Chapter 19

Eukaryotic Genomes: Organization, Regulation, and Evolution

Key Concepts

19.1 Chromatin structure is based on successive levels of DNA packing

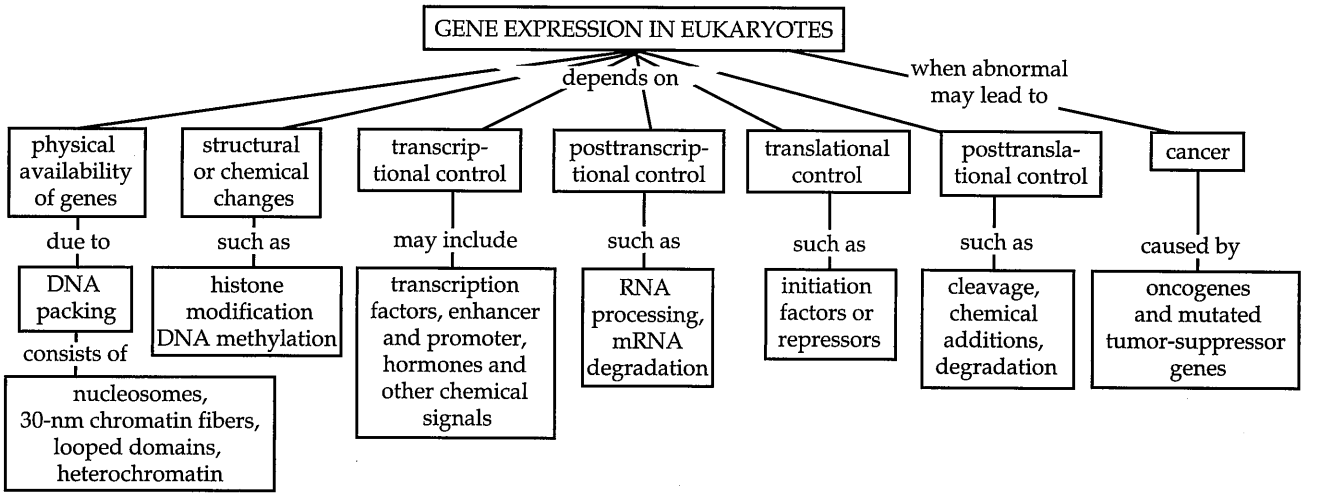
19.2 Gene expression can be regulated at any stage, but the key step is transcription

19.3 Cancer results from genetic changes that affect cell cycle control

19.4 Eukaryotic genomes can have many noncoding DNA sequences in addition to genes

19.5 Duplications, rearrangements, and mutations of DNA contribute to genome evolution

Framework



Chapter Review

The control of gene expression is more complex in eukaryotes due to the much greater size of the genome and the cell specialization found in multicellular eukaryotes. **Chromatin** is the DNA-protein complex, which is ordered into multiple levels of structural organization.

19.1 Chromatin structure is based on successive levels of DNA packing

In eukaryotes, each chromosome consists of a single, extremely long DNA double helix precisely complexed with a large amount of protein. In interphase, chromatin is extended in the nucleus, whereas discrete, condensed chromosomes appear during mitosis.

Nucleosomes, or "Beads on a String" **Histones** are small, positively charged proteins that bind tightly to the negatively charged DNA to make up chromatin. Unfolded chromatin appears as a string of beads, each bead a **nucleosome** consisting of the DNA helix wound around a protein core of four pairs of different histone molecules. The amino end *(histone tail)* of each of the eight histones extends outward. Also

called the *10-nm fiber* because of its diameter, a nucleosome is the basic unit of DNA packing, remaining intact even during transcription. A fifth histone, H1, attaches to the DNA near the bead for the next level of packing.

Higher Levels of DNA Packing The *30-nm fiber* is a tightly coiled cylinder of nucleosomes organized with the aid of histone H1, the histone tails, and the linker DNA between nucleosomes. The *looped domain* is a loop of the 30-nm chromatin fiber attached to a nonhistone protein scaffold. During prophase, looped domains form a *300-nm fiber.* In a metaphase chromosome, looped domains coil and fold, further compacting the chromatin.

