

THE COMPREHENSIVE BOTTOM LINE

BIOLOGY REVIEW:

Cellular Organization:

1. Cells are compartmentalized: protects from the environment; prevents diffusion of contents & maintain specific pH.
2. Prokaryotes (bacteria) lack a nucleus & have few organelles.
3. Eukaryotes (plants, animals, protozoa) have a nucleus & a bunch of different organelles.
4. Hierarchy of biological structure: (a) inorganic precursors (b) metabolites (DHAP; pyruvate; citrate, etc) (c) building blocks (amino acids, glucose, fatty acids, etc) (d) macromolecules = biopolymers (proteins, polysaccharides, nucleic acids) (e) Supra-molecular complexes (Multi-enzyme complexes, ribosomes, etc) (f) organelles (g) cell
5. Organelle Function: Mitochondria = ATP; oxidating power plant; Golgi = protein sorting; Rough ER = protein synthesis; Smooth ER = phospholipid synthesis; lipid modifications; Peroxisome = H_2O_2 ; problem molecule oxidation; Nucleus = DNA; RNA synthesis; Cilia & Flagella = motility & sensory organelle.

CHEMISTRY REVIEW:

Bonding & H_2O :

1. Covalent bonds: strong bonds formed by sharing electrons
2. Ionic bonds: opposite charges attract
3. Hydrogen bonds: 2 electronegative atoms (ie O or N) share a proton
4. Van der Waals interactions: weak attractions & repulsion that occur when molecules are very close
5. Hydrophobic interactions: occur because H_2O attempts to exclude nonpolar molecules.
6. Bond strength: Covalent > ionic & hydrogen > Van der Waals
7. H_2O is polar & due to tetrahedral electron orbitals, is capable of sharing in 4 hydrogen bonds.

pH & Buffers:

8. $pH = -\log [H^+]$
9. Acidic if $pH < 7$; Basic if $pH > 7$
10. K_a is the acid dissociation constant; $K_a = [H^+][A^-]/[HA]$
11. $pK_a = -\log K_a$;
12. Buffers resist change in pH when the pH is within ± 1.0 pH unit of the pK_a of the buffer. Weak acids & bases act as buffers when $pH = pK_a \pm 1.0$
13. Henderson-Hasselbalch eqn: $pH = pK_a - \log [HA]/[A^-]$; allows prediction of pH based on the concentrations of the acid & base forms of a weak acid or base. The pK_a is the pH at which $[HA] = [A^-]$.

Carbon Chemistry:

14. The chemistry of living organisms is aqueous organic chemistry
15. A saturated carbon has 4 single bonds with tetrahedral geometry; the single bonds freely rotate
16. Carbonyl carbons are susceptible to nucleophilic attack
17. Carboxylic acids, amides, & esters are susceptible to nucleophilic acyl substitution (example: the peptide bond amides are susceptible to attack by H_2O = hydrolysis)

AMINO ACIDS & PEPTIDES:

Amino Acids:

1. Amino acids contain both an amine & a carboxylic acid
2. Only 20 α -amino acids (aa) are used to synthesize proteins (only the L-form). Know the structures of the 20 aa, each complete name, three letter code & 1 letter code.
3. The side group of each amino acid determines the chemical behavior specific to that aa.
4. Hydrophobic side groups are found inside proteins & hydrophilic polar or charged side groups are found at the surface of proteins.
5. Polar, uncharged aa side groups: Ser, Thr, Gln, Asn, & ~Tyr (if the pH gets high, the alcohols & Cys can lose a proton)
6. Polar charged aa side groups: Basic = Lys, Arg, His; Acidic = Glu, Asp; these do depend on pH
7. Hydrophobic: Leu, Val, Ala, Ile, Cys, Met, Aromatic = Phe, Trp, & ~Tyr

Peptides:

8. Peptides are linear polymers of amino acids linked to one another by amide bonds.
9. The peptide bond is an amide bond that has partial double bond character; it is not free to rotate. Geometry of the peptide bond is trans configuration.
10. The peptide unit contains the peptide bond and all the atoms bonded directly to the carbonyl carbon and the amide nitrogen; the peptide unit is planar.
11. Hydrolysis of the peptide bond is almost always thermodynamically favorable.

