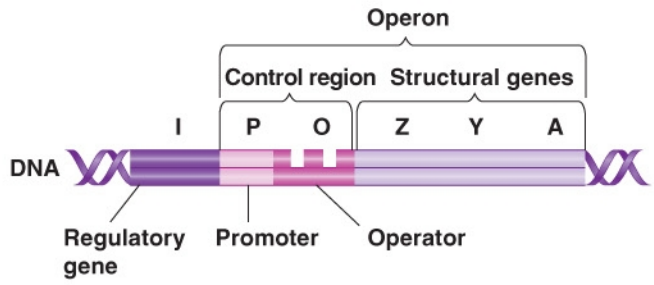
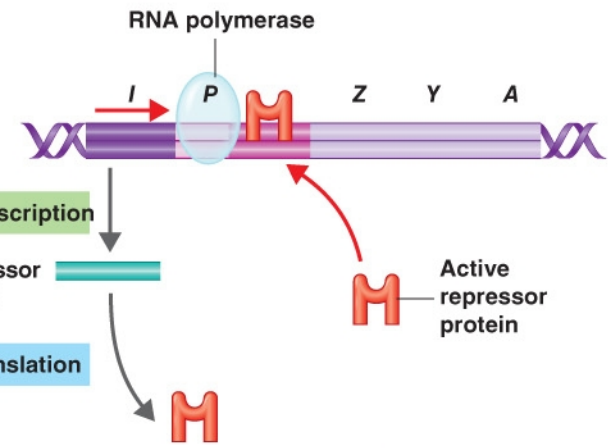


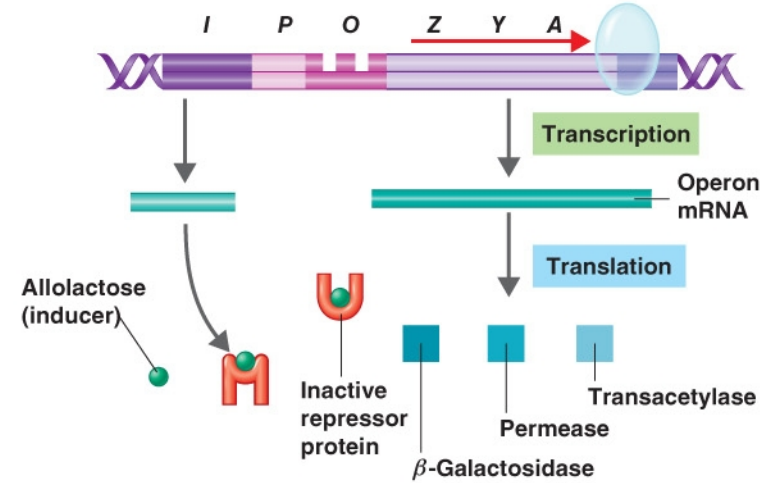
Operon model: Negative inducible



1 Structure of the operon. The operon consists of the promoter (P) and operator (O) sites and structural genes that code for the protein. The operon is regulated by the product of the regulatory gene (I).



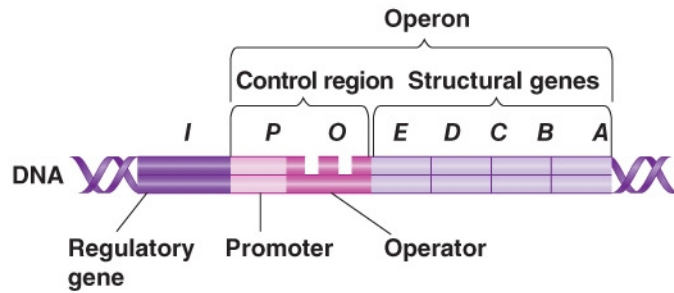
2 Repressor active, operon off. The repressor protein binds with the operator, preventing transcription from the operon.



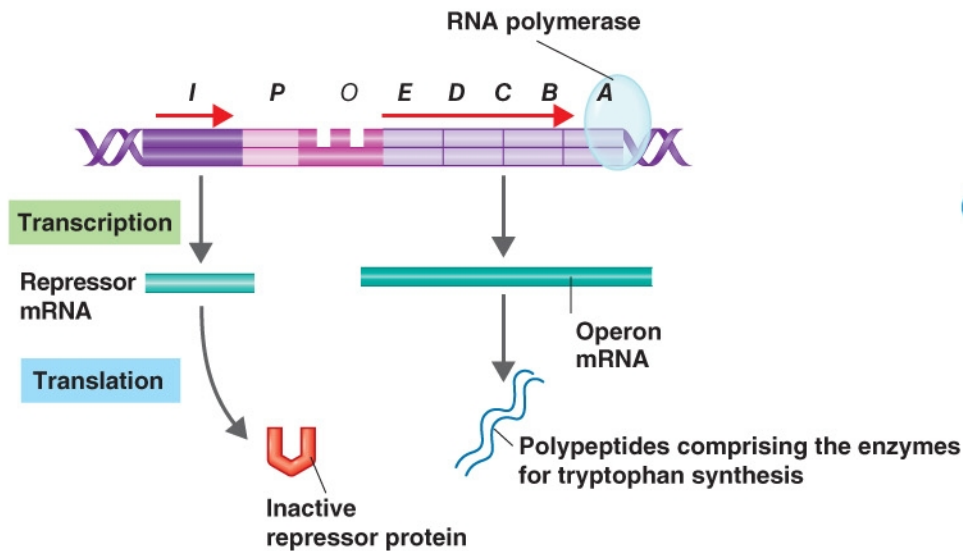
3 Repressor inactive, operon on. When the inducer allolactose binds to the repressor protein, the inactivated repressor can no longer block transcription. The structural genes are transcribed, ultimately resulting in the production of the enzymes needed for lactose catabolism.

Fig. 8.12

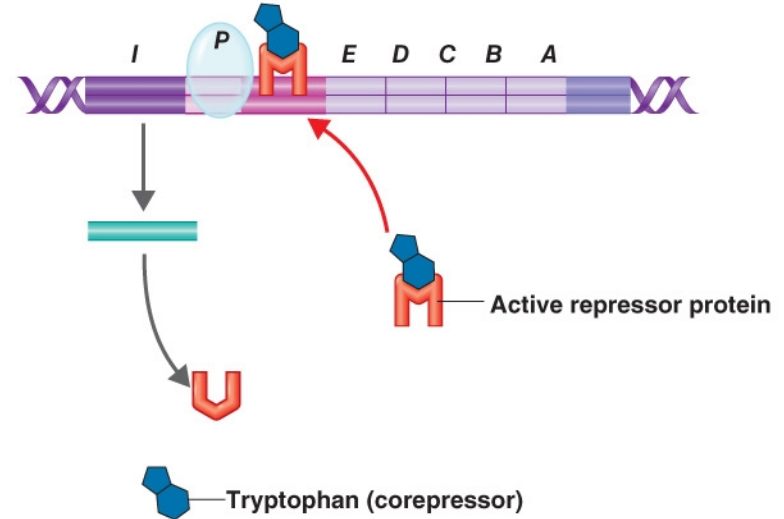
Operon model: negative repressible



- 1 Structure of the operon.** The operon consists of the promoter (P) operator (O) sites and structural genes that code for the protein. The operon is regulated by the product of the regulatory gene (I).