The looped domains of interphase chromosomes appear to be attached to discrete locations of the nuclear lamina inside the nuclear envelope, perhaps helping to organize areas of transcription. Certain regions of chromatin, called **heterochromatin,** are in a highly condensed state during interphase. The more open form of interphase chromatin, called **euchromatin,** is available for transcription.

■ INTERACTIVE QUESTION 19.1

List the multiple levels of packing in a metaphase chromosome in order of increasing complexity.

19.2 Gene expression can be regulated at any stage, but the key step is transcription

Eukaryotic cells in multicellular organisms control the expression of their genes in response to their external and internal environments and also to direct the **cell differentiation** necessary to create specialized cells.

Differential Gene Expression Differences in cells with the same genome are the result of **differential gene expression.** Only a small portion of the DNA codes for proteins; some codes for RNA, and most is noncoding DNA (although some of this is transcribed into RNAs of unknown function). Gene expression can be regulated at various stages, although, as in prokaryotes, it is most commonly regulated at transcription.

Regulation of Chromatin Structure DNA packing and the location of genes relative to nucleosomes and attachment to the protein scaffold may help control which genes are available for transcription.

The attachment of acetyl groups ($-COCH_3$) to amino acids in histone tails, called **histone acetylation,** appears to change the binding of a nucleosome's histones such that transcription proteins may more easily access genes. Some enzymes that acetylate or deacetylate histones are associated with or part of transcription factors. The addition of methyl groups ($-CH_3$) to histone tails leads to condensation of the chromatin. According to the *histone code hypothesis,* the specific patterns of modifications determine chromatin shape and influence transcription.

In a process called DNA methylation, methyl groups are added to DNA bases (usually cytosine) after DNA synthesis. Genes are generally more heavily methylated in cells in which they are not expressed. Some proteins that bind to methylated DNA also interact with histone deacetylation enzymes, reinforcing the transcription repression.

DNA methylation may reinforce gene regulatory decisions of early development as enzymes act during DNA replication to methylate daughter DNA strands and pass on methylation records. Such methylation patterns account for **genomic imprinting** in mammals in which either a maternal or paternal allele of certain genes is permanently regulated.

Epigenic inheritance is the transmission of traits by mechanisms that do not involve changes in DNA sequences, such as chromatin modifications that affect gene expression in future generations of cells.

■ INTERACTIVE QUESTION 19.2

a. Give an example of highly methylated and inactive DNA common in mammalian cells.

b. Would histone tail deacetylation increase or decrease the transcription of a gene located in that nucleosome?

Regulation of Transcription Initiation A typical gene consists of a promoter sequence, where a transcription initiation complex that includes RNA polymerase II attaches, and a sequence of introns interspersed among the coding exons. After transcription, RNA processing of the pre-mRNA removes the introns and adds a 5′ cap and a poly-A tail at the 3′ end. **Control elements** are noncoding sequences to which transcription factors bind that help regulate transcription.

General **transcription factors** bind to RNA polymerase and each other to initiate the transcription of all protein-coding genes. The binding of *specific* transcription factors to control elements,

however, greatly increases transcription rates of particular genes.

Proximal control elements are located close to the promoter. **Enhancers** are groups of *distal control elements* located far upstream or downstream of a gene or in an intron. According to a current model, a protein-mediated bend in the DNA brings **activators** bound to enhancers into contact with *mediator proteins*, which interact with proteins at the promoter. These protein-protein interactions assemble the initiation complex. Hundreds of transcription activators have been identified. Most have a DNA-binding domain and one or more activation domains that bind other regulatory proteins.

Some specific transcription factors act as **repressors,** which can inhibit gene expression in several ways, such as blocking the binding of activators or binding to specific control elements in an enhancer. Some activators and repressors influence chromatin structure by recruiting proteins that either acetylate or deacetylate histones. Most eukaryotic gene repression may occur by this *silencing* at the level of chromatin modification.

