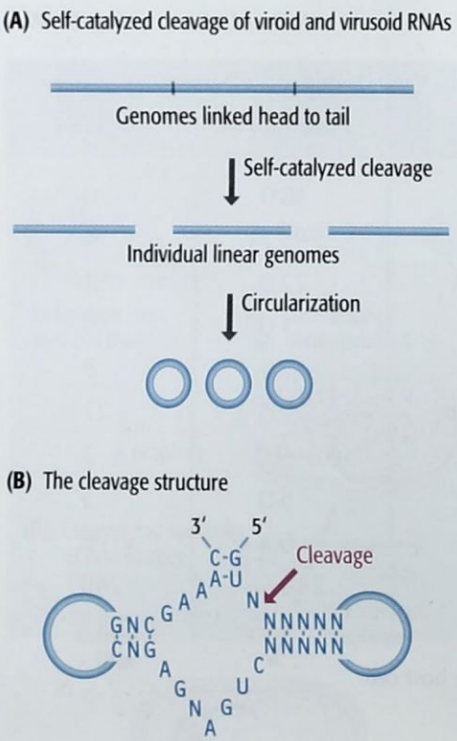


Figure 9.9 Self-catalyzed cleavage of linked genomes during replication of viroids and virusoids. (A) The replication pathway. (B) The "hammerhead" structure, which forms at each cleavage site and which has enzymatic activity. N, any nucleotide.

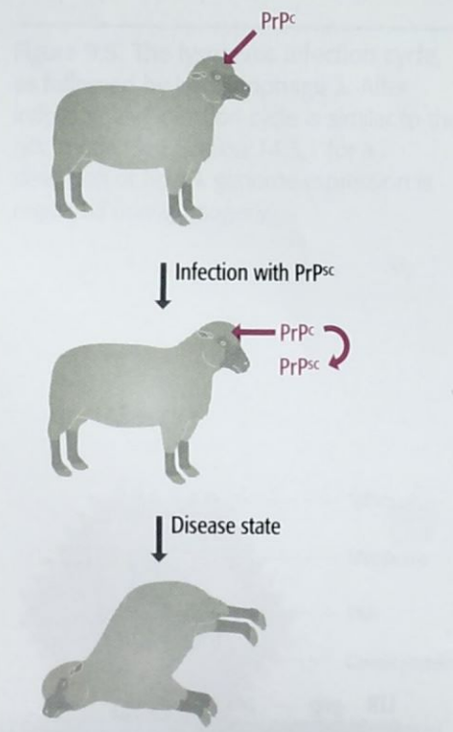


Figure 9.10 The mode of action of a prion. A normal, healthy sheep has PrP^{C} proteins in its brain. Infection with PrP^{SC} molecules leads to conversion of newly synthesized PrP^{C} proteins into PrP^{SC}, leading to the disease state—scrapie in sheep.

by a self-catalyzed reaction in which the RNA molecule acts as an enzyme (Figure 9.9). We will study these RNA enzymes in greater detail in Section 12.2.4.

Nucleic acid molecules that replicate within plant cells can perhaps be looked on as genomes even if they contain no genes. The same cannot be said for **prions**, as these infectious, disease-causing particles contain no nucleic acid. Prions are responsible for scrapie in sheep and goats and their transmission to cattle has led to the new disease called BSE—bovine spongiform encephalopathy. Whether their further transmission to humans causes a variant form of Creutzfeldt–Jakob disease (CJD) is controversial but accepted by many biologists. At first, prions were thought to be viruses but it is now clear that they are made solely of protein. The normal version of the prion protein, called PrP^{C}, is coded by a mammalian nuclear gene and synthesized in the brain, although its function is unknown. PrP^{C} is easily digested by proteases whereas the infectious version, PrP^{SC}, has a more highly β-sheeted structure that is resistant to proteases and forms fibrillar aggregates that are seen in infected tissues. Once inside a cell, PrP^{SC} molecules are able to convert newly synthesized PrP^{C} proteins into the infectious form, by a mechanism that is not yet understood, resulting in the disease state. Transfer of one or more of these PrP^{SC} proteins to a new animal results in accumulation of new PrP^{SC} proteins in the brain of that animal, transmitting the disease (Figure 9.10). Infectious proteins with similar properties are known in lower eukaryotes, examples being the Ure3 and Psi^{+} prions of *Saccharomyces cerevisiae*. It is clear, however, that prions are *gene products* rather than genetic material and despite their infectious properties, which led to the initial confusion regarding their status, they are unrelated to viruses or to subviral particles such as viroids and virusoids.

9.2 Mobile Genetic Elements

In Chapters 7 and 8 we learnt that eukaryotic genomes, and to a lesser extent those of prokaryotes, contain genome-wide or interspersed repeats, some with copy numbers of several thousand per genome, with the individual repeat units distributed in an apparently random fashion (Section 7.2.4). For many interspersed repeats, the genome-wide distribution pattern is set up by **transposition**, the process by which a segment of DNA can move from one position to another in a genome. These movable segments are called transposable elements, or **transposons**. Some types move by a **conservative** process, which involves the excision of the sequence from its original position followed by its reinsertion elsewhere. Conservative transposition therefore results in the transposon simply changing its position in the genome without increasing its copy number (Figure 9.11). **Replicative transposition**, on the other hand, results in an increase in copy number, because during this process the original element remains in place while a copy is inserted at the new position. This replicative process can therefore lead to a proliferation of the transposon at interspersed positions around the genome.