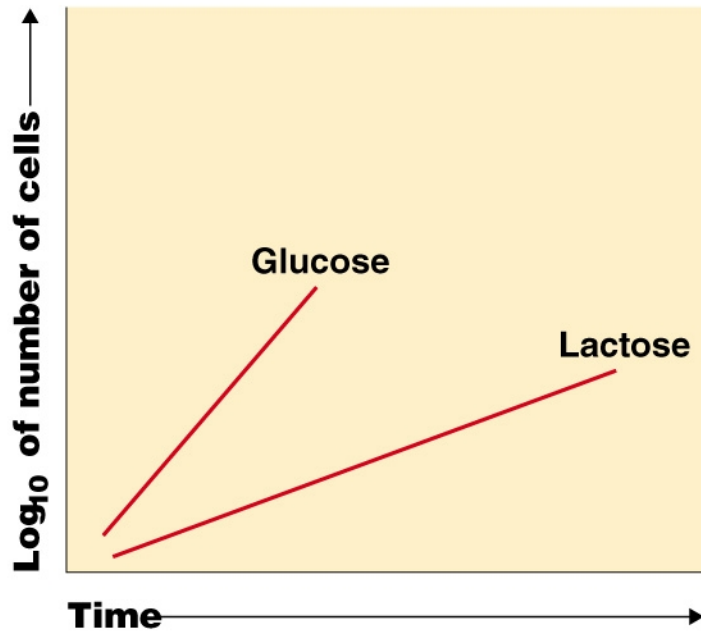


- 2 Repressor inactive, operon on.** The repressor is inactive, and transcription and translation proceed, leading to the synthesis of tryptophan.

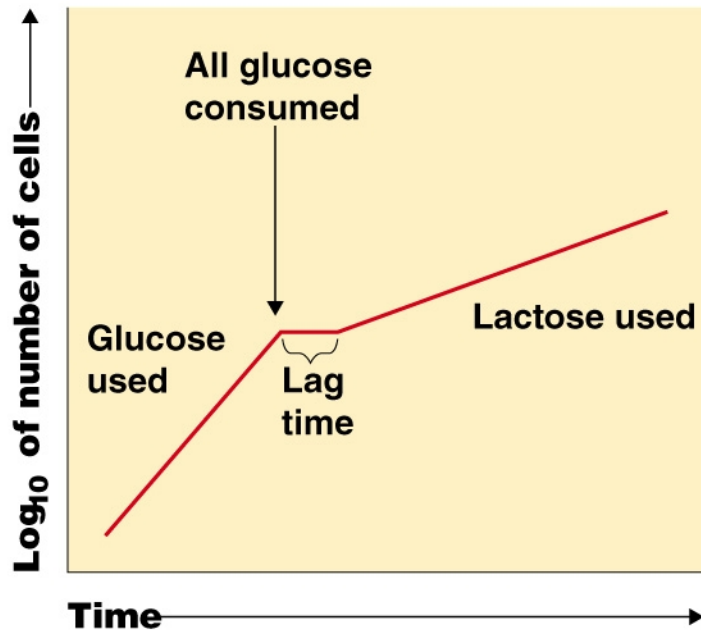


- 3 Repressor active, operon off.** When the corepressor tryptophan binds to the repressor protein, the activated repressor binds with the operator, preventing transcription from the operon.

Operon model

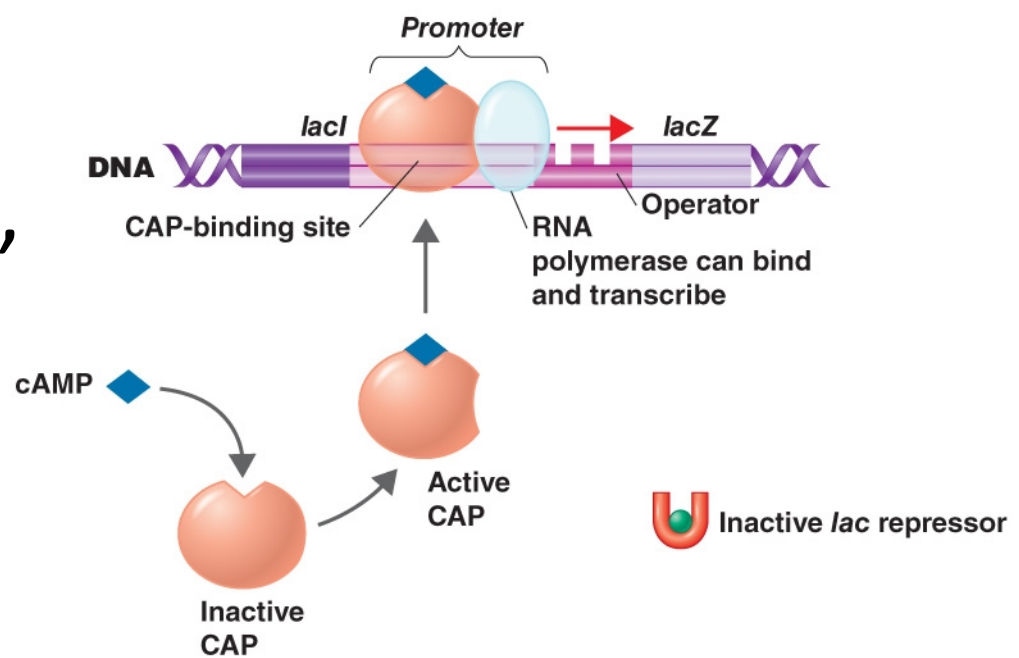


(a) Bacteria growing on glucose as the sole carbon source grow faster than on lactose.

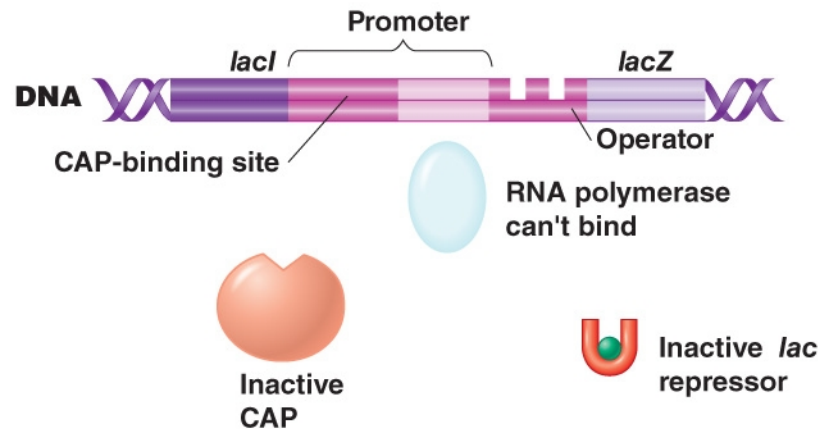


(b) Bacteria growing in a mixture of glucose and lactose first consume the glucose and then, after a short lag time, the lactose. During the lag time, intracellular cAMP increases, the operon is transcribed, more lactose is transported into the cell, and β -galactosidase is synthesized to break down lactose.

Operon model: Positive inducible, complex



(a) Lactose present, glucose scarce (cAMP level high). If glucose is scarce, the high level of cAMP activates CAP, and the *lac* operon produces large amounts of mRNA for lactose digestion.

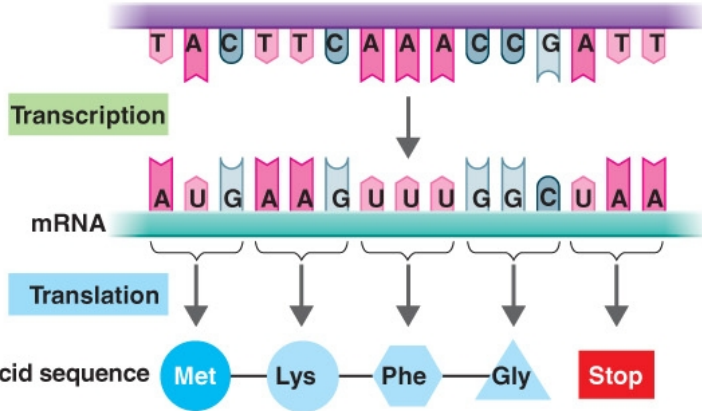


(b) Lactose present, glucose present (cAMP level low). When glucose is present, cAMP is scarce, and CAP is unable to stimulate transcription.