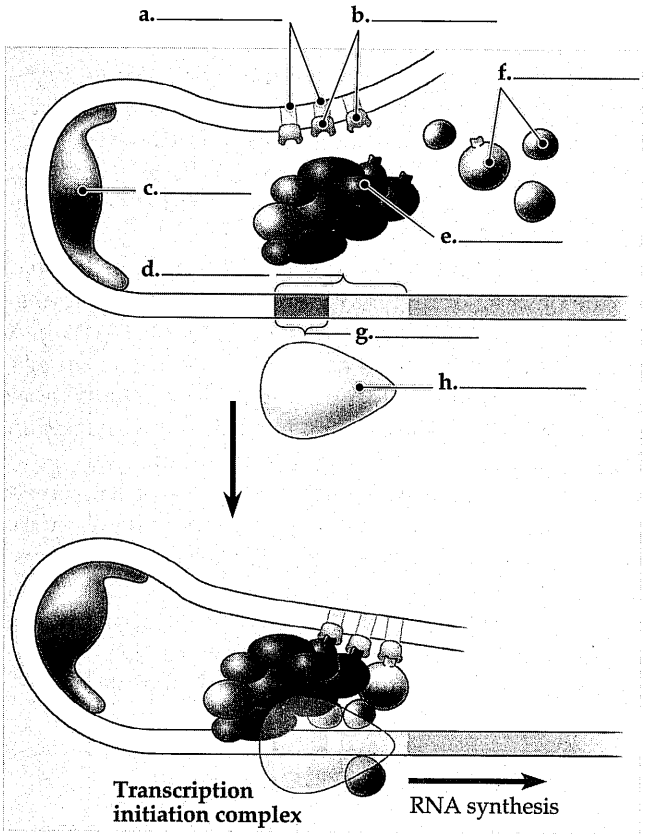
Only a dozen or so short nucleotide sequences have been found in control elements, but each enhancer contains about ten different control elements. The combination of these elements ensures that specific activator proteins must be present to activate transcription. Thus, the specific activators and repressors made in a cell determine which genes are expressed.

Prokaryotic genes are often organized into operons that are controlled by the same promoter and control elements and transcribed together. In a few cases, coexpressed eukaryotic genes (each with its own promoter) are clustered together and their regulation involves changes in chromatin structure making them available for transcription. Most eukaryotic genes coding for enzymes in the same metabolic pathway are scattered on the chromosomes. The integrated control of these genes may involve a specific collection of control elements associated with each related gene and requiring the same activators.

For example, steroid hormones may activate multiple genes when they bind to specific receptor proteins within a cell that function as transcription activators for several genes. Other signal molecules bind to cell surface receptors and initiate signal transduction pathways that activate particular transcription activators or repressors. The same chemical signal will activate genes with the same control elements and thus coordinate gene expression.

■ INTERACTIVE QUESTION 19.3

Label the components of this diagram of how enhancers, mediator proteins, and transcription factors facilitate formation of a transcription initiation complex.



Mechanisms of Post-transcriptional Regulation Gene expression, measured by the amount of functional protein that is made, can be regulated at any posttranscriptional step. At the level of RNA processing, **alternative RNA splicing** may produce different mRNA molecules when regulatory proteins control which segments of the primary transcript are chosen as introns and which as exons.

In contrast to prokaryotic mRNA that is degraded after a few minutes, eukaryotic mRNA can last hours or even weeks. The hydrolysis of mRNA by nucleases is usually preceded by the enzymatic shortening of the poly-A tail and removal of the 5′ cap. The untranslated region (UTR) at the 3′ end of mRNA may contain nucleotide sequences that affect stability.

MicroRNAs (miRNAs) are small, single-stranded RNA molecules that associate with a complex of proteins and then base-pair with complementary sequences on target mRNA. This miRNA-protein complex either blocks translation or degrades the mRNA. Long RNA molecules fold on themselves, and an enzyme called Dicer cuts this double-stranded RNA into short fragments. One strand of the fragment becomes an miRNA. This inhibition of gene expression by RNA molecules was first observed experimentally and called **RNA interference (RNAi).** Injection of double-stranded RNA into a cell produced **small interfering RNAs (siRNAs)** that appear to fuction as miRNAs to inhibit expression of genes with the same sequence.

