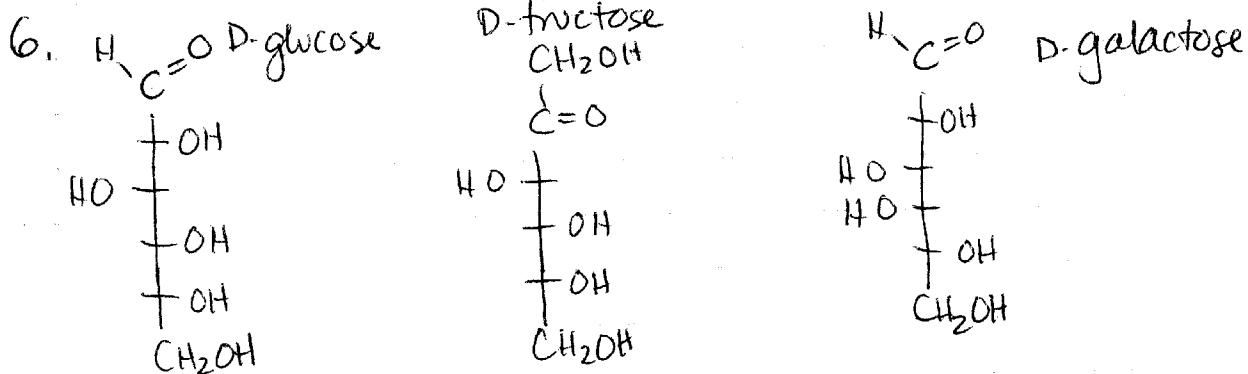
Protein digestion starts in stomach. Pepsin hydrolyzes some peptide bonds.

In small intestine, digestion of carbs, proteins, fats continues or starts.

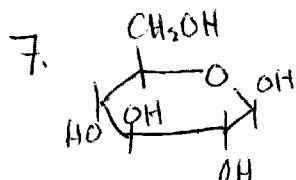
carbs hydrolysis sugars \rightarrow absorbed into bloodstream.

fats have to be hydrolyzed, transported across intestinal lining, re-attach to form TAG's, then coated w/ proteins for transport to cells. Cells hydrolyze TAG's as needed.

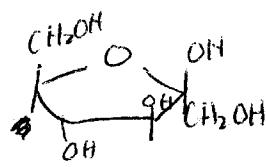
Proteins hydrolysis aa's \rightarrow absorbed into bloodstream
 (similar to carbs. Monomers are water-soluble.)



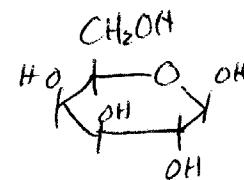
(4)



β -D-glucose

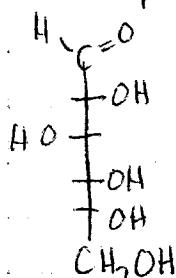


β -D-fructose

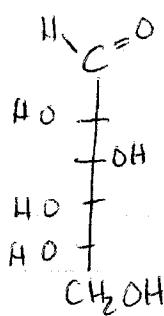


β -D-galactose

8. D-glucose and L-glucose are enantiomers of each other (nonsuperimposable mirror images)



D-glucose

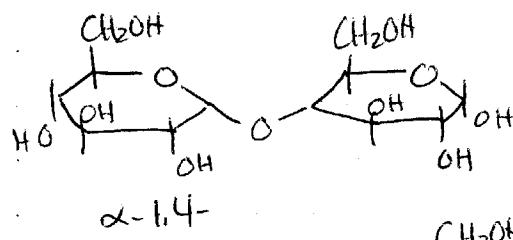


L-glucose

opposite @ each chiral C.

9. galactose - 4 chiral C's.