Paper: Toledo-Arana *et al.* 2009

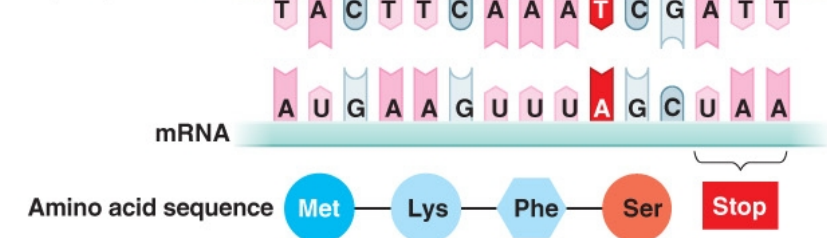
Mutations

DNA (template strand)

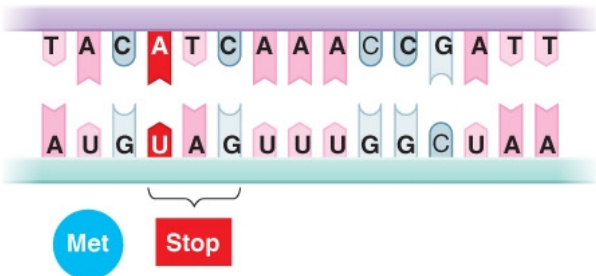


(a) Normal DNA molecule

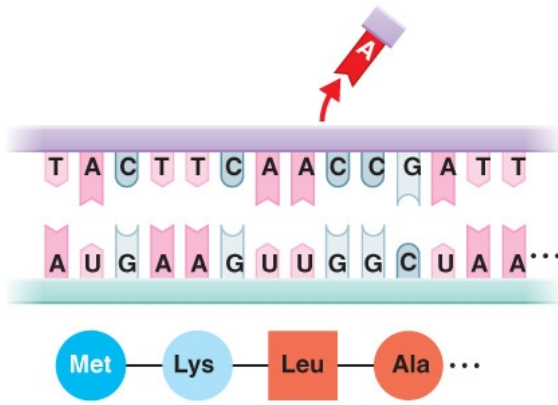
DNA (template strand)