The translation of eukaryotic mRNA can be delayed by the binding of regulatory proteins to the 5′ untranslated region (5′ UTR) that prevent ribosome binding. A great deal of mRNA is synthesized and stored in egg cells; translation may begin when enzymes add more A residues to poly-A tails or when translation initiation factors are activated following fertilization.

Following translation, polypeptides are often cleaved or chemical groups added to yield an active protein. Some proteins must be transported to target locations. Selective degradation of proteins may serve as a control mechanism in the cell. Molecules of the protein ubiquitin are added to mark proteins for destruction. **Proteasomes** recognize and degrade the marked proteins.

■ INTERACTIVE QUESTION 19.4

a. The untranslated regions (UTR) at both the 5′ and 3′ ends of an mRNA may contribute to regulation of gene expression. Describe their different effects.

b. Does the action of microRNAs increase or decrease gene expression? Explain.

19.3 Cancer results from genetic changes that affect cell cycle control

Types of Genes Associated with Cancer Carcinogens, x-rays, or viruses most often cause the changes in the genes that regulate cell growth and division that lead to cancer. Viruses that can cause cancer in animals are called *tumor viruses*. Cells become transformed into cancer cells when viral nucleic acid becomes integrated into host DNA. Viruses have been linked to certain types of human cancer.

Oncogenes, or cancer-causing genes, were first found in certain retroviruses. Similar genes were later recognized in the genomes of humans and other animals. Cellular **proto-oncogenes,** which code for proteins that stimulate cell growth and division, may become oncogenes by several mechanisms. Mutations may result in more copies of the gene being present than normal (amplification), transposition or chromosomal translocation (both of which may bring the gene under the control of a more active promoter or control element), or a change in a nucleotide sequence in either a promoter or enhancer that increases gene expression or in the gene that creates a more active or resilient protein.

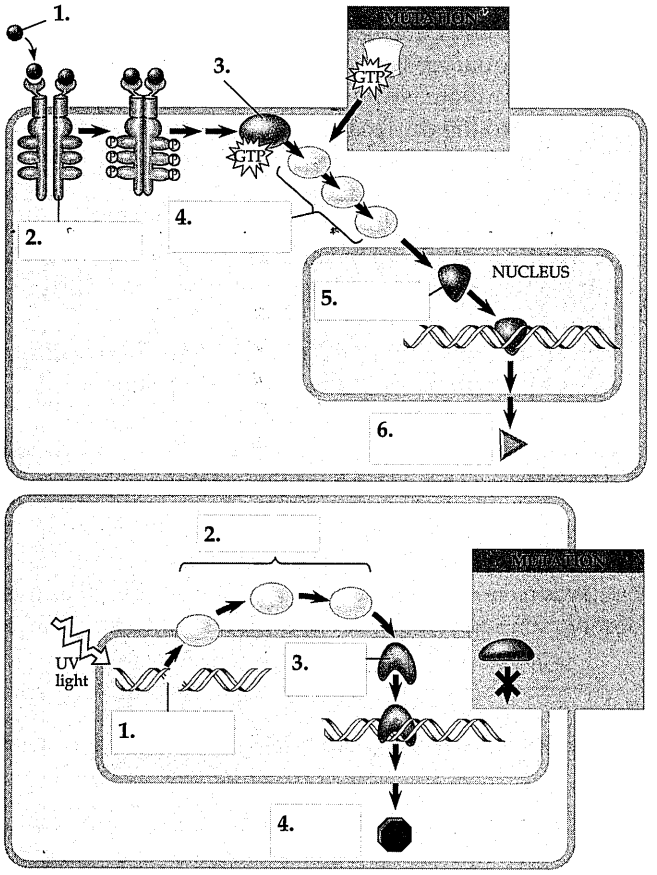
Mutations in tumor-suppressor genes can contribute to the onset of cancer when they result in a decrease in the activity of proteins that prevent uncontrolled cell growth. Tumor-suppressor proteins have various functions, such as repair of damaged DNA, control of cell adhesion, and inhibition of the cell cycle.