PROTEINS:

Electrophoresis of Proteins:

1. Electrophoresis separates by size & charge
2. Isoelectric focusing separates by isoelectric point = pH at which net charge is zero.
3. SDS PAGE separates proteins by size due to homogenous binding of SDS (~1 SDS/2 aa)
4. β -ME, DTT, or DTE are used to reduce disulfide bridges back to free sulfhydryls.
5. 2-Dimensional gel electrophoresis combines (1st) isoelectric focusing & (2nd) SDS PAGE

Protein Structure:

6. Primary protein structure of a protein is the linear sequence of amino acids
7. Secondary protein structure refers the local 3-D structure adopted by an aa sequence (ie α -helix & β -sheet).
8. α -helix is tightly coiled; 3.6 aa residues/turn; each residue is H-bonded to the 4th residue above & below in the same helix; the aa side groups stick out away from the helix axis.
9. β -sheet polypeptide strands are nearly fully extended; Hydrogen bonding occurs *between* strands; multiple strands are usually fairly flat & known as β -sheets; aa side groups stick out on either side of the sheet.
10. β -turns are tight, compact turns that typically connect β -strands;
11. Proline breaks up α -helix & β -sheet structure; proline & glycine are frequently in β -turns.
12. Tertiary protein structure: spatial arrangement of all the amino acids in a protein.
13. Quaternary protein structure: spatial arrangement of proteins in a protein complex.
14. Cystines = disulfide bridges between 2 cys residues (disulfide = oxidized form).
15. Insulin = 1st protein sequenced; tedious chemical analysis carried out by Fred Sanger.
16. Amino acid analysis identifies which & how much of each amino acid is present.
17. Edman degradation = automated amino acid sequencing.
18. Collagen consists of a triple helix that is rich in proline & hydroxyproline; hydroxyproline synthesis requires Vitamin C; without Vitamin C, collagen is defective (Scurvy).
19. Prions (slow viruses) are infectious proteins; normal forms of prion proteins are expressed in the organism; the normal form (PrP^C) & the infectious form (PrP^{Sc}) have different 3-D conformations. It is believed that the infectious form binds to the normal form & converts the normal form to the infectious form. The infectious form persists & forms plaques because the cells & tissue are unable to degrade the very stable infectious form.

MYOGLOBIN & HEMOGLOBIN:

1. Many plants & animals have similar O₂ binding proteins with a heme prosthetic group.
2. The heme group is close to planar with a central Fe⁺² ion chelated by 4 heme nitrogens.
3. The 3D structure of myoglobin was the 1st for any protein (John Kendrew, 1957). Showed the protein was (a) compact (b) contained no H₂O molecules inside (c) only hydrophobic amino acids were packed inside (the 2 exceptions are the proximal & distal His).
4. O₂ only binds the ferrous (Fe⁺²) form & not the ferric (Fe⁺³) form.
5. Myoglobin/Hemoglobin have to perform 2 tricks:
(1) Bind O₂ without having the electronegative O₂ oxidize Fe⁺² to Fe⁺³
(2) Prevent tight, linear binding of CO₂ to Fe⁺²; the distal His prevents linear CO₂ binding
6. The 3-D structure of myoglobin, one hemoglobin subunit, leghemoglobin (plants) and erythrocyruorin (insect) are nearly superimposable but the amino acid sequence of these proteins vary considerable: "Different aa sequences can give rise to proteins with the same 3D structure which functions similarly in different organisms."
7. Allosteric behavior: binding of a molecule at one site on a protein affects the binding or activity at a separate site on the protein.
8. Hemoglobin is allosteric because of (1) Cooperative binding of O₂ {the binding of the 1st O₂ increases the affinity for O₂ at a 2nd site} (2) H⁺ binding lowers affinity for O₂ (3) CO₂ binding lowers affinity for O₂ (4) one molecule of 2,3-BPG binds between the two β-subunits which results in lower O₂ affinity in all 4 subunits.
9. Bohr Effect: Higher acid (H⁺) & CO₂ enhance release of O₂.

ENZYME KINETICS & A LITTLE THERMODYNAMICS:

1. The change in Gibb's free energy, ΔG , identifies if a reaction is favorable.
 $\Delta G < 0$; favorable as written (left to right)
 $\Delta G > 0$; the reverse rxn is favored (right to left)
2. Enzymes do not affect ΔG or the direction of the favored rxn. Enzymes only speed it up.
3. Enzymes lower the energy of activation, ΔG^\ddagger , required to reach the transition state.
4. Enzymes: (1) required in trace amounts (2) unchanged by the net rxn (3) enhance rxn rate but do not affect equilibrium conc (4) catalyze the forward & reverse rxn equally
5. Michaelis-Menten Eqn: $v = V_{\max} [S]/([S] + K_M)$ The initial rate of a catalyzed rxn is dependent on the substrate conc, [S], & the Michaelis constant, K_M , for that enzyme.
6. If $[S] \ll K_M$; $v = V_{\max} [S]/K_M$
If $[S] = K_M$; $v = \frac{1}{2} V_{\max}$
If $[S] \gg K_M$; $v = V_{\max}$
7. For most enzymes, $10^{-7} \text{ M} < K_M < 10^{-1} \text{ M}$; a low K_M (say, 10^{-7} M) indicates stronger binding than a higher K_M (say, 10^{-5} M).
8. The K_M indicates the substrate conc, [S], that the enzyme is working at half max. If the [S] is much lower than K_M , the enzyme won't be able to turn over the rxn very fast.
9. The fraction of sites filled in an enzyme, $f_{ES} = [S]/(K_M + [S])$
10. In the cell, [S] is typically between 1-100% K_M .
11. Irreversible inhibitors essentially kill the enzyme covalently & do not dissociate or leave.
12. Competitive inhibitors compete for the "substrate" binding site. K_M gets bigger, V_{\max} stays the same. The factor is $(1 + [I]/K_i)$
13. Noncompetitive inhibitors bind at a site distinct from the substrate binding site. K_M stays the same, V_{\max} gets slower (smaller). The factor is $(1 + [I]/K_i)$
14. There are also uncompetitive and mixed mode inhibitors in which both the V_{\max} & K_M change.

CATALYSIS:

1. **Induced Fit Model (Dan Koshland, 1958):** substrate binding site is not shaped exactly like the substrate. Instead, the active site and, sometimes even the substrate, alter their shape upon binding to fit one another. Also, upon binding, the active site tends to match the transition state of the reaction which functions to lower the transition state energy.
2. **Transition state analogs** are very good competitive inhibitors: they mimic the transition state of the reaction but are unable to be converted to product. These analogs bind to the enzyme stronger than either the natural substrate or product.
3. **Penicillin** is an irreversible inhibitor glycopeptide transferase, an enzyme that catalyzes a critical peptide cross-linking reaction for bacterial peptidoglycan cell wall synthesis.
4. **Lysozyme** hydrolyzes the polysaccharide linkages of the peptidoglycan cell wall.
5. **Lysozyme** catalysis involves the formation of a covalent bond between enzyme and substrate; this is known as an 'adduct'.
6. **Lysozyme**, like most enzymes, operates best in a fairly narrow pH range.
7. **Proteases** catalyze the hydrolysis of peptide bonds; specificity depends on the aa side groups. Trypsin cuts after Lys & Arg; Chymotrypsin cuts after Phe, Trp, & Tyr. Elastase cuts after Gly & Ala (& Cys & Ser if given enough time).
8. **Zymogens** = proenzymes = precursor proteins that are activated by proteolysis. This is a form of regulation that allows the protein to be made & stored in an inactive form where it can be accessed quickly.
9. **Other enzymatic regulation**
 - (1) reversible effectors: (a) inhibitors; (b) activators
 - (2) binding of regulatory proteins
 - (3) reversible posttranslational modifications: (a) phosphorylation of Ser, Thr, & Tyr
(b) O-GlcNAc modification of Ser & Thr
10. The catalytic triad of the serine proteases are composed of Ser, His, & Asp. The His & Asp act in concert to accept a proton from Ser, thus generating a powerful nucleophile which can attack the peptide bond of a protein. The mechanism involves a stable Serine-substrate adduct.
11. In the Aspartic proteases, 2 Asp residues stabilize the peptide carbonyl while H₂O attacks.
12. Trypsin, Chymotrypsin & elastase share the same catalytic triad; they differ in specificity because trypsin has a negatively charged binding pocket that accepts Lys & Arg side groups, while chymotrypsin has a pocket which binds large hydrophobic side groups of Phe, Tyr, Trp.
13. **Ribozymes:** (1) made of RNA (2) follow Michaelis-Menten kinetics (3) show substrate specificity.

MOLECULAR MOTORS:

1. **Molecular motors** are mechanochemical enzymes capable of transforming chemical energy into work.
2. The traditional eukaryotic motor proteins move cargo from point A to point B.
3. **Myosin:** moves along actin filaments; responsible for muscle contraction, cytokinesis & some cell motility.
4. **Dynein:** moves toward minus-end of microtubules; responsible for ciliary/flagellar beat & organelle & vesicle movement.
5. **Kinesin:** most move to plus, a few to the minus end of microtubules; organelle & vesicle motor.
6. The power stroke of myosin follows ATP hydrolysis but not immediately; it is the release of inorganic phosphate that triggers the power stroke. If no nucleotide is bound to myosin, myosin binds very tightly to actin in a way that is referred to as 'rigor.'