(b) Missense mutation



(c) Nonsense mutation

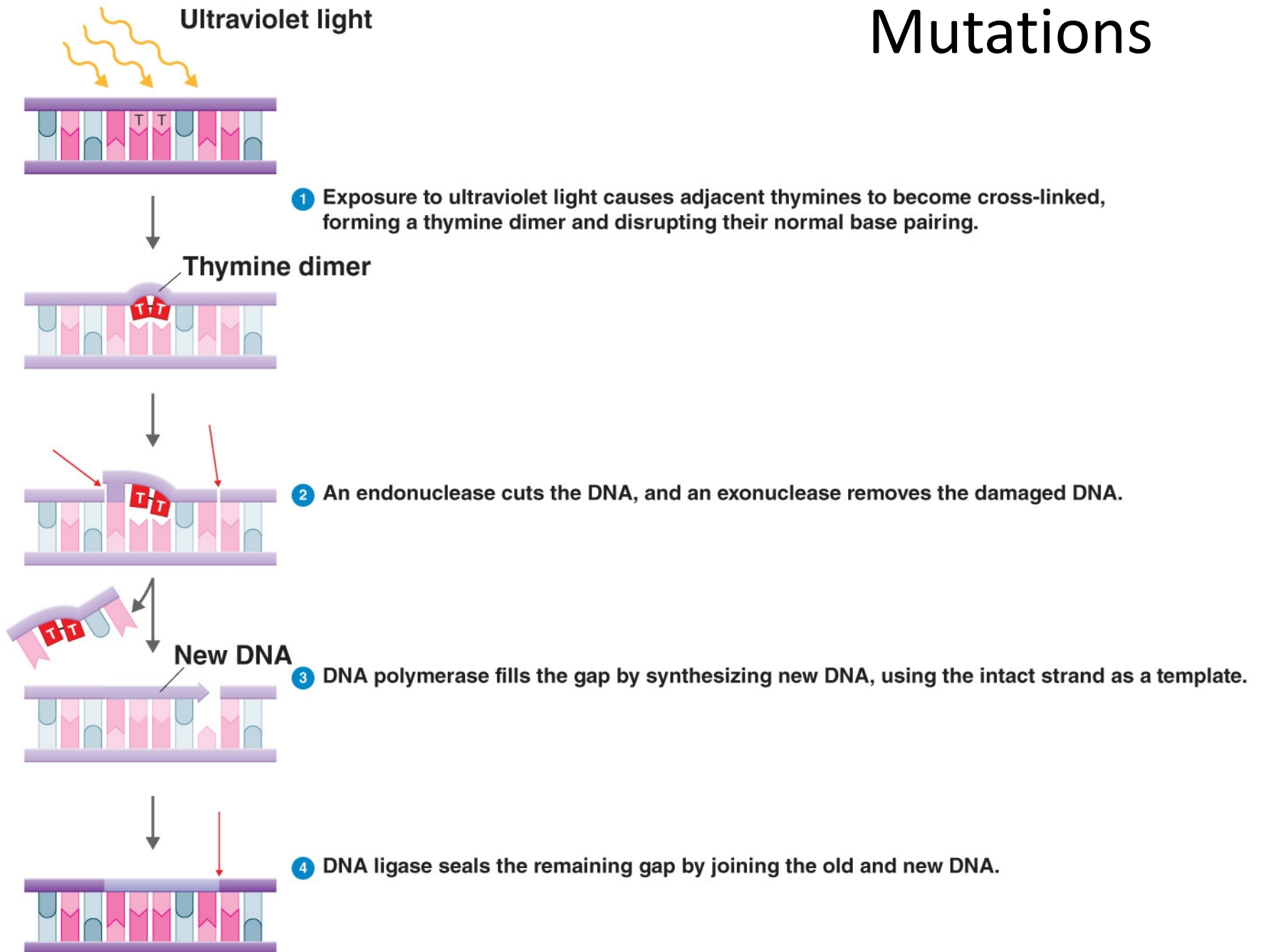


(d) Frameshift mutation

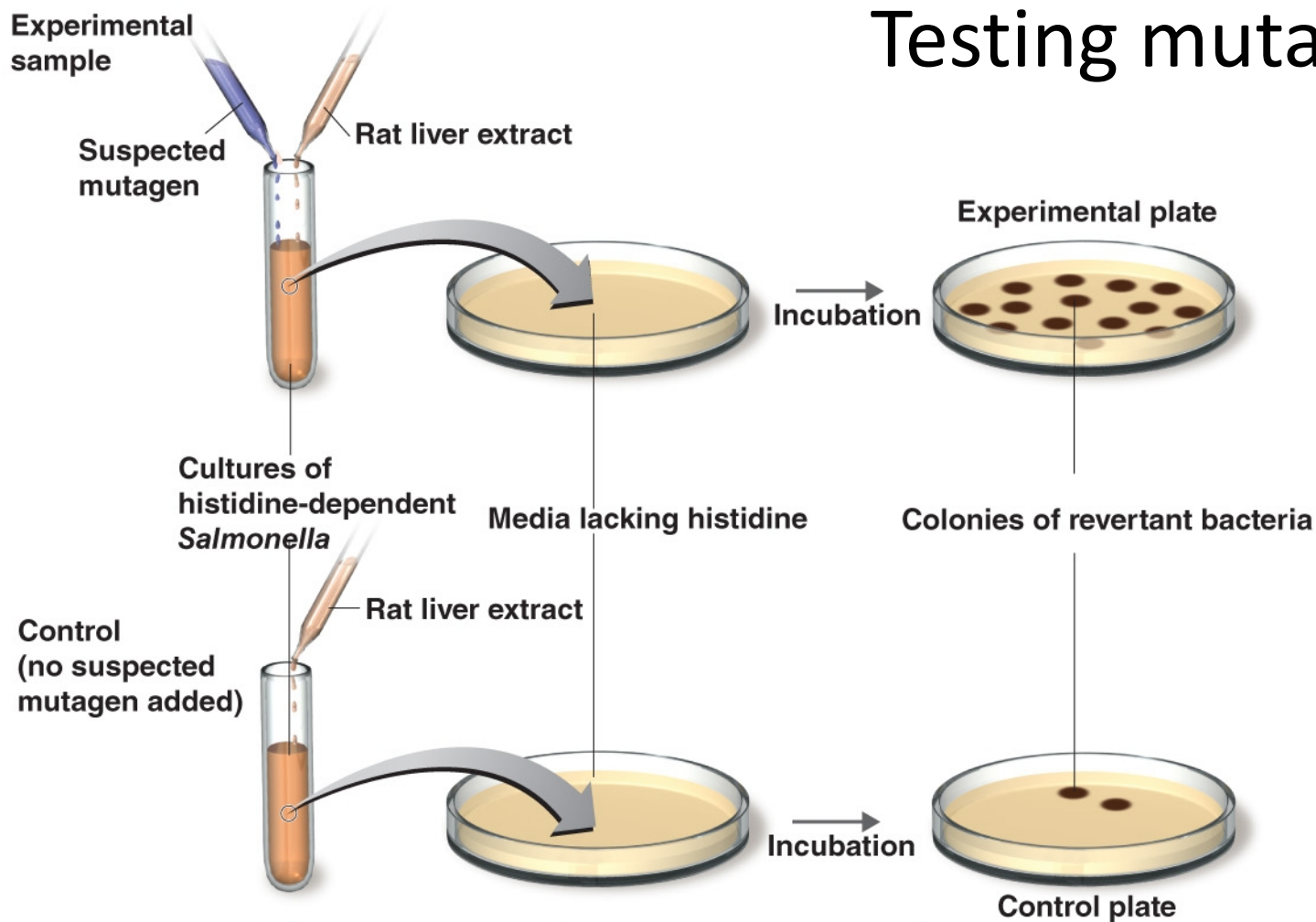
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Fig. 8.18

Mutations



Testing mutagens

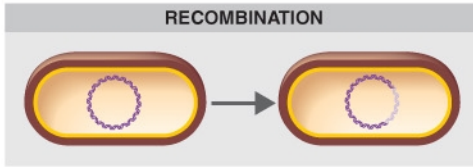


- 1 Two cultures are prepared of *Salmonella* bacteria that have lost the ability to synthesize histidine (histidine-dependent).
- 2 The suspected mutagen is added to the experimental sample only; rat liver extract (an activator) is added to both samples.
- 3 Each sample is poured onto a plate of medium lacking histidine. The plates are then incubated at 37° C for two days. Only bacteria whose histidine-dependent phenotype has mutated back (reverted) to histidine-synthesizing will grow into colonies.
- 4 The numbers of colonies on the experimental and control plates are compared. The control plate may show a few spontaneous histidine-synthesizing revertants. The test plates will show an increase in the number of histidine-synthesizing revertants if the test chemical is indeed a mutagen and potential carcinogen. The higher the concentration of mutagen used, the more revertant colonies will result.

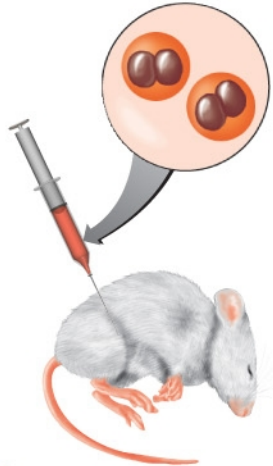
Cyclophosphamide:
a nitrogen mustard
alkylating agent



A sign of recombination



- 1 Living encapsulated bacteria injected into mouse.



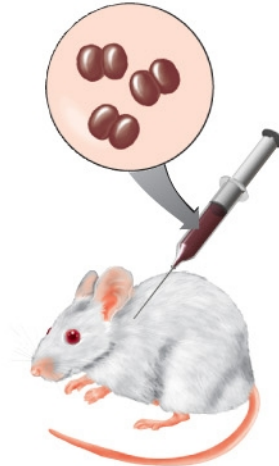
- 2 Mouse died.



- 3 Colonies of encapsulated bacteria were isolated from dead mouse.

(a)

- 1 Living nonencapsulated bacteria injected into mouse.



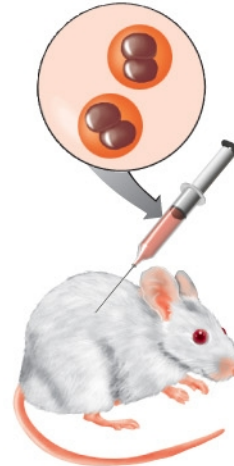
- 2 Mouse remained healthy.



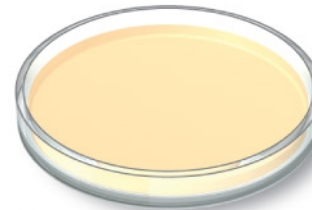
- 3 A few colonies of nonencapsulated bacteria were isolated from mouse; phagocytes destroyed nonencapsulated bacteria.

(b)

- 1 Heat-killed encapsulated bacteria injected into mouse.



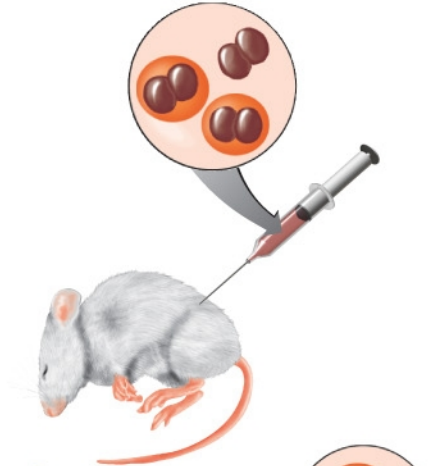
- 2 Mouse remained healthy.



- 3 No colonies were isolated from mouse.

(c)

- 1 Living nonencapsulated and heat-killed encapsulated bacteria injected into mouse.



- 2 Mouse died.



- 3 Colonies of encapsulated bacteria were isolated from dead mouse.