Interference with Normal Cell–Signaling Pathways In about 30% of human cancers, the *ras* proto-oncogene is mutated. The ***ras* gene** codes for a G protein that connects a growth-factor receptor on the plasma membrane to a cascade of protein kinases that leads to the production of a cell cycle stimulating protein. A mutation may create a hyperactive version of the Ras protein that relays a signal without binding of a growth factor.

The ***p53* gene** is mutated in about 50% of human cancers. It codes for a tumor-suppressor protein that is involved in the synthesis of several growth-inhibiting proteins. Such proteins may activate the *p21* gene, whose product binds to cyclin-dependent kinases, halting the cell cycle and allowing time for the cell to repair damaged DNA. The p53 transcription factor can also activate genes involved in DNA repair. Should DNA damage be irreparable, p53 activates "suicide genes" that initiate apoptosis.

■ INTERACTIVE QUESTION 19.5

These diagrams represent signaling pathways that stimulate (top diagram) or inhibit (bottom) the cell cycle. Describe the numbered steps and then explain the effect of mutations that make a hyperactive Ras protein (top) or a defective p53 transcription factor (bottom).



The Multistep Model of Cancer Development More than one somatic mutation appears to be needed to produce a cancerous cell. Mutation of a single proto-oncogene can stimulate cell division, but both alleles for several tumor-suppressor genes must be defective to allow uncontrolled cell growth. In many malignant tumors, the gene for telomerase becomes activated and cells become able to divide indefinitely.

Virus-associated cancers are thought to account for about 15% of human cancer cases. The virus might add an oncogene or its insertion may affect a proto-oncogene or tumor-suppressor gene.

Inherited Predisposition to Cancer A genetic predisposition to certain cancers may involve the inheritance of an oncogene or a recessive mutant allele for a tumor-suppressor gene. Approximately 15% of colorectal cancers involve inherited mutations, often in the tumor-suppressor gene *APC*, which regulates cell migration and adhesion.

The 5–10% of breast cancer cases linked to family history have been linked to mutant alleles for either *BRCA1* or *BRCA2*, both of which appear to be tumor-suppressor genes.

19.4 Eukaryotic genomes can have many noncoding sequences in addition to genes

The Relationship Between Genomic Composition and Organismal Complexity Unlike the prokaryote genome, in which most of the DNA contains uninterrupted codes for proteins (or RNA), the eukaryote genome contains mostly noncoding DNA, including regulatory sequences, introns that interrupt coding sequences, nonfunctional genes, and sequences present in many copies, called **repetitive DNA.** More than half of this repetitive DNA (44% of the human genome) is either made up of or related to transposable elements.

Transposable Elements and Related Sequences **Transposons** move about a genome as a DNA intermediate. **Retrotransposons,** which make up the majority of transposable elements, always move by the "copy and paste" mechanism because they are first transcribed into an RNA intermediate. This RNA transcript is converted back to DNA by reverse transcriptase, coded by the retrotransposon itself. Transposable elements are present as multiple (although not identical) copies of transposons or as related sequences that have lost the ability to move. In primates, perhaps 10% of the genome is made up of *Alu elements*, 300-nucleotide-long sequences, many of which are transcribed into RNA but are of unknown function.

Other Repetitive DNA, Including Simple Sequence DNA About 5% of the human genome is repetitive DNA not related to transposable elements, which consists of large-segment duplications. *Simple-sequence DNA*, on the other hand, makes up 3% and consists of multiple copies of tandemly repeated sequences, often of fewer than 15 nucleotides. Much of this so-called *satellite DNA* is located at centromeres and telomeres.