Both types of transposition involve recombination, and we will therefore deal with the details of the processes when we study recombination and related types of genome rearrangement in Chapter 17. What interests us here is the variety of structures displayed by the transposable elements found in eukaryotic and prokaryotic genomes, and the link that exists between these elements and viral genomes.

Figure 9.11 Conservative and replicative transposition.

9.2.1 Transposition via an RNA intermediate

Replicative transposons can be further subdivided into those that transpose via an RNA intermediate and those that do not. The process that involves an RNA intermediate, which is called **retrotransposition**, begins with synthesis of an RNA copy of the transposon by the normal process of transcription (Figure 9.12). The transcript is then copied into double-stranded DNA, which initially exists as an independent molecule outside of the genome. Finally, the DNA copy of the transposon integrates into the genome, possibly back into the same chromosome occupied by the original unit, or possibly into a different chromosome. The end result is that there are now two copies of the transposon, at different points in the genome.

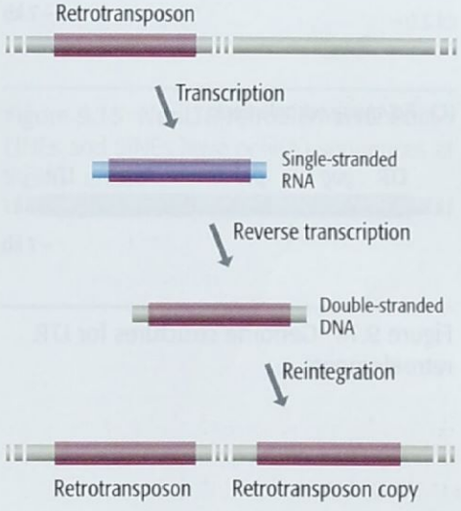


Figure 9.12 **Retrotransposition.** Compare with Figure 7.20 and note that the events are essentially the same as those that result in a processed pseudogene.

If we compare the mechanism for retrotransposition with that for replication of a viral retroelement, as shown in Figure 9.7, then we see that the two processes are very similar, the one significant difference being that the RNA molecule that initiates the process is transcribed from an endogenous genomic sequence during retrotransposition, and an exogenous viral genome during replication of a viral retroelement. This close similarity alerts us to the relationships that exist between these two types of element.

RNA transposons with long terminal repeats are related to viral retroelements

RNA transposons, or **retroelements**, are features of eukaryotic genomes but have not so far been discovered in prokaryotes. They can be broadly classified into two types: those that possess **long terminal repeats** (**LTRs**) and those that do not. Long terminal repeats, which play a central role in the process by which the RNA copy of an LTR element is reverse transcribed into double-stranded DNA (Section 17.3.2), are also possessed by viral retroelements (see Figure 9.8). It is now clear that these viruses are one member of a superfamily of elements that also includes endogenous LTR transposons. The first of the endogenous elements to be discovered was the *Ty* sequence of yeast, which is 6.3 kb in length and has a copy number of 25–35 in most *Saccharomyces cerevisiae* genomes—recall that one such element was present in the 50 kb segment of the yeast genome that we examined in Section 7.2.2 (see Figure 7.15B). Yeast genomes also contain 100 or so additional copies of the 330 bp LTRs of *Ty* elements, these solo "delta" sequences probably arising by homologous recombination between the two LTRs of a *Ty* element, which could excise the bulk of the element leaving a single LTR (Figure 9.13). This excision event is probably unrelated to transposition of a *Ty* element, which occurs by the RNA-mediated process shown in Figure 9.12.

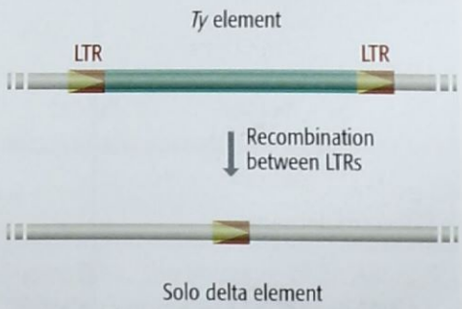


Figure 9.13 Homologous recombination between the LTRs at either end of a *Ty* element could give rise to a delta sequence.