(d)

Recombination: Transformation

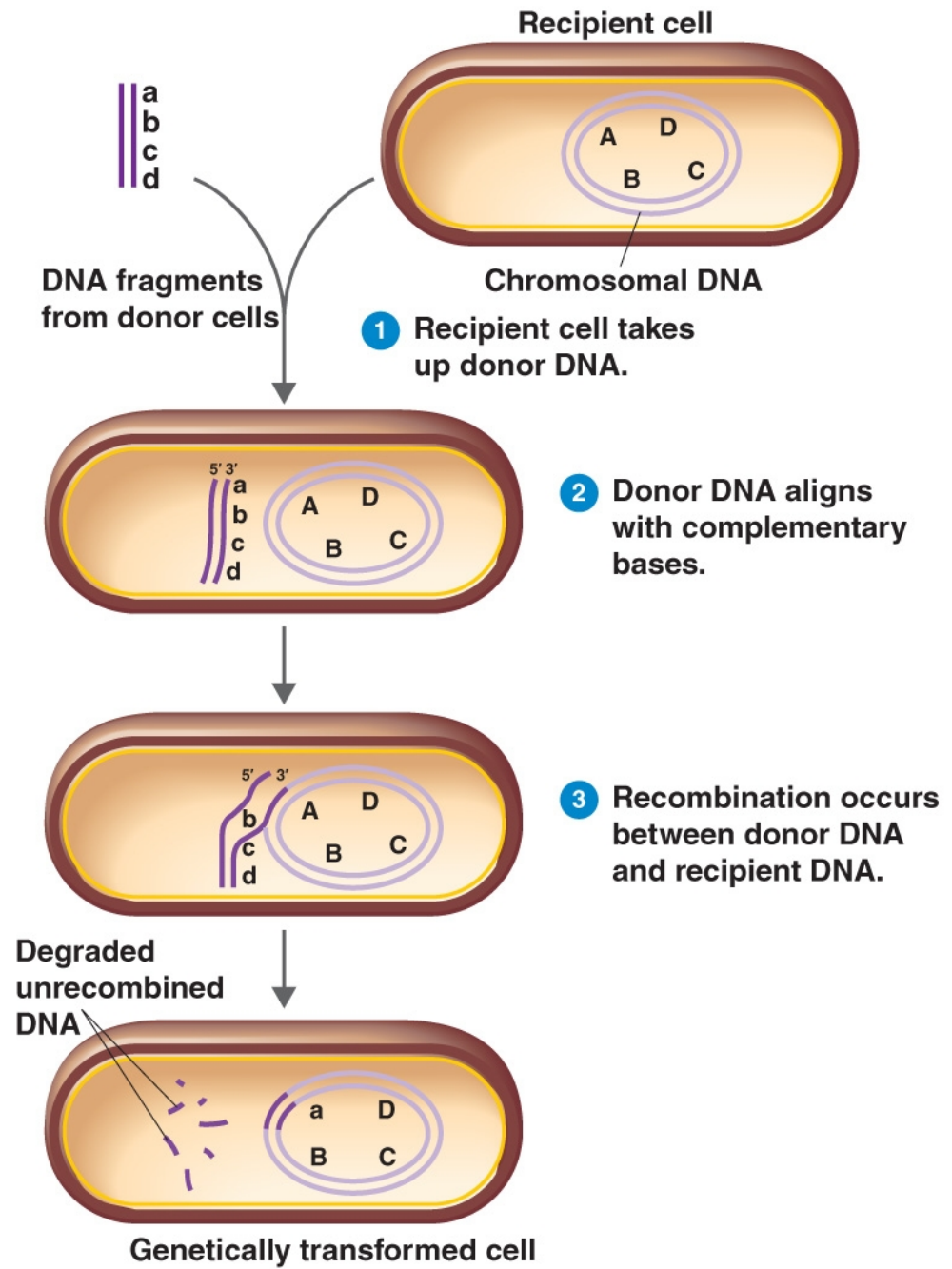
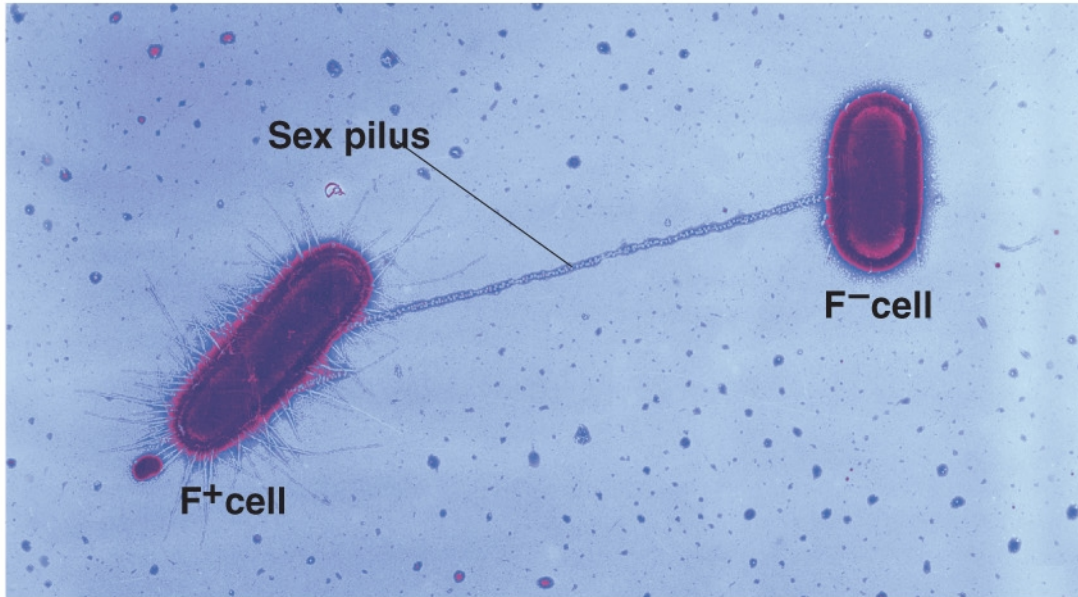