METABOLISM OF CARBOHYDRATES:

Introduction:

1. **Carbohydrates** = sugars; used for (1) energy storage (glycogen, starch, dextran); (2) metabolic intermediates; (3) ribose in DNA/RNA; (4) structure (cell walls); (5) to modify proteins & lipids

(solubility & recognition).

2. 5 & 6 carbon aldoses & ketoses cyclize to form hemiacetal & hemiketal rings. Closed ring of glucose is favored ~ 100 to 1 in aqueous soln.
3. Sucrose is a disaccharide of glucose & fructose.
4. Glucose is stored for long term use as polymers. Animals: glycogen; plants: starch; bacteria: dextran.
5. Cellulose is a linear polymer of glucose which is stabilized by hydrogen bonds.
6. The exoskeleton of insects consists of polymers of a glucose derivative (*N*-acetyl glucosamine).

Metabolism Overview:

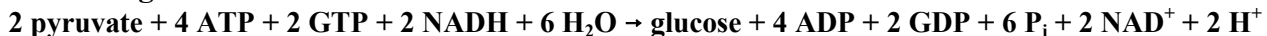
1. Catabolism: degradation pathways; generates energy.
2. Anabolism: biosynthetic pathways; consume energy.
3. Universal free energy currency = ATP; $\text{ATP} + \text{H}_2\text{O} \rightarrow \text{ADP} + \text{P}_i$ $\Delta G^{\circ\prime} = -30.5 \text{ kJ/mol}$; average cellular $\Delta G = \sim 50 \text{ kJ/mol}$.
4. Average human at rest turns over 40 kg ATP/day; average ATP molecule $t_{1/2} < 1 \text{ min}$.
5. Creatine~P provides short term reservoir to replenish ATP (abundant in muscle & sperm flagella).
6. ΔG for a reaction is dependent on the concentration of reactants & products:

$$\Delta G = \Delta G^{\circ} + 5700 \text{ J/mol} \log \frac{[\text{products}]}{[\text{reactants}]}$$

Glycolysis:

7. $\text{Glucose} + 2 \text{ P}_i + 2 \text{ ADP} + 2 \text{ NAD}^+ \rightarrow 2 \text{ pyruvate} + 2 \text{ ATP} + 2 \text{ NADH} + 2 \text{ H}^+ + 2 \text{ H}_2\text{O}$
 $\Delta G = \sim 100 \text{ kJ/mol}$; thus, this pathway is favorable.
8. Where: occurs in all living cells; eukaryotes: in the cytosol.
9. Why: generates energy & metabolites for (1) more energy via Krebs's if O_2 is available & (2) biosynthesis.
10. Regulation: hexokinase (inhib: Glc-6-P); phosphofructokinase (activ: AMP, Frc-2,6-BP; inhib: ATP, citrate); pyruvate kinase (Activ: AMP, Frc-1,6-BP; Inhib: ATP, Acetyl CoA, Alanine)
11. Phosphofructokinase is the key enzyme: the commitment step.
12. The 3 regulated enzymes catalyze reactions with fairly large negative ΔG . Doesn't do much good to regulate a reaction that is near equilibrium (small ΔG).
13. Erythrocytes have no nuclei or mitochondria & depend entirely on glycolysis for energy.
14. Fates of pyruvate: (1) enter Krebs's cycle as Acetyl CoA; (2) make OAA (to replenish Krebs's intermediates or start gluconeogenesis; (3) lactate (to replenish NAD^+ if O_2 is scarce).

Gluconeogenesis:



15. Formation of glucose from lactate or pyruvate.
16. Where: Liver, kidney & muscle of higher animals. Occurs in various organelles.
17. Why: to replenish glucose levels when glycogen stores are depleted.
18. What: the 3 steps of glycolysis with large negative ΔG values must be bypassed; all other glycolytic enzymes are used.
19. 1st pyruvate enters mitochondria & is carboxylated to form OAA; OAA is reduced to form malate; malate passes thru membrane into the cytosol; malate is oxidized back to OAA; OAA is then decarboxylated to form PEP. Cost = 1 ATP, 1 GTP/pyruvate.
20. Frc-1,6-BP & Glc-6-P are both simply hydrolyzed to remove a phosphate
21. Net cost for gluconeogenesis: 6 ATP equivalents & 2 NADH
22. Net gain for glycolysis: 2 ATP & 2 NADH
23. Carboxylation of pyruvate: pyruvate carboxylase utilizes biotin to add the CO_2 group.
24. Reciprocal regulation: AMP & Frc-2,6-BP (\uparrow glycolysis; \downarrow glucone.); Acetyl CoA, Glc-6-P (\downarrow glycolysis; \uparrow glucone.).