There are several types of *Ty* element in yeast genomes. The most abundant of these, *Ty1*, is similar to the *copia* retroelement of the fruit fly. These elements are therefore now called the *Ty1/copia* family. If we compare the structure of a *Ty1/copia* retrotelement with that of a viral retroelement, then we

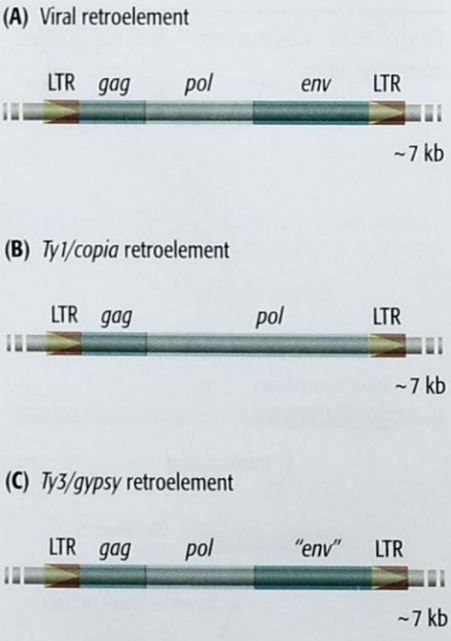


Figure 9.14 Genome structures for LTR retroelements.

see clear family relationships (Figure 9.14A and B). Each *Ty1/copia* element contains two genes, called *TyA* and *TyB* in yeast, which are similar to the *gag* and *pol* genes of a viral retroelement. In particular, *TyB* codes for a polyprotein that includes the reverse transcriptase that plays the central role in transposition of a *Ty1/copia* element. Note, however, that the *Ty1/copia* element lacks an equivalent of the viral *env* gene, the one that codes for the viral coat proteins. This means that *Ty1/copia* retroelements cannot form infectious virus particles and therefore cannot escape from their host cell. They do, however, form virus-like particles (VLPs) consisting of the RNA and DNA copies of the retroelements attached to core proteins derived from the TyA polyprotein. In contrast, the members of a second family of LTR retroelements, called *Ty3/gypsy* (again after the yeast and fruit fly versions), do have an equivalent of the *env* gene (Figure 9.14C) and at least some of these can form infectious viruses. Although classed as endogenous transposons, these infectious versions should be looked upon as viral retroelements.

LTR retroelements make up substantial parts of many eukaryotic genomes, and are particularly abundant in the larger plant genomes, especially those of grasses such as maize (see Figure 7.15D). They also make up an important component of invertebrate and some vertebrate genomes, but in the genomes of humans and other mammals all the LTR elements appear to be decayed viral retroelements rather than true transposons. These sequences are called **endogenous retroviruses (ERVs)** and with a copy number of approximately 240,000 they make up 4.7% of the human genome (Table 9.3). Human ERVs are 6–11 kb in length and have copies of the *gag*, *pol*, and *env* genes. Although most contain mutations or deletions that inactivate one or more of these genes, a few members of the human ERV group HERV-K have functional sequences. By comparing the positions of the HERV-K elements in the genomes of different individuals, it has been inferred that at least some of these are active transposons. The majority of human ERVs are, however, inactive sequences that are not capable of additional proliferation.

Table 9.3 Transposable elements in the human genome

Class	Family	Approximate number of copies	Fraction of genome (%)
SINE	Alu	1,200,000	10.7
	MIR	450,000	2.5
	MIR3	85,000	0.4
LINE	LINE-1	600,000	17.3
	LINE-2	370,000	3.3
	LINE-3	44,000	0.3
LTR retroelements	ERV	240,000	4.7
	MaLR	285,000	3.8
DNA transposons	MER-1	213,000	1.4
	MER-2	68,000	1.0
	Others	60,000	0.4

RNA transposons that lack LTRs

Not all types of RNA transposon have LTR elements. In mammals the most important types of nonLTR retroelements, or **retroposons**, are the **LINEs** (**long interspersed nuclear elements**) and **SINEs** (**short interspersed nuclear elements**). SINEs have the highest copy number for any type of interspersed repetitive DNA in the human genome, with over 1.7 million copies comprising 14% of the genome as a whole (Table 9.3). LINEs are less frequent, with just over 1 million copies, but as they are longer they make up a larger fraction of the genome—over 20%. The abundance of LINEs and SINEs in the human genome is underlined by their frequency in the 50 kb segment that we looked at in Section 7.2.2 (see Figure 7.12).

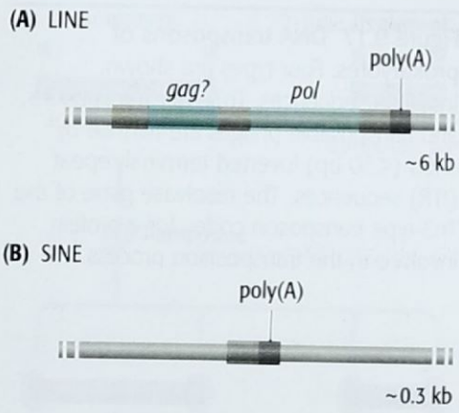


Figure 9.15 NonLTR retroelements. Both LINEs and SINEs have poly(A) sequences at their 3′ ends.