Fig. 8.26

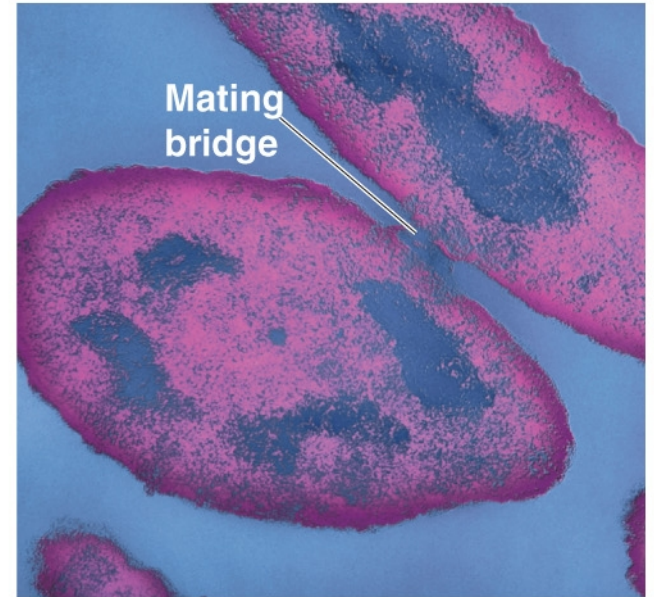
Recombination: Conjugation



(a) Sex pilus

TEM | 1 μ m

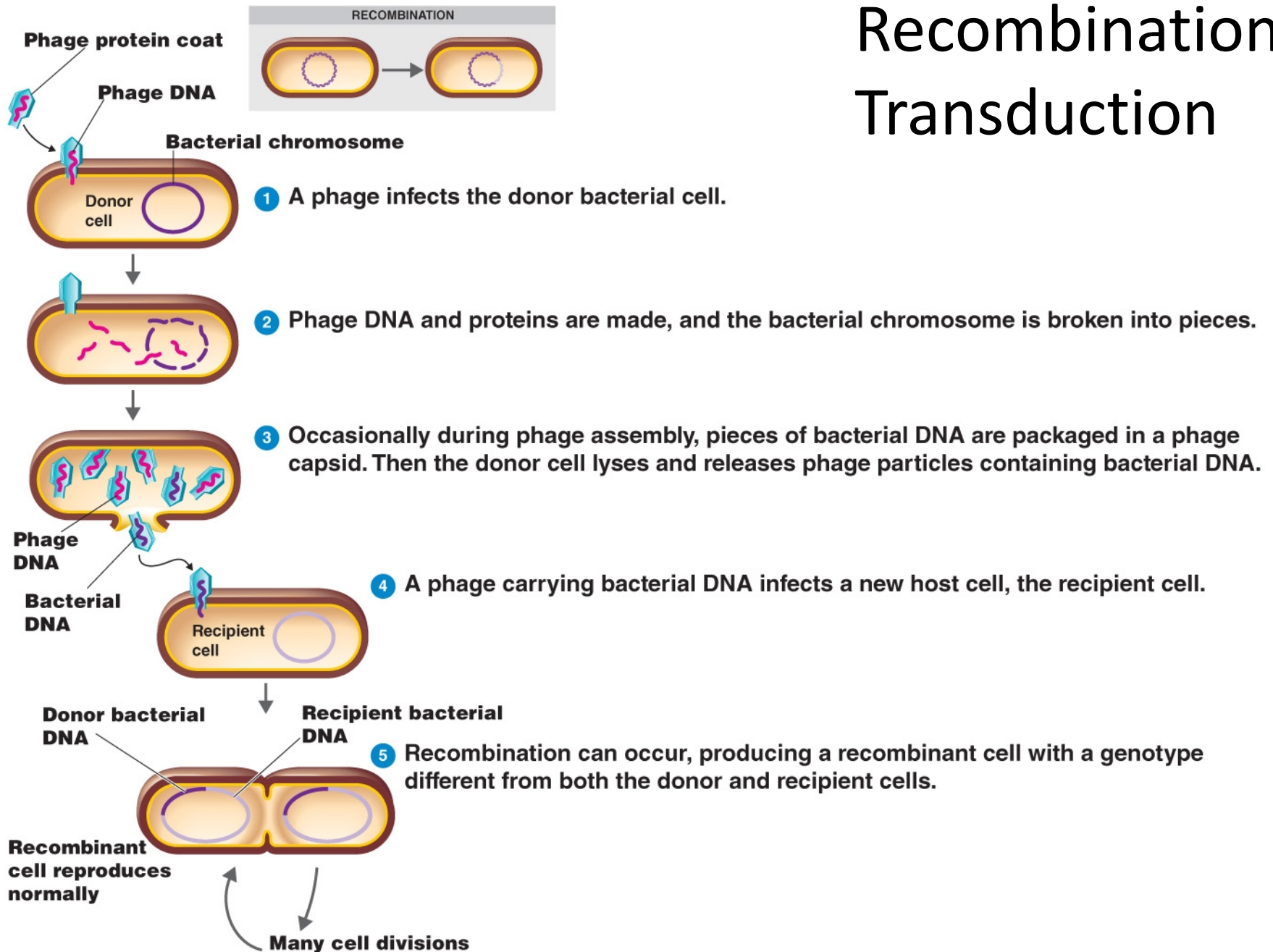
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(b) Mating bridge

TEM | 0.3 μ m

Recombination: Transduction

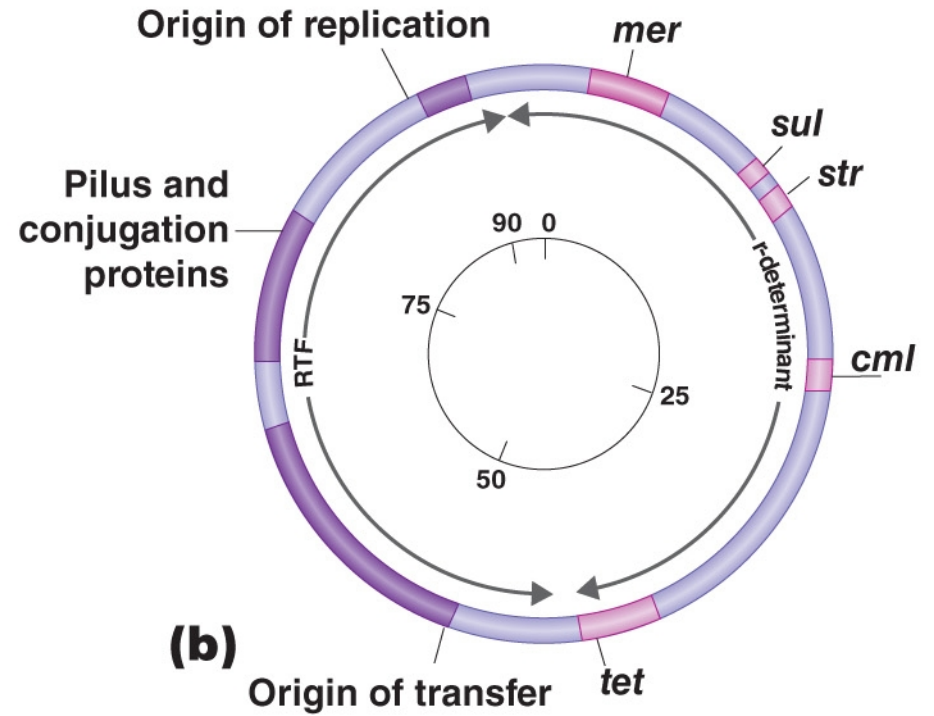


Plasmids



(a)

SEM 20 nm

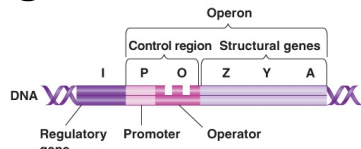


(b)

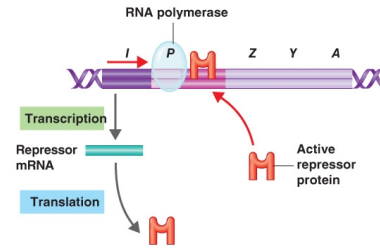
Operon model

Operon model

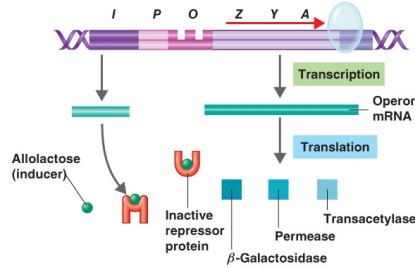
Operon model: Negative inducible



1 **Structure of the operon.** The operon consists of the promoter (P) and operator (O) sites and structural genes that code for the protein. The operon is regulated by the product of the regulatory gene (I).

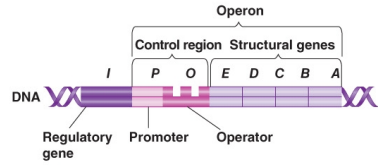


2 **Repressor active, operon off.** The repressor protein binds with the operator, preventing transcription from the operon.

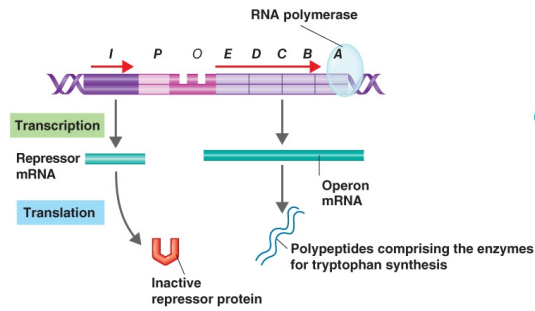


3 **Repressor inactive, operon on.** When the inducer allolactose binds to the repressor protein, the inactivated repressor can no longer block transcription. The structural genes are transcribed, ultimately resulting in the production of the enzymes needed for lactose catabolism.