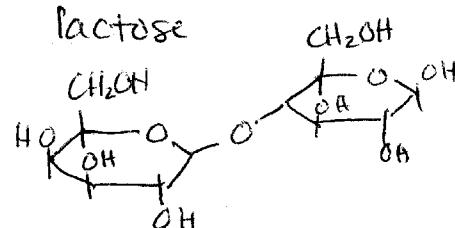
10. maltose



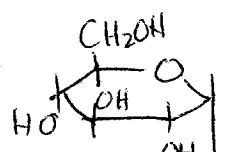
α -1,4-

- Fructose - 3 chiral C's.

- lactose

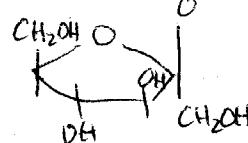


β -1,4-

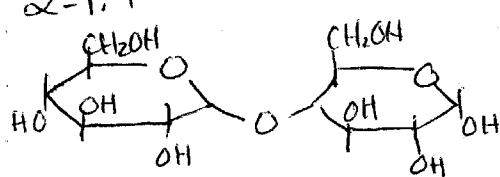


sucrose

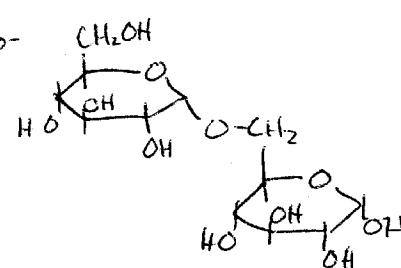
α, β -1,2-



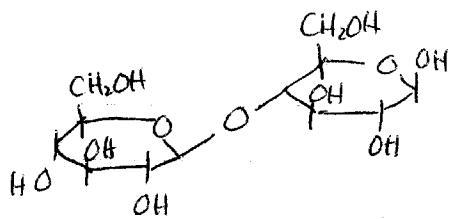
11. α -1,4-



- α -1,6-



(5)

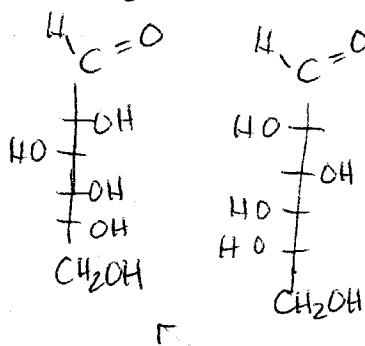
 β -1,4

12. cellulose, amylose, and amylopectin are found in plants. glycogen is present in animals. All consist of D-glucose subunits. Cellulose and amylose are unbranched, while amylopectin + glycogen are branched. Amylose is helical. Cellulose is in parallel strands. Cellulose contains β -1,4 glycosidic bonds, which can't be digested by humans. The others contain α -1,4 glycosidic bonds (and α -1,6 if branched). These can be hydrolyzed by humans.

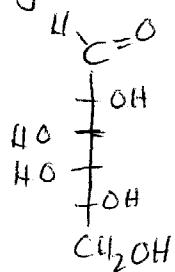
13. enantiomer - nonsuperimposable mirror image.

diastereomer - a stereoisomer that isn't an enantiomer.

ex: D-glucose and L-glucose are enantiomers



D-glucose and D-galactose are diastereomers:



14. Mutarotation is the following process:

β anomer \rightleftharpoons open chain \rightleftharpoons α anomer
inter conversion of α and β anomers for sugars.

Only sugars that can mutarotate are able to be oxidized.

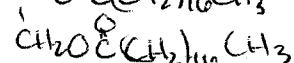
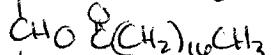
The open chain form can be oxidized. (reducing sugar)

Sugars with a free anomeric C can undergo mutarotation.

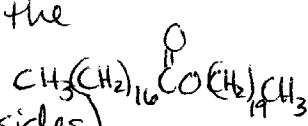
If ~~the~~ anomeric C's are tied up in glycosidic bonds, the sugar can't oxidize.

15. Wax - ester w/ ~~too~~ long hydrocarbon chains (on both sides)

triacylglycerol - triester of glycerol + 3 fatty acids.