There are three families of LINEs in the human genome, of which one group, LINE-1, is both the most frequent and the only type that is able to transpose, the LINE-2 and LINE-3 families being made up of inactive relics. A full-length LINE-1 element is 6.1 kb and has two genes, one of which codes for a polyprotein similar to the product of the viral *pol* gene (Figure 9.15A). There are no LTRs, but the 3′ end of the LINE is marked by a series of A–T base pairs, giving what is usually referred to as a poly(A) sequence (though of course it is a poly(T) sequence on the other strand of the DNA). Not all copies of LINE-1 are full length, because the reverse transcriptase coded by LINEs does not always make a complete DNA copy of the initial RNA transcript, meaning that part of the 3′ end of the LINE may be lost. This truncation event is so common that only 1% of the LINE-1 elements in the human genome are full-length versions, with the average size of all the copies being just 900 bp. Although LINE-1 transposition is a rare event, it has been observed in cultured cells and appears to be responsible for hemophilia in some patients, due to movement of a LINE-1 sequence into the factor VIII gene, disrupting the gene and hence preventing synthesis of this important blood clotting protein.

SINEs are much shorter than LINEs, being just 100–400 bp and not containing any genes, which means that SINEs do not make their own reverse transcriptase enzymes (Figure 9.15B). Instead they "borrow" reverse transcriptases that have been synthesized by LINEs. The commonest SINE in primate genomes is **Alu**, which has a copy number of approximately 1.2 million in humans (Table 9.3). An Alu element comprises two halves, each half made up of a similar 120 bp sequence, with a 31–32 bp insertion in the right half (Figure 9.16). The mouse genome has a related element, called B1, which is 130 bp in length and equivalent to one half of an Alu sequence. Some Alu elements are actively copied into RNA, providing the opportunity for proliferation of the element.

Alu is derived from the gene for the 7SL RNA, a noncoding RNA involved in movement of proteins around the cell. The first Alu element may have arisen by the accidental reverse transcription of a 7SL RNA molecule and integration of the DNA copy into the human genome. Other SINEs are derived from tRNA genes which, like the gene for the 7SL RNA, are transcribed by RNA polymerase III in eukaryotic cells (Section 11.2.1), suggesting that some feature of the transcripts synthesized by this polymerase make these molecules prone to occasional conversion into retroposons.

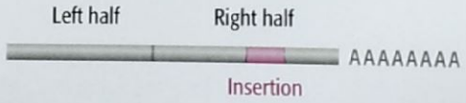


Figure 9.16 The structure of an Alu element. The element consists of two halves, each of 120 bp, with a 31–32 bp insertion in the right half, and a poly(A) tail at the 3′ end. The two halves (excluding the insertion) have about 85% sequence identity.

9.2.2 DNA transposons

Not all transposons require an RNA intermediate. Many are able to transpose in a more direct DNA-to-DNA manner. In eukaryotes, these DNA transposons

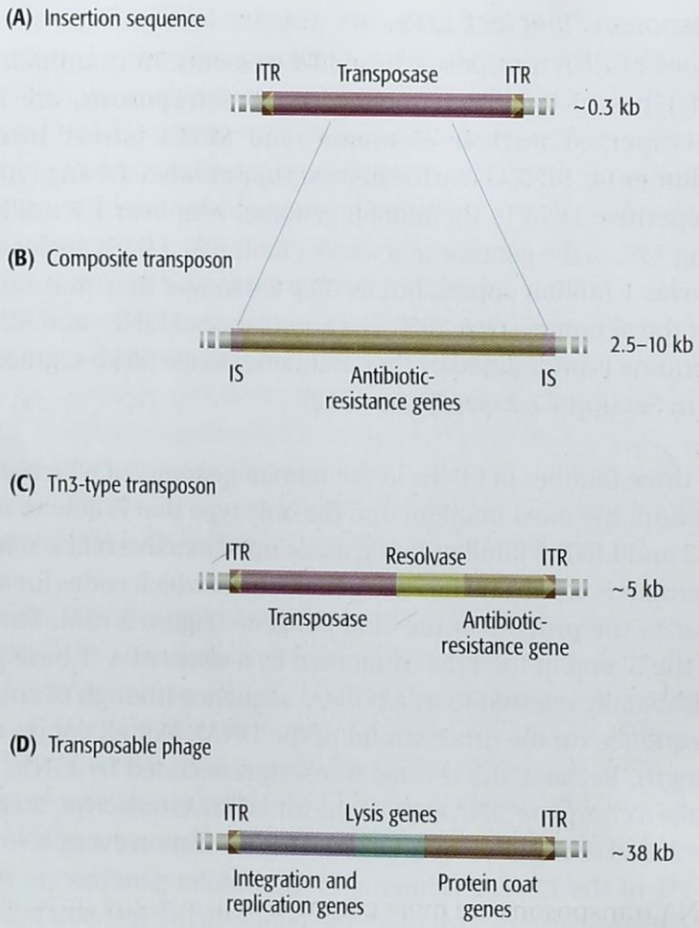


Figure 9.17 DNA transposons of prokaryotes. Four types are shown. Insertion sequences, Tn3-type transposons, and transposable phages are flanked by short (<50 bp) inverted terminal repeat (ITR) sequences. The resolvase gene of the Tn3-type transposon codes for a protein involved in the transposition process.

are less common than retrotransposons, but they have a special place in genetics because a family of plant DNA transposons—the Ac/Ds elements of maize—were the first transposable elements to be discovered, by Barbara McClintock in the 1950s. Her conclusions—that some genes are mobile and can move from one position to another in a chromosome—were based on exquisite genetic experiments, the molecular basis of transposition not being understood until the late 1970s.