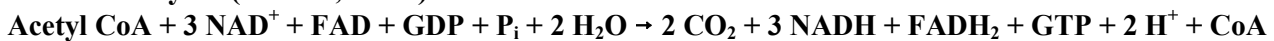
Glycogen Degradation & Synthesis:

25. Glycogen is a retrievable storage of glucose in animals; linear α -1,4 glycosidic bonds with α -1,6 branches.
26. Glucose units are released as Glc-1-P via glycogen phosphorylase.
27. The Glc-1-P has 2 fates: (1) if needed in the blood, Glc-1-P loses the P_i so that free glucose can pass through the membrane; (2) phosphoglucomutase catalyzes Glc-1-P \rightarrow Glc-6-P.
28. The lion's share of glycogen synthesis belongs to glycogen synthase:
 $UDP\text{-Glc} + \text{glycogen}_{(n \text{ residues})} \rightarrow \text{glycogen}_{(n+1)} + UDP$
Note the use of UDP-glucose (UDP-Glc).
29. Glycogen granule size is limited to the reach of glycogen synthase, which is only active when bound to glycogenin at the granule core. Number of glycogen particles = # of glycogenin.
30. Synthesis & degradation are reciprocally controlled by a number of hormones. One of these, epinephrine, stimulates cAMP production; cAMP activates protein kinase A (PKA); PKA directly inactivates glycogen synthase, while indirectly activating (via phosphorylase kinase) glycogen phosphorylase.

Pentose Phosphate Pathway:

31. Oxidative branch transforms Glc-6-P to Ribulose-5-P. This generates NADPH (which will be used for reductive biosynthesis) and CO_2 .
32. The Ribulose-5-P is one simple rxn away from forming ribose-5-P which is needed to make all of the nucleosides and nucleotides.
33. The non-oxidative branch of the PPP can transform excess ribulose-5-P/ribose-5-P into Gly-3-P & Frc-6-P (glycolytic intermediates) at no cost. This part of the pathway can also be used to synthesize ribose-5-P when NADPH is not needed by the cell.

Citric Acid Cycle (Kreb's, TCA):



34. Final common pathway for oxidation of all fuel molecules.
35. What: oxidizes Acetyl CoA (which often comes from pyruvate) to CO_2 .
36. Where: all living cells of all organisms; in eukaryotes: mitochondria.
37. Why: generates lots of high-energy compounds: 3 NADH; 1 $FADH_2$; 1 GTP. The high energy electrons feed into the ETS.
38. CoA = Coenzyme A; has a reactive thiol (can form thioester with carboxylic acids) at the end of a long leash.
39. Pyruvate DH is a very large, 60 subunit multienzyme (E1, E2, E3) complex responsible for the formation of Acetyl CoA. Lipoamide (E2) is a long leash that transfers an acetyl group from TPP (E1) to CoA. The E2 lipoamide is regenerated (oxidized) by FAD in the E3 active site.
40. OAA is replenished after one complete turn of the cycle. The acetyl CoA that enters the cycle is not lost (as CO_2) in the first pass through the cycle.
41. Regulated at citrate synthase, isocitrate synthase, & α -KG DH (& also pyruvate DH). ATP inhibits all but α -KG DH.

Electron Transport System (ETS):

42. Where: all living cells. Eukaryotes: at the inner membrane of the mitochondria.
43. What: accepts high energy electrons from NADH & $FADH_2$; ultimately donates the electrons to O_2 to make H_2O . During the process, electrons are sequentially passed along electron carriers; sometimes the energy from the process is harnessed.
44. Why: uses energy from e- transfer to pump H^+ out to form a $[H^+]$ gradient across membrane.