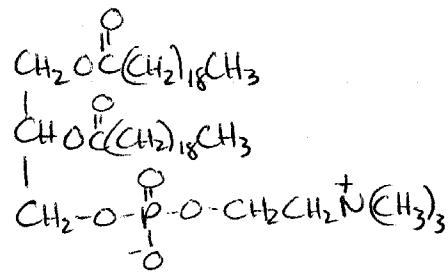
used for food/energy



used for protective coatings

(6)

glycerophospholipids
 contain glycerol,
 2 fatty acids,
 phosphate, amino
 alcohol

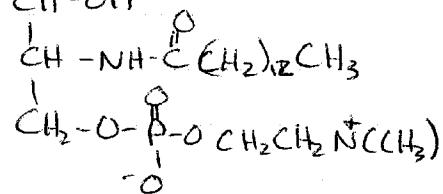
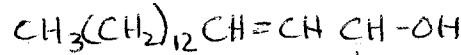


present in cell membranes - part of the lipid bilayer.

sphingolipids - in brain, nerve tissue

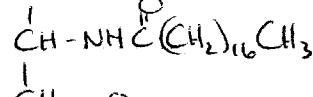
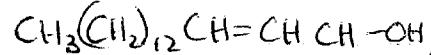
contain sphingosine, 1
 fatty acid, phosphate,
 amino alcohol

also in
 cell membranes.

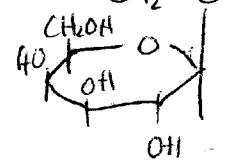
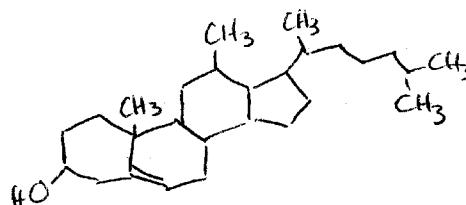


glycosphingolipids

contain sphingosine,
 1 fatty acid, sugar



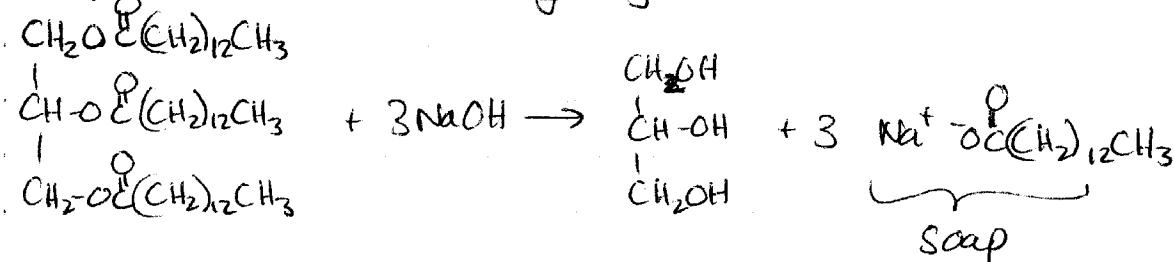
Steroid
 ex: cholesterol



cholesterol is in cell membranes

other steroids: hormones — testosterone, estrogen, progesterone, etc.

16. soaps made from base hydrolysis of a fat or oil.

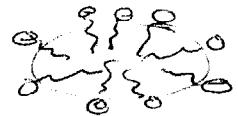


17. Soaps have a charged "head" and a long nonpolar tail.
 many soap molecules can form micelles around grease
 or oil droplets in solution, with the nonpolar tail of the
 soap embedded in the grease. The charged head is on

(7)

the surface of the micelle, in contact with water.

The entire micelle is soluble in water ~~so~~ and can be washed away.



18. could add acid or base - this would change charges on some of the acidic and/or basic residues, and would therefore disrupt the salt bridges that help hold the 3^o structure in place. For example, if you add base, all of the + sidechains would become deprotonated, changing their charges to - charge. A + charge is no longer attracted to a - charge, so no salt bridge at this location. Protein unfolds partially.
- could add an organic solvent - protein would fold very differently. Normally, a protein folds so that its nonpolar sidechains are pointing inside and ^{many} ~~most~~ of the polar / charged groups are on the outside. In a nonpolar solvent, the nonpolar side chains would face the outside of the protein and the polar ones point inside. The hydrophobic and hydrophilic interactions would be disrupted.