Genes and Multigene Families Sequences coding for proteins and structural RNAs makes up 1.5% of the human genome. Including introns and regulatory sequences, the amount of gene-related DNA is 25% of the genome. About half of the coding DNA is for genes present in single copies of unique sequences. **Multigene families** are collections of similar or identical genes. With the exception of the genes for histone proteins, identical multigene families code for RNA products. The genes coding for the three largest rRNA molecules are arranged in a single transcription unit repeated in huge tandem arrays, enabling cells to produce the millions of ribosomes needed for protein synthesis.

Examples of multigene families of nonidentical genes are the two families of genes that code for the α and β polypeptide chains of hemoglobin. Different versions of each globin subunit are clustered together on two different chromosomes and are expressed at the appropriate time during development. **Pseudogenes,** nonfunctional DNA sequences similar to real genes, are also found in the globin gene family clusters.

■ INTERACTIVE QUESTION 19.6

Match the letter of the description and fill in the percentage (listed below) for each type of DNA sequence found in the human genome.

	Types of DNA	Description	%
1.	Exons & RNA-coding	_____	_____
2.	Introns & regulatory	_____	_____
3.	Transposable elements and related repetitive sequences	_____	_____
4.	Simple sequence repeats	_____	_____
5.	Large-segment duplications	_____	_____
6.	Unique noncoding DNA	_____	_____

	Descriptions
A.	satellite DNA in centromeres and telomeres
B.	multiple copies of moveable sequences
C.	gene fragments, nonfunctional genes
D.	protein and RNA-coding sequences
E.	multiple copies of large sequences
F.	DNA related to gene expression

Percentages to choose from: 1.5, 3, 5, 15, 24, and 44

19.5 Duplications, rearrangements, and mutations of DNA contribute to genome evolution

Duplication of Chromosome Sets Extra sets of chromosomes may arise by accidents in meiosis. The resulting extra genes might diverge through mutation, leading to genes with novel functions.

Duplication and Divergence of DNA Segments Errors such as unequal crossing over during meiosis and slippage of template strands during DNA replication might lead to the duplication of individual genes.

The α-globin and β-globin gene families appear to have evolved from a common ancestral globin, which was duplicated and diverged about 500 million years ago. Multiple duplications and mutations within each family have led to the current family of genes with related functions.

In other cases, mutations of a duplicated gene may lead to a protein product with a new function.

■ INTERACTIVE QUESTION 19.7

Lysozyme and α-lactalbumin have similar sequences but different functions. Both genes are found in mammals, but birds have only the gene for lysozyme. What does this observation suggest about the evolution of these genes?

Rearrangements of Parts of Genes: Exon Duplication and Exon Shuffling Unequal crossing over can lead to a gene with a duplicated exon. Exons often code for domains of a protein. Their duplication could provide a protein with enhanced structure and function. Errors in meiotic recombination could also lead to *exon shuffling* within a gene or between nonallelic genes. For instance, the gene for TPA has three different exons that may have originated in the genes for epidermal growth factor, finbronectin, and plasminogen.

How Transposable Elements Contribute to Genome Evolution Transposable elements may contribute to the evolution of a genome by promoting recombination, disrupting cellular genes or control elements, or moving genes or exons to new locations. Recombination events can take place between homologous transposable element sequences that are scattered throughout the genome, causing chromosomal mutations that may occasionally be beneficial to the organism.

Transposable elements that insert within a gene may disrupt its functioning; those that insert within

regulatory sequences may increase or decrease gene expression. A transposable element can also move a copy of a gene or an exon to a new location. The transposition of an *Alu* element into introns may provide an alternative splice site in the RNA transcript, producing a new portion of a protein that may result in alternative functions. The increased genetic diversity provided by these mechanisms provides raw material for natural selection.

Word Roots

eu- = true (*euchromatin:* the more open, unraveled form of eukaryotic chromatin)

hetero- = different (*heterochromatin:* nontranscribed eukaryotic chromatin that is highly compacted and is visible with a light microscope during interphase)

nucleo- = the nucleus; **-soma** = body (*nucleosome:* the basic beadlike unit of DNA packaging in eukaryotes)

proto- = first, original; **onco-** = tumor (*proto-oncogene:* a normal cellular gene corresponding to an oncogene)

pseudo- = false (*pseudogenes:* DNA segments very similar to real genes but which do not yield functional products)

retro- = backward (*retrotransposons:* transposable elements that move in a genome as an RNA intermediate, a transcript of the retrotransposon DNA)

Structure Your Knowledge

1. Fill in the table below to help you organize the major mechanisms that can regulate the expression of eukaryotic genes.

