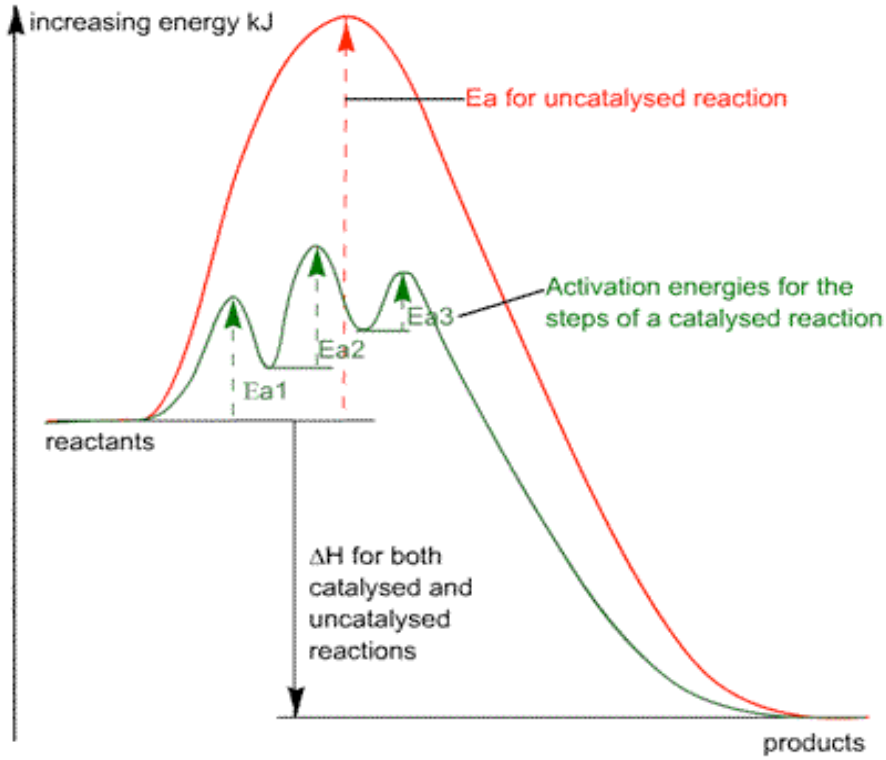
Molecular Biology – Enzymes and Metabolism

I. ENZYME STRUCTURE AND FUNCTION

a. Catalytic Proteins – enzymes change the rate of a rxn, but aren't consumed in the rxn - lower the *activation energy* of the rxn so that the rxn can take place under normal biological conditions (ie, without extremely temps, pH, etc) and not damage the cells in which the rxn is occurring. This occurs via H-bonds, ionic interactions, etc.



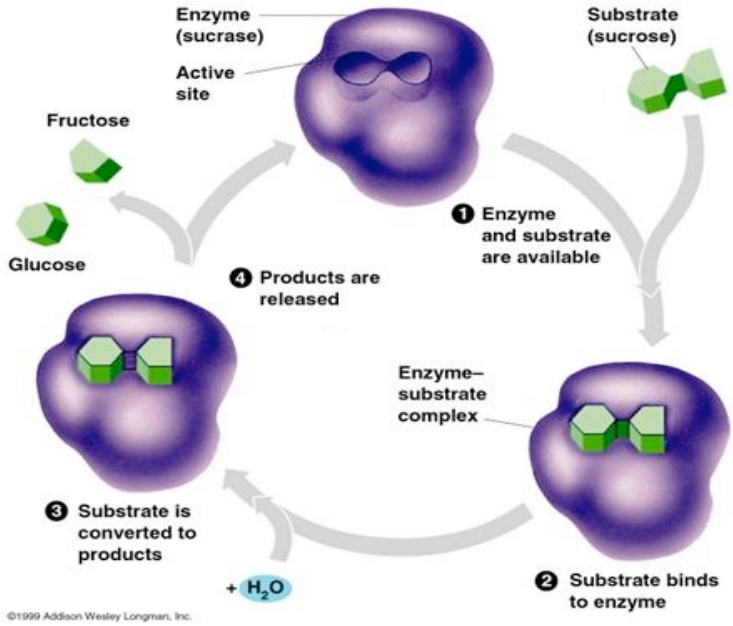
**Remember that transition states are at the top of an energy curve and CANNOT be isolated, while intermediates exist in troughs within the rxn curve and they CAN be isolated.

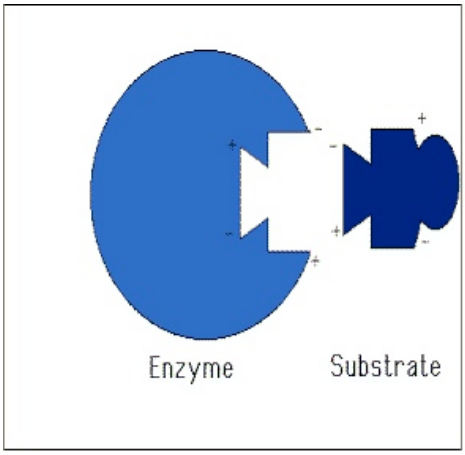
b. Active Sites/Specificity

i. Active sites are pockets or grooves on the protein (enzyme) surface that bind the substrate/reactant very specifically. These active sites house the amino acids that provide the non-ionic interactions that stabilize the transition state of a reaction and lower the activation energy.

ii. Lock and key vs. induced fit

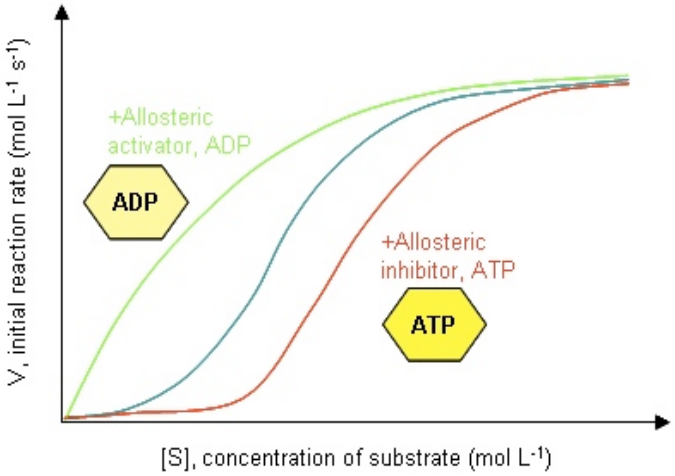
1. Lock and key – old theory – idea was that the structure of the reactant fit perfectly in the space provided by the active site
2. Induced Fit – newly accepted theory – similar to a two hands meeting in a handshake, the non-covalent interactions between the reactant and the enzyme cause their structures to mold around one another as they come together.

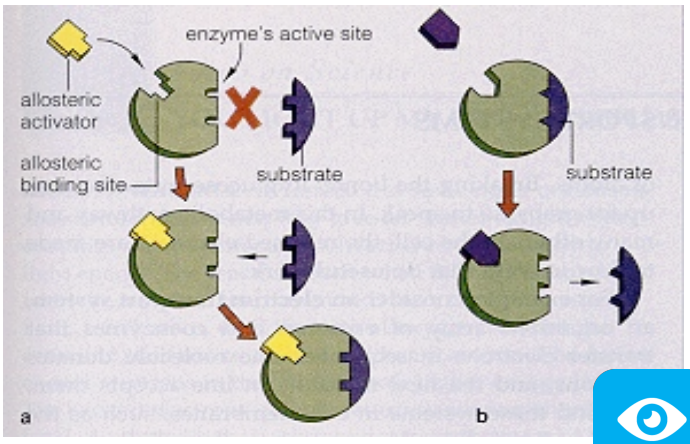




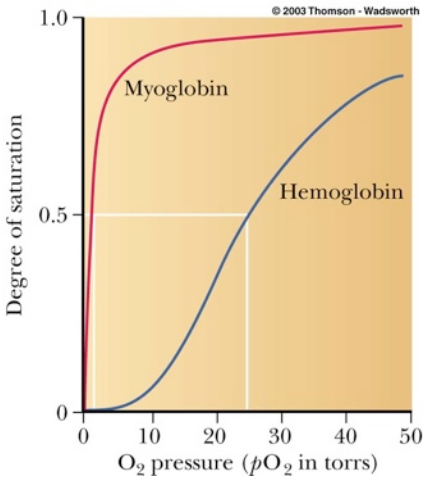
c. Coenzymes
   i. Used by some enzymes to help them function
   ii. Bind to the active site and assist in stabilization of the rxn
   iii. Inorganic ions – vitamins, metal cations, usually obtained in diet (iron, zinc, copper, Vitamin A, B vitamins, etc)

II. Control of Enzyme Activity
   a. Allosteric Regulation
      i. Binding of substrate at one site on the enzyme affects binding at another site elsewhere on the same enzyme molecule
      ii. Occurs in 2 different ways:
         1. Binding of one type of substrate affects the binding of a different type of substrate at another site on the same enzyme molecule. Usually a metabolite within the metabolic cascade binds the allosteric site and acts as either an inhibitor or activator for the active site.
            a. This is called an <u>Allosteric Enzyme</u> – a regulatory enzyme with its catalytic activity modulated by the non-covalent binding of a specific metabolite at a site other than an active site. An activating metabolite increases the velocity of a rxn, while an inhibiting metabolite decreases the velocity of a rxn (see figures below

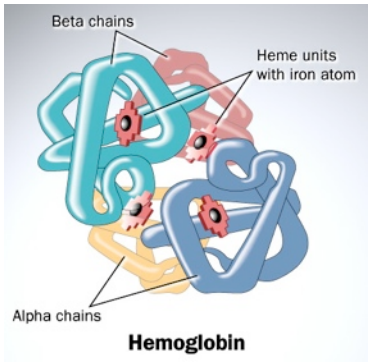




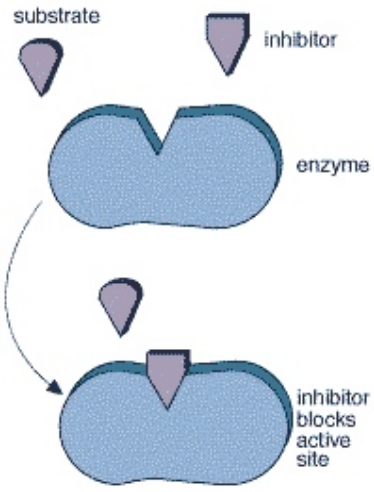
2. Cooperative Regulation

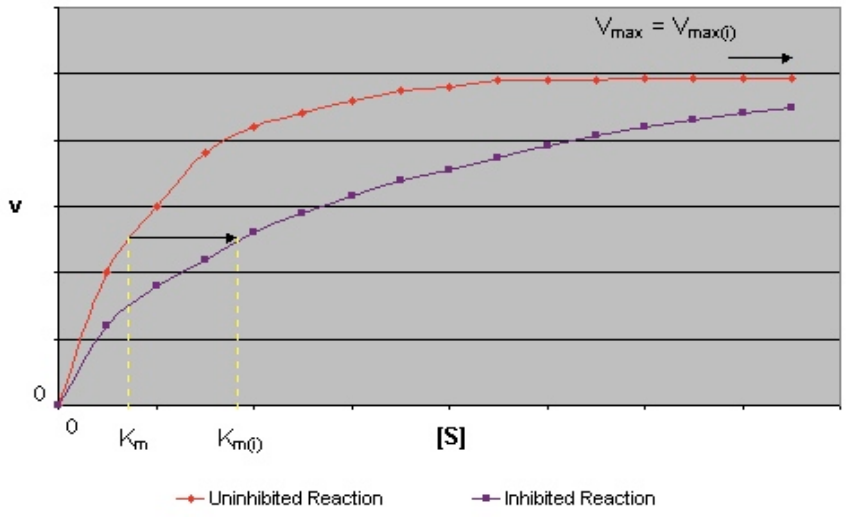


   a. The other type of allosteric regulation in which the enzyme has greater than one active site for the SAME substrate. When one molecule of the substrate binds to an active site, it increases the likelihood that the same substrate will bind to other active sites on the same enzyme molecule.
   b. Hemoglobin uses cooperative regulation to bind oxygen, and while it doesn't function as an enzyme per se, its kinetics are a good way to demonstrate cooperative regulation. Note the sigmoid-shaped velocity curve (shown in hemoglobin as "degree of saturation."
      i. When one molecule of oxygen binds to one of 4 hemoglobin active sites, it changes conformation of the other active sites, making it more likely that subsequent oxygen molecules will bind to the remaining, unfilled active sites.

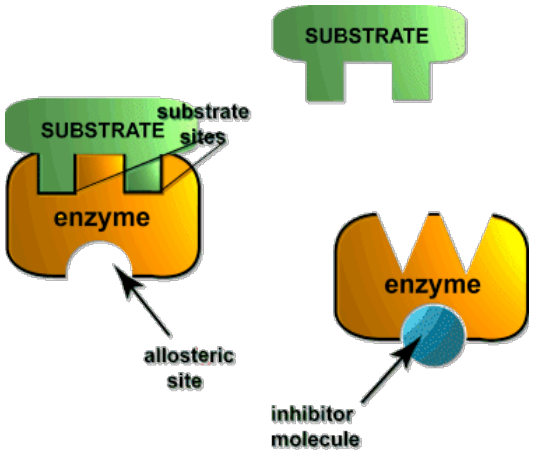


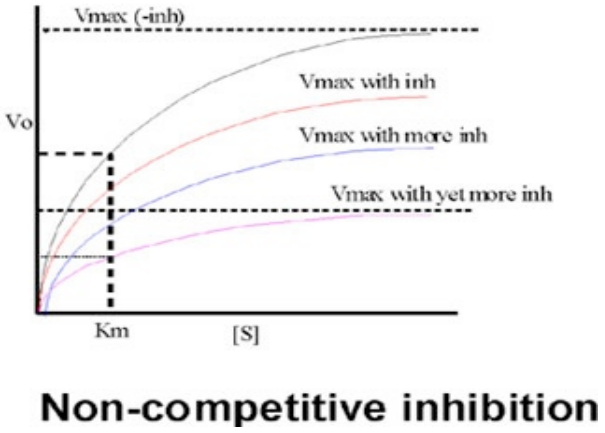
b. Inhibitive Regulation – decreases an enzyme's activity (two types):
   i. Competitive Inhibition
      1. inhibitor binds at active site and prevents regular substrate from binding
      2. carbon monoxide is a competitive inhibitor of oxygen binding hemoglobin
      3. increasing the concentration of the substrate can increase the Vmax of the reaction because it makes it more likely that the substrate will displace some of the competitive inhibitor and get into the active site – this is why victims of carbon monoxide poisoning are placed in a hyperbaric chamber with high concentration, high pressure oxygen.





ii. Non-Competitive Inhibition
    1. inhibitor binds somewhere other than the active site and changes the shape of the enzyme so that it binds the substrate with lower affinity.
    2. Lowers the Vmax of the rxn, which cannot be overcome by increasing the concentration of a substrate because the inhibitor is bound somewhere other than the active site
    3. This is a type of allosteric regulation which was discussed above.





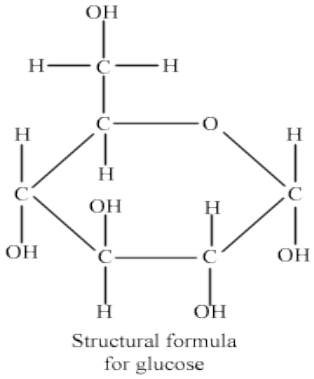
iii. Feedback Inhibition
    1. Similar to situations discussed above in which the end product of an enzymatic cascade acts as an inhibitor to stop the action of the enzymes in the cascade.
    2. Used in almost all biological systems to control the enzyme activity – esp. in glycolysis, Kreb's cycle, etc

III. Basic Metabolism

a. Oxidative Catabolism of Glucose
    i. Overall rxn:

$$C_6H_{12}O_6 + 6\,O_2 \rightarrow 6\,CO_2 + 6\,H_2O + \text{Energy (ATP)}$$

    ii. Be able to recognize the structure of glucose:

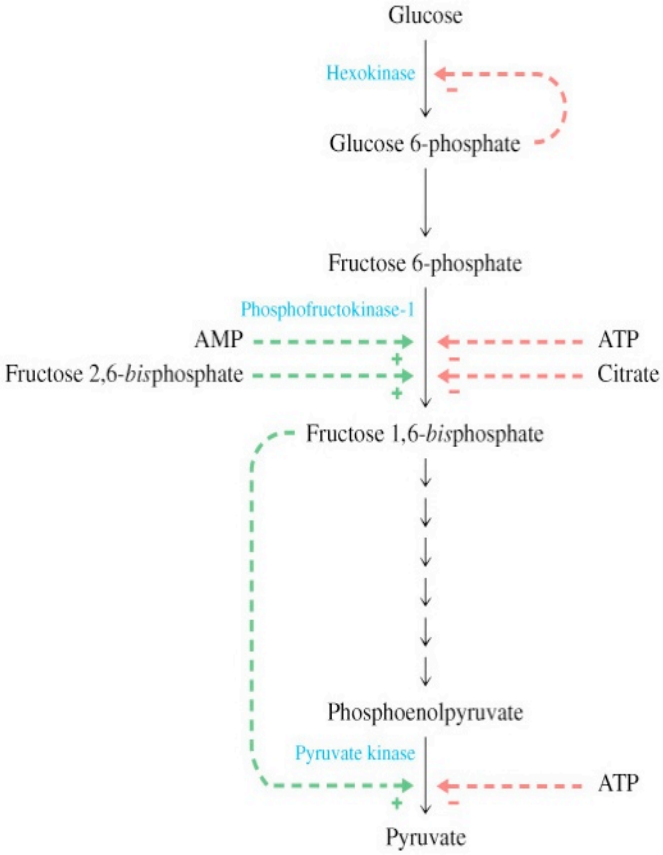

Structural formula for glucose

b. Glycolysis
    i. Occurs in *cytoplasm*
    ii. Occurs with or without oxygen
    iii. One molecule of glucose is broken into 2 molecules of pyruvate
    iv. Overall rxn:

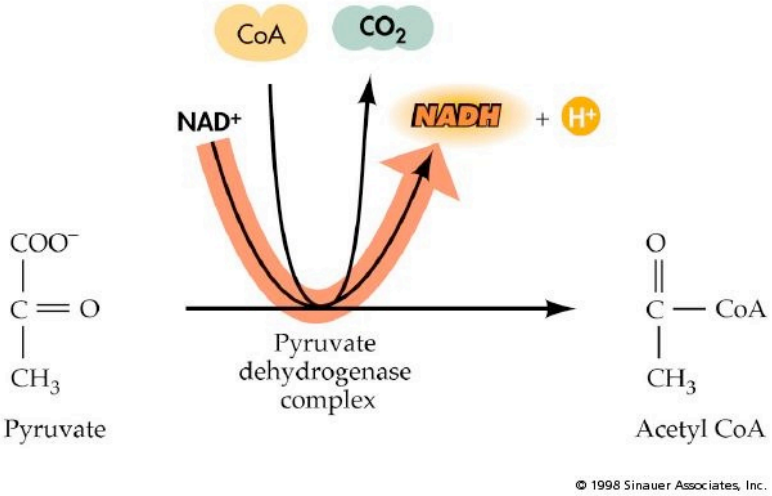
$$\text{Glucose} + 2\,ADP + 2\,P_i + 2\,NAD^+ \rightarrow 2\,\text{Pyruvate} + 2\,ATP + 2\,NADH + 2\,H_2O + 2\,H^+$$

    v. When oxygen is present, pyruvates enter the Pyruvate Dehydrogenase Complex
    vi. Without oxygen, the Krebs cycle and Electron Transport do not occur
        1. Some organisms use fermentation to produce alcohol from the pyruvates
        2. Humans and most animals convert the pyruvates to lactic acid which is sent to the liver and converted back to glucose

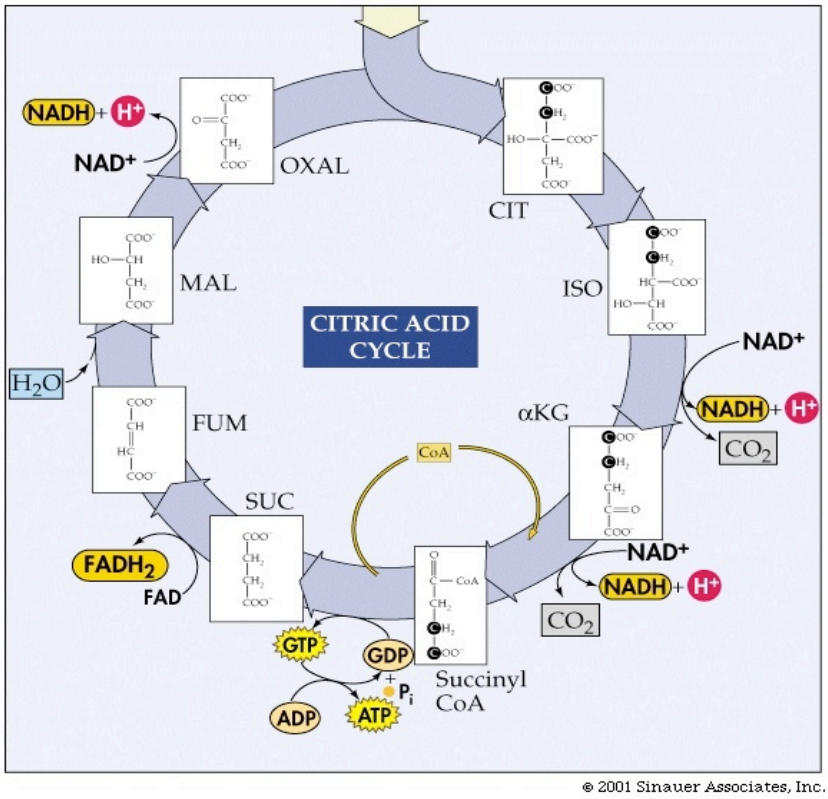
vii. In figure to the right, don't worry about the specific enzyme names and intermediate names, but notice how allosteric regulation is used to regulate the cycle



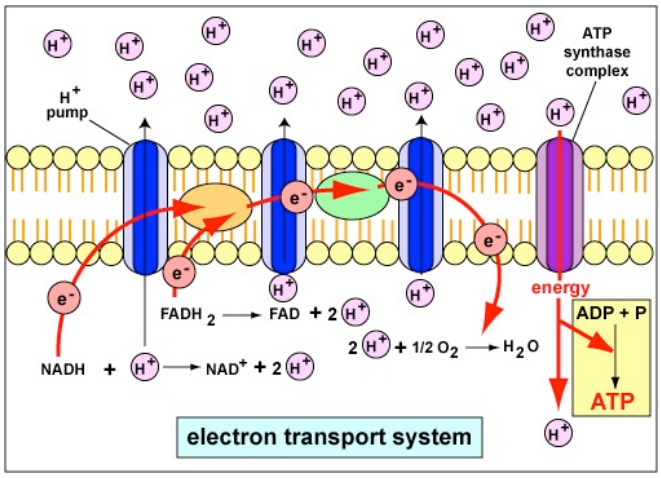
c. Gluconeogenesis
   i. Basically the reverse of glycolysis. Serves to convert the intermediates of glycolysis back into glucose.
   ii. Occurs when the products of glycolysis build up and then act to shut down glycolysis and activate the gluconeogenesis enzymes.
   iii. For example – Build up of Acetyl CoA activates gluconeogenesis, but a build up of ADP (which is converted to ATP) activates glycolysis
d. Pyruvate Dehydrogenase Complex
   i. Enzymes present in the *mitochondrial matrix*
   ii. The pyruvates are decarboxylated to form Acetyl CoA
   iii. Acetyl CoA then moves on to the Kreb's Cycle



e. Kreb's/TCA/Citric Acid Cycle
   i. Oxygen is not actually utilized during the TCA cycle, but it won't without oxygen because the pyruvate won't be converted to Acetyl CoA
   ii. Produces 6 NADH, 2 $FADH_2$ and 2 GTP per glucose molecule (per 2 Acetyl CoA)
   iii. For the exam, there is not need to know the individual steps in the TCA cycle. Do know that it occurs in MITOCHONDRIA (both matrix and on the inner mitochondrial membrane) and the products of the rxn



f. Electron Transport Chain
   i. Occurs on inner mitochondrial membrane (bacteria use plasma membrane)
   ii. 2 things occur:
      1. Electron carriers are re-oxidized (remember mnemonic LEO GER)
      2. Energy is stored in the form of phosphate bonds as ADP is converted to ATP



   iii. Basic process:
      1. Protons from NADH and FADH2 are pumped across the inner mitochondrial membrane from the matrix to the inter-membrane space. This is made possible by electrons being moved between electron carriers on the membrane. The protons form an electrochemical gradient across the membrane. The protons flow down their gradient through a channel in the ATP synthase molecule. This movement of protons drives the ATP synthase motors which convert ADP to ATP.
      2. ATP produced:
         a. Conversion of NADH to NAD produces 2.5 ATP
         b. Conversion of FADH2 to FAD produces 1.5 ATP

IV. Overall ATP Production in the catabolism of one molecule of glucose:

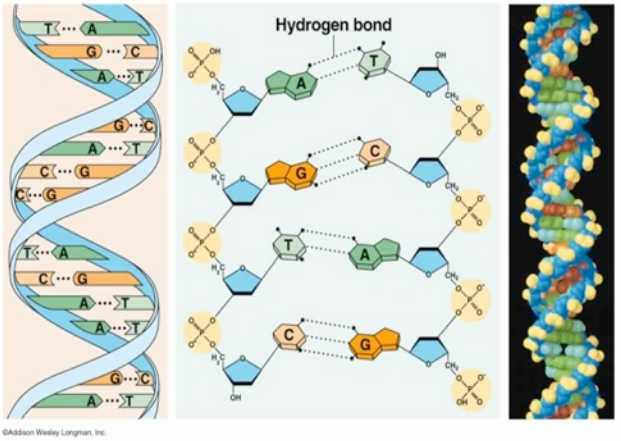
Process	**Molecules formed/used**	**ATP Equivalents**
Glycolysis	-2 ATP	-2 ATP
	+4 ATP	+4 ATP
	2 NADH	+5 ATP
Pyruvate Dehydrog Complex	2 NADH	+5 ATP
TCA Cycle	6 NADH	15 ATP
	2 GTP	2 ATP
	2 FADH2	3 ATP
TOTAL		**32 ATP**

Molecular Biology – DNA and Protein Synthesis

I. DNA Structure and Function

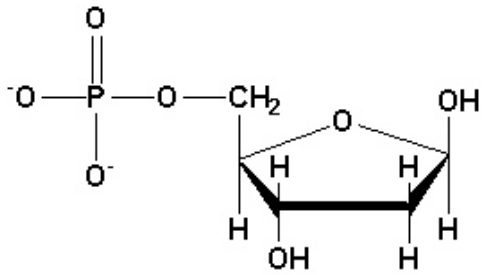
a. Watson-Crick Model

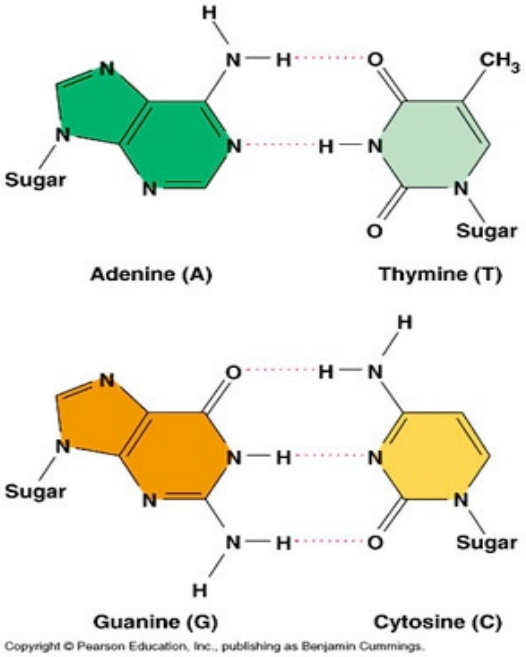
- "right-handed" double helix (think reaching up to change a light bulb)
- Sugar phosphate backbone makes up exterior of helix
- Nitrogenous base pairs hydrogen bonded together make up interior of helix
- Watson-Crick elucidated structure at Cambridge in 1953

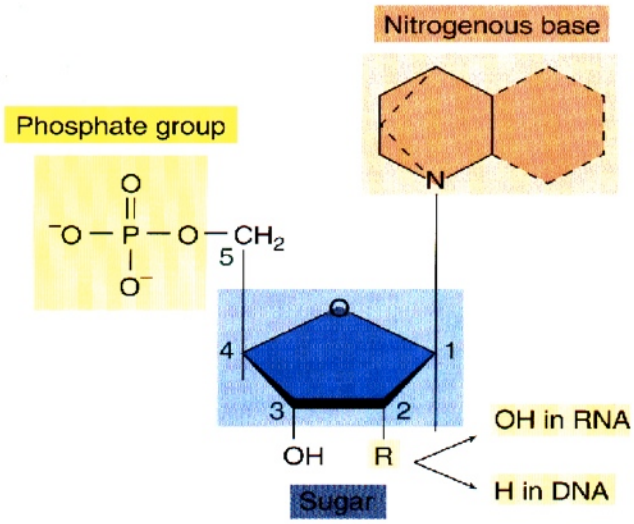


○ DNA transmits its genetic information via its nitrogenous base sequences

- Genes coded in triplet sequences known as codons
- 64 possible sequences
- Each codon will code for a specific amino acid during translation





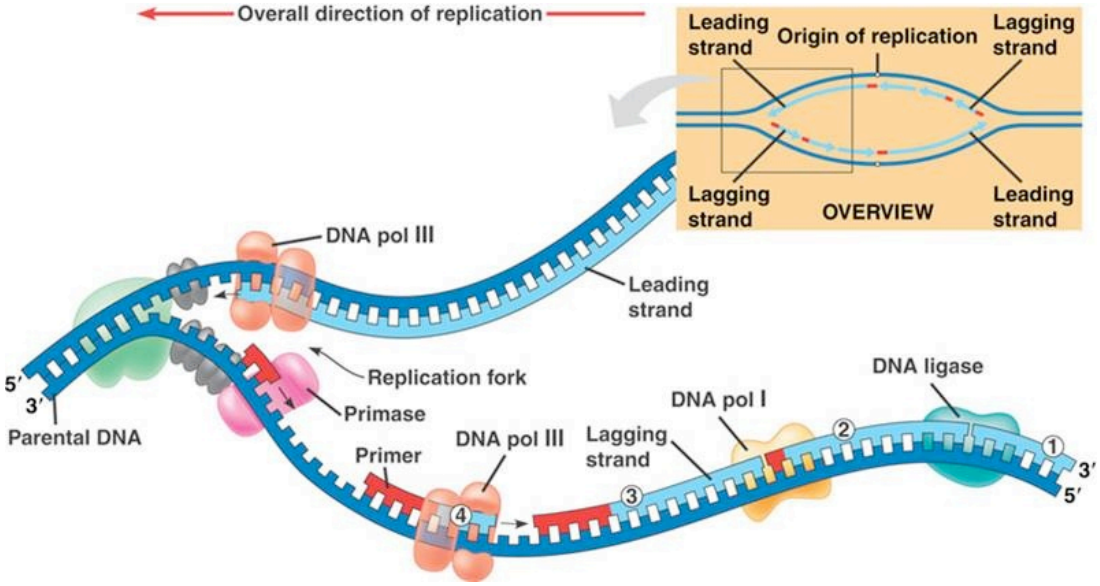


Nitrogenous Bases

1. Purines
   a. Adenine and Guanine
2. Pyrimidines
   a. Thymine, Cytosine and Uracil (RNA only)
3. Specific Base Pairing
   a. A=T

b. G≡C
c. Remember the mnemonic: "**A**t **T**he **G**irls' **C**lub"

b. DNA Replication
   i. Occurs during the S-phase of the cell cycle
   ii. Semi-conservative process
      1. each new daughter molecule contains one strand from the parent strand and one newly synthesized strand
   iii. Steps:
      1. Helicase unwinds strand
      2. Topoisomerases cut strands as needed to prevent supercoils
      3. Primer lays down a small segment of RNA that can be identified by DNA polymerase
      4. DNA polymerase synthesizes new strand in **5' → 3'** direction.
         a. This occurs in one continuous process on the "leading strand"
         b. On the "lagging strand" the daughter strand must be synthesized in segments as the parent molecule is unwound because replication can only occur in a **5' → 3'** direction. These segments are known as "Okazaki Fragments"
      5. Ligase removes all primers and fills in the gaps with DNA to complete the process

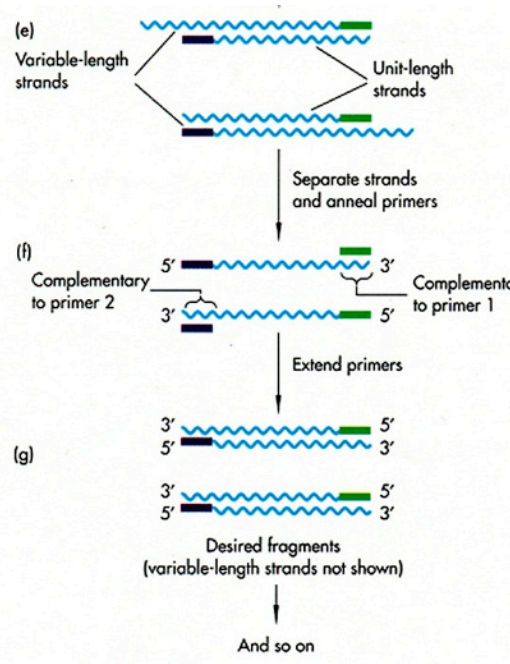


c. Recombinant DNA
   i. Restriction Enzymes (restriction endonucleases)
      1. enzymes that recognize specific nucleotide sequences and cleave the DNA molecule at those sites (this is usually a palindromic sequence). The cleavage leaves a "sticky end" that is able to re-anneal with another sticky end. This allows segments of DNA that are cut out by endonucleases to be spliced into other DNA molecules.
   ii. Hybridization

1. Denatured double-stranded DNA will re-anneal in an attempt to reform a double stranded molecule. This allows segments of DNA to be identified or labeled in a lab. If a sequence of DNA is known, but the location of that sequence within a genome is unknown, the genome can be denatured, the complement of the known sequence can be added and the segments will anneal with the gene in the genome allowing the gene locus to be identified.

iii. Gene Cloning

1. Amplification of genes for study
2. Can be spliced into a bacteria's genome so that it will be replicated whenever the bacteria divides
3. PCR is now the most common
   a. DNA is placed in the synthesizer
   b. The DNA is denatured
   c. All the components necessary for replication are added (enzymes, nitrogenous bases, etc) so that the denatured strands are replicated
   d. This process is repeated over and over to allow exponential replication of the original sequence

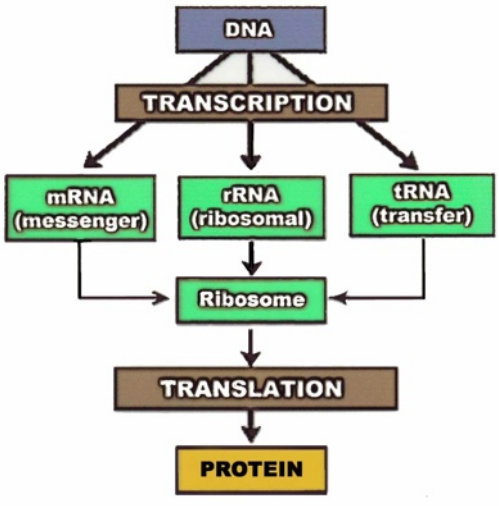


II. Protein Synthesis

a. Central Dogma:

1 2
DNA → RNA → Protein

1 = transcription
2 = translation

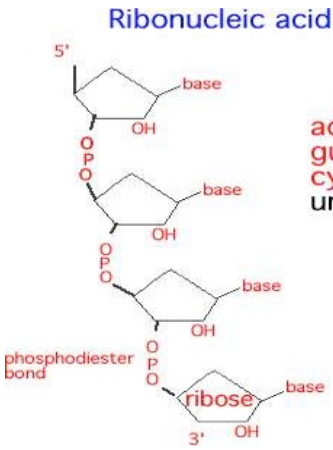


b. Transcription

i. Description

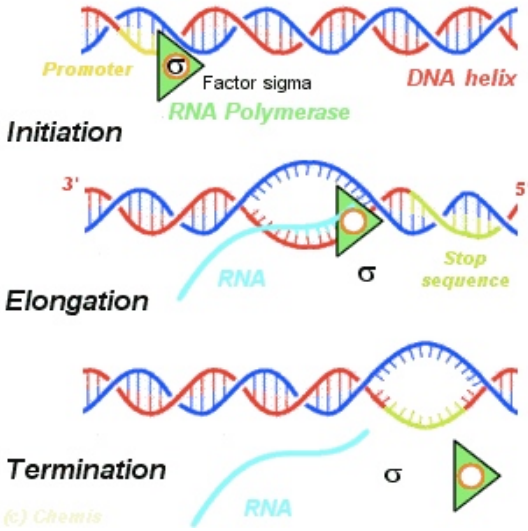
1. conversion of DNA sequence to RNA so that the code can be transmitted from the nucleus to the cytoplasm for protein synthesis

2. RNA
   a. Single-stranded
   b. Uracil used instead of thymine
   c. Ribose used instead of deoxyribose (hydroxyl group present at 2' position in ribose, absent in deoxyribose"
   d. Three types of RNA



i.mRNA – "messenger" – carries the codons to the cytoplasm
ii.tRNA – "transfer" – carries the "anti-codon" and the corresponding amino acid
iii.rRNA – "ribosomal" – along with other proteins, forms ribosomes, which serve as docking sites for mRNA and tRNA during translation

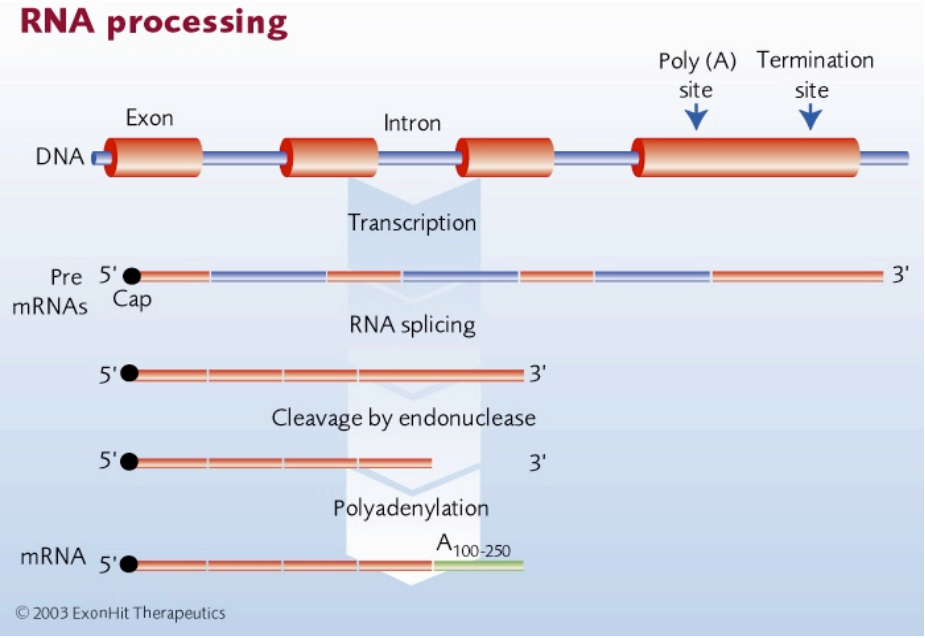
ii. 3 Phases of Transcription
1. Initiation
   a. RNA polymerase binds to promoter sequence on template strand of DNA
      i. Promoter is a sequence of DNA that signals where a gene begins (the TATA box is one example)
      ii. Transcription factors assist RNA polymerase in finding the promoter region
   b. DNA strands separate



2. Elongation
   a. mRNA molecule is synthesized using the DNA template
3. Termination
   a. Nucleotide termination sequence signals the end of the gene, transcription stops and the mRNA molecule is released
   b. The piece of segment of DNA that is transcribed in a single round of transcription is known as a "transcription unit." In eukaryotes, this includes only one gene, in prokaryotes, a transcription unit can contain several separate genes.

iii. RNA Processing
1. occurs post-transcriptionally to prepare the mRNA molecule to leave the nucleus for the cytoplasm.
2. Rids the mRNA molecule of unneeded portions and prevents the mRNA from being degraded by enzymes in the cytoplasm
3. Steps
   a. 5' methyl cap is added
   b. 3' poly-adenine (poly-A) tail is added
   c. Splicing occurs (introns are removed, exons are joined together)

4. modified mRNA is now ready to leave the nucleus for the cytoplasm



III. Translation

a. Description

i. Conversion of the genetic code into amino acid sequences that form functional proteins necessary for organism's structure and function

ii. The reading frame of an mRNA molecule gives a certain set of codons.

1. wrong reading frame gives an entirely different set of codons, different amino acids and a non-functional protein, so it is crucial that the correct reading frame be identified:

U**AUG**AGCGGCGA**AUG**GCGAUGAG

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Correct start *Incorrect* start point gives different set of codons

iii. each codon codes for a specific amino acid

1. 64 possible combinations (4*4*4), but only 20 different amino acids are used

a. Genetic code is redundant → there is greater than one codon for each amino acid

b. ie, CGU, CGC, CGA and CGG all code for the amino acid arginine

iv. Anti-codons on the tRNA molecules complement the condones along the mRNA and transfer the corresponding amino acid to the protein chain growing from the ribosome

b. Three Steps in Translation

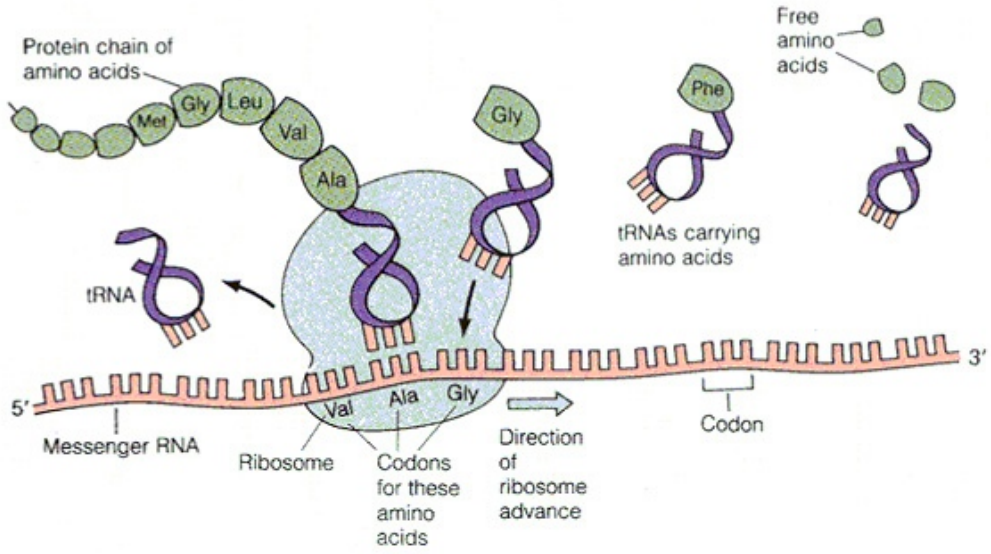
i. Initiation

1. ribosome attaches slightly upstream from the AUG start codon (codes for methionine)

2. initiation factors bring mRNA, initiator tRNA and ribosomal subunits together

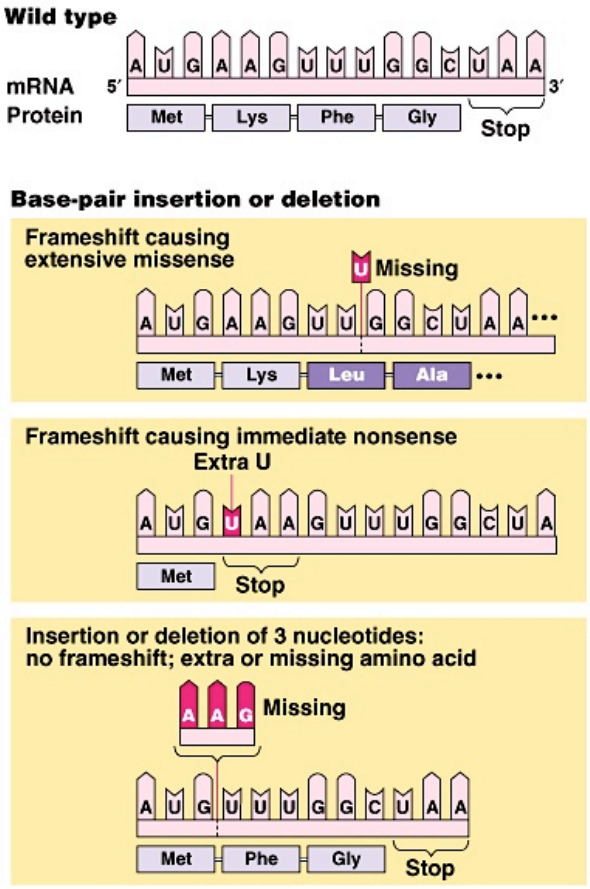
ii. Elongation – 3 step cycle
1. Codon recognition by complementary tRNA
2. Peptide bond formation between new amino acid and nascent protein chain
3. Translocation – ribosome uses GTP to move itself downstream on the mRNA to the next codon

iii. Termination
1. stop codon signals end of gene
a. stop codons are UAA, UAG and UGA
i. mnemonic: "U Are Away, U Are Gone, U Go Away)
ii. stop codons do NOT code for an amino acid, they just signal



the translation complex to disassemble and release the newly synthesized protein

IV. Mutations
a. Point Mutations
i. Change in one nucleotide pair in a single gene
ii. Types:
1. Base-Pair Substitution
a. Replacement of one nucleotide and its complementary partner with another pair of nucleotides
i. Silent mutations
1. have no effect of the amino acid coded by the codon – remember the redundancy of the genetic code
2. most amino acids have very similar codons
a. Arginine is coded by CGU, CGC, CGA and CGG, so if a mutation occurs at the third position, no change in amino acid will occur
ii. Missense mutations
1. Changes the amino acid coded for



2. This is especially concerning if the amino acid is located in the "active portion" of a protein (ie, the active site of an enzyme)

iii. Nonsense mutations
1. Changes the codon to a stop codon
2. Results in a truncated protein and usually renders the protein non-functional unless the mutation is very near the end of the gene

iv. Insertions and Deletions
1. Loss or addition of one or more nucleotide pairs in a gene
2. Usually disastrous because they alter the reading frame of the gene → a "frameshift mutation"

V. DNA Repair
- a. Mismatch repair
  - i. incorrect base recognized almost immediately after synthesis on new strand.
  - ii. Process: a stretch of DNA behind and in front of the mismatch is clipped by endonucleases, excised by helicase, and exonucleases, and replaced with the correct sequence by DNA Pol I (then sealed with ligases).
- b. Nucleotide-excision repair (NER)
  - i. tends to repair more severe modifications that alter the helical pattern of the affected DNA (e.g. thymine dimers from UV radiation).
  - ii. Process: recognition, clipping the backbone by endonucleases, excision of the affected part, replacing by Pol I, resealing by DNA ligase.
- c. Base-excision repair (BER)
  - i. tends to repair more subtle modifications, like a mismatched base pair not caught by either proofreading or mismatch repair (e.g. accidental uracil in DNA).
  - ii. Process: recognition, clipping off the inappropriate base by glycosylases, clipping the backbone by endonucleases, chewing off by exonucleases of the affected part, replacing by Pol I, resealing by DNA ligase.
- d. Recombinatorial repair (homologous recombination):
  - i. Repairs double-stranded breaks in DNA.
  - ii. Process: partially degrades both sides of the break to create primers for DNA synthesis, attracts intact, homologous sequence from other chromosome, each strand aligns itself with a strand on homologue and fills in its gap from that strand.
- e. Non-Homologous-End-Joining (NHEJ):

    i. A form of double-stranded break repair that doesn't involve the homologous chromosomes. Essentially you unwind the two ends with helicases, pair up a few matching bases, and reseal the phosphodiester backbone. Note that this can be inaccurate, as you often lose a few bases off the unpaired strands during the resealing.
f. Lesion bypass polymerization
    i. Usually occurs when the cell doesn't have enough resources to fix all the thymine dimers occasioned by UV exposure. Effectively it allows replication to proceed, despite the fact that the dimer interferes with normal processing, by eliminating the polymerase's ability to do base proofreading (ie. 3' to 5' exonuclease activity).
    ii. The error rate is 2 to 4 orders of magnitude higher than normal replication, thus frequently results in cancers, etc.

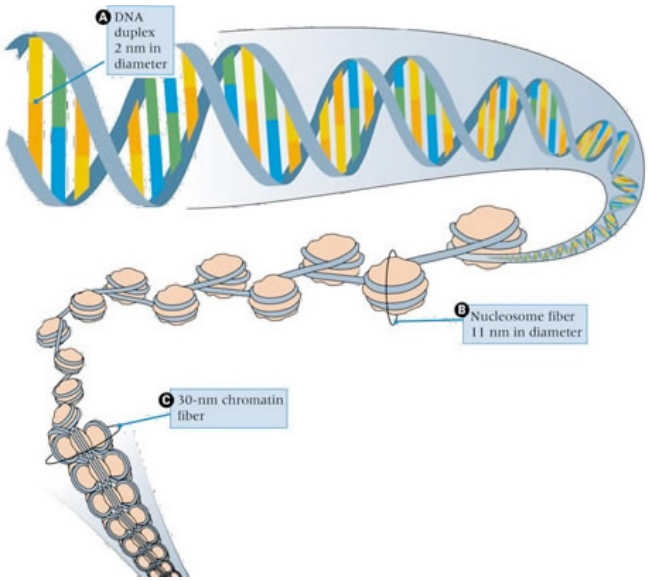
BIOLOGY - EUKARYOTES

I. Eukaryotic Chromosome Organization

a. Chromosomal proteins

i. Histones

1. Four core histone subunits: H2A, H2B, H3, H4, each with two copies
2. Make up an octomer
3. DNA wrapped around histone octomer = *Nucleosome*
4. Nucleosomes bundled tightly together = *Chromatin*



ii. Euchromatic regions

1. More relaxed, less repeats. Makes up most of the genome.
2. Usually what is sequenced

iii. Heterochromatic regions

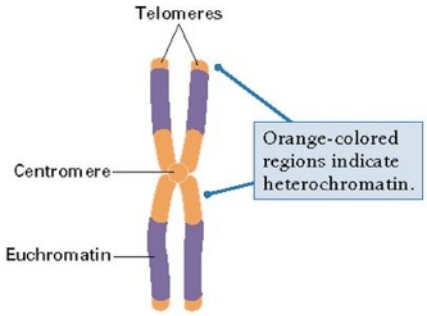
1. More condensed, more repeats. Tends to be near centromeres and makes up less of the genome.

b. Telomeres

i. Sequence at the ends of chromosomes, consisting of a large number of repeating segments.

ii. Shortened very time the chromosome is replicated, since the RNA primer on the very last Okazaki fragment can't be replaced by Pol I (Pol I needs to have a nearby 3' OH from the next fragment to bind and replace the RNA primer)

iii. After a certain point, the telomeres get short enough that the cell becomes unstable and is destroyed.



c. Centromeres

i. DNA near middle of the chromosome

ii. Point where sister chromatids contact, also point of mitotic spindle (later lecture)

II. Control of Gene Expression in Eukaryotes

a. Transcription regulation

i. VERY complicated

ii. Essentially, proteins can bind DNA to switch transcription on or off, or simply enhance or inhibit transcription (see image next page)

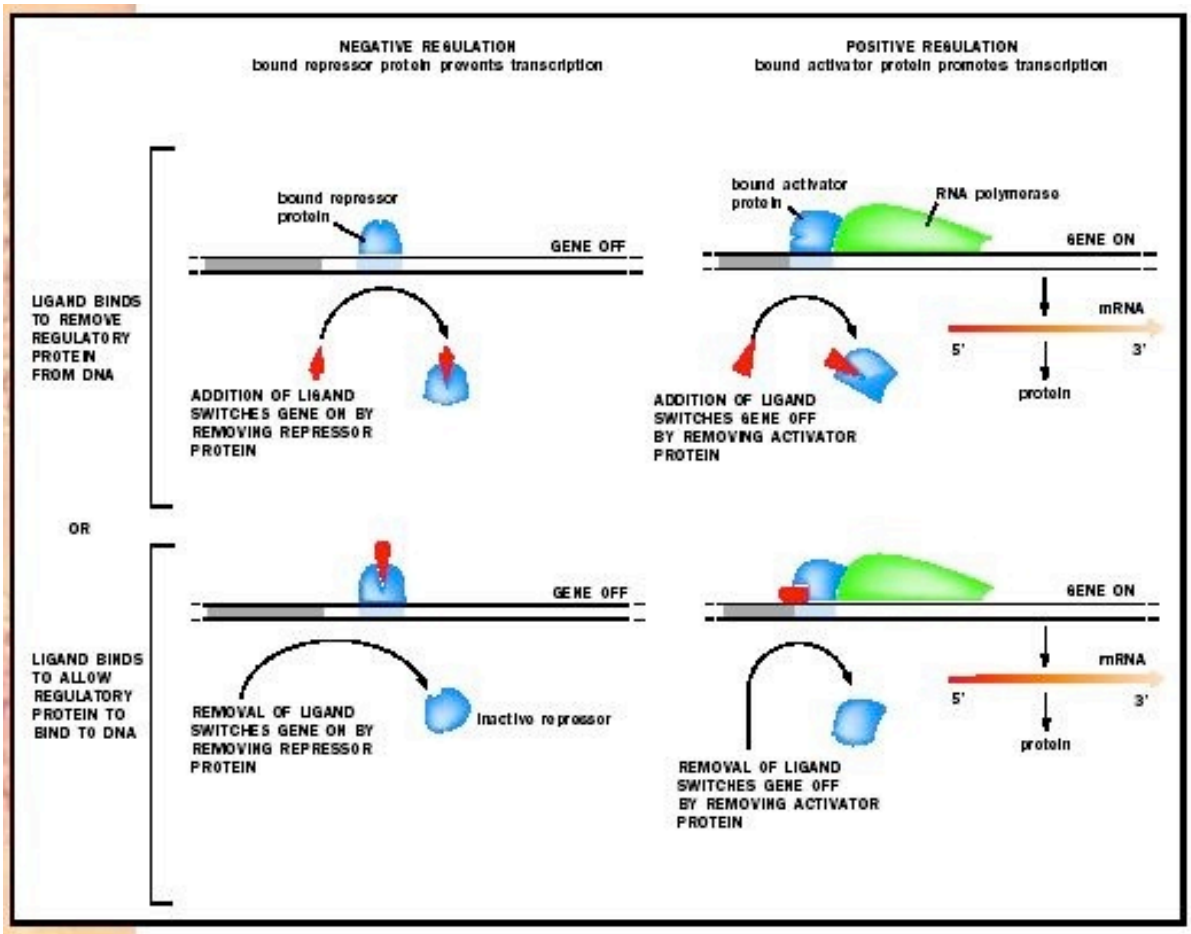
iii. DNA control elements

1. transcription-influencing segments of DNA on or associated with the gene being transcribed.
2. TATA box/initiator sequence
   a. usually 25-35 bp upstream from start site. Determines site of transcription initiation and directs binding of RNA Pol II.
3. Promoter

a. usually within 200 bp upstream of start site, about 20 bp long. Bound by transcription factors to regulate transcription.

4. Enhancers
    a. usually much farther upstream, or downstream, than promoters, although still fairly short in themselves (8-20 bp). Can be upstream of the start site, downstream of the last exon, or within introns in the gene itself. Similar function to promoters.
5. Activators/Repressors

b. DNA binding proteins
    i. Homeodomain proteins
        1. Helix-turn-helix structure



2. Regulators of development and affect many genes at once.

ii. Zinc-finger proteins
    1. have a "finger" made up of two antiparallel beta sheets and a helix, held together by a zinc ion. This finger is what binds with the DNA.
    2. The largest family of SSDBPs. Include androgen and estrogen receptors.

iii. Basic leucine zipper proteins (bZIP)
    1. The "basic" here refers to the fact that they have a high-pH region that binds to the DNA.
    2. Often form homodimers to bind DNA.

iv. Basic helix-loop-helix proteins (bHLH)
    1. also has basic region for DNA binding.

c. Cancer
    i. Three things usually need to happen to get cancer.
        1. Mutation or mismatching event has to occur in DNA.
        2. Repair mechanisms have to either miss it or be overwhelmed by too many such events (ie. exposure to lots and lots of UV radiation).
        3. Self-destruction pathways (ie. apoptosis) in the cell need to misfire.
    ii. Couple of examples of diseases resulting from mutations in DNA repair mechanisms: Cockayne's syndrome, Xeroderma pigmentosum (generally involved with light sensitivity, neurodegeneration, premature aging, and cancer)

BIOLOGY - MICROBIOLOGY

I. Viral Structure and Life History

a. Viruses

i. Considered non-living

ii. Contain either DNA or RNA

1. renegade hypothesis suggests that pieces of DNA or RNA escaped from their cells and were able to survive by acting as parasites on other cells

iii. no metabolic machinery of their own

1. cannot perform their own energy production or protein synthesis

iv. depend upon host for reproduction

v. no regulatory membranes to control entry and exit of substances or to control their internal environment

vi. smaller than bacteria – the smallest are 20 nm (smaller than ribosomes)

vii. attack very specifically – plant viruses can't infect animals

viii. viruses are generally antigenic – our bodies produce antibodies when exposed to them

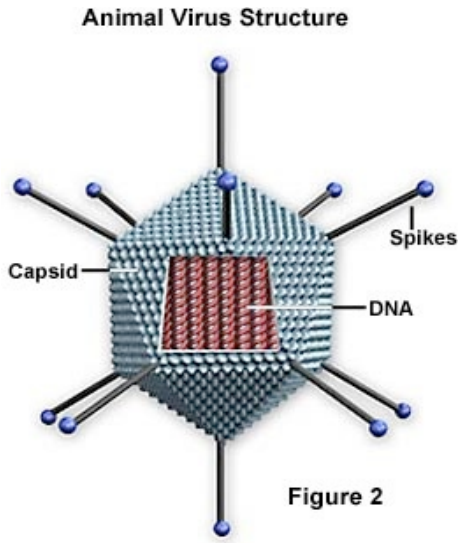
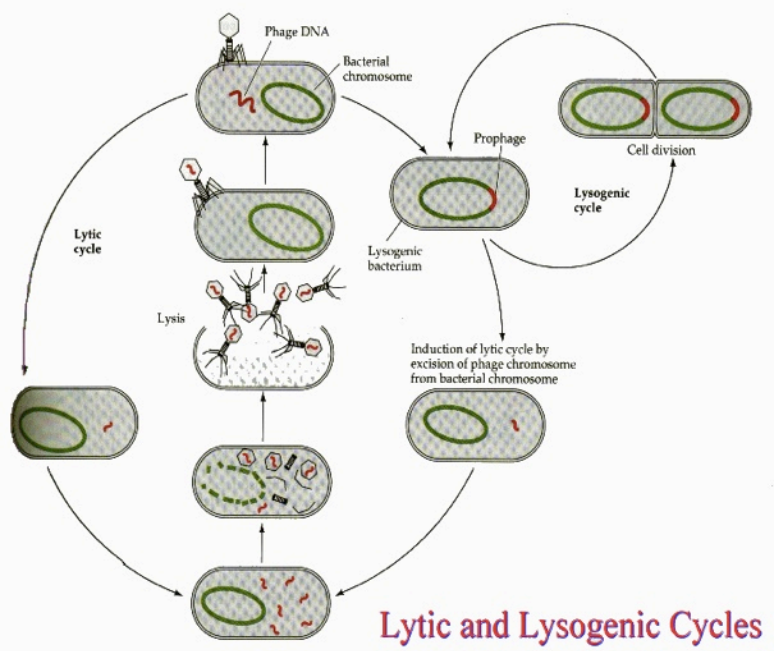


Figure 2

b. Virus Lifecycle

i. Lytic Lifecycle – virus inserts itself into a host cell, hijacks its metabolic machinery to begin reproducing itself and quickly multiplies and kills the cell

ii. Lysogenic Lifecycle – virus inserts itself in the host cell's genome where it can lie dormant for an indeterminate period of time before reactivating an entering a lytic cycle



c. Common Vocabulary encountered with viruses

i. Virion – form of virus that exist outside of cells

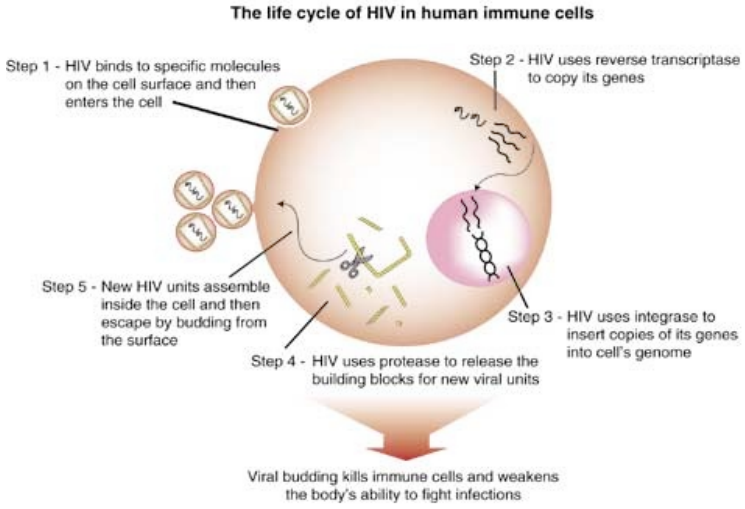
ii. Viroid – virus that infects plants

iii. Bacteriophage – virus that infects bacteria

1. head – protein coat and core

2. tail – proteins specialized for attaching to bacteria

iv. Pathogenesis – the process by which an entity (e.g., a virus) causes a disease



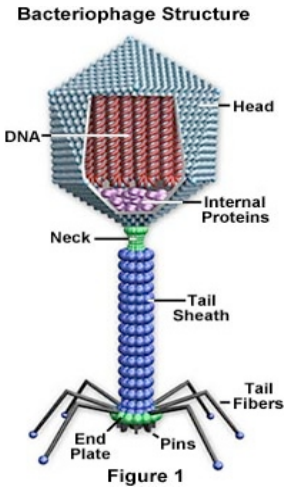
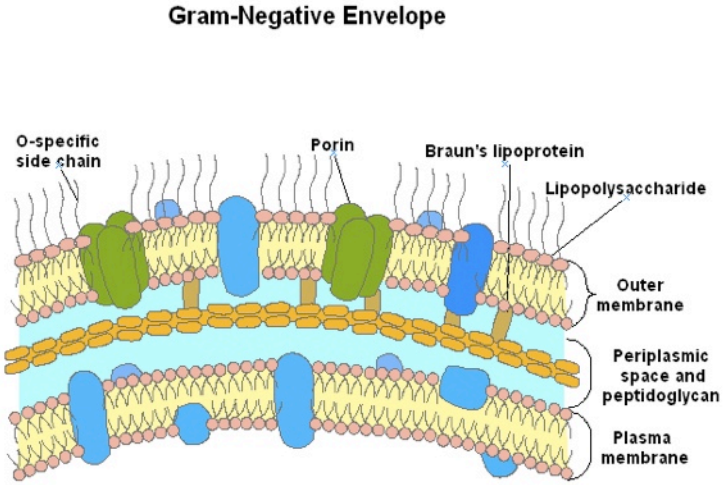


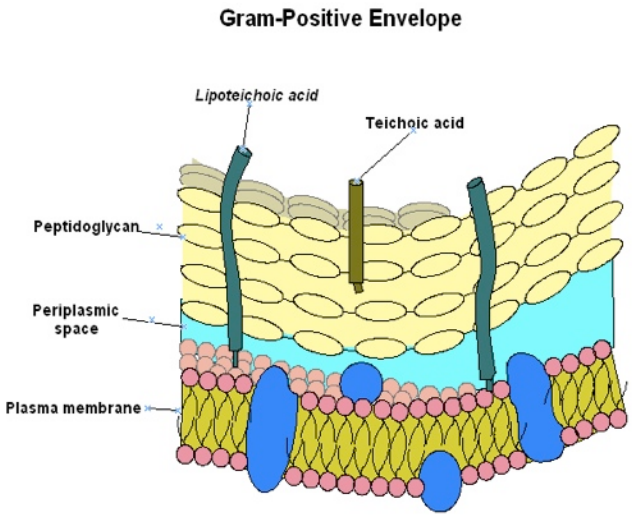
Figure 1

v. Virulence – Capacity for an infective organism (a virus, bacteria or fungus) to cause a disease. For example, an extremely virulent virus would be able to cause a disease if a host were expose to only a few virions
vi. Viremia – presence of virus in the blood

d. Possible viral treatment modalities
   i. Disrupt virus binding to host
   ii. Interfere with DNA/RNA replication by virus – could affect other cells in the body (side effects)

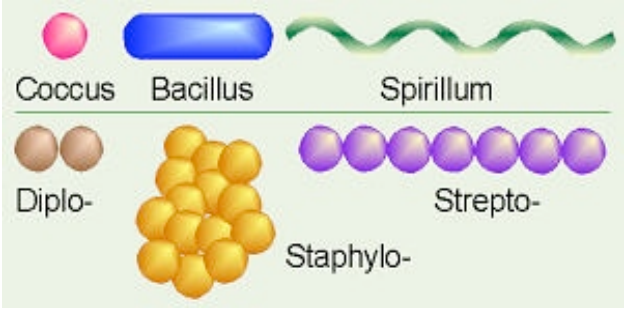
II. Prokaryotic Cells
   a. Bacteria
      i. Description
         1. circular DNA resides in nucleoid region in the cytoplasm because prokaryotes have no nucleus
         2. all metabolic enzymes are in the cytoplasm because there are no membrane-bound organelles
      ii. Gram Positive vs Gram Negative





1. Gram Negative
    a. Peptidoglycan cell wall enclosed by two plasma membranes
    b. Outer lipid bilayer prevents Gram stain from penetrating the cell wall – bacteria appear pink under microscope
    c. Tend to be more resistant to antibiotics that attack the cell wall
2. Gram Positive
    a. Thick peptidoglycan layer with no outer lipid bilayer – Gram stain penetrates easily and stains these bacteria purple
    b. Susceptible to antibiotics that attack the cell wall

iii. Bacteria Shapes
1. Bacillus - rods
2. Cocci – spherical
    a. Diplococci - pairs
    b. Streptococci - chains
    c. Staphylococci - clusters
3. Spirilla/Spirochetes – helically coiled



iv. Aerobic vs Anaerobic
1. Obligate anaerobes
    a. Cannot live in the presence of oxygen
    b. Examples include certain skin infections (Tetanus)
2. Facultative Anaerobes
    a. Prefer to live in the presence of oxygen, but can tolerate anaerobic environments as well
    b. Can switch to fermentation in an anaerobic environment
    c. Most bacteria fit this category
3. Obligate Aerobes
    a. Can't live without oxygen for cellular respiration

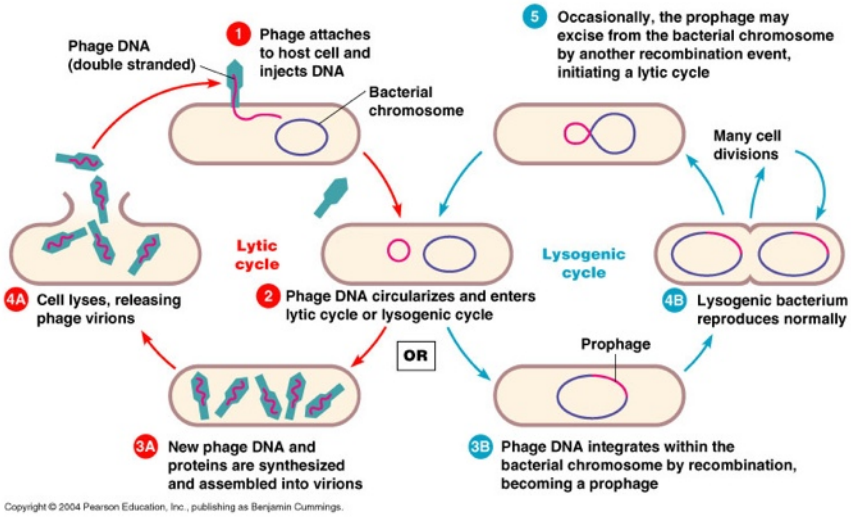
v. Feeding/ Major Modes of Nutrition
1. Photoautotrophs
    a. Form organic compounds from carbon dioxide using light energy
2. Chemoautotrophs
    a. Use carbon dioxide as carbon source but obtain energy from inorganic substances (NH3, FE++, H2S)
3. Photoheterotrophs
    a. Use light to generate ATP, but obtain carbon from organic sources
4. Chemoheterotrophs
    a. Must consume organic molecules for both energy and carbon
    b. Found widely among prokaryotes, protists, fungi, animals and even some plants

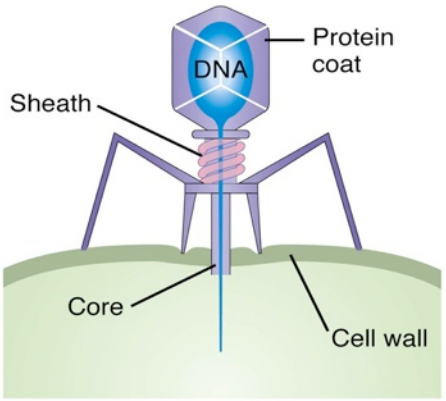
**These classifications apply not only to prokaryotes, but also to all forms of life

vi. Reproduction
1. Asexual reproduction through binary fission → results in exponential growth potential
    a. In binary fission, the DNA replicates and the contents of the cytoplasm is split evenly into two new cells

vii. Prokaryotic Cell Genetics/Methods of Gene Recombination

1. Plasmids
    a. Circular DNA that exists in bacteria separate from the bacteria's main genome
    b. Replication of plasmid is independent from replication of the main genome
    c. Plasmids often contain genes that allow for conjugation
        i. In conjugation, the bacterium with the conjugation gene grows a sex pilus which is able to attach to another bacterium. The plasmid then replicates and is passed through the pilus to the recipient bacterium.
2. Transformation/Transduction
    a. Transformation
        i. Bacteria will randomly pick up DNA floating in the environment
        ii. Very inefficient, but if bacteria are exposed to enough free DNA, they will pick it up and incorporate it into their genome
    b. Transduction
        i. Transfer of DNA to a bacterium by a bacteriophage



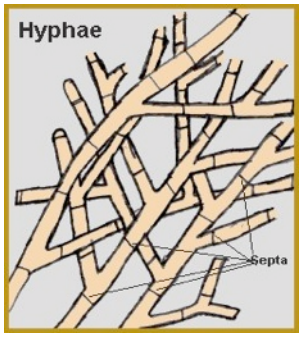


3. Acquisition of Virulence/Antibiotic Resistance
    a. The ability of a bacteria strain to cause disease or to be resistant to antibiotics is encoded in their genes, especially on plasmids
    b. These genes can be passed from one bacterium to another, thereby increasing its virulence or making it resistant to antibiotics
        i. Can be passed by conjugation, transduction or transformation

III. Fungi – Eukaryotes
    a. Description
        i. Unicellular – yeasts

ii. Multi-cellular – molds, mushrooms
iii. Contain cell walls constructed from chitin
iv. All fungi digest food outside their bodies by secreting powerful hydrolytic enzymes and them absorbing them

b. Modes of Nutrition
   i. Saprobic Fungi
      1. absorb nutrients from non-living organic material
   ii. Parasitic Fungi
      1. absorb nutrients from the cells of living hosts
   iii. Mutualistic Symbionts
      1. absorb nutrients from another organism, but reciprocate with function beneficial to the partner in some way – ie, some fungi break down soil minerals so that plants can absorb them and then, in turn, get carbon the plant secures through photosynthesis



c. Multicellular Fungi
   i. The basic building units are thread-like structures known as hyphae
      1. hyphae are divided into "cells" by cross walls known as septae
   ii. Mycelia – interwoven mats of hyphae that form a larger structure

d. Fungal Lifecycle
   i. Fungi begin life as haploid spores
   ii. The spores grow into a mature complete fungus that is also haploid
   iii. Fertilization of gametes occurs within the mature fungus to form zygotes
   iv. The zygotes undergo meiosis to form haploid spores that grow within a structure called an ascus
   v. The spores are released to disperse and grow into new fungi