Other denaturing agents: heat, agitation, heavy metals, high conc. of salts, detergents etc. (see notes for a brief explanation why.)

19. The primary structure of a protein is the amino acid sequence, typically written from N-terminus to C-terminus. The ~~is~~ sequence affects how the protein will fold (but it's not exactly predictable). The secondary structure consists of sections of regular and repeating structures like α -helix or β -sheet sections. These structures are held in place by H-bonds between C=O and N-H groups along the backbone of the protein. (Doesn't involve R groups at all)
- Tertiary structure - overall 3-D shape/folding of protein, influenced by interactions between R groups. Types of interactions: salt bridges (+ and - charged side chains attracted), disulfide bridges (-S-S- bonds - covalent) , groups on cysteines react + form -S-S- bonds - covalent) ,

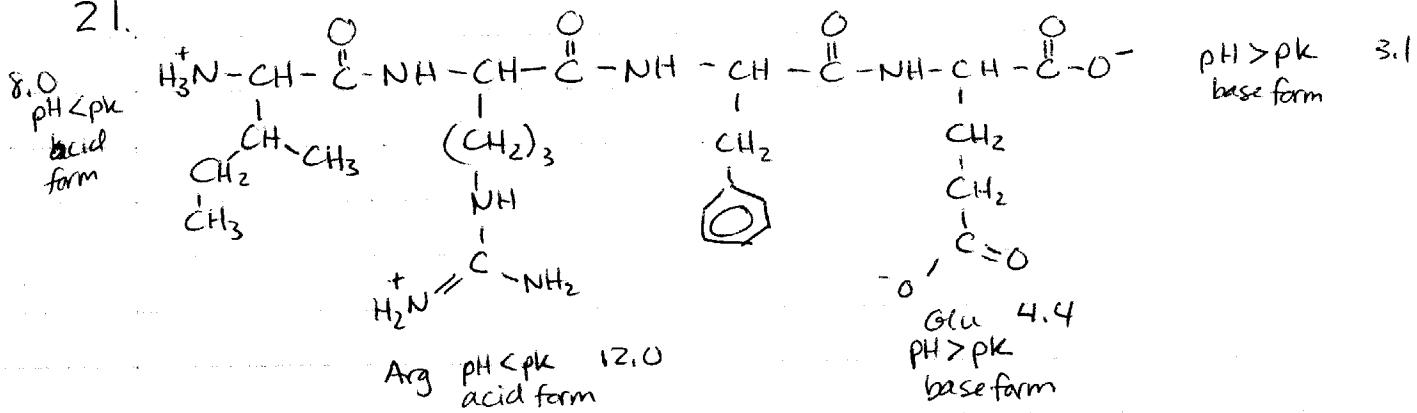
(8)

hydrogen bonding, hydrophobic interactions (nonpolar groups cluster together on inside to avoid water), hydrophilic interactions (lots of polar, charged groups on exterior of protein).

quaternary structure - interactions between R-groups on different polypeptide chains in a multi-subunit protein. Same types of interactions as 3^o structure: H-bonding, disulfide bridges, salt bridges, hydrophobic + hydrophilic interactions.

20. If an enzyme folds differently, its active site will probably be disrupted / will have a different shape or will be gone! If there's no active site, it can't bind substrate, so no rxn will occur.

21.