Fig. 8.12

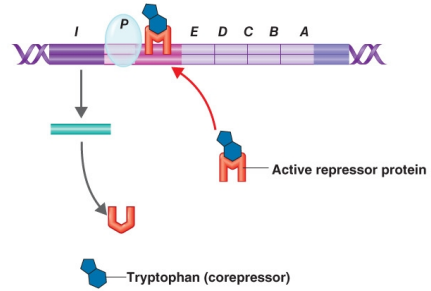


1 Structure of the operon. The operon consists of the promoter (P) operator (O) sites and structural genes that code for the protein. The operon is regulated by the product of the regulatory gene (I).



2 Repressor inactive, operon on. The repressor is inactive, and transcription and translation proceed, leading to the synthesis of tryptophan.

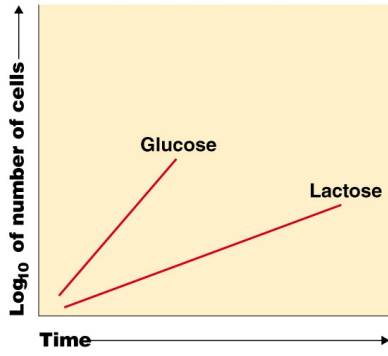
Operon model: negative repressible



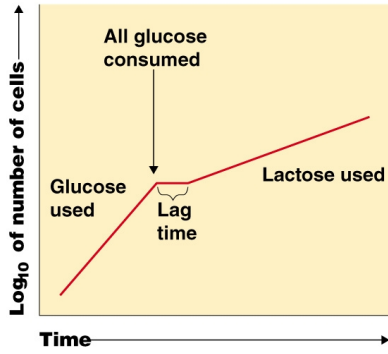
3 Repressor active, operon off. When the corepressor tryptophan binds to the repressor protein, the activated repressor binds with the operator, preventing transcription from the operon.

Fig. 8.13

Operon model



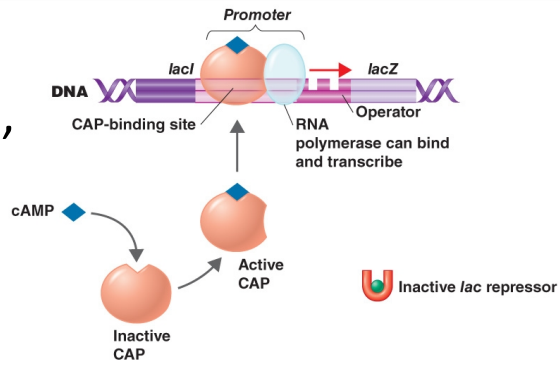
(a) Bacteria growing on glucose as the sole carbon source grow faster than on lactose.



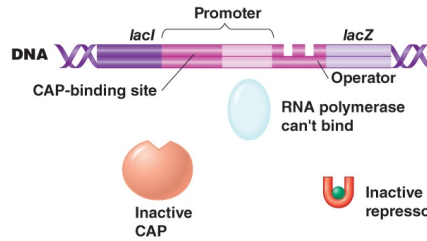
(b) Bacteria growing in a mixture of glucose and lactose first consume the glucose and then, after a short lag time, the lactose. During the lag time, intracellular cAMP increases, the operon is transcribed, more lactose is transported into the cell, and β -galactosidase is synthesized to break down lactose.

Fig. 8.14

Operon model: Positive inducible, complex



(a) Lactose present, glucose scarce (cAMP level high). If glucose is scarce, the high level of cAMP activates CAP, and the *lac* operon produces large amounts of mRNA for lactose digestion.



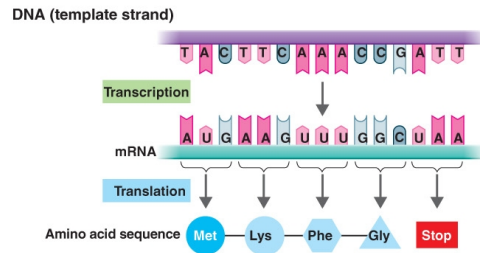
(b) Lactose present, glucose present (cAMP level low). When glucose is present, cAMP is scarce, and CAP is unable to stimulate transcription.

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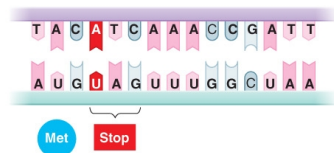
Fig. 8.15

Paper: Toledo-Arana *et al.* 2009

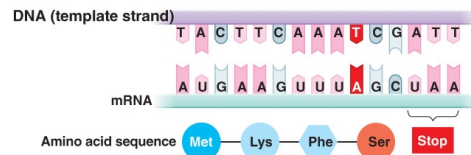
Mutations



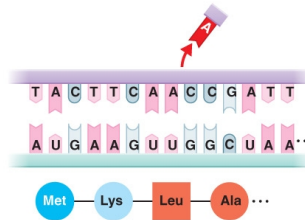
(a) Normal DNA molecule



(c) Nonsense mutation



(b) Missense mutation

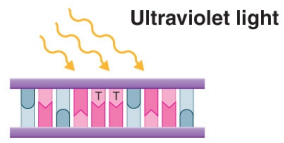


(d) Frameshift mutation

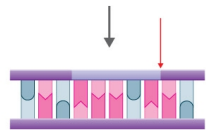
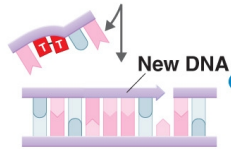
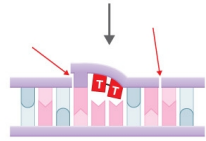
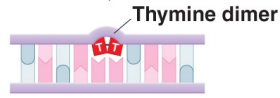
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Fig. 8.18

Mutations



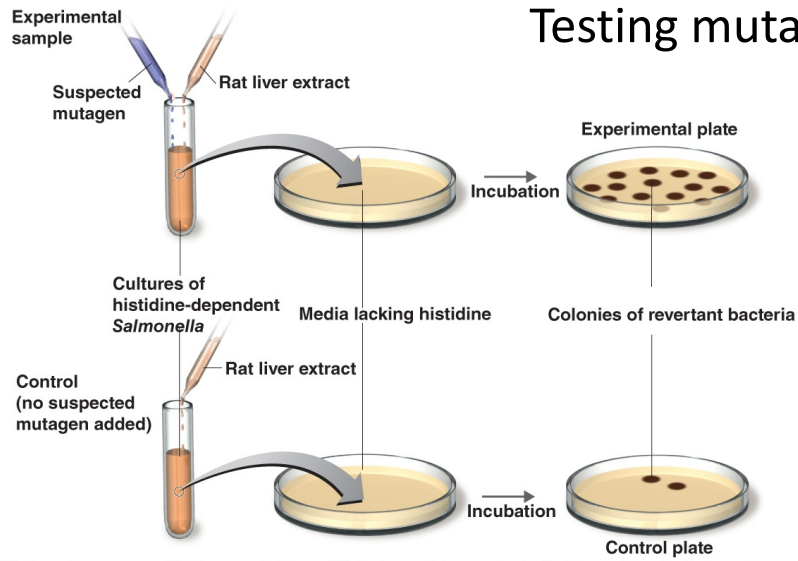
- 1 Exposure to ultraviolet light causes adjacent thymines to become cross-linked, forming a thymine dimer and disrupting their normal base pairing.



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Fig. 8.21

Testing mutagens



- 1 Two cultures are prepared of *Salmonella* bacteria that have lost the ability to synthesize histidine (histidine-dependent).
- 2 The suspected mutagen is added to the experimental sample only; rat liver extract (an activator) is added to both samples.
- 3 Each sample is poured onto a plate of medium lacking histidine. The plates are then incubated at 37° C for two days. Only bacteria whose histidine-dependent phenotype has mutated back (reverted) to histidine-synthesizing will grow into colonies.
- 4 The numbers of colonies on the experimental and control plates are compared. The control plate may show a few spontaneous histidine-synthesizing revertants. The test plates will show an increase in the number of histidine-synthesizing revertants if the test chemical is indeed a mutagen and potential carcinogen. The higher the concentration of mutagen used, the more revertant colonies will result.

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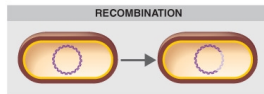
Fig. 8.23



Cyclophosphamide:
a nitrogen mustard
alkylating agent

en.wikipedia.org

A sign of recombination



- 1 Living encapsulated bacteria injected into mouse.



- 2 Mouse died.



- 3 Colonies of encapsulated bacteria were isolated from dead mouse.

(a)

- 1 Living nonencapsulated bacteria injected into mouse.



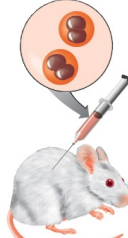
- 2 Mouse remained healthy.



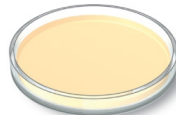
- 3 A few colonies of nonencapsulated bacteria were isolated from mouse; phagocytes destroyed nonencapsulated bacteria.

(b)

- 1 Heat-killed encapsulated bacteria injected into mouse.



- 2 Mouse remained healthy.



- 3 No colonies were isolated from mouse.

(c)

- 1 Living nonencapsulated and heat-killed encapsulated bacteria injected into mouse.



- 2 Mouse died.



- 3 Colonies of encapsulated bacteria were isolated from dead mouse.

(d)

Fig. 8.25

Recombination: Transformation

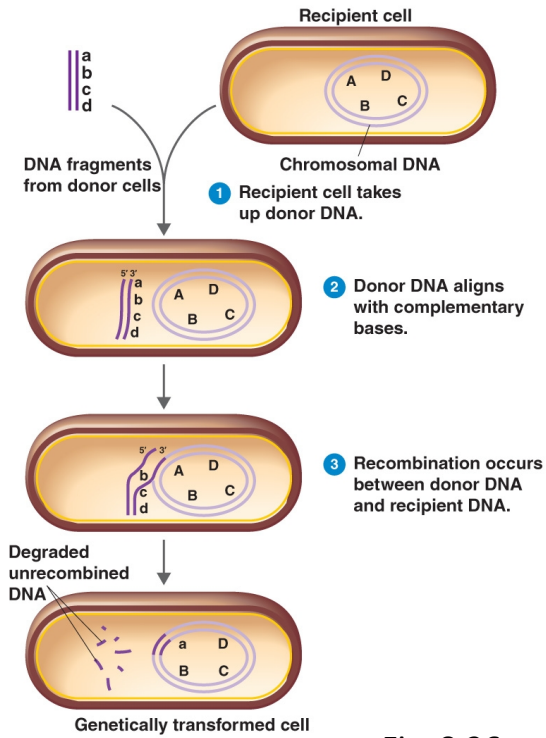
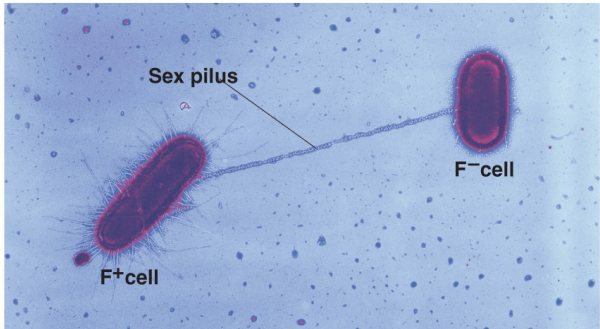


Fig. 8.26

Recombination: Conjugation



(a) Sex pilus

TEM 1 μ m



(b) Mating bridge

TEM 0.3 μ m

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Fig. 8.27

Recombination: Transduction

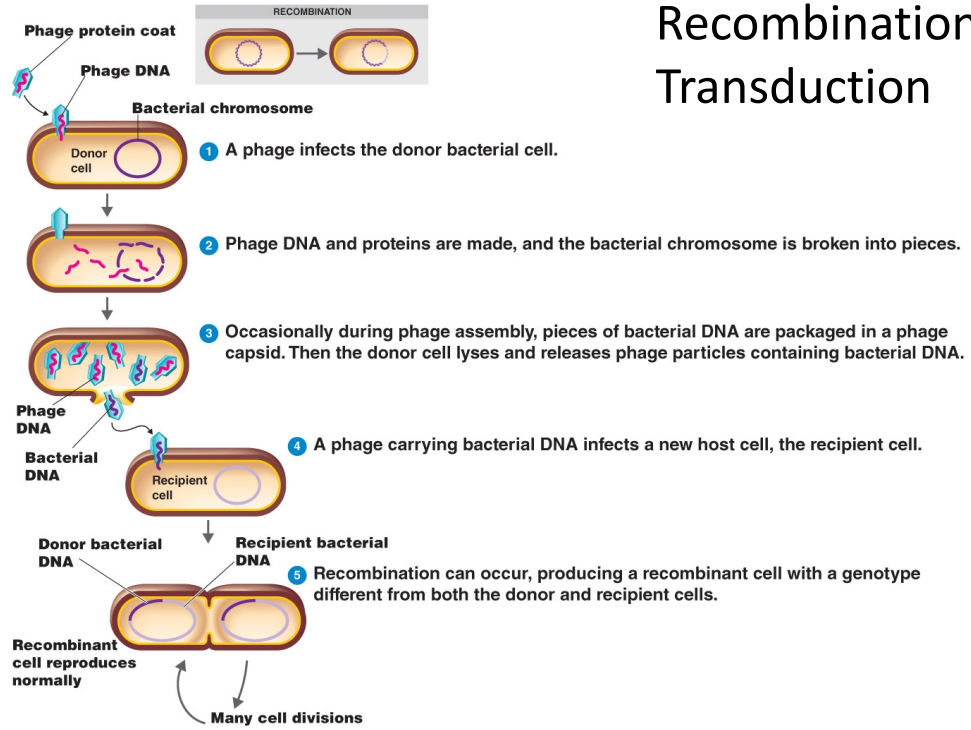
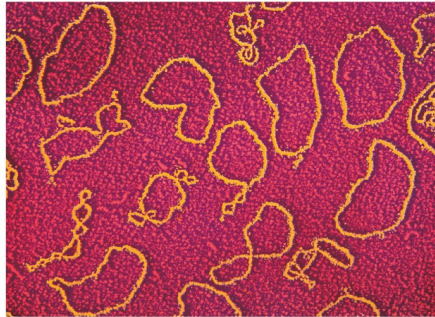


Fig. 8.29

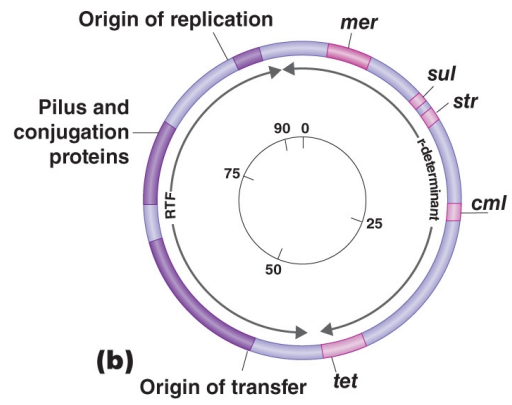
Plasmids



(a)

SEM 20 nm

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(b)

Fig. 8.30

Operon model

Fig. 8.13

Operon model

Fig. 8.13