Genetics/ 3rd year

Lecture 1

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Outlines

- Introduction
- Importance of genetics
- Basic Concepts of Genetics
- Branches of genetics
- Characteristics of Model Organisms suitable for genetic studies

Introduction:

Genetics is defined as the science that deals with heredity and variation in organisms, including the genetic features and constitution of a single organism, species, or group, and with the mechanisms by which they are affected.

The term genetics was proposed by the English biologist William Bateson In 1906. The classical principles of genetics were deduced by Gregory Mendel in 1865, on the basis of the results of breeding experiments with peas. Mendel studied the inheritance of a number of well-defined traits, such as seed color, and was able to deduce general rules for their transmission.

In all cases, he could correctly interpret the observed patterns of inheritance by assuming that each trait is determined by a pair of inherited factors, which are now called genes. These factors are transmitted from one plant generation to the next in a predictable pattern, each factor being responsible for an observable trait. The trait one can observe is the phenotype. The underlying genetic information is the genotype. The term gene for this type of a heritable factor was introduced in 1909 by the Danish biologist Wilhelm Johannsen (1857 1927). Theodor Boveri (1862–1915), recognized the genetic individuality of chromosomes in 1902.

- Genetics became an independent scientific field in 1910 when Thomas H. Morgan introduced the fruit fly (Drosophila melanogaster) for systematic genetic studies and showed that genes are arranged on chromosomes in sequential order. Morgan summarized this in 1915 in the chromosome theory of inheritance. This theory proposed that hereditary factors responsible for Mendelian inheritance are located on the chromosomes within the nucleus. This field is known as a Classic (Mendelian) genetics.

- Population genetics as a new genetic field was found as a result of the work of the English mathematician G.H. Hardy and the German physician W. Weinberg independently who recognized, in 1908 that Mendelian inheritance accounts for certain regularities in the genetic structure of populations. Their work contributed to the successful introduction of genetic concepts into plant and animal breeding.

	Seed shape	Seed color	Flower color	Flower position	Pod shape	Pod color	Plant height
One form of trait (dominant)	round	yellow	violet-red	axial	inflated	green	tall
A second form of trait (recessive)	wrinkled	green	white	terminal	pinched	yellow	short

Figure (1): Phenotype of several traits in pea.(Weinberg, Robert A. , 2014, The biology of cancer , 2nd edition)

The double helix structure of DNA molecule was deduced by Watson and crick in 1953, this structure elucidated the replication of DNA and how could the DNA store the genetic information.

Importance of genetics:

In the last century, genetics has become an important biological tool, using mutants to gain an understanding of specific processes. This work has included:

a. Analyzing heredity in populations.

b. Analyzing evolutionary processes.

c. Identifying genes that control steps in processes.

d. Mapping genes.

e. Determining products of genes.

F. Analyzing molecular features of genes and regulation of gene expression.

Basic Concepts of Genetics:

1. Genetic material of both eukaryotes and prokaryotes is DNA (deoxyribonucleic acid). Many viruses also have DNA, but some have RNA genomes instead.

2. DNA has two chains, each made of nucleotides composed of a deoxyribose sugar, a phosphate group and a base. The chains form a double helix.

3. There are four bases in DNA: A (adenine), G (guanine), C (cytosine) and T (thymine).

a. In RNA, U (uracil) replaces T.

b. The sequence of bases determines the genetic information.

c. Genes are specific sequences of nucleotides that pass traits from parents to offspring.

4. Genetic material in cells is organized into chromosomes (literally “colored body” because it stains with biological dyes).

a. Prokaryotes generally have one circular, super coiled chromosome.

b. Eukaryotes generally have:

i. Linear chromosomes in their nuclei, with different species having different numbers of chromosomes.
ii. DNA in organelles (e.g., mitochondria and chloroplasts) that is usually a circular molecule

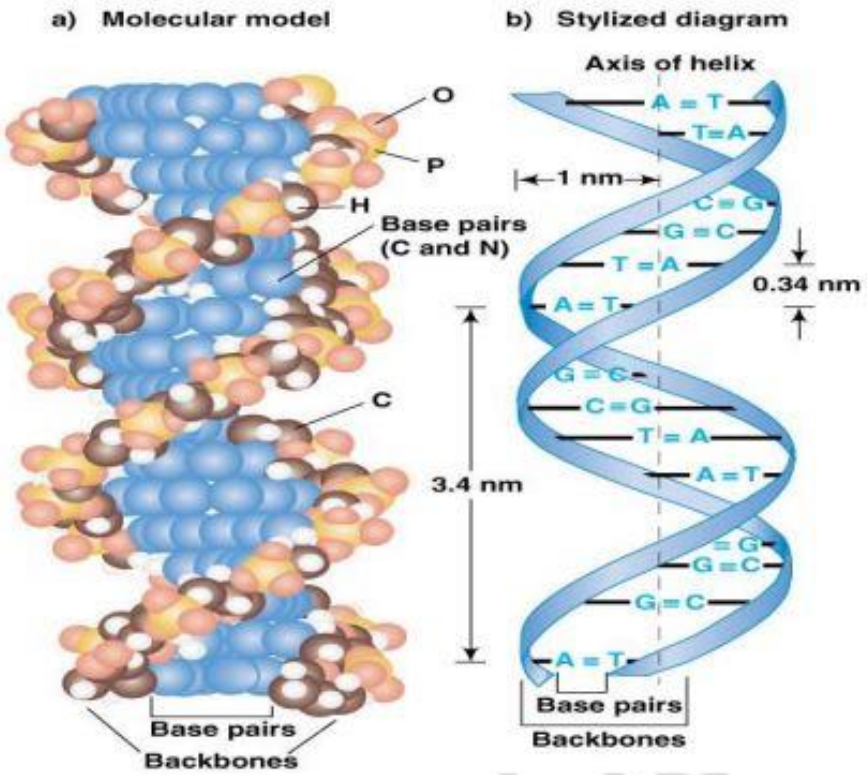

Figure (2): the double helix structure of DNA proposed by Watson and Crick in 1953. (Russell ,P. , *I* Genetics, a molecular approach , 3rd edition , 2009, edited by Yue-Wen Wang. Pearsons,USA).

Branches of genetics

1- Mendelian (classic) genetics: deals with transmission (movement) of genes and genetic traits from parents to offspring, and with genetic recombination.

2- Population genetics: studies heredity in groups for traits determined by one or a few genes.

3- Quantitative genetics: studies group hereditary for traits determined by many genes simultaneously. 4- Human genetics: is the study of inheritance as it occurs in human beings.

5- Medical genetics is the branch of medicine that involves the diagnosis and management of hereditary disorders.

a. Cytogenetic is the study of the structure, the behavior of the chromosomes and chromosome abnormalities.
b. Biochemical genetics: Metabolic (or biochemical) genetics involves the diagnosis and of inborn errors of metabolism in which patients have enzymatic deficiencies that involved in metabolism of carbohydrates, amino acids, and lipids.
c. Cancer genetics.
d. Mitochondrial genetics: its deal with the diagnosis of mitochondrial disorders, which have a molecular basis but often result in biochemical abnormalities due to deficient in energy production.

e- Developmental genetics: is the study of the process by which animals and plants grow and develop.

f. Genetic counseling: Genetic counseling is the process of providing information about genetic conditions, diagnostic testing, and risks in other family members.

6- Molecular genetics: deals with the molecular structure and function of genes.

7- Microbial genetics. It studies the genetics of very small (micro) organisms; bacteria, archaea, viruses and some protozoa and fungi. This involves the study of the genotype of microbial species the phenotypes.

8- Genetic engineering: is the direct manipulation of an organism's genome using biotechnology.

Characteristics of Model Organisms suitable for genetic studies:

Many organisms are used in genetic research. Desirable qualities for an experimental organism include:

a. A well-known genetic history.

b. b. A short life cycle so generations can be studied in a relatively.

c. A large number of offspring from each mating.

d. Ease of growing and handling the organism.

e. Marked genetic variation within the population.

Genetics/ 3rd year

Lecture 2

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Outlines

- **Identify the genetic material**
- **Griffith's experiment**
- **Oswald Avery’s experiment**

Identify the genetic material

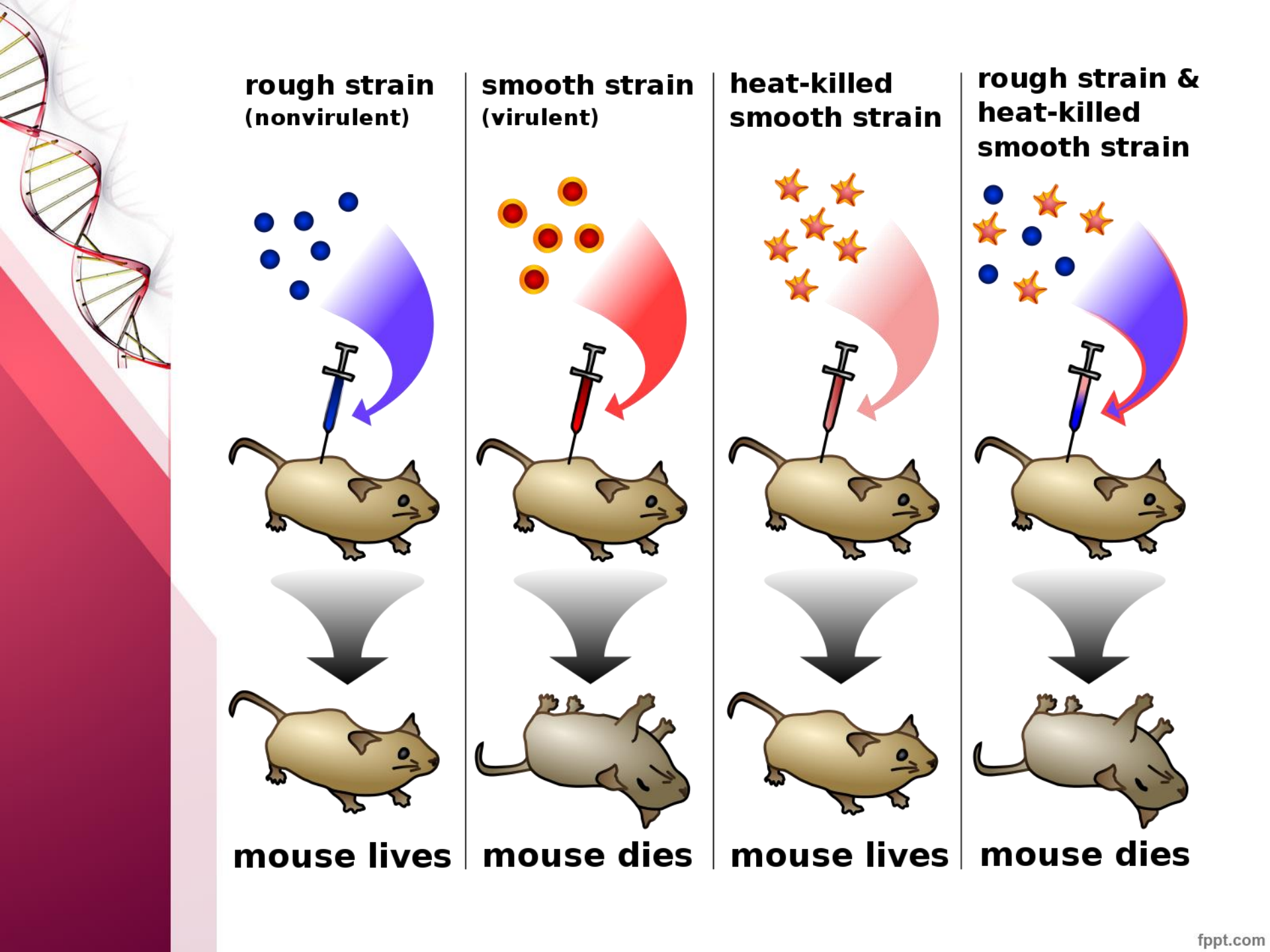
Frederick Griffith (1877–1941) is a British bacteriologist whose focus was the epidemiology and pathology of bacterial pneumonia. In January 1928 he reported what is now known as Griffith's Experiment, the first widely accepted demonstrations of bacterial transformation, whereby a bacterium clearly changes its form and function. He showed that Streptococcus pneumonia, implicated in many cases of lobar pneumonia, could transform from one strain into a different strain. The observation was attributed to an unidentified underlying principle, later known in the Avery laboratory as the "transforming principle" (abbreviated as T. P.) and identified as DNA.

Griffith's experiment

Pneumococci has two general forms—rough (R) and smooth (S). The S form is more virulent, and bears a capsule, which is a slippery polysaccharide coat—outside the peptidoglycan cell wall common among all classical bacteria—and prevents efficient phagocytosis by the host's essential immune cells.

Injected subcutaneously with S form, mice succumbed to pneumonia and death within several days. However, the R form, lacking a capsule—its outer surface being cell wall—is relatively avirulent, and does not cause pneumonia as often.

When Griffith injected heat-killed S into mice, as expected, no disease caused. When mice were injected with a mixture of heat-killed S and live R, however, pneumonia and death caused. The live R had transformed into S—and replicated as such—often characterized as Griffith's Experiment. More accurately, Griffith reported that R tended to transform into S if a large amount of live R, alone, were injected, and that adding much heat-killed S made transformation reliable. The scientific explanation for these results is that the non-pathogenic rough bacteria (R) received part of the genetic material from the pathogenic smooth bacteria (S) and transformed into a pathogenic smooth strain that killed the injected mice. This process is called transformation. The figure below shows an experiment of Frederick Griffith.

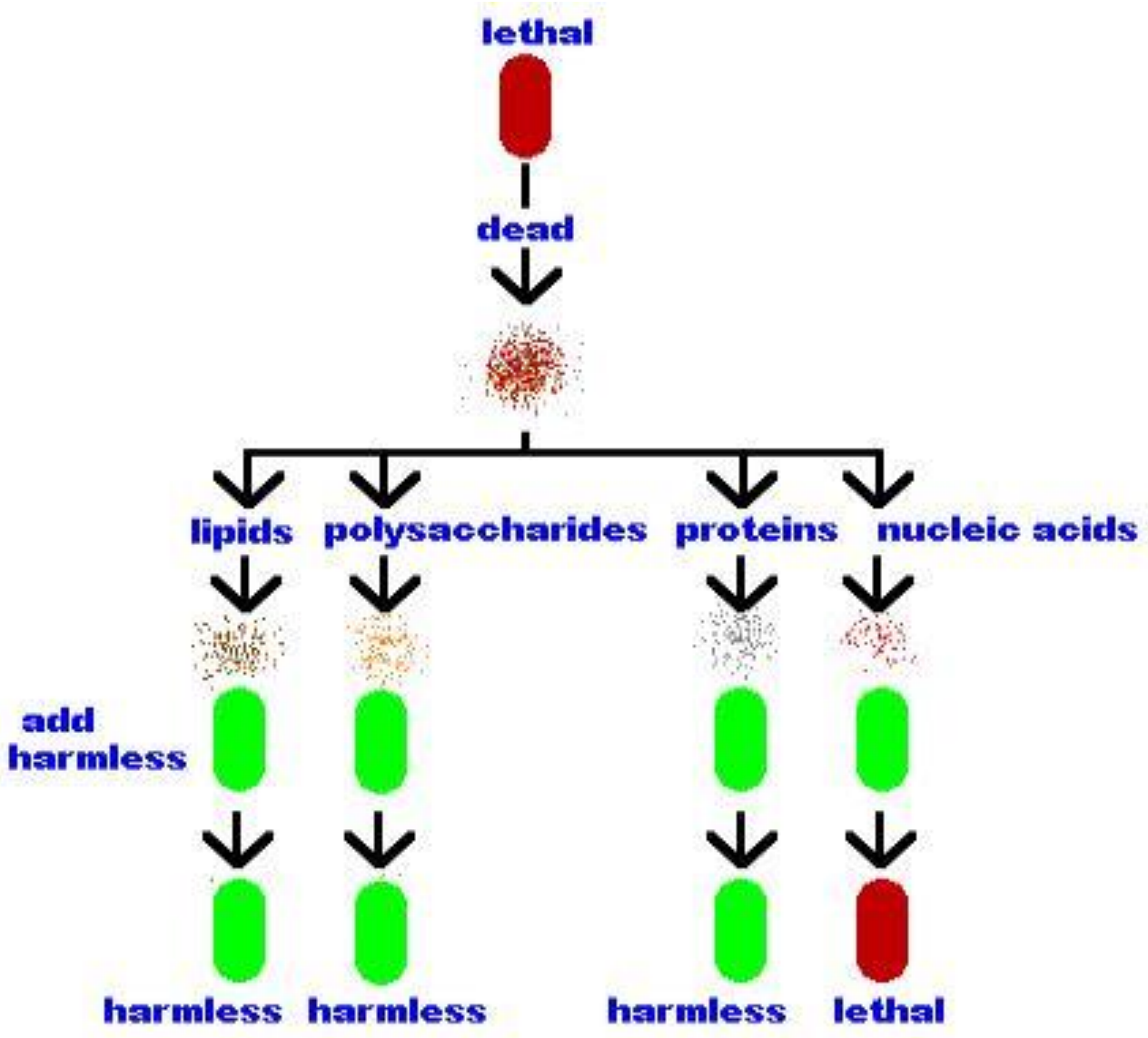

rough strain
(nonvirulent)
smooth strain
(virulent)
heat-killed
smooth strain
rough strain &
heat-killed
smooth strain
mouse lives
mouse dies
mouse lives
mouse dies

Oswald Avery's experiment

In 1934 AD, the scientist Oswald Avery and his group repeated the Griffith experiment using modern technical methods. They extracted the components of bacteria from proteins, fats, polysaccharides, and nucleic acid. Then they mixed the components of the rough and smooth bacteria with each other. They found that the only substance capable of transforming the rough bacteria (R strain) non-pathogenic to the smooth bacteria (S strain) pathogenic is the DNA of smooth bacteria. The purer DNA is more effective to the process of bacterial transformation. It was also discovered that adding a DNA-digesting enzyme, DNAase, would stop the process of bacterial transformation, and that the presence of other enzymes that do not affect on DNA and bacterial transformation. Thus, Avery and his group in 1944 AD were reached to the following fact:

"The DNA is the genetic material of the cell, and the DNA of a specific cell with a specific genotype can combine with the DNA of another cell with a different genotype, which leads to changing the genotype of the new cell to the genotype of the old cell.

The following figure shows the experiment of Avery Oswald and his group:



Genetics/ 3rd year

Lecture 3

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Outlines

- **Gene structure**
- **Common features**
- **Eukaryotes**
- **Prokaryotes**

Gene structure

Gene structure is the organisation of specialised sequence elements within a gene. Genes contain most of the information necessary for living cells to survive and reproduce. In most organisms, genes are made of DNA, where the particular DNA sequence determines the function of the gene. A gene is transcribed (copied) from DNA into RNA, which can either be non-coding (ncRNA) with a direct function, or an intermediate messenger (mRNA) that is then translated into protein. Each of these steps is controlled by specific sequence elements, or regions, within the gene. Every gene, therefore, requires multiple sequence elements to be functional. This includes the sequence that actually encodes the functional protein or ncRNA, as well as multiple regulatory sequence regions. These regions may be as short as a few base pairs, up to many thousands of base pairs long.

Much of gene structure is broadly similar between eukaryotes and prokaryotes. These common elements largely result from the shared ancestry of cellular life in organisms over 2 billion years ago. Key differences in gene structure between eukaryotes and prokaryotes reflect their divergent transcription and translation machinery. Understanding gene structure is the foundation of understanding gene annotation, expression, and function.

Common features

The structures of both eukaryotic and prokaryotic genes involve several nested sequence elements. Each element has a specific function in the multi-step process of gene expression. The sequences and lengths of these elements vary, but the same general functions are present in most genes. Although DNA is a double-stranded molecule, typically only one of the strands encodes information that the RNA polymerase reads to produce protein-coding mRNA or non-coding RNA..

This 'sense' or 'coding' strand, runs in the 5' to 3' direction where the numbers refer to the carbon atoms of the backbone's ribose sugar. The open reading frame (ORF) of a gene is therefore usually represented as an arrow indicating the direction in which the sense strand is read

Regulatory sequences are located at the extremities of genes. These sequence regions can either be next to the transcribed region (the promoter) or separated by many kilobases (enhancers and silencers). The promoter is located at the 5' end of the gene and is composed of a core promoter sequence and a proximal promoter sequence. The core promoter marks the start site for transcription by binding RNA polymerase and other proteins necessary for copying DNA to RNA. The proximal promoter region binds transcription factors that modify the affinity of the core promoter for RNA polymerase. Genes may be regulated by multiple enhancer and silencer sequences that further modify the activity of promoters by binding activator or repressor proteins. Enhancers and silencers may be distantly located from the gene, many thousands of base pairs away.

The binding of different transcription factors, therefore, regulates the rate of transcription initiation at different times and in different cells.

Regulatory elements can overlap one another, with a section of DNA able to interact with many competing activators and repressors as well as RNA polymerase. For example, some repressor proteins can bind to the core promoter to prevent polymerase binding. For genes with multiple regulatory sequences, the rate of transcription is the product of all of the elements combined. Binding of activators and repressors to multiple regulatory sequences has a cooperative effect on transcription initiation.

Although all organisms use both transcriptional activators and repressors, eukaryotic genes are said to be 'default off', whereas prokaryotic genes are 'default on'. The core promoter of eukaryotic genes typically requires additional activation by promoter elements for expression to occur. The core promoter of prokaryotic genes, conversely, is sufficient for strong expression and is regulated by repressors.

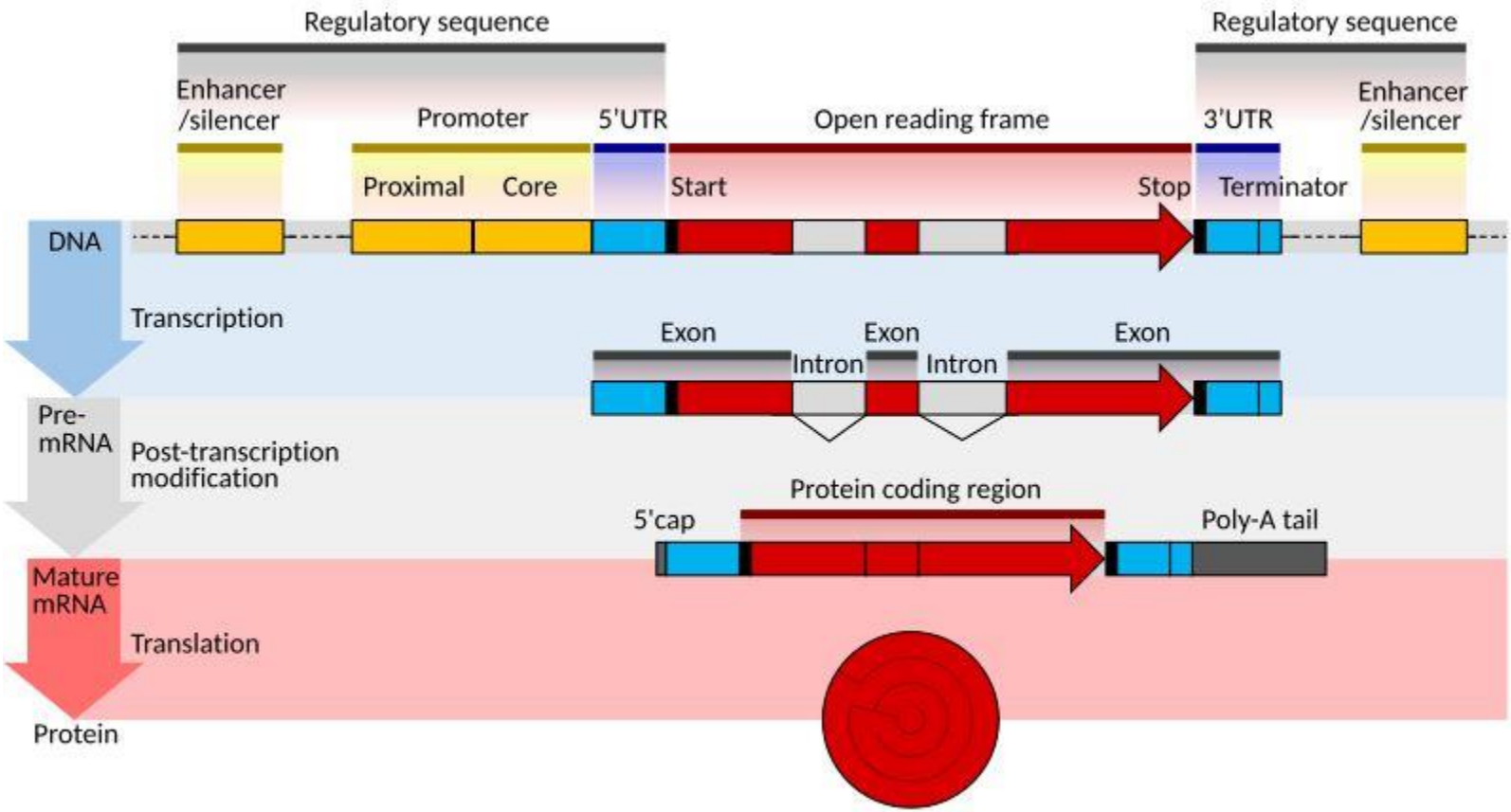
An additional layer of regulation occurs for protein coding genes after the mRNA has been processed to prepare it for translation to protein. Only the region between the start and stop codons encodes the final protein product. The flanking untranslated regions (UTRs) contain further regulatory sequences. The 3' UTR contains a terminator sequence, which marks the endpoint for transcription and releases the RNA polymerase. The 5’ UTR binds the ribosome, which translates the protein-coding region into a string of amino acids that fold to form the final protein product. In the case of genes for non-coding RNAs, the RNA is not translated but instead folds to be directly functional.

Eukaryotes

The structure of eukaryotic genes includes features not found in prokaryotes. Most of these relate to post-transcriptional modification of pre-mRNAs to produce mature mRNA ready for translation into protein. Eukaryotic genes typically have more regulatory elements to control gene expression compared to

prokaryotes. This is particularly true in multicellular eukaryotes, humans for example, where gene expression varies widely among different tissues.

A key feature of the structure of eukaryotic genes is that their transcripts are typically subdivided into exon and intron regions. Exon regions are retained in the final mature mRNA molecule, while intron regions are spliced out (excised) during post-transcriptional processing. Indeed, the intron regions of a gene can be considerably longer than the exon regions. Once spliced together, the exons form a single continuous protein-coding regions, and the splice boundaries are not detectable. Eukaryotic post-transcriptional processing also adds a 5' cap to the start of the mRNA and a poly-adenosine tail to the end of the mRNA. These additions stabilise the mRNA and direct its transport from the nucleus to the cytoplasm, although neither of these features are directly encoded in the structure of a gene.

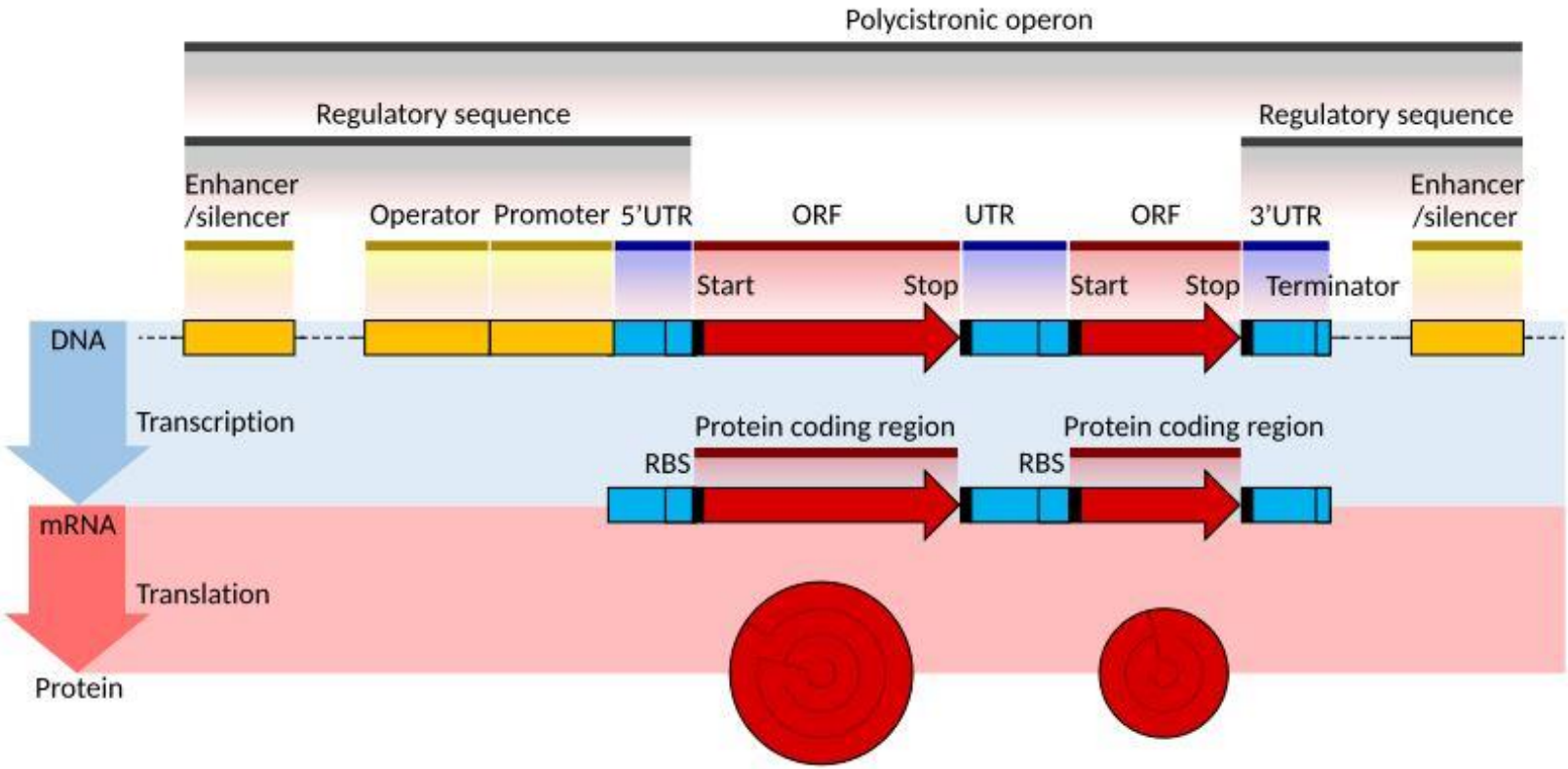


The structure of a eukaryotic protein-coding gene. Regulatory sequence controls when and where expression occurs for the protein coding region (red). Promoter and enhancer regions (yellow) regulate the transcription of the gene into a pre-mRNA which is modified to remove introns (light grey) and add a 5' cap and poly-A tail (dark grey). The mRNA 5' and 3' untranslated regions (blue) regulate translation into the final protein product.

Prokaryotes

The overall organisation of prokaryotic genes is markedly different from that of the eukaryotes. The most obvious difference is that prokaryotic ORFs are often grouped into a polycistronic operon under the control of a shared set of regulatory sequences. These ORFs are all transcribed onto the same mRNA and so are co-regulated and often serve related functions. Each ORF typically has its own ribosome binding site (RBS) so that ribosomes simultaneously translate ORFs on the same mRNA. Some operons also display translational coupling, where the translation rates of multiple ORFs within an operon are linked. This can occur when the ribosome remains attached at the end of an ORF and simply translocates along to the next without the need for a new RBS. Translational coupling is also observed when translation of an ORF affects the accessibility of the next RBS through changes in RNA secondary structure. Having multiple ORFs on a single mRNA is only possible in prokaryotes because their transcription and translation take place at the same time and in the same subcellular location.

The operator sequence next to the promoter is the main regulatory element in prokaryotes. Repressor proteins bound to the operator sequence physically obstructs the RNA polymerase enzyme, preventing transcription. Riboswitches are another important regulatory sequence commonly present in prokaryotic UTRs. These sequences switch between alternative secondary structures in the RNA depending on the concentration of key metabolites. The secondary structures then either block or reveal important sequence regions such as RBSs. Introns are extremely rare in prokaryotes and therefore do not play a significant role in prokaryotic gene regulation.



The structure of a prokaryotic operon of protein-coding genes. Regulatory sequence controls when expression occurs for the multiple protein coding regions (red). Promoter, operator and enhancer regions (yellow) regulate the transcription of the gene into an mRNA. The mRNA untranslated regions (blue) regulate translation into the final protein products.

Genetics/ 3rd year

Lecture 4

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Outlines

- **DNA Replication**
- **General feature of DNA replication**
- **DNA Replication in Prokaryotic cells**
- **Mechanism of Lagging strand synthesis**
- **DNA Replication in Eukaryotes**

DNA Replication

DNA replication is fundamental process occurring in all living organism to copy their DNA. The process is called replication in sense that each strand of double strand DNA (dsDNA) serve as template for reproduction of complementary strand.

General feature of DNA replication

- DNA replication is semi conservative - It is bidirectional process

- It proceed from a specific point called origin - It proceed in 5'-3' direction

- It occur with high degree of fidelity - It is a multi-enzymatic process

DNA replication occurs by three steps

1. Initiation: • Initiation complex formation • Closed complex formation • Open complex formation

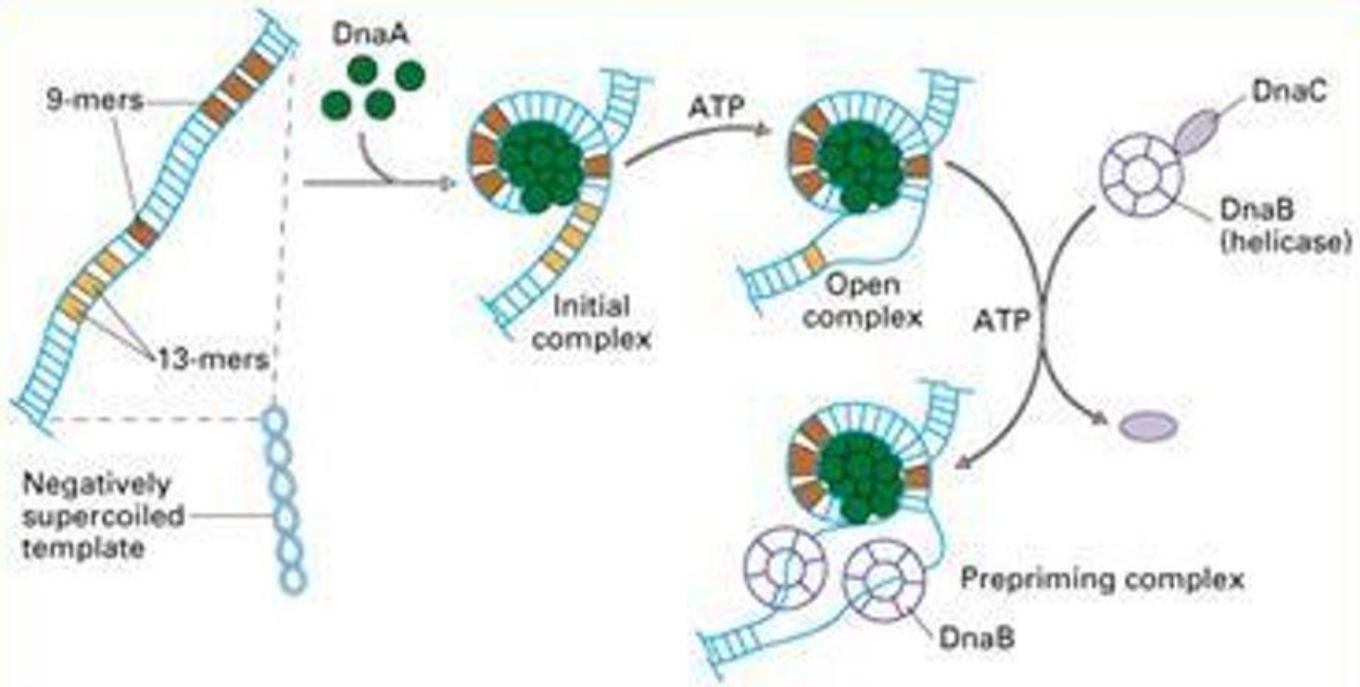
2. Elongation: • Leading strand synthesis • Lagging strand synthesis

3. Termination.

DNA Replication in Prokaryotic cells :

1. Initiation:

DNA replication begins from origin. In *E coli*, replication origin is called OriC which consists of 245 base pair and contains DNA sequences that are highly conserved among bacterial replication origin. Two types of conserved sequences are found at OriC, three repeats of 13 bp (GATCTNTTTATTT, GATCTNTTNTATT, GATCTCTTATTAG) and four repeats of 9 bp (TTATCCACA, TTTGGATAA, TTATACACA, TGTGGATAA) called 13 mer and 9 mer respectively.



♣ About 20 molecules of Dna A proteins binds with 9 mer repeats along with ATP which causes DNA to wraps around dnaA protein forming initial complex. The dna A protein and ATP trigger the opening of 13 mer repeats froming open complex.

♣ Two copies of dnaB proteins (helicase) binds to 13 mer repeats. This binding is facilitated by another molecule called dnaC. The dnaB and dnaC interaction causes dnaB ring to open which binds with each of the DNA strand. The hydrolysis of bound ATP release dnaC leaving the dnaB bound to the DNA strand.

♣ The binding of helicase is key step in replication initiation. dnaB migrates along the single stranded DNA in 5'-3' direction causing unwinding of the DNA.

♣ The activity of helicase causes the topological stress to the unwinded strand forming supercoiled DNA. This stress is relieved by the DNA topoisomerase (DNA gyrase) by negative supercoiling. Similarly, single stranded binding protein binds to the separated strand and prevents reannaeling of separated strand and stabilize the strand.

♣ The DNA polymerase cannot initiate DNA replication. So, at first primase synthesize 10±1 nucleotide (RNA in nature) along the 5'-3' direction. In case of *E. coli* primer synthesized by primase starts with ppp-AG-nucleotide. Primer is closely associated with dnaB helicase so that it is positioned to make RNA primer as a single stand DNA (ssDNA) of lagging strand.

2. Elongation:

A . Leading strand synthesis:

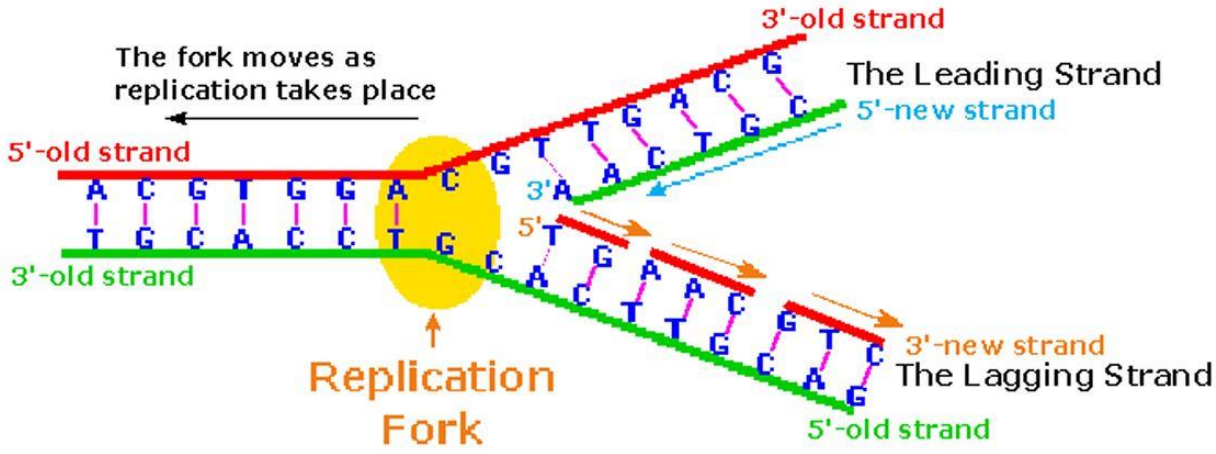
♣ Leading strand synthesis is more a straight forward process which begins with the synthesis of RNA primer by primase at replication origin.

♣ DNA polymerase III then adds the nucleotides at 3’end. The leading strand synthesis then proceed continuously keeping pace with unwinding of replication fork until it encounter the termination sequences.

B. Lagging strand synthesis:

♣ The lagging strand synthesized in short fragments called Okazaki fragments. At first RNA primer is synthesized by primase and as in leading strand DNA polymerase III binds to RNA primer and adds dNTPS.

♣ On this level the synthesis of each okazaki fragments seems straight forward but the reality is quite complex.



Mechanism of Lagging strand synthesis:

♣ The complexicity lies in the co-ordination of leading and lagging strand synthesis. Both the strand are synthesized by a single DNA polymerase III dimer which accomplished the looping of template DNA of lagging strand synthesizing Okazaki fragments.

♣ Helicase (dnaB) and primase (dnaG) constitute a functional unit within replication complex called **primosome**.

♣ DNA pol III use one set of core sub unit (Core polymerase) to synthesize leading strand and other set of core sub unit to synthesize lagging strand.

♣ In elongation steps, helicase in front of primase and pol III, unwind the DNA at the replication fork and travel along lagging strand template along 5'-3' direction.

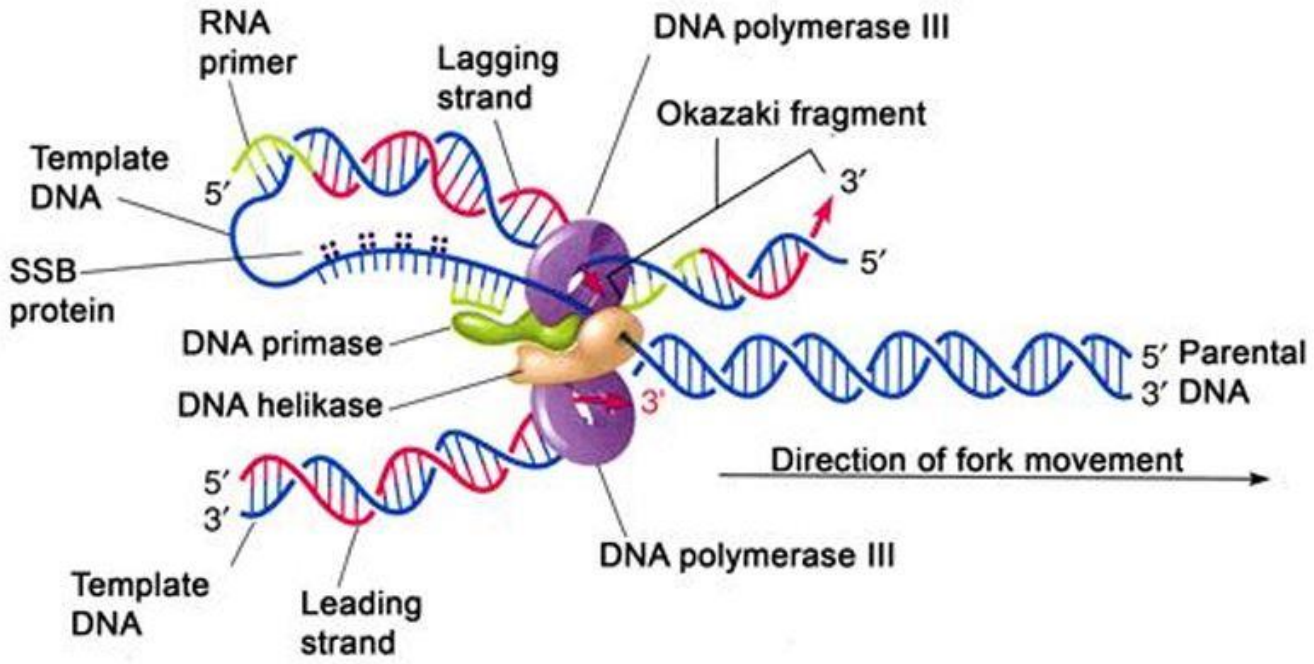
♣ DnaG primase occasionally associated with dnaB helicase synthesizes short RNA primer. A new B-sliding clamp is then positioned at the primer by B-clamp loading complex of DNA pol III.

♣ When the Okazaki fragments synthesis is completed, the replication halted and the core sub unit dissociates from their sliding clamps and associates with new clamp. This initiates the synthesis of new Okazaki fragments.

♣ Both leading and lagging strand are synthesized co-ordinately and simultaneously by a complex protein moving in 5’-3’ direction. In this way both leading and lagging strand can be replicated at same time by a complex protein that move in same direction.

♣ Every so often the lagging strands must dissociates from the replicosome and reposition itself so that replication can continue.

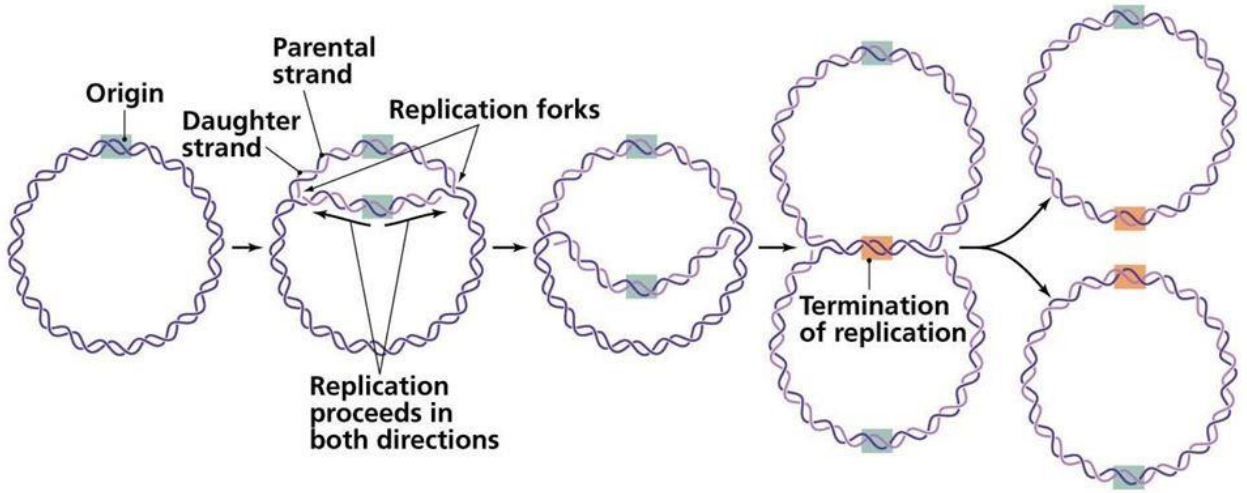
♣ Lagging strand synthesis is not completes until the RNA primer has been removed and the gap between adjacent Okazaki fragments are sealed. The RNA primer are removed by exonuclease activity (5’-3’) of DNA pol-I and replaced by DNA. The gap is then sealed by DNA ligase using NAD as co-factor.



3. Termination:

- Evantually the two replication forks of circular *E. coli* chromosome meet at termination recognizing sequences (ter).

- The Ter sequence of 23 bp are arranged on the chromosome to create trap that the replication fork can enter but cannot leave. Ter sequences function as binding site for Terminus utilization substance (TUS) protein.



- Ter-TUS complex can arrest the replication fork from only one direction. Ter-TUS complex encounter first with either of the replication fork and halt it. The other opposing replication fork halted when it collide with the first one. This seems the Ter-TUS sequences is not essential for termination but it may prevent over replication by one fork if other is delayed or halted by a damage or some obstacle.

- When either of the fork encounter Ter-TUS complex, replication halted.

- Final few hundred bases of DNA between these large protein complexes are replicated by not yet known mechanism forming two interlinked chromosome.

- In *E. coli* DNA topoisomerase IV (type II) cut the two strand of one circular DNA and segrate each of the circular DNA and finally join the strand. The DNA finally transfer to two daughter cell.

DNA Replication in Eukaryotes:

1. Eukaryotic chromosomes generally contain much more DNA than those of prokaryotes, and their replication forks move much more slowly. If they were like typical prokaryotes, with only one origin of replication per chromosome, DNA replication would take many days.

2. Instead, eukaryotic chromosomes contain multiple origins, at which DNA denatures and replication then proceeds bidirectionally until an adjacent replication fork is encountered. The DNA replicated from a single origin is called a replicon, or replication unit.

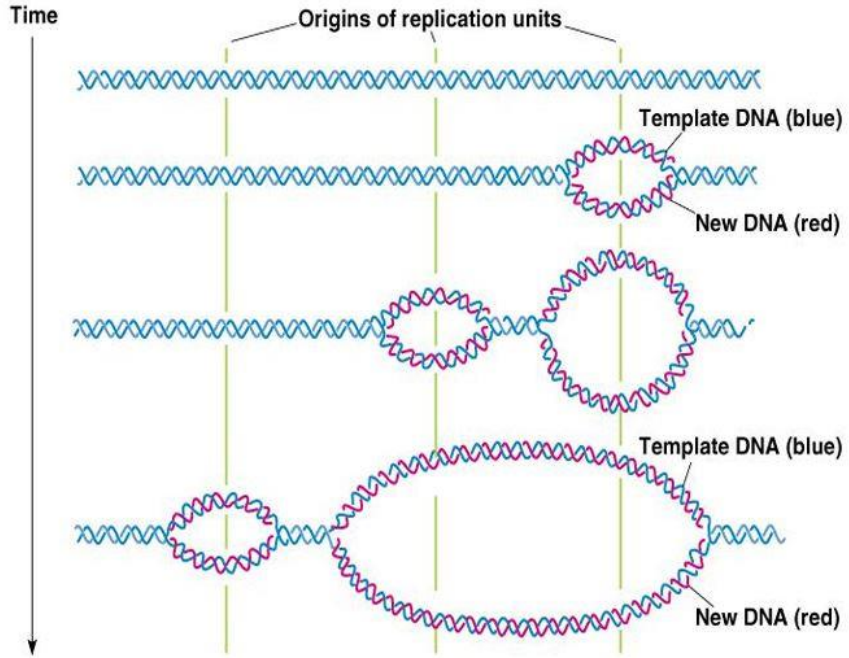


Figure 4: Temporal ordering of DNA replication initiation events in replication units of eukaryotic chromosomes.

Eukaryotic replication enzymes:

a. Three DNA polymerases are used to replicate nuclear DNA. Pol α (alpha) extends the 10-nt RNA primer by about 30nt. Pol δ(delta) and Pol ε(epsilon) extend the RNA/DNA primers, one on the leading strand and the other on the lagging (it is not clear which synthesizes which).

b. Other DNA pols replicate mitochondrial or chloroplast DNA, or are used in DNA repair.

Genetics/ 3rd year

Lecture 5

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Outlines

- **Heritability**
- **Heritability in the broad sense (H^2)**
- **Heritability in the narrow sense (h^2)**
- **Importance of Heritability**

Heritability

is a statistic used in the fields of breeding and genetics that estimates the degree of variation in a phenotypic trait in a population that is due to genetic variation between individuals in that population.

- **Heritability in the broad sense (H^2):** is the proportion of the phenotypic variance that is due to all genetic effects (additive, dominance and epistasis):

$$H^2 = \frac{V_G}{V_P} = \frac{V_A + V_D + V_I}{V_P}$$

It measures the strength of the relationship between the phenotypic values of the individuals and their genotypic values.

Where: VP= VG + VE

Heritability in the narrow sense (h^2): is the proportion of the phenotypic variance that is due to additive genetic effects only.

$$h^2 = \frac{V_A}{V_P}$$

It measures two things:

1. The degree to which the offspring resemble their parents in the phenotype for a trait.
2. The strength of the relationship between the phenotypic values and the additive genetic effects (the relationship between P and A).

- **Notes:**

1. Heritability is a measure on a population of individuals in a given environment for a given character. It is NOT measured on one individual.
2. Heritability can be estimated for each quantitative trait.
3. It varies from population to another and from environment to another for the same traits.

Example

- Suppose that for lamb weight at six months for the sheep population in Palestine: $V_A = 40$, $V_D = 8$, $V_I = 2$, $V_E = 50$. Calculate the heritability in the broad sense and heritability in the narrow sense for this trait.

Heritability in the broad sense:

$$H^2 = \frac{V_A + V_D + V_I}{V_P} = \frac{40 + 8 + 2}{40 + 8 + 2 + 50} = \frac{50}{100} = 0.50$$

Heritability in the narrow sense:

$$h^2 = \frac{V_A}{V_P} = \frac{40}{100} = 0.40$$

- **Heritability is always positive ranging from 0 to 1.0.**
- Traits with low heritability ($h^2 < 0.20$):
  - reproductive traits like days open calving interval, litter size, and conception rate
  - longevity or productive live (about 0.10)
  - weaning weight in swine (about 0.10)
- Moderately heritable traits (h^2 of 0.2 to 0.4):
  - Milk yield, fat yield and protein yield (0.25-0.35)
  - Birth weight in sheep
  - Yearling weight in sheep
- Highly heritable traits ($h^2 > 0.4$):
  - Carcass traits and traits related to skeletal dimensions like mature body weight
  - Fat and protein% in milk.

Importance of heritability

1) Heritability is very important in selection (in genetic improvement) ➔It determines if phenotypic selection would be efficient or not:

   - Low heritability: phenotypic selection is not efficient (low accuracy of selection).

   - High heritability: phenotypic selection is efficient (high accuracy of selection)

2) Heritability is important in prediction of breeding values