22. a. Ala and Val, Leu and Ile, Met and Phe, etc (choose 2 nonpolar)

b. Ser and Thr, Asn and Ser, Tyr and Gln, etc.

c. Cys and Cys

d. Ser and H_2O , Asn and H_2O etc (polar + H_2O solvent)

e. Asp and Lys, Glu and Arg etc. (choose 1 \oplus , one \ominus)

23. structural (collagen, keratin)

contractile (muscles contract ... actin, myosin)

transport (hemoglobin transports O_2 through bloodstream)

Storage, hormones, enzymes! antibodies etc. See p. 643

24. Enzymes speed up reactions by: ① lowering activation energy

by destabilizing the substrate (has to change shape slightly to fit)

② holding substrate in correct orientation ③ providing acidic, basic, other groups for catalysis ④ bringing substrate + catalytic sites together

(9)

25. The active site of a particular enzyme has a specific shape, and it's only complementary to one type of substrate. The catalytic groups in the active site are also specific to the substrate and type of reaction being catalyzed. Other substrates won't fit in the active site and/or the catalytic groups could be inappropriate for the rxn being catalyzed.

26. Competitive inhibition - the inhibitor is similar (in structure) to the substrate. It can bind to the enzyme at the active site, but for some reason (something is missing in the structure) it can't react. So it takes up active sites and doesn't do anything once bound. Therefore, fewer substrate molecules are able to bind to enzymes because fewer active sites are available. If you increase [S], you increase the likelihood of substrate binding instead of inhibitor, so rate of rxn increases.

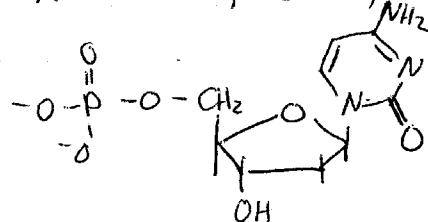
Noncompetitive inhibition - the inhibitor doesn't look like the substrate. It binds to the enzyme but not at the active site. When it's bound, it causes the shape to change in such a way that the active site is no longer functional (^{its} shape changes). Substrate can no longer bind to ^(w/ inhibitor attached) enzyme, so rate is slowed.

Adding more S has no effect. Decreasing [I] would help - this decreases the chances that E will have I bound.

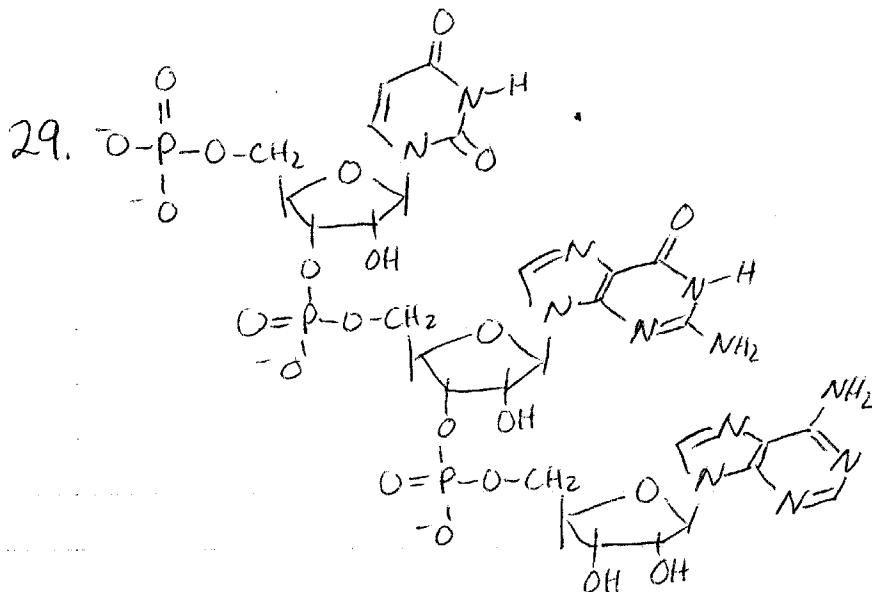
Inreversible inhibition - inhibitor forms a covalent bond to enzyme + can't be removed.