DNA transposons are common in prokaryotic genomes

DNA transposons are an important component of many prokaryotic genomes. The insertion sequences, IS1 and IS186, present in the 50 kb segment of *E. coli* DNA that we examined in Section 8.2.1 (see Figure 8.7), are examples of DNA transposons, and a single *E. coli* genome may contain as many as 20 of these of various types. Most of the sequence of an IS is taken up by one or two genes that specify the **transposase** enzyme that catalyzes its transposition (Figure 9.17A). There are a pair of inverted repeats at either end of each IS element, between 9 bp and 41 bp in length depending on the type of IS, and insertion of the element into the target DNA creates a pair of short (4–13 bp) *direct* repeats in the host genome. IS elements can transpose either replicatively or conservatively.

IS elements are also components of a second type of DNA transposon first characterized in *E. coli* and now known to be common in many prokaryotes. These **composite transposons** are made up of a pair of IS elements flanking a segment of DNA, usually containing one or more genes—often ones coding for antibiotic resistance (Figure 9.17B). Tn10, for example, carries a gene for tetracycline resistance, and Tn5 and Tn903 both carry a gene for resistance to

kanamycin. Some composite transposons have identical IS elements at either end, and others have one element of one type and one of another. In some cases the IS elements are orientated as direct repeats and sometimes as inverted repeats. These variations do not appear to affect the transposition mechanism for a composite transposon, which is conservative in nature and catalyzed by the transposase coded by one or both of the IS elements.

Various other classes of DNA transposon are known in prokaryotes. Two additional important types from *E. coli* are:

- **Tn3-type transposons**, which have their own transposase gene and so do not require flanking IS elements in order to transpose (Figure 9.17C). Tn3 elements transpose replicatively.
- **Transposable phages**, which are bacterial viruses that transpose replicatively as part of their normal infection cycle (Figure 9.17D).

DNA transposons are less common in eukaryotic genomes

The human genome contains approximately 350,000 DNA transposons of various types (Table 9.3), all with terminal inverted repeats and all containing a gene for a transposase enzyme that catalyzes the transposition event. However, the vast majority of these elements are inactive, either because the transposase gene is nonfunctional or because sequences at the ends of the transposon, which are essential for active transposition, are missing or mutated.

Active DNA transposons are more common in plants, and include the Ac/Ds transposon, the first one to be discovered by McClintock, and the Spm element, both of which are found in maize. An interesting feature of these plant transposons is that they work together in family groups. For example, the Ac element codes for an active transposase that recognizes both Ac elements and Ds sequences. The latter are versions of Ac that have internal deletions that remove part of the transposase gene, meaning that a Ds element cannot

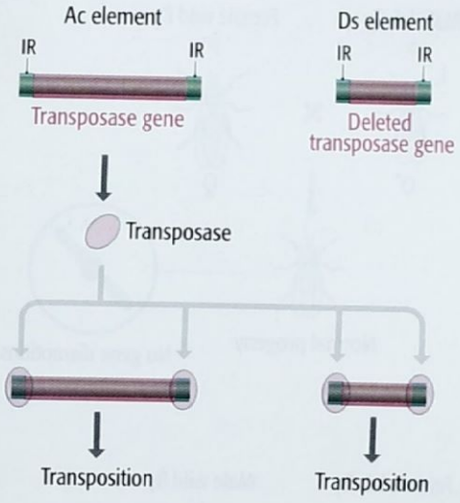


Figure 9.18 The Ac/Ds transposon family of maize. The full-length Ac element is 4.2 kb and contains a functional transposase gene. The transposase recognizes the 11 bp inverted repeats (IRs) at either end of the Ac sequence and catalyzes its transposition. The Ds element has an internal deletion and so does not synthesize its own transposase. But it still has the IR sequences, which are recognized by the transposase made by the Ac element. Hence the Ds element is also able to transpose. There are approximately ten different types of Ds element in the maize genome, with deletions ranging in size from 194 bp to several kilobases.

Figure 9.19 Variegated pigmentation in maize kernels caused by transposition in somatic cells. The highly colored forms of *Zea mays* are popularly known as "Indian corn." Image courtesy of Lena Struwe.