Level of Control	Examples
Chromatin structure	**a.**
Transcriptional regulation	**b.**
Post-transcriptional regulation	**c.**
Translational regulation	**d.**
Post-translational regulation	**e.**

2. **a.** What are proto-oncogenes? How do they become oncogenes?
   b. What is the role of tumor-suppressor genes in the development of cancer?
3. **a.** Describe a retrotransposon.
   b. How do transposable elements contribute to genome evolution?

Test Your Knowledge

MULTIPLE CHOICE: *Choose the one best answer.*

1. The control of gene expression is more complex in eukaryotic cells because
   a. DNA is associated with protein.
   b. gene expression differentiates specialized cells.
   c. the chromosomes are linear and more numerous.
   d. operons are controlled by more than one promoter region.
   e. inhibitory or activating molecules may help regulate transcription.

2. Histones are
   a. small, positively charged proteins that bind tightly to DNA.
   b. small bodies in the nucleus involved in rRNA synthesis.
   c. basic units of DNA packing consisting of DNA wound around a protein core.
   d. DNA bending proteins that facilitate formation of transcription initiation complexes.
   e. proteins responsible for producing repeating sequences at telomeres.
3. Heterochromatin
   a. has a higher degree of packing than does euchromatin.
   b. is visible with a light microscope during interphase.
   c. is not actively involved in transcription.
   d. makes up metaphase chromosomes.
   e. is all of the above.
4. DNA methylation of cytosine residues
   a. can be induced by drugs that reactivate genes.
   b. may be a mechanism of exogenic inheritance when methylation patterns are repeated in daughter cells.
   c. occurs in the promoter region and enhances binding of RNA polymerase.
   d. makes satellite DNA a different density so it can be separated by ultracentrifugation.
   e. may be related to the transformation of proto-oncogenes to oncogenes.
5. Which of the following appears to be attached to the nuclear lamina in a precise and organized fashion?
   a. nucleosomes
   b. heterochromatin
   c. 30-nm fiber
   d. looped domains of interphase chromosomes
   e. enhancer regions of actively transcribed genes
6. Which of the following is *not* true of enhancers?
   a. They may be located thousands of nucleotides upstream from the genes they affect.
   b. When bound with activators, they interact with the promoter region and other transcription factors to increase the activity of a gene.
   c. They may complex with steroid-activated receptor proteins and thus selectively activate specific genes.
   d. They may coordinate the transcription of enzymes involved in the same metabolic pathway.
   e. They are located within the promoter, and when complexed with a steroid or other small molecule, they release an inhibitory protein and thus make DNA more accessible to RNA polymerase.
7. Which of the following is *not* an example of the control of gene expression that occurs after transcription?
   a. mRNA stored in the cytoplasm needing activation of translation initiation factors
   b. the length of time mRNA lasts before it is degraded
   c. rRNA genes amplified in tandem arrays
   d. alternative RNA splicing before mRNA exits from the nucleus
   e. splicing or modification of a polypeptide
8. Pseudogenes are
   a. tandem arrays of rRNA genes that enable actively synthesizing cells to create enough ribosomes.
   b. genes that can become oncogenes when mutated by carcinogens.
   c. genes of multigene families that are expressed at different times during development.
   d. sequences of DNA that are similar to real genes but lack regulatory sequences necessary for gene expression.
   e. both c and d.
9. Which of the following might a proto-oncogene code for?
   a. DNA polymerase
   b. reverse transcriptase
   c. receptor proteins for growth factors
   d. an enhancer
   e. transcription factors that inhibit cell division genes

10. A gene can develop into an oncogene when it
    a. is present in more copies than normal.
    b. undergoes a translocation that removes it from its normal control region.
    c. develops a mutation that creates a more active or resistant protein.
    d. is transposed to a new location where its expression is enhanced.
    e. does any of the above.