27. Cofactor - an organic molecule or metal ion needed for certain enzymes to be functional. Enzyme can't catalyze rxn without it.
ex: Fe^{2+} , Co^{2+} , heme, some vitamins

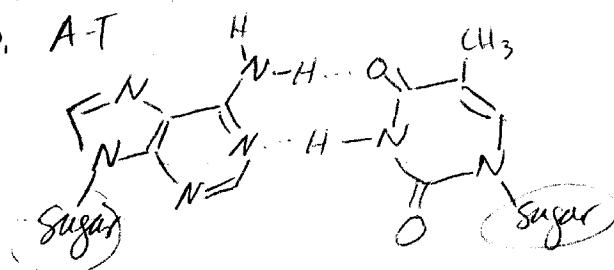
28.



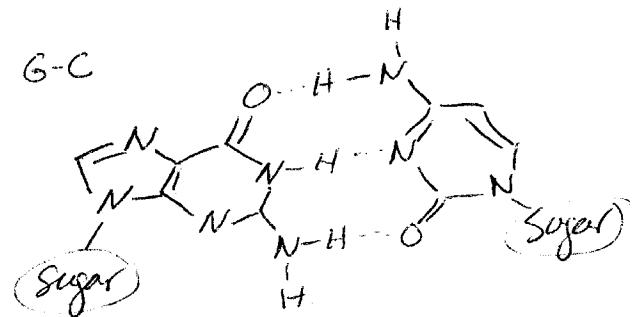
10



30 A-T



6-0



31. 3' AATTGCGCTATAAGGTCA - 5'

5' TTAACGCGATATTCCAGT- 3'

32. Complementary RNA

5' UUAACGCGAUAAUCAGU-3'
 protein - Leu-Thr-Arg-Tyr-Ser-Ser -
 N-term C-term

33. significant ^{added} change: see frameshift mutation - add a base

ex: 3' ACATTGCGCTATAAGGTCA 5'

mRNA S' UGUAAACGCGAUAUUCCAGU-3'

protein Cys - Asn - Ala - Ile - Phe - Gln -

insignificant change : silent mutation

or one that changes a sidechain to a very similar sidechain.

maybe a change in the UUA codon to UUG
(Leu) (also Leu)

all aa's after the addition
are different! This will
affect the folding of the
protein. Will probably be
inactive.

(11)

or maybe a change in UUA codon to AUA
 (Leu) (Ile)

Leu and Ile are pretty similar - both nonpolar, approx.

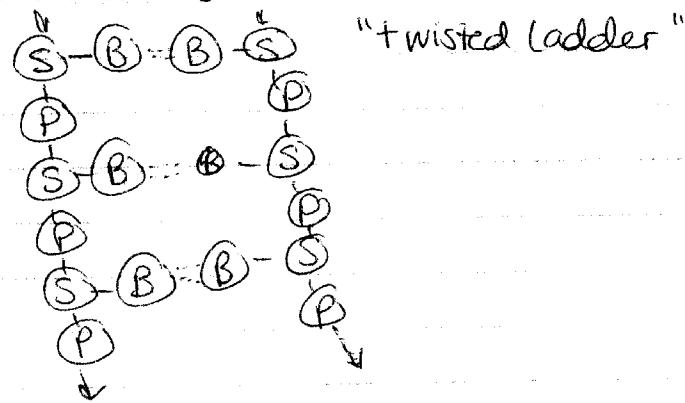
Same size.

so ^{new} RNA (mutated) might be: ${}^5' \text{AUAAACGGCAUAUUCCAGU} {}^{3'}$
 mutated DNA would be: ${}^3' \text{TATTGCCGTATAA} \overset{\uparrow}{\text{GGTCA}} {}^{-5'}$
 this base changed

34. DNA - Double helix - 2 strands twisted together.



alternating sugar-phosphate-sugar-phosphate
 backbone, Bases hydrogen bonded together
 on the inside



35. Anticodon - a 3-base sequence on one end of a tRNA molecule. The anticodon sequence is complementary to a codon sequence on mRNA. The tRNA w/ complementary anticodon also carries an amino acid that corresponds to the codon. For example, ^{are} mRNA codon for Leu is UUA. The anticodon ^{in tRNA} would have the sequence AUU, and would also have the amino acid Leucine attached to ~~it~~ the tRNA molecule (if the tRNA is "activated" or "charged")