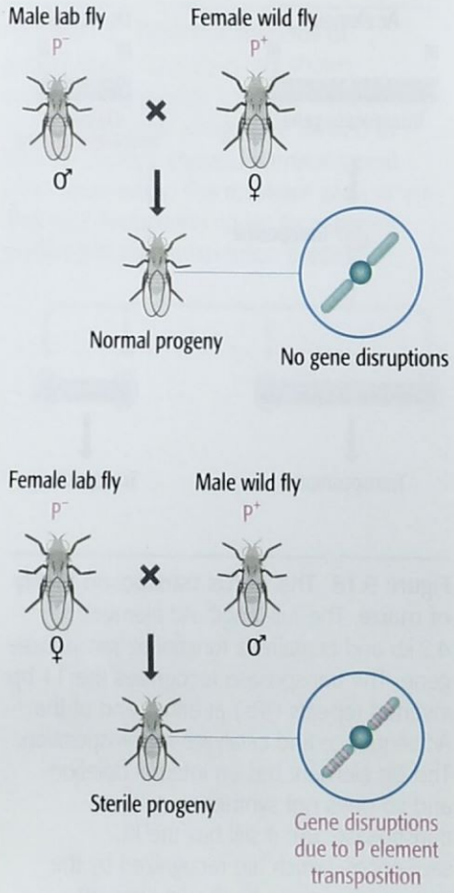


Figure 9.20 Hybrid dysgenesis. Crosses between male lab flies and female wild flies give normal progeny, but when the male partner is a wild fly the offspring are sterile. One possible explanation of hybrid dysgenesis is that the cytoplasm of flies with P elements (P^+ in this diagram) contains a repressor that prevents P element transposition. The fertilized egg resulting from a cross between a female P^+ fly and male P^- fly will contain this repressor and so the progeny are normal. However, the repressor will not be carried in the sperm from a male P^+ fly, so the fertilized egg from a cross between a male P^+ and a female P^- fly will lack the repressor, allowing P element transposition to occur and resulting in progeny displaying hybrid dysgenesis.

make its own transposase and can move only through the activity of the transposase synthesized by a full-length Ac element (Figure 9.18). Similarly, full-length Spm elements are accompanied by deleted versions which transpose through use of the transposase enzymes coded by the intact elements. The activity of Ac elements is apparent during the normal life cycle of a maize plant, transposition in somatic cells resulting in changes in gene expression which are manifested in, for example, variegated pigmentation in maize kernels (Figure 9.19).

McClintock's realization that the maize genome contains transposable elements resulted from her studies into the genetic basis of the different color patterns displayed by kernels. The P element, a DNA transposon in *Drosophila melanogaster*, was similarly discovered from studies of an unusual genetic event which, as it turns out, arises from transposition. This event is called **hybrid dysgenesis** and occurs when females from laboratory strains of *D. melanogaster* are crossed with males from wild populations. The offspring resulting from such crosses are sterile and have chromosomal abnormalities along with a variety of other genetic malfunctions. The explanation is that the genomes of wild fruit flies contain inactive versions of P elements—typical DNA transposons comprising a transposase gene flanked by inverted terminal repeats—but that laboratory strains lack these elements. After crossing, the elements inherited from the wild flies become active in the fertilized eggs, transposing into various new positions and causing the gene disruptions that characterize hybrid dysgenesis (Figure 9.20). Exactly why this activation occurs is not known, but a more interesting question is why the genomes of wild populations of *D. melanogaster* contain P elements whereas laboratory strains do not. Most of the laboratory strains are descended from flies collected by Thomas Hunt Morgan some 90 years ago, and used by Morgan and his colleagues in the first gene mapping experiments (Section 3.2.3). It appears that wild populations at that time lacked P elements, which have somehow proliferated in wild genomes during the last 90 years. The inability of wild and laboratory flies to produce viable offspring means that these two populations fail one of the main criteria used to identify biological species—the ability of all individuals to mate productively. This raises the intriguing possibility that speciation might, at least in some organisms, be driven by differential proliferation of transposable elements within the genomes of members of different populations.

Summary

Early studies of viruses focused largely on the bacteriophages—viruses that infect bacteria. Bacteriophages are constructed of protein and nucleic acid, the protein forming a capsid that encloses the genome. There are three basic types of capsid structure and many types of genome organization, different phages having single- or double-stranded DNA or RNA genomes, some with the entire genome contained in a single molecule, and some with segmented genomes. Bacteriophages follow two distinct infection cycles. All phages can infect via the lytic cycle, which results in the immediate synthesis of new bacteriophages, usually accompanied by death of the host cell. Some can also follow the lysogenic cycle, during which a copy of the phage genome becomes inserted into the host DNA, where it may remain in quiescent form for many cell generations. Eukaryotic viruses are equally diverse in terms in genome organization but display just two capsid structures. Most eukaryotic viruses

follow a lytic infection cycle but this does not always result in the immediate death of the host cell. A number of DNA and RNA viruses can integrate their genomes into eukaryotic chromosomes in a manner similar to a lysogenic bacteriophage. The viral retroelements, which include HIV, the causative agent of AIDS, are examples of integrative RNA viruses. Satellite RNAs and virusoids are different types of infective RNA molecule that contain no genes and depend on other viruses for their transmission. Viroids are small, infective RNA molecules that never become encapsidated, and prions are infective proteins. Some mobile genetic elements, which are DNA sequences that can transpose within a genome but cannot escape from the cell, are related to RNA viruses. These elements transpose via an RNA intermediate in a pathway similar to the infection process of viral retroelements. The *Ty1/copia* and *Ty3/gypsy* retroelements, and the endogenous retroviruses of mammals, are the mobile elements most closely related to RNA viruses. Mammalian genomes also contain other types of RNA transposon, called LINEs and SINEs, most of which have lost their ability to transpose. DNA transposons do not make use of an RNA intermediate in their transposition pathway. These transposons are common in bacteria, within which they are responsible for the spread of genes coding for antibiotic resistance. DNA transposons are less widespread in eukaryotes but include some important examples, such as the Ac/Ds transposon of maize, the first transposon of any kind to be studied in detail, and the P element of *Drosophila melanogaster*, which is responsible for the hybrid dysgenesis that occurs when female laboratory fruit flies are crossed with male wild flies.