	<u>e- Donor</u>	<u>e- Acceptor</u>	<u>Function</u>
Complex I (NADH CoQ Reductase):	NADH	Q	pump 4 H^+
Complex II (Succinate DH)	$FADH_2$	Q	$2e^-$'s enter ETS
Complex III (Cytochrome Reductase)	QH_2	Cyt $c_{(ox)}$	pump 4 H^+
Complex IV (Cytochrome Oxidase)	Cyt $c_{(red)}$	O_2	pump 2 H^+ ; make H_2O

- 46. Co Q = Coenzyme Q = Q = ubiquinone: lipid (membrane) soluble e⁻ carrier.
- 47. Cytochrome c: H₂O soluble e⁻ carrier: exists in periplasmic space (bacteria) or intermembrane space of mitochondria. Carries one electron at a time.

Oxidative Phosphorylation:

- 48. **What:** H⁺ flow down the gradient (ie into the mito matrix) thru the F₀ portion of ATP Synthase causing the F₀ particle & the γ-subunit of the F₁ particle to spin like a rotary engine. The spinning γ-subunit causes conformational changes within the F₁ subunit so that ATP is released.
- 49. **Where:** at the mitochondrial inner membrane; ATP generation occurs in matrix; H⁺ flow into the matrix.
- 50. **Why:** This is how cells/organisms make the majority of the ATP that result from consuming nutrients. (Some cells/organisms use light energy to pump a H⁺ gradient)
- 51. Mitchell's chemiosmotic theory proposed that the proton flow could be used as an energy source to run the F₁F₀ ATP synthase. This is because, in the average mitochondrion, allowing protons to flow back across the inner mito membrane, from the intermembrane space to the mito matrix, 23 kJ of energy is liberated per mole of protons (H⁺).

NET YIELD OF GLUCOSE OXIDATION:

For the complete oxidation of Glucose to form 6 CO₂, the yield is 36 ATP (bacterial) or 30-32 ATP for mitochondrial oxidation. The lower yield in eukaryotes is due primarily to the cost in transport of ATP & NADH across the inner mitochondrial membrane.

Photosynthesis in Plants:

- 52. **What: Light reaction:** Light energy is used to move electrons through an ETS which results in oxidation of H₂O, pumping of protons (into thylakoid lumen), production of NADPH; the proton gradient is used to run the CF₁CF₀ ATP synthase.
- 53. **Where:** Chloroplast; Most of the machinery is found in the thylakoids; ATP is made on the stroma side of the thylakoid membrane.
- 54. **Why:** The ATP & NADPH synthesized in the light reaction is used to run the Calvin Cycle which fixes CO₂ - this is known as the dark reaction.

LIPIDS:

β-Oxidation:

- 55. **What:** Mitochondrial oxidation of fatty acids - oxidation, hydration, oxidation, thiolysis. Yields Acetyl CoA units.
- 56. **Where:** Mitochondrial matrix; problem oxidations occur in the peroxisome.
- 57. **Why:** Good source of energy. Acetyl CoA units often undergo TCA cycle to generate ATP for the cell.
- 58. Complete oxidation(including TCA, ETS & Ox Phos) of palmitate (C₁₆:0) yields 108 ATP and 130 H₂O.

Fatty Acid Synthesis:

- 59. **What:** Acetyl CoA units are used to synthesize fatty acids. Most common source of Acetyl CoA is metabolism of sugars (carbohydrates).
- 60. **Where:** Cytosol; the liver is most common organ for FA synthesis in higher animal.
- 61. **Why:** FA's are used to make various lipids including those in the lipid bilayers of membranes; FA's are also stored as triacylglycerol for long term storage.
- 62. FA synthesis & β-oxidation are controlled by epinephrine, glucagon & insulin in much the same way that glycogen metabolism is controlled.

NUCLEIC ACIDS:

Nucleotides:

- 63. Nucleotides contain nitrogenous bases, ribose and phosphate. Purines = A, G; pyrimidines = C, T, U**
- 64. A pairs with T or U (2 H-bonds) & G pairs with C (3 H-bonds).**
- 65. Chargaff's Rules: A = T, C = G, purine = pyrimidine**
- 66. Watson & Crick model = B-form of DNA**
The B form is a right handed helix, bases separated by 3.4 Å;
One turn of the helix = 10 residues = 34 Å.
- 67. Ribozymes: (1) made of RNA (2) follow Michaelis-Menten kinetics (3) show substrate specificity.**
- 68. DNA polymerases are machines that are responsible for making copies of DNA.**
- 69. Transcription = making RNA using DNA templates; catalyzed by RNA polymerases.**
- 70. Translation = protein synthesis using RNA template; catalyzed by ribosomes.**