11. What is the main reason that prokaryotic genes average 1,000 nucleotide base pairs, whereas human genes average about 27,000 base pairs?
    a. Prokaryotes have smaller, but many more, individual genes.
    b. Prokaryotes are more ancient organisms; longer genes arose later in evolution.
    c. Prokaryotes are much simpler organisms; humans have many types of differentiated cells.
    d. Prokaryotic genes do not have introns; human genes have many.
    e. Human proteins are much larger and more complex than prokaryotic proteins.

12. A tumor-suppressor gene could cause the onset of cancer if
    a. both alleles have mutations that decrease the activity of the gene product.
    b. only one allele has a mutation that alters the gene product.
    c. it is inherited in mutated form from a parent.
    d. a proto-oncogene has also become an oncogene.
    e. both a and d have happened.

13. What is apoptosis?
    a. a cell suicide program that may be initiated by p53 protein in response to DNA damage
    b. metastasis, or the spread of cancer cells to a new location in the body
    c. the transformation of a normal cell to a cancer cell
    d. the mutation of a G protein into a hyperactive form
    e. the transformation of a proto-oncogene to an oncogene by a point mutation

14. Which of the following would you expect to find as part of a receptor protein that binds with a steroid hormone?
    a. a TATA box located within the promoter region
    b. a domain that binds to DNA and protein-binding domains
    c. an activated operator region that allows attachment of RNA polymerase
    d. an enhancer sequence located at some distance upstream or downstream from the promoter
    e. transmembrane domains that facilitate its localization in a plasma membrane

15. A eukaryotic gene typically has all of the following associated with it *except*
    a. a promoter.
    b. an operator.
    c. enhancers.
    d. introns and exons.
    e. control elements.

16. What are proteasomes?
    a. complexes of proteins that excise introns
    b. single-stranded RNA molecules complexed with proteins that block translation of or degrade mRNA
    c. small, positively charged proteins that form the core of nucleosomes
    d. enormous protein complexes that degrade unneeded proteins in the cell
    e. complexes of transcription factors whose protein–protein interactions are required for enhancing gene transcription

17. Tissue plasminogen activator (TPA) is a protein with three types of domains. One of each of these types is found in the protein's epidermal growth factor, fibronectin, and plasminogen. What is a likely explanation for this?
    a. All four genes are members of a multigene family involved in cell signaling.
    b. TPA was the first gene to evolve; the other three genes each lost two domains.
    c. The gene for TPA arose by several instances of exon shuffling from the other three genes.
    d. TPA must have many *Alu* elements that allow for alternative splice sites to incorporate these exons.
    e. Several duplication events led to the evolution of the TPA gene.

18. Which of the following would most likely account for a family history of colorectal cancer?
    a. inheritance of a proto-oncogene
    b. a family diet that is low in fats and high in fiber
    c. a family history of breast cancer
    d. inheritance of the *ras* oncogene that locks the G protein in an active configuration
    e. inheritance of one mutated *APC* allele that regulates cell adhesion and migration

19. Which of the following best describes what pseudogenes and introns have in common?
    a. They are RNA molecules that are not translated into proteins.
    b. They are DNA segments that lack a promoter but do have other control regions.
    c. They are not expressed—they do not produce a functional product.
    d. They code for RNA products, not proteins.
    e. They appear to have arisen from retrotransposons.

20. How is the coordinated transcription of genes involved in the same pathway regulated?
    a. The genes are transcribed in one transcription unit, although each gene has its own promoter.
    b. The genes are located in the same region of the chromosome, and enzymes deacetylate the entire region so that transcription may begin.
    c. The genes all respond to the same general transcription factors, although they may respond to different specific transcription factors.
    d. A steroid hormone selectively binds to the promoters for all the genes.
    e. The genes have the same combination of control elements in the enhancer that bind with the particular activators present in the cell.