Multiple Choice Questions

*Answers to odd-numbered questions can be found in the Appendix

9.1.* Which type of bacteriophage capsid structure comprises polypeptide subunits arranged in a specific structure that surrounds a nucleic acid core, and a filamentous tail that facilitates entry into cells?

a. Icosahedral.
b. Filamentous.
c. Head-and-tail.
d. Segmented.

9.2. Which type of bacteriophage capsid structure comprises polypeptide subunits arranged in a helix resulting in a rod-like structure?

a. Icosahedral.
b. Filamentous.
c. Head-and-tail.
d. Segmented.

9.3.* In which type of bacteriophage life cycle is the host cell killed shortly after the initial infection?

a. Lytic.
b. Lysogenic.
c. Temperate.
d. Prophage.

9.4. A prophage is defined as:

a. A new phage particle that is assembled inside a host cell during infection.
b. An RNA molecule that does not encode its own capsid proteins.
c. A phage with an RNA genome that is converted to DNA by the enzyme reverse transcriptase.
d. A quiescent form of a bacteriophage that is integrated into the host cell genome.

9.5.* How do eukaryotic viruses acquire lipid membranes?

a. The lipids are synthesized by proteins coded by viral genes.
b. The viral capsid acquires the membrane when it leaves the host cell.
c. The viral capsid acquires the membrane when it is assembled inside the host cell.
d. The viral capsid acquires the membrane when it first binds to a host cell.

9.6. The enzyme reverse transcriptase is present in which type of viruses?

a. Prions.
b. Prophages.
c. Retroviruses.
d. Virusoids.

9.7.* Which of the following are RNA molecules that do not encode their own capsid proteins and move from cell to cell with the assistance of helper viruses?

a. Prions.
b. Prophages.
c. Retroviruses.
d. Virusoids.

9.8. How can viroids replicate and move from cell to cell if they contain no genes and never become encapsidated?

a. They are replicated and transferred from cell to cell with the assistance of a helper virus.
b. They are replicated by host cell enzymes and move from cell to cell with the assistance of a helper virus.
c. They are replicated by host cell or helper virus enzymes and move from cell to cell as naked RNA.
d. They are replicated with the assistance of a helper virus and move from cell to cell as naked DNA.

9.9.* Prions are defined as infectious, disease-causing particles that:

a. Contain only RNA.
b. Contain only DNA.
c. Contain only proteins (no nucleic acids).
d. Contain only lipids (no nucleic acids).

9.10. Conservative transposition is characterized by which of the following?

a. Excision of a transposon from one location and its subsequent insertion at a different location.
b. Replication of a transposon such that the original sequence remains in place and the new sequence is inserted at a different location.
c. Movement of a transposon from one cell to another.
d. Replication of repeated DNA sequences due to slippage of DNA polymerase.

9.11.* Which of the following enzymes is specified by a gene present in RNA transposons?

a. DNA polymerase.
b. RNA polymerase.
c. Reverse transcriptase.
d. Telomerase.

9.12. Which of the following are RNA transposons that lack long terminal repeats (LTRs) and are unable to synthesize their own reverse transciptases?

a. Retroelements.
b. Endogenous retroviruses (ERVs).
c. Long interspersed nuclear elements (LINEs).
d. Short interspersed nuclear elements (SINEs).

Multiple Choice Questions (continued)

*Answers to odd-numbered questions can be found in the Appendix

9.13.* What is thought to be the origin of the Alu RNA transposon?

a. It is thought to be derived from a retrovirus.
b. It is thought to be derived from a protein-coding gene.
c. It is thought to be derived from a cellular noncoding RNA molecule.
d. It is thought to be derived from a DNA virus.

9.14. Which enzyme is specified by a gene present in DNA transposons?

a. DNA polymerase.
b. RNA polymerase.
c. Reverse transcriptase.
d. Transposase.

9.15.* Name the researcher who first identified transposons and the organism he or she studied.

a. David Baltimore and retroviruses.
b. Barbara McClintock and maize.
c. Thomas Hunt Morgan and fruit flies.
d. Craig Venter and humans.

Short Answer Questions

*Answers to odd-numbered questions can be found in the Appendix

9.1.* How are viruses different from cells? Is it appropriate to look on viruses as living organisms?

9.2. How do the genomes of viruses differ from cellular genomes?

9.3.* What are overlapping genes, as found in some viral genomes?

9.4. How long does it take a lytic bacteriophage to lyse a host cell following the initial infection? What is the time line for the lytic infection cycle of T4 phage?

9.5.* Discuss the differences between the capsids of bacteriophages and eukaryotic viruses.

9.6. Discuss the life cycle of retroviruses.

9.7.* What is a transposon?

9.8. What are the characteristics of the LTR retroelements present in the human genome?

9.9.* Discuss the properties and types of retroposons present in the human genome.

9.10. What are the general properties of composite transposons?

9.11.* What are the important features of the DNA transposons found in plants?

9.12. Describe the basis to hybrid dysgenesis in fruit flies.

In-depth Problems

*Guidance to odd-numbered questions can be found in the Appendix

9.1.* To what extent can viruses be considered a form of life?

9.2. Bacteriophages with small genomes (for example, ϕX174) are able to replicate very successfully in their hosts. Why then should other bacteriophages, such as T4, have large and complicated genomes?

9.3.* Genetic elements that reproduce within or along with a host genome, but confer no benefit on the host, are sometimes called "selfish" DNA. Discuss this concept, in particular as it applies to transposons.

9.4. Some bacteriophages, such as T4, modify the host RNA polymerase after infection, so that this polymerase no longer recognizes *E. coli* genes, but transcribes bacteriophage genes instead. How might this modification be carried out?

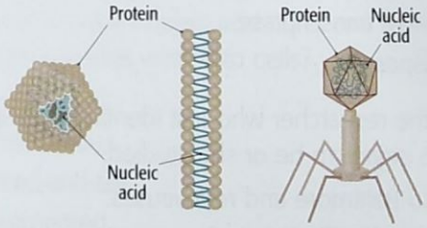
9.5.* Why do LTR retroelements have long terminal repeats?

continued ...

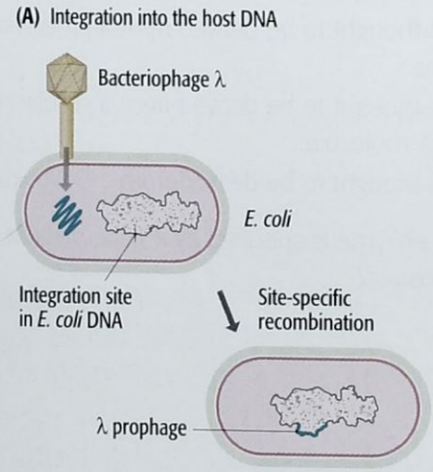
Figure Tests

*Answers to odd-numbered questions can be found in the Appendix

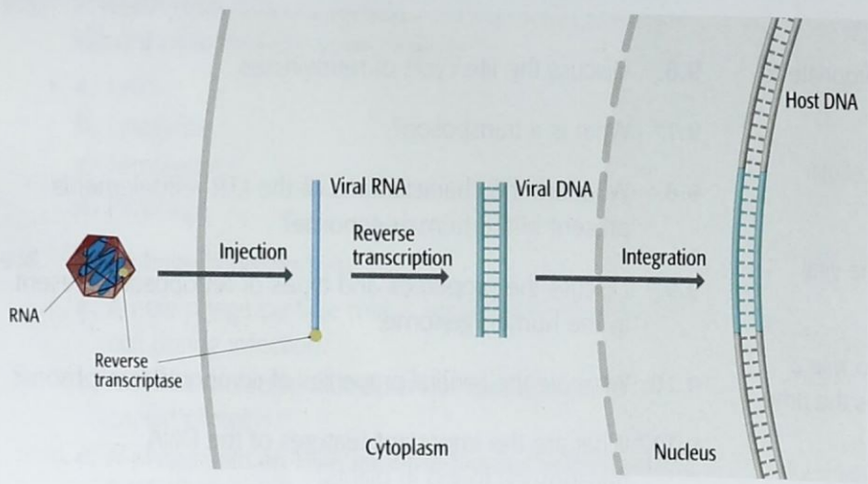
9.1* Identify the three types of bacteriophage capsid structure.



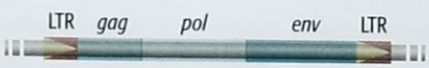
9.2. What type of bacteriophage life cycle is represented in the figure?



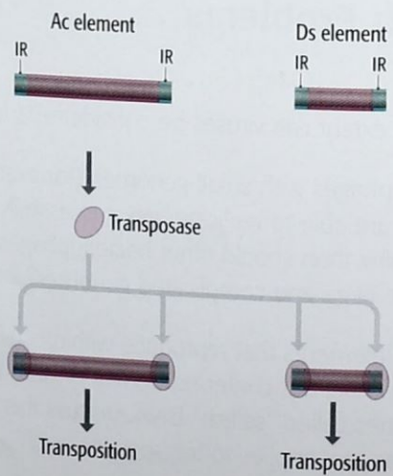
9.3* What type of viral infection is shown in the figure?



9.4. This figure shows the genome of what type of virus? What are the functions of the LTR sequences?



9.5* Name the researcher who first described the Ac and Ds elements. What is the difference between these elements?



Further